formed first in patients with massive upper gastrointestinal haemorrhage. If this is unsuccessful in locating the bleeding point, then arteriography should follow. Vasoconstricting drugs may then be infused if the bleeding site is demonstrated. This may be either the definitive treatment or may allow the patient to undergo surgery in a more stable condition. Those patients in whom stress ulcers[6] or varices can be demonstrated by endoscopy are particularly suitable candidates for selective vasoconstrictor infusions if other medical methods fail, since surgery is particularly hazardous in these patients.

The performance of these procedures is dependent upon the availability of trained radiologists and technicians and the proper facilities. These requirements are becoming more easily met and are within the reach of most larger hospitals. We feel that the present place and future potential of angiography in gastrointestinal bleeding need to be emphasized.—We are, etc.,

ROBERT K. GRAY
J. DUNCAN CRAVEN

Department of Radiology,
University of California,
Center for the Health Sciences,
Los Angeles

1 Baum, S., *et al.*, *Surgery*, 1965, **58**, 797.
2 Nusbaum, M., *et al.*, *Archives of Surgery*, 1968, **97**, 1005.
3 Rosch, J., *et al.*, *Gastroenterology*, 1970, **59**, 341.
4 Rosch, J., Dotter, C. T., and Antonovic, R., *American Journal of Roentgenology*, 1972, **116**, 279.
5 Baum, S., *et al.*, *Advances in Surgery*, 1973, **7**, 149.
6 Athanasoulis, C. A., Baum, S., and Waltman, A. C., *New England Journal of Medicine*, 1974, **290**, 597.

SIR,—We were interested to note the inclusion of stress ulceration of the stomach among the causes of massive upper gastrointestinal bleeding in your leading article (9 March, p. 403), as we have recently had under our care a patient who died as a result of haemorrhage from stress ulceration of the stomach six weeks after partial gastrectomy. As far as we are aware this complication has not previously been reported following gastric surgery.

In December 1973 a 71-year-old man was admitted to this hospital with symptoms of severe iron deficiency anaemia but no previous record of haemorrhage. Subsequent investigations revealed a large benign ulcer on the lesser curve of the stomach. He was discharged home after blood transfusion but was readmitted in January 1974 with melaena. At operation on 16 January a Bilroth I partial gastrectomy was carried out, excising approximately 60% of the stomach including the area of ulceration. There was an eroded atherosclerotic vessel in the base of the ulcer, which was found histologically to be benign. He made an uneventful recovery and was discharged home on the 11th postoperative day taking ferrous sulphate 200 mg three times a day as his haemoglobin level was only 9·3 g/100 ml. He was seen on 18 February in a follow-up clinic when he complained of lack of energy. His haemoglobin level was 9·1 g/100 ml. He was eating satisfactorily but had a small capacity for food.

On the morning of 4 March the patient collapsed at home and was dead when the family practitioner arrived 15 minutes later.

Necropsy revealed 800 ml of blood in the stomach with multiple superficial erosions straddling the lesser curve of the gastric remnant; the recent surgical anastomoses were soundly healed. The brain was normal; there was evidence of chronic bronchitis and emphysema; the heart was enlarged and though the coronary arteries were narrowed by atheroma there was no evidence of myocardial infarction.

It was assumed that in the absence of alcohol or any medication except ferrous sulphate the gastric erosions could be explained only by stress ulceration. It is said that stress ulceration is produced by the action of gastric acid on an ischaemic mucosa. This patient may have developed gastric ischaemia as a result of his atheroma, but it is surprising that there was sufficient acid produced following gastric resection to cause multiple erosions.—We are, etc.,

ALEXANDER P. ROSS
G. C. BRILL

Royal Hampshire County Hospital,
Winchester

Intrathoracic Foregut Cysts

SIR,—Regarding your leading article on this subject (20 April, p. 132), I would like to warn against the discovery at operation that this posterior mediastinal radiological opacity is in fact that other rarity an intrathoracic meningocele.[1] When this is so the importance of obtaining a completely watertight closure of the dural defect cannot be overemphasized. A substantial morbidity and even mortality may otherwise result from leakage of cerebrospinal fluid into the chest.

Clinical clues to this diagnosis should exist in the form of some stigmata of neurofibromatosis. Radiological evidence in the form of adjacent bony dysplasia is also usually present, but more often is misinterpreted as being secondary to local pressure changes.—I am, etc.,

JOHN MILES

Regional Neurosurgical Centre,
Walton Hospital,
Liverpool

1 Miles, J. B., Pennybacker, J. B., and Sheldon, P., *Journal of Neurology, Neurosurgery and Psychiatry*, 1969, **32**, 99.

Streptokinase and Heparin in Treatment of Pulmonary Embolism

SIR,—We thank Professor W. A. L. MacGowan (13 April, p. 119) for his observations on our trial (2 March, p. 343). His point concerning the incompatibility of heparin and hydrocortisone is valid[1]: the statement in the paper implying that we added hydrocortisone to the loading dose of heparin was an error in writing; hydrocortisone was added only to the initial streptokinase infusion.

We do not agree that the acidity of 5% glucose for intravenous infusion reduces the potency of heparin to a significant extent in clinical practice. There were no marked differences among our patients in terms of plasma thrombin clotting times and plasma heparin concentrations whether the diluent was 0·9% saline or 5% glucose. Stock and Warner[2] have shown with in-vitro studies that heparin does not deteriorate in glucose solution kept at room temperature for at least 24 hours. This has been confirmed in clinical practice by a cross-over trial by Chessells *et al.*[3] They compared 5% glucose (pH 3·8-4·5) and 5% sorbitol (pH 6·2) as diluents for heparin infusion in patients with myocardial infarction: the partial thromboplastin times with kaolin and the plasma heparin concentrations when glucose was used were not significantly different from those when sorbitol was used. On the basis of this evidence our study is unlikely to have been biased in favour of streptokinase.—We are, etc.,

D. A. TIBBUTT
A. A. SHARP
J. M. HOLT

Radcliffe Infirmary,
Oxford

GRAHAM MILLER
GEORGE SUTTON

The Brompton Hospital,
London

1 Engelberg, H., *Heparin: Metabolism, Physiology and Application*, p. 45. Springfield, Thomas, 1963.
2 Stock, S. L., and Warner, N., *British Medical Journal*, 1971, **3**, 307.
3 Chessells, J. M., Braithwaite, T. A., and Chamberlain, D. A., *British Medical Journal*, 1972, **2**, 81.

Smoking in Pregnancy and Child Development

SIR,—Professor N. R. Butler and Mr. H. Goldstein (8 December 1973, p. 573) have shown that the children of mothers who smoked during pregnancy had small but significant impairments in physical and mental development at the ages of 7 and 11 years. They took into account the possibly confounding effects of maternal age and height, social class, birth order, family size, and sex. They interpret their results as showing that smoking in pregnancy has a long-term adverse effect on the surviving offspring.

We have recently demonstrated that the offspring of West Jerusalem mothers who smoked in pregnancy had an increased incidence of admissions to hospital in the first year of life.[1] The effect of smoking was specific to bronchitis and pneumonia, while there were no significant increases in episodes of gastroenteritis and other causes of admission. The findings were independent of birth weight, social class, and birth order and were related in a dose-response effect to the number of cigarettes smoked by the mother in pregnancy. We interpret these findings as being due to the effect of the passive inhalation of tobacco smoke by the infants after birth.

The possible retarding effects both of repeated episodes of chest infections in young children and of admissions to hospital need no elaboration. We agree that smoking in pregnancy should be strongly discouraged but urge caution against the assumption that long-term damage is due to prenatal influence alone. It is necessary to take into account the possibility that the damage, or part of it, is due to a continued insult by environmental tobacco smoke.—We are, etc.,

SUSAN HARLAP
A. MICHAEL DAVIES

Department of Medical Ecology,
Hebrew University-Hadassah Medical School,
Jerusalem

1 Harlap, S., and Davies, A. M., *Lancet*, 1974, **1**, 529.

SIR,—It is usual, when discussing the inauspicious characteristics of the children of smoking mothers, to consider whether these characteristics may be ascribed to the mother or to the smoking. I would not wish to argue the innocence of cigarettes in regard to