It seems clear that the only spastic infant in our series would have been equally likely to be handicapped had it been delivered by caesarean section and, in our hands, prolonged resuscitation did not keep alive spastics caused by the mode of delivery, so depressing our perinatal loss rate. I thank Mr. E. O. Williams and Mr. S. S. F. Pooley for permission to follow up the patients referred to, who were under their care.—I am, etc.,

F. ANN MUSSON

Topsham, Devon

1 Meeting of the Royal Society of Medicine, Obstetrics and Gynaecology Section, 30 May 1975.
2 Neligan, G., *et al., The Formative Years; Birth, Family and Development in Newcastle upon Tyne.* Nuffield Provincial Hospitals Trust. London, Oxford University Press, 1974.

Adrenal Tumours and Hypertension

SIR,—We were interested to read the comments of Dr. V. Tchertkoff and colleagues (24 May, p. 444), though we have reservations in accepting their statement that adrenocortical nodules "have no relation whatsoever to hypertension." Others have described a higher incidence of such lesions in hypertensive patients compared with normotensives coming to necropsy.[1-4]

All the patients in our series (18 January, p. 135) had hypertension with aldosterone excess. The pathological findings were as we reported. However, we did not conclude that the adrenocortical nodules in these patients had an aetiological role in either the hypertension or the aldosterone excess. Indeed, as previously reported,[5] nodules were not identified in all patients, even among those in whom no tumour was found.—We are, etc.,

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J. J. BROWN
D. L. DAVIES
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E. HAYWOOD
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1 Dobbie, J. W., *Journal of Pathology*, 1969, **99**, 1.
2 Symington, T., *Functional Pathology of the Human Adrenal Gland.* Edinburgh, Livingstone, 1969.
3 Neville, A. M., Mackay, A. M., *Clinics in Endocrinology and Metabolism*, 1972, **1**, 361.
4 Granger, P., and Genest, J., *Canadian Medical Association Journal*, 1970, **103**, 34.
5 Ferriss, J. B., *et al., Lancet*, 1970, **2**, 995.

Orbital Bruits in Patients on Maintenance Haemodialysis

SIR,—Referring to the investigation by Mr. S. R. Lancer and others (31 May, p. 481) of orbital bruits in patients on maintenance haemodialysis, we want to report an investigation we made of this subject.

We used the same method as Mr. Lancer and his colleagues. Two groups of patients were studied: 11 children on maintenance haemodialysis with an A.V. fistula and six children after renal transplantation with a still-functioning fistula. Their ages ranged from 5 to 17 years. All had a proximal and large A.V. fistula consisting of a saphenous loop inserted between the brachial artery and the cephalic vein. Seven of the patients on maintenance haemodialysis had an orbital bruit. After occlusion of the fistula the bruit disappeared in one patient but appeared in two others without bruit with open fistula. The presence of the bruit was clearly related to the level of the packed cell volume. None of the patients with a functioning kidney transplant had an orbital bruit despite the presence of a large A.V. fistula. All patients had a packed cell volume greater than 35%.

We can endorse the importance of anaemia in relation to the appearance of an orbital bruit. The influence of the A.V. fistula is of less importance.—We are, etc.,

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An Easy Death—An Uneasy Argument

SIR,—Dr. M. A. Simpson's letter (19 July, p. 155) does indeed clarify the position in that he tacitly admits that good terminal care and active euthanasia are becoming synonymous. It only needs a breakthrough in consensus thinking, like that which made the Abortion Act of 1967 possible, to introduce "voluntary dying" as a respectable and accepted alternative to "natural dying" in circumstances defined by law. I introduce the subject of abortion because both abortion and euthanasia are expressions of active interference with natural processes to the denial of unwanted life—in the one case not yet born, in the other not yet concluded.

The frequency of suicide in dying patients is open to further study. One wonders how accurate are statistics in this connexion, since any compassionate doctor would record the killing disease as "cause of death" under such circumstances, not that the patient had stopped eating or breathing of his own accord. Other, more obvious methods would be beyond the strength of many terminal patients, though cases do occur of poor, distracted sufferers who have embraced death by more obvious means.

The diseases Dr. Simpson quotes are often associated with euphoria. For many people an organic disease is a relief from the unendurable tensions of life. It is when the disease has reached its terminal stage that the protracted business of dying has to be suffered to the advantage of none but simply out of deference to outworn conventions of the sanctity of life.—I am, etc.,

S. L. HENDERSON SMITH

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Increased Serum Iron in Acute Leukaemia

SIR,—We should like to correct Dr. F. Rosner's misconception (12 July, p. 100) regarding ferritin in acute leukaemia. Our paper (1 February, p. 245) was concerned only with serum ferritin protein concentrations. Ferritin is indeed an iron-containing protein and its concentration in the serum correlates well with total body iron stores in normal subjects. In leukaemic patients this relationship does not appear to exist, and in fact we found no correlation between serum ferritin and serum iron concentration.[1]

In patients with acute myeloblastic leukaemia a gross rise in both serum and white cell ferritin concentrations was observed but the white cell ferritin appeared to be mainly apoprotein[2]—that is, ferritin with very little iron in it. Furthermore, our preliminary studies on serum ferritin, at least in states of iron overload, show that it also is mainly apoferritin.[2]

We think it is important to appreciate the difference between serum iron (that is, transferrin-bound iron) and serum ferritin, especially in states such as acute leukaemia where the serum ferritin concentration probably bears little relationship to the iron stores of the patient.—We are, etc.,

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M. WORWOOD
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Cardiff

1 Worwood, M., *et al., British Journal of Haematology*, 1974, **28**, 27.
2 Worwood, M., *et al., Clinical Science and Molecular Medicine*, 1975, **48**, 441.

Infection after Insertion of I.U.D.

SIR,—The report by Dr. M. de Swiet and others (12 July, p. 76) of bacterial endocarditis diagnosed six weeks after the insertion of an intrauterine contraceptive device (I.U.D.) suggests that a causal relationship might exist and a recommendation is made regarding prophylactic chemotherapy. The bacteriological assessment of the case is admittedly incomplete and I suggest that the relationship was a causal one.

From 1964 I established a clinic at the Birmingham and Midland Hospital for Women where I.U.D.s were fitted to over 2000 patients whose immediate follow-up was excellent, though it lapsed subsequently. It has been a matter of surprise that clinical evidence of infection has been so trivial after such a comparatively unsterile procedure. No serious inflammatory complication that could reasonably be attributed to the I.U.D. has been noted. An exacerbation of pre-existing venereal infection was diagnosed in two patients. One woman developed gastroenteritis within 12 hours of fitting and the device was removed but refitted uneventfully afterwards. One patient evacuated a pyometra two years after fitting, but her uterine cavity was sterile and another device was fitted at her request. One patient was operated on for acute pelvic peritonitis two years after fitting, when the device was removed and bacterial swabs from it and from the pelvic peritoneum both showed haemolytic streptococci. Subsequent bacteriology, however, proved them to be of different types.

The successful use of the I.U.D. must depend largely on the astonishing resistance to infection of the endometrial cavity in the absence of pregnancy. I would like to emphasize my personal opinion, however, that an I.U.D. should be removed whenever possible as soon as any pregnancy should be diagnosed.—I am, etc.,

WILFRID MILLS

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Metastatic Carcinoma Causing Haematemesis

SIR,—We were very interested in the description by Dr. E. Edward and Mr. G. Royle of a case of haematemesis due to gastric metastases from bronchial carcinoma (14 June, p. 598). We wish to report a similar case recently presenting with profound anaemia and widespread gastrointestinal