mothers had low serum α fetoprotein had left sided diaphragmatic defects: one died before surgery; two had surgery, of whom one died and one survived.

It has been suggested that low maternal serum α fetoprotein in congenital diaphragmatic hernia may be due to liver herniation into the chest resulting in decreased hepatic production or secretion of α fetoprotein.² Liver herniation occurred in only two of our three patients whose mothers had low serum α fetoprotein and therefore is unlikely to be the sole explanation of this observation. None of our three patients whose mothers had low serum α fetoprotein had antenatal diagnosis of congenital diaphragmatic hernia by ultrasonography. Our experience suggests that low maternal serum α fetoprotein has a definite but limited role as an adjunct investigation to ultrasonography in the antenatal diagnosis of congenital diaphragmatic hernia.

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Clinical monitoring of steatorrhoea in cystic fibrosis

SIR,—Walters and colleagues, comparing methods of assessing steatorrhoea in cystic fibrosis,¹ attributed the steatocrit test to Colombo.² The correct attribution is Phuapradit³ and so their claim to have used the method 'exactly as originally described' is not substantiated.

Phuapradit *et al* published the steatocrit as a method for assessing fat excretion in the newborn. If applied to the older child on a mixed diet, particularly if the subject is malabsorbing (the authors did not say whether or not their subjects were receiving pancreatic enzyme supplements during the study), the stool homogenisation technique becomes crucial. A fine tolerance Potter-Elvehjem homogeniser is essential for satisfactory dispersal of stool solids and the homogenate water content must be higher than that used by Phuapradit and colleagues. Once established, the conditions for homogenisation and centrifugation must be scrupulously observed. Walters *et al* thought them insufficiently important to give details of either step and it is not surprising therefore that serious technical difficulties were experienced. The steatocrit cannot be dismissed on this evidence.

The other semiquantitative stool fat method assessed was microscopy of stool homogenate after lipid staining (a subjective assessment but apparently not done blind). It produced a specificity of 55% or 46% depending on which of the two normal fat excretion limits quoted is accepted (20 mmol/day in the text, 17.5 mmol/day in figs 1 and 2). Most children with cystic fibrosis achieve normal or near normal fat excretion when given microencapsulated enzyme supplements but half such children

would be indicated for further investigation or increased enzyme dosage by the microscopy method. The authors' enthusiasm for the test is therefore not warranted and it would not, as was suggested, identify the non-compliant for there would be no way of differentiating the latter from a poor responder. Those who spend their working days contemplating stools understand the need for an alternative to the 72 hours fat excretion test. The 'bluebird' still eludes us.

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Drs Walters, Kelleher, and Littlewood comment.

Thank you for the opportunity of replying to the letter of Drs Brown and Booth relating to our recent publication.¹ We are grateful to them for pointing out our error in stating that the steatocrit method was carried out as originally described. In fact, what should have been stated was that the method was carried out as described by Colombo *et al*, though it must be stated that methodological details in that paper were indeed sparse.² All homogenisations, after addition of two volumes of water, were carried out in a Waring blender at 12 500 rpm for five minutes. Centrifugation details are given in our paper and both these conditions were standardised for the results published. As stated in our paper, various technical changes were attempted to improve the method, but without success, and we have no explanation for this. We have not dismissed the steatocrit method but would repeat that, whatever the reason, 'in our hands this method would not serve as a screening test'.

We do agree that microencapsulated pancreatic enzyme preparations greatly improve the control of fat malabsorption in cystic fibrosis. In the trials so far published, however, a considerable proportion of patients remain with appreciable steatorrhoea, and identification of these patients is important. In our clinic, where over 160 patients taking these enzyme preparations have had regular faecal fat assessments, about 20% remain with significant steatorrhoea. Whether continuing malabsorption is due to a poor response to treatment or non-compliance is difficult or impossible to assess, but the presence of continuing severe steatorrhoea, which will be reliably demonstrated by the simple microscopic screening test, will alert the physician to investigate both possibilities.

Happily we do not spend our working days contemplating stools, but we would agree that an alternative to timed faecal collections is needed, particularly in cystic fibrosis where regular monitoring of fat malabsorption may be required. However, in our hands the steatocrit method was certainly not the elusive 'bluebird'. The microscopic fat method does, however, have some flight in it and does

exclude severe steatorrhoea but neither this indirect method, nor any other presently available, will quantify the severity of fat loss and we would certainly not make this claim.

Patients who do not come¹

SIR,—Stamps are cheaper than visits. Instead of a further appointment, mothers can often be invited to write with news of the child, most effectively on an easily remembered day such as a birthday, half term, or Guy Fawkes, but at any time if there is concern. An addressed envelope, perhaps stamped, may be given as a reminder. The least literate letters are often the most informative, and no news is good news. The letter of thanks (from the doctor, not a functionary), can offer another appointment if necessary.

New patients who fail to come can be invited the next week by a personal letter asking them to confirm the booking by telephone so that it may be offered to another child if necessary. If there is no response, the referring doctor is told.

Paediatricians rarely have unplanned waiting lists for admission, but if they do, it is worth telephoning to ask if a proposed admission date is convenient before sending it.

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- 1 Andrews R, Morgan JD, Addy DP, McNeish AS. Understanding non-attendance in outpatient paediatric clinics. *Arch Dis Child* 1990;65:192-5.

Monitoring and sudden infant death syndrome

SIR,—We are disappointed with your recent article on home monitoring and sudden infant death syndrome.¹ On average between five and 10 infants at risk of sudden death are referred each week to our department by their consultant paediatrician or family practitioner for investigation and management. The latter includes a home monitoring programme that involves the use of transcutaneous oxygen pressure (PO₂) monitors and this is well known to the Foundation for the Study of Infant Death and to the other authors of your article. We have presented our results at national (paper presented at joint meeting of British Paediatric Respiratory Group and Foundation for Study of Infant Death, Liverpool 16 September 1988) and international² conferences concerning sudden infant death.

The single 'throw away' sentence in the article dismissing transcutaneous PO₂ monitoring in the home is not based on experience but on prejudice following its use in neonatal units. In contrast, we have experience in over 250 infants with the use of this monitor in the home for periods up to 13 months (mean (SD) 6.4 (2.6) months). Unlike breathing movement detectors, this monitor is 100% sensitive to hypoxaemic episodes. It has infrequent false alarms (one every six days) compared with an average of six per day for monitors based on impedance pneumography and electrocardiography.³

In our experience, the PO₂ monitor does not cause skin burns (sensor temperature 43°C, recommended resting interval eight hours), has a median total response time of 21