

- <sup>2</sup> Little, P. J., and Bailey, R. R., *New Zealand Medical Journal*, 1970, 72, 183.  
<sup>3</sup> Mahon, W. A., Wittenberg, J. V. P., and Tuffnel, P. G., *Canadian Medical Association Journal*, 1970, 103, 1031.  
<sup>4</sup> Fabre, J., Pitton, J. S., and Kunz, J. P., *Chemotherapy*, 1966, 11, 73.

### Hypoxic Newborn Infants

SIR,—The paper by Dr. M. A. Chadd and others (27 November, p. 516) appears to lend support to other work on the effects of birth asphyxia on haemostasis in the new born.<sup>1</sup> However, in fairness to themselves I feel that the authors should correct a number of inconsistencies which occur in their paper as published.

The study purports to be on full-term infants, but yet in Table I the mean gestation of the hypoxic group is given as 35.1 weeks and that of the controls as 35.5 weeks. Antenatal complications (Table I) are recorded as  $46 \pm 10.2\%$  (hypoxic) and  $17 \pm 7.8\%$  (control). Surely antenatal complications were or were not present in each case? There is no mention of what antenatal complications were recorded.

Rectal temperature below  $35^{\circ}\text{C}$  in the first 24 hours (Table I) is recorded as  $4 \pm 4.1\%$  for the hypoxic group and  $25 \pm 8.8\%$  for the control group. Again it is difficult to see what these figures mean and any interpretation put on them seems incompatible with the text statement that a rectal temperature below  $35^{\circ}\text{C}$  was detected in a greater proportion of hypoxic babies (as compared with control babies).

The statement in the discussion that this work suggests that "disseminated intravascular coagulation is a frequent factor of major importance in infants who die shortly after birth with haemorrhage" hardly seems justified in a paper devoted to babies with low Apgar scores in whom "there was no overt evidence of haemorrhage." Finally, the statement in the summary that "the consumption of clotting factors . . . led to the appearance of an haemorrhage diathesis" is at variance with the clinical findings reported.—I am, etc.,

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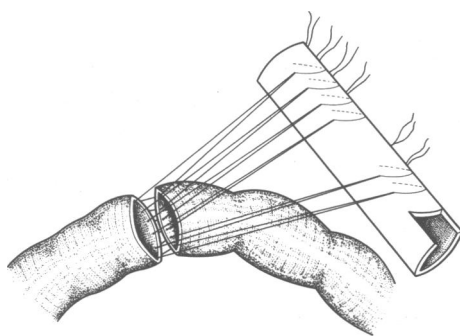
- <sup>1</sup> Chessells, J. M., and Wigglesworth, J. S., *Archives of Disease in Childhood*, 1971, 46, 253.

### Plastic Stitch Holder

SIR,—During the operation of anterior resection of the rectum interrupted stitches are usually used for anastomosing the bowel. The ends of each stitch are left long and held with artery forceps, to be tied at the end when all the stitches are in position. The assistant has to hold the forceps in order and on a slight stretch so that the thread does not get tangled or twisted.

A plastic stitch holder may be used for this purpose. This will leave the assistant's hand free. The stitch holder is pinned to the green sheet covering the patient, using a safety-pin and placed in a suitable position near the edge of the wound. The ends of each stitch are slipped in a slit in the stitch holder (Fig.).

The plastic stitch holder can be made from the disposable plastic container of a Foley's catheter; approximately 18-20 cm length from the sealed end of the plastic container is ideal for this purpose. The sealed



end is more suitable than the open end, because it is less malleable. Slits are cut in one of the rounded surfaces of the tube with a scalpel blade. Each slit should be slightly curved and slanting, or oblique so that the thread will not slip out from it easily.

I should like to thank Mr. D. V. Kneafsey for trying the device during operations and Dr. M. Bodkin for the original drawing.

—I am, etc.,

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### Size of Record Folder

SIR,—Dr. J. K. Hawkey and others (11 December, p. 667) favour the A4 folder, and their project, financed by the Department of Health and Social Security, was enhanced by a motion supporting the A4 size for general practice at the annual general meeting of the Royal College of General Practitioners on 20 November, as a reference to council.

The present M.R.E. (5 or 6) takes all sheets including quarto, or foolscap, or A4, folded once or twice only. The trouble in using it is not its size but the organization and methods of filing.

Some hospitals still fold letters to fit a window in the envelope to save typing time. This causes much unfolding and refolding when received, or none at all and stuffing in the M.R.E. as it is. There is no positive policy, such as described in the college's journal,<sup>1</sup> adopted by the Department of Health at executive council level, and such negative policies as there are (selective destruction, etc.) have never been published to show why the present M.R.E. was adopted in 1948, from its German original of 1910.

It needs expert attention to detail but is the right size and shape for practical politics for 50 million people.—I am, etc.,

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- <sup>1</sup> Jameson, M. J., *Journal of the College of General Practitioners*, 1966, 11, 336.

### Sickle-cell Trait and Altitude

SIR,—The diagnosis of intestinal infarction during flight in a Ghanaian nurse with positive sickling is so important that it is surprising to find that all Dr. R. L. Green and colleagues (4 December, p. 593) could offer by way of laboratory confirmatory tests was the statement: "Haemoglobin studies later showed that she was a sickle-cell trait carrier."

I would like to record for the information of your readers that unless satisfactory answers are given by the authors to the following questions the diagnosis of "intestinal infarction in a straightforward sickle-cell trait" is at best very shaky indeed.

(1) Apart from "acute abdominal pain" and "board-like rigidity of the whole abdomen" what were the other clinical features? Was there history of cold-season rheumatism? Was the patient jaundiced or clinically anaemic?

(2) How much of  $A_2$ , A and S haemoglobins were there? How did they exclude sickle-cell beta-thalassaemia?

(3) Was there a tendency to peculiar rouleaux formation of the red cells? What, in fact, were the haemoglobin level and platelet count? Was she polycythaemic?

(4) What about her G-6-PD? Was she deficient in this enzyme or normal?

While I wondered seriously how the three authors, who to my knowledge have never worked in Ghana, obtained such a poorly investigated case from Ghana, I recognized from the acknowledgements the name of a British surgeon who worked in the same hospital as myself. Could the authors please give further details about the patient described in Case I? Perhaps we could trace the patient and undertake a fuller investigation including a family study. This would help clear up a matter which has serious international repercussions.—I am, etc.,

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### Gastritis and L-Dopa

SIR,—Though the use of L-dopa in Parkinson's disease is now established in Britain its effectiveness is frequently limited by side effects, of which gastrointestinal disturbances are the most common. While gastrointestinal bleeding is not mentioned in most recorded series of patients on levo-dopa, Keenan's<sup>1</sup> report of five examples of this complication in the Eaton collaborative study of 485 cases suggests that it may be a rare sequel to L-dopa therapy. I have recently observed gastric bleeding in a patient taking this drug.

A man of 56, whose past history was unexceptional except for a myocardial infarction in 1958, developed Parkinson's disease in 1964. The disorder gradually progressed and by the time of his admission to hospital in 1970 he was severely incapacitated, and showed the typical face, voice, and posture of advanced Parkinsonism with rigidity and bradykinesia predominating. He was started on a small dose of L-dopa, which was gradually increased to 2 g a day, on which dose he was discharged together with orphenadrine which he had been taking for several years. Five weeks later he had an acute painless melaena. There was no evidence of either haemorrhage from other sites or a bleeding diathesis. Laparotomy revealed diffusely haemorrhagic and swollen gastric mucosa and a pyloroplasty was performed. The histological picture was that of a non-specific gastritis. He made an uninterrupted recovery and while continuing on orphenadrine was given amantadine in place of the levo-dopa. Five months later he died of a myocardial infarct. Postmortem examination of the stomach revealed that the previous inflammation had been replaced by diffuse mucosal scarring and submucosal fibrosis.