BRITISH MEDICAL JOURNAL

SATURDAY 6 JANUARY 1968

LEADING ARTICLES

PAPERS AND ORIGINALS

MEDICAL MEMORANDA

MIDDLE ARTICLES

CORRESPONDENCE 53

NEWS AND NOTES

CURRENT PRACTICE

BOOK REVIEWS 41

OBITUARY NOTICES 60

SUPPLEMENT

No. 5583 Brit. med. J., 1968, Vol. 1, 1–68 Weekly. Price 5s.

BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON W.C.1 TEL: 01–387 4499

Correspondence

Letters to the Editor should not exceed 500 words.

Pulmonary Embolectomy

SIR,—Your leading article on pulmonary embolectomy (9 December, p. 566) gives helpful guidance covering the indications for an emergency operation. Confusion between pulmonary embolism and myocardial infarction will not be very frequent, and indeed failure to improve after an hour or two's intensive treatment with heparin, oxygen, digitalis, and pressor agents is a sound indication for operative intervention by surgical teams who are experienced in open heart surgery. The decision is difficult, for some such patients may still recover. Bedside consultation between physician and cardiac surgeon is obligatory.

The advice preferred for management of the less desperate case may, however, be much less wise. It is absolutely untrue that 30% of patients with pulmonary embolism suffer a second episode if adequately treated with heparin or oral anticoagulants.[1] Your reference for this statement dates back to 1942,[2] when anticoagulant treatment was just being introduced, and is as irrelevant as would be the reassessment of collapse therapy for pulmonary tuberculosis based on the literature of a quarter of a century ago. Similarly, Phear's paper[3] is misused to suggest that prolonged breathlessness is common after treated pulmonary embolism. Most of the patients to whom he referred had not received anticoagulant treatment or even been recognized in life as suffering from pulmonary embolism.

Likewise, treatment of thrombosis in leg veins should usually be medical rather than surgical. Sevitt's[4] classical study based on extensive post-mortem evidence showed the effectiveness of oral anticoagulant therapy in preventing venous thrombosis, and any claims for a different therapeutic approach have to be compared in a controlled fashion with this regimen rather than with the untreated disease.—We are, etc.,

D. W. BARRITT.
S. C. JORDAN.
Cardiac Department,
United Bristol Hospitals.

REFERENCES
1 Barritt, D. W., and Jordan, S. C., *Lancet*, 1960, 1, 1309.
2 Barker, N. W., and Priestley, J. T., *Surgery*, 1942, 12, 411.
3 Phear, D., *Lancet*, 1960, 2, 832.
4 Sevitt, S., and Gallagher, N. G., ibid., 1959, 2, 981.

Localization of Intracranial Tumours

SIR,—I quote from your leading article (16 December, p. 631) on localization of intracranial tumours: "If the patient shows signs and symptoms of grossly raised intracranial pressure it is usually considered safer to undertake a ventriculogram, but if these signs are absent more information can be obtained by undertaking a lumbar encephalogram."

Although the author is no doubt writing about patients in special units, nevertheless the sentence might be interpreted by some readers as inferring that lumbar puncture in cases of suspected intracranial tumour is a safe procedure unless there are signs and symptoms of *grossly* raised intracranial pressure. It should be emphasized that, unless in special units with facilities for emergency neurosurgical procedures, the only safe advice is that lumbar puncture should *never* be carried out if an intracranial tumour is suspected, even if signs and symptoms of raised intracranial pressure are absent. I would think there are few neurosurgeons who have not been presented with a comatose or moribund patient who, suffering from a nearly silent cerebellar tumour, has been precipitated into foraminal impaction by injudicious lumbar puncture.—I am, etc.,

COLIN GLEADHILL.
Department of Neurological Surgery,
Royal Victoria Hospital,
Belfast.

Budd-Chiari Syndrome after Oral Contraceptives

SIR,—We were interested to read the case report by Drs. K. Sterup and J. Mosbech (16 December, p. 660). We have been treating a patient whom we believed to have this syndrome and who improved after stopping oral contraceptives.

A 39-year-old housewife was admitted to Dudley Road Hospital under the care of Mr. L. W. Aldridge in December 1966. Three months before she had had an episode of abdominal swelling which lasted two weeks. It subsequently recurred and was accompanied by severe epigastric pain, nausea, and anorexia. Examination revealed a thin woman with a hard, tender, irregular mass in the epigastrium and moderate ascites. There was no splenomegaly. Abdominal carcinomatosis was suspected and laparotomy was performed. The liver was found to be enlarged and irregular, and looked cirrhotic. A biopsy was taken, and the patient was referred to us for further investigation and treatment.

She had had ten pregnancies and had eight living children. She had taken Anovlar (norethisterone and ethinyl oestradiol) for two and a half years, and on questioning said she thought it had "upset" her recently. Liver function tests showed: serum bilirubin 1 mg./100 ml.; alkaline phosphatase 35.7 King-Armstrong units; aspartate aminotransferase 5 i.u./l.; alanine aminotransferase 3.5 i.u./l.; thymol turbidity 1.1 units; albumin 2.52 g./100 ml.; globulins 4.38 g./100 ml.; gammaglobulin 1.66 g./100 ml.; bromsulphalein retention 26% at 30 minutes.

Wedge biopsy of the liver showed considerable fibrosis with disruption of the lobular architecture but no regeneration nodules. The sinusoids were markedly congested in some areas. There was an infiltration of the fibrous septa with lymphocytes and histiocytes. No lesions could be demonstrated in hepatic veins.

She was advised to stop the oral contraceptive pill, and her general well-being steadily improved without the use of diuretics