Correspondence

Parenteral nutrition compared with transpyloric feeding

Sir,

We should like to comment on two recent articles on the feeding of low birthweight infants. The paper by Yu *et al*¹ was a retrospective case-control study of perinatal risk factors for necrotising enterocolitis, which the authors refer to as a 'controlled study'. The authors state that the timing, type, and volume of milk feeding were not different in infants with necrotising enterocolitis and matched controls. This is scarcely surprising, since during the relevant years (1979 and 1980) 'there was a standard feeding regimen for very low birthweight infants'. Unless there are at least two different feeding regimens, no study will tell us which regimen is the less hazardous. Could the slight increase in the incidence of necrotising enterocolitis during the study have been caused by a trend to earlier feeding?

The paper by Glass *et al*² is a prospective, controlled trial of two feeding policies: parenteral *v* early transpyloric feeding. Unfortunately there are three grave defects to this study. Firstly, the two groups of infants are not truly comparable: whereas one third of the 'parenteral' group were outborn, this is true of only one tenth of the 'enteral' group. As one would expect, the parenteral group had the worse outcome—perhaps because these infants are at much greater risk of hypothermia³ as well as probably being sicker infants in the first place. This fact could fully account for the apparent difference in outcome between the two feeding policies.

The second defect is that the two groups entered into the trial do not have their outcomes compared directly. Instead, the enteral group is divided into two subcategories. Only the 'successful' enteral subcategory was consistently superior to the parenteral group: had the analysis compared the two initial groups, the conclusions might have been different. The whole purpose of a study such as this should be to help predict which infants will both tolerate early feeding and avoid necrotising enterocolitis.

The third defect in this study is that neither of the feeding regimens 'on trial' could ever sensibly be adopted as a uniform policy by any special care baby unit for all its infants of less than 1.5 kg. Thus:

- (1) Fluid input was built up to 200 to 225 ml/kg/day which may be considered excessive, with risks of patent ductus arteriosus and necrotising enterocolitis as well as congestive cardiac failure.^{3 4}
- (2) Many of the infants in the 'parenteral' group must have had central and peripheral intravenous lines as well as gastric and duodenal tubes, and perhaps arterial lines as well. We are not surprised at the high rate of sepsis, and do not think that many units would wish to subject all their very low birthweight

infants to this risk. Intravenous feeding can be performed without the use of central venous lines.

- (3) There is no one policy that will apply to all very low birthweight infants: the severely asphyxiated 800 g infant who has severe respiratory distress syndrome and persistent apnoea may not tolerate enteral feeds for weeks or months. On the other hand, a relatively mature but growth retarded infant of 1.4 kg may tolerate feeds from the first day of life. Any feeding policy must recognise that these infants are very different: any study of feeding practices must do so too.

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Dr Glass and co-workers comment:

As pointed out in our paper² patients were allocated alternately to groups and chance determined that more of the parenterally fed group were outborn than inborn. This does not carry the implication which Clarke *et al* seek to put on it. Our outborn infants came from nearby maternity units linked with the Simpson Memorial Maternity Pavilion and served by an integrated neonatal paediatric staff based on the regional neonatal referral unit in the Simpson. These outborn infants were transferred immediately after birth in transport incubators with paediatric staff in attendance. They did not suffer from hypothermia. In these circumstances, weight for weight, the outcome for inborn and outborn infants is similar. Further, in the patients reported, adverse complications were not confined to one group.

Dr Clarke and his colleagues do not seem to have read our paper properly. Table 4 analyses the two initial groups indicating the relative mortalities and infection, necrotising enterocolitis, and hyperbilirubinaemia rates. In respect of the long term comparisons of growth we did not think it necessary to explain why this had to be confined to those who had survived.

Amid the complexities of neonatal care for infants of very low birthweight it is surely somewhat naïve to imply that there can be a 'uniform policy' on feeding for all such infants. We were not advocating any policy. We were examining recognised methods of feeding low birthweight infants critically, with a view to understanding better their drawbacks and their advantages. We commented on the fact that a high fluid intake could be associated with an increased risk of necrotising enterocolitis in the context of the paper by Bell *et al*³ which Dr Clarke and his colleagues quote. At the same time judgement on an appropriate fluid intake for low birthweight infants must take account