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.

M P SAMUELS
C F POETS
D P SOUTHALL
National Heart and Lung Institute,
Dovehouse Street,
London SW3 6LY

- 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.

L H P WILLIAMS
S J BODDEN
Bassellaw District
General Hospital,
Kilton,
Workshop,
Notts S81 0BD

- 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