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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- 1 Joint study group on fetal abnormalities. Recognition and management of fetal abnormalities. *Arch Dis Child* 1989;64:971-6.
- 2 Resta DG, Luthy DA, Karp LE, Hickok DE. Low maternal serum alpha-fetoprotein levels and congenital diaphragmatic defects. *American Journal of Genetics*, 1987;26:991-44.
- 3 Glick PL, Pohlson GC, Resta R, et al. Maternal serum alpha-fetoprotein is a marker for anomalies in paediatric surgery. *J Pediatr Surg* 1988; 23:16-20.

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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- 1 Walters MP, Kelleher J, Gilbert J, Littlewood JM. Clinical monitoring of steatorrhoea in cystic fibrosis. *Arch Dis Child* 1990;65:99-102.
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- 3 Phuapradit P, Narang A, Mendonca P, Harris DA, Baum JD. The steatocrit: a simple method for estimating stool fat content in newborn infants. *Arch Dis Child* 1981;56:725-7.

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

seconds to hypoxaemia (oxygen saturation less than 60%) and baseline transcutaneous PO₂ values are reproducible from siting to siting. In addition this monitor may also detect other potential causes of life threatening events where there is peripheral vasoconstriction due to metabolic shock or low cardiac output, although this function has yet to be fully validated.

Pulse oximetry was given more support than transcutaneous PO₂ in your article. In our experience, the former is much too prone to movement artefact to use at home. Excessive alarms are common and occur when the infant has normal body movements (false positives). Of more concern is the failure to alarm which may occur when these movements arise in association with cyanotic episodes (false negatives).⁴

All infants in our programme are supervised closely by doctors and clinical nurse specialists. The project is funded by three charities as well as the Department of Health and the National Heart and Chest Hospitals. The Foundation for the Study of Infant Death is only one of a number of charities that help parents and supports research into the mechanisms responsible for sudden infant death. In our opinion, the views given in your article on home monitoring should have been balanced by the experience of charities and departments who have used transcutaneous PO₂ monitors in the home.

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- 1 Davies PA, Milner AD, Silverman M, Simpson H. Monitoring and sudden infant death syndrome: an update. *Arch Dis Child* 1990;65:238-40.
- 2 Samuels MP, Levene P, Southall DP. Home monitoring of transcutaneous PO₂ in the management of infants at increased risk of sudden death. *Pediatr Pulmonol* 1989;7:280.
- 3 Weese-Mayer DE, Brouillette RT, Morrow AS, Conway LP, Klemka-Walden LM, Hunt CE. Assessing validity of infant monitor alarms with event recording. *J Pediatr* 1989;115:702-8.
- 4 Samuels MP, Southall DP. Oxygen monitoring in paediatric practice. *Intensive Care World* 1989;6:140-3.

Drs Davies, Milner, Silverman, and Simpson comment:

Dr Southall and his colleagues have missed the point. Our report 'Monitoring and sudden infant death syndrome: an update' considered the published information that was available to us at the time, and attempted to relate that information to the topic of sudden infant death. Their letter has failed to provide any objective evidence, as opposed to opinion, that transcutaneous oxygen monitoring is more effective than pulse oximetry.

Early diet in preterm babies and developmental status in infancy

SIR,—We would like to comment on the analysis of the data presented by Lucas *et al.*¹ The article purports to show that developmental status is affected by diet, with a disadvantage being found in the group given human donor milk when compared with a group getting preterm formula. Their conclusion,

however, was based only on the analysis of those babies who were not neurologically impaired. It is unsatisfactory to exclude the neurologically impaired babies in an analysis of developmental outcome. This is especially so as there were more neurologically impaired babies in the group fed preterm formula. The difference between the developmental quotients of the two groups was no longer significant when the neurologically impaired babies were added.

The manufacturers are keen to quote this article to support their promotion of preterm formula. Perhaps the most important bit of this paper is the demonstration that there is absolutely no difference between the group fed only breast milk and those fed only preterm formula.

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- 1 Lucas A, Morley R, Cole TJ, *et al.* Early diet in preterm babies and developmental status in infancy. *Arch Dis Child* 1989;64:1570-8.

Drs Lucas and Morley comment:

Drs Bodden and Williams suggest that exclusion of neurologically impaired infants from our analysis invalidated the results and our most important finding was a negative one. We challenge these views most strongly.

Assessment of cognitive development in 9 month old babies is highly dependent on age appropriate motor skills, and overall developmental quotient (DQ) can be worthless in children with, say, cerebral palsy. Of such impaired children now assessed at 7-8 years, mean DQ at 9 months was <3rd centile (around 70), yet subsequent mean verbal intelligence quotient was on the 50th centile and up to the 80th. There was no significant difference in the incidence of neurological impairment between feed groups; indeed it seems implausible that a diet (preterm formula) that promoted improved developmental scores should also cause frank neurological damage.

Clearly inclusion of invalid test scores is likely to obscure valid findings. Nevertheless, we wish to reassure Drs Bodden and Williams that for the major subgroup analyses (small for gestational age infants and those consuming >50% of intake as trial diet), differences between diet groups were robust enough to remain significant when neurologically impaired children were included (unpublished).

As pointed out, in one limb of our trial, including only 30% of the cohort, a significant dietary effect did not emerge. Yet there were clear and potentially important dietary effects over the trial as a whole. This just emphasises the need for adequate trial size, particularly in an unselected cohort, as in our study, where so many other factors, including low consumption of the trial diet itself, operated against detecting any dietary effect. That such an effect emerged, despite great variation in clinical course and social background of the infants, provides compelling evidence that early diet in premature babies does indeed influence later development, and our unpublished data obtained at 18 months reaffirm this view.

Access for peritoneal dialysis in neonates and infants

SIR,—Lewis *et al* describe the use of a tapered polyurethane catheter which can be introduced into the abdomen over a 'j' guide wire by the Seldinger technique to provide access for acute peritoneal dialysis in small infants.¹ They argue that the method of introducing the cannula makes the technique safer than using a stylet or trocar, but describe a high incidence of problems including the death of one baby after the catheter eroded through the gut wall.

We agree that the Seldinger technique is the safest method of introducing peritoneal dialysis catheters at any age, but we believe the Tenckhoff catheter which we use has major advantages over other designs.² Although Lewis *et al* describe the 'Pendlebury' catheter as being 'soft and pliable', it is designed to be stiff enough to be 'forced through the skin with a "screwing" action' and it tapers to a tip of about 1.5 mm diameter. By contrast, the Tenckhoff is a very soft Silastic catheter with no taper; the risk of it causing perforation must be negligible. They are designed for permanent peritoneal dialysis and are available as 16 French gauge (FG) 'straight' catheters with side holes extending to between 3 and 15 cm from the tip, and as 'curled' catheters with holes extending 20.5 cm along a helical end of approximately 6 cm diameter.

Although Tenckhoff catheters are usually inserted surgically,² they may be introduced percutaneously using a technique similar to that described by Lewis *et al* but with some modifications. We apply local anaesthetic (as EMLA cream, Astra) to the site one hour before the procedure which is done with sedation, analgesia, and infiltration of local anaesthetic. A small skin incision is made using a number 15 scalpel blade used vertically to produce a cut of no more than 3.5 mm, to avoid leakage. The opening is then gently dilated using artery forceps. It is not necessary to use two needles in order to introduce fluid and pass a wire as Lewis *et al* describe, as there are cannulas that are designed to fulfill both roles (two part needle, Kimal). With about 30 ml/kg fluid in the peritoneum the peel away sheath and its introducer are simply inserted into the abdomen in a similar way to the Pendlebury catheter. The sheath itself is a thin walled plastic outer catheter rather like a drinking straw which has a longitudinal groove each side along its inside surface, and two moulded wings at the top. Once the introducer is in the abdominal cavity the sheath can be slid onward over its tip, and the introducer and the wire removed. The Tenckhoff is then slipped through the lumen of the sheath into the abdomen. At this point the wings of the sheath can be snapped apart and the two halves of the sheath easily torn apart along their length. The Tenckhoff is advanced simultaneously, until the sheath is pulled completely out of the patient as two separate halves.

Tenckhoff catheters are available with and without Teflon cuffs which are designed to be buried beneath the peritoneum and in subcutaneous tunnels, when used as a permanent catheter. We use a cuffed design to aid safe anchorage of the catheter by inserting a skin stitch about 1 cm from the entry site and tying the thread around the cuff (which remains outside the patient); this provides a firm grip for the tie. The catheter can then be cut to any convenient length and the connector inserted.

We have used standard (16 FG) diameter Tenckhoffs in newborns, but not in the