considering the criteria involved, one cannot avoid this thorny problem of the quality of life.

If Dr McGucken accepts that he faces taxing ethical problems in neonatal practice, then he must have some concept of a life so awful and so full of misery that it would be morally wrong to support it intensively. It is merely a matter of an alternative definition. I cannot believe that because of the experience of rewarding relationships between parents and their handicapped child he thinks life should be preserved whatever the cost.

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Serum albumin concentrations and oedema in the newborn

Sir,

The paper by Cartlidge and Rutter contains much valuable information on albumin concentrations and oedema in the preterm neonate.¹ Early (<7 days) oedema does not usually cause undue concern if attention is focused on fluid and electrolyte balance as part of the overall care at that stage.

It is the occurrence of later oedema in the relatively well, rapidly growing preterm baby that is the more interesting physiological (?) phenomenon, even if it is the less worrying clinically. The authors reported a significant relation between albumin concentration and oedema, but with a poor correlation. No relation between nutritional state and albumin concentrations existed at 3 weeks of age. Which measures of nutritional state were used to support this statement?

It is tempting to find similarities between this late neonatal oedema and some forms of nutritional oedema. The role of protein deficiency and secondary hypoalbuminaemia as the principal cause of oedema in kwashiorkor has been questioned.^{2,3} Some malnourished children develop oedema during nutritional recovery at a time when their albumin concentrations are rising.⁴

If these phenomena are paralleled in preterm babies oedema and hypoalbuminaemia in the late neonatal period may reflect nutritional inadequacies. Dismissal of the nutritional state, particularly if assessed by anthropometry alone, may be premature. Thus the excellent data presented should encourage us to look for nutritional problems rather than dismissing them.

Eventually, only meticulous balance studies on energy, protein, and 'accessory nutrients'³ may lead to an understanding of which, if any, of these dietary factors is deficient in the growing baby with oedema. It would be interesting to know whether Drs Cartlidge and Rutter found hypoalbuminaemia and oedema (independently) to be related to previous nutrition in these babies. Did the type of milk used or the parenteral route of feeding have any influence on the incidence of these problems?

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Sir,

The recent paper by Cartlidge and Rutter is an important objective attempt to try to find whether hypoalbuminaemia is a cause of oedema in the preterm infant.¹ The study oversimplifies the problem and cannot justify the conclusion that plasma albumin measurement in preterm infants is not helpful. The causes of oedema in an acutely ill infant with severe respiratory distress are likely to be different from those in an infant several weeks old requiring total parenteral nutrition. Did the authors analyse such groups separately? Plasma albumin is just one factor influencing oedema formation; others include fluid intake, fluid output, and capillary leakiness. If a low plasma albumin concentration is found in conjunction with oedema plasma albumin concentration is one factor that can be influenced. In my experience oedema in babies of more than 2 weeks of age with a plasma albumin concentration of less than 20 g/l often respond to an albumin infusion with a loss of oedema. I would therefore suggest that measurement of plasma albumin concentration should not be abandoned as suggested by the authors.

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Drs Cartlidge and Rutter comment:

We agree with Dr Miller that the cause of oedema in the two situations he cites is likely to be different; we found no correlation, however, between oedema and hypoalbuminaemia in well and ill infants during both the early and late neonatal periods. As the mean arterial blood pressure is lower in preterm infants than in adults it is likely that capillary hydrostatic pressure is also considerably less. It is therefore not surprising that the preterm infant can tolerate hypoalbuminaemia without becoming oedematous. In view of this, we find Dr Miller's experience with albumin infusions surprising. Indeed, if capillary leakiness is important in oedema formation as he suggests the albumin given would merely leak into the interstitial fluid and oedema may thereby be increased.

Dr Watkinson makes an interesting suggestion, which links late onset oedema to poor nutrition. The nutritional state of 3 week old infants in our study was determined by their weight. They were divided into three groups depending on whether their weight was less than 95%, between 95% and 105%, or more than 105% of their birth weight. There was no significant difference in serum albumin concentration between these groups. Oedema was not influenced by the type of nutrition received, but serum albumin concentrations tended to be lower in those infants fed parenterally.

References

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