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 hypothermia\(^3\) 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 paper\(^5\) 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 naive 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.\(^1\) which Dr Clarke and his colleagues quote. At the same time judgement on an appropriate fluid intake for low birthweight infants must take account...
of all the factors linked to hydration, whether over- or underhydration. In the era of delayed feeding in the 1950s neurological and intellectual handicap were the all too frequent outcome of a regimen which often led to underhydration. Necrotising enterocolitis and patent ductus arteriosus are not the sole hazards of low birthweight.

Our paper refers to the ordered and selective use of central and peripheral venous lines, nasogastric, and nasoduodenal feeding. It is difficult to understand why Dr Clarke and his colleagues seem anxious to imply a casual and excessive use of these techniques and then to dissociate themselves from their own misrepresentation.

References

Hyponatraemia in preterm infants—arginine vasopressin secretion

Sir,

We read with interest the report by Rees et al on the role of excessive arginine vasopressin secretion and subsequent water retention in the development of early hyponatraemia in sick preterm infants. 1

We would like to point out that the increased rate of arginine vasopressin secretion may also be implicated in the aetiology of late hyponatraemia frequently seen in healthy low birthweight preterm infants, which was thought to be due to renal salt wasting.

In a recent study we measured simultaneously the urinary excretion of aldosterone and arginine vasopressin along with sodium balance, plasma and urine sodium, and osmolality in nine healthy preterm neonates during the first five weeks of life (Sulyok et al, unpublished data). It could be shown that both urinary aldosterone and arginine vasopressin excretion increased with advancing postnatal age from the initial values of mean (SD) 0-94 (0-16) µg/day and 0-38 (0-08) ng/day in the first week to a maximum of 4-30 (0-76) µg/day and 1-19 (0-26) ng/day (P<0-01), respectively in weeks 4 and 5, in spite of the declining plasma sodium concentration. Moreover, significant positive correlation was found between urinary aldosterone and arginine vasopressin excretion in seven of the nine infants studied.

On the basis of these observations it seems to be relevant to assume that in salt-losing preterm infants the increasing rate of aldosterone and arginine vasopressin secretion occurs in response to the same stimulation, that is to the protracted contraction of extracellular fluid compartments. The higher arginine vasopressin secretion rate results in more efficient water reabsorption and may contribute to restoring body fluids to normal. In support of this assumption antipyrine and bromide space studies by Roy et al did not show any difference in the fluid compartments in preterm infants with or without late hyponatraemia. 2

References

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Dr Shaw comments:

Drs Kovács and Sulyok draw attention to an important point. It has been generally believed for many years that in sodium depletion the development of hyponatraemia represents an appropriate secretion of arginine vasopressin due to a fall in blood volume and mediated through atrial volume receptors. There have, however, been few published data to support this proposition, and none in preterm babies. It is therefore valuable that their data support the current theories. It is true, as they say, that an 'increased rate of arginine vasopressin secretion is implicated in the aetiology of late hyponatraemia . . .', but it must be emphasised that the increased secretion of arginine vasopressin is, as far as we know, an entirely physiological response to volume contraction and it should not be thought that late hyponatraemia is caused by water overload due to inappropriate release of arginine vasopressin. It is usually due to sodium depletion and should be treated with sodium supplements, not water restriction.

Urinary albumin excretion in school children

Sir,

We read with interest the recent paper on urinary albumin excretion in normal children, 1 which has provided valuable normal ranges for this measurement. The authors quote their results both as albumin excretion rates and urine albumin:creatinine ratio. We note that the authors had to