Correspondence

institution of total parenteral feeding during the first 2 or 3 weeks of life is owing to the tendency of the disorder to appear in clusters is a misjudgement; the simultaneous increase in the incidence of the disease in preterm infants with weight >1500 g (in whom no change in feeding policy was made) rules out this explanation. Unfortunately, Richard Cooke has failed to reduce the incidence of NEC in his unit, despite ‘more extensive use’ of parenteral nutrition. This may be owing to the high prevalence of the disease in the preterm infants in whom this ‘extensive use’ was abandoned.

It is true that some very low birthweight infants develop NEC once enteral feeding takes over the initial regimen of parenteral nutrition, but this occurrence is rare and the severity of this late onset disease seems less. Concerning the volume of feed and rate of increase these were already demonstrated, retrospectively and prospectively, not to be significant factors in the pathogenesis of NEC.  

References


Sir,

In their historically controlled trial of parenteral feeding in the very low birthweight infant Eyal et al.1 claim that an observed decrease in the incidence of NEC between 1977 and 1979 from 18·2% to 3·5% was unlikely to be explained by ‘the epidemic cluster of cases characteristic of some outbreaks’ and was owing to the change in feeding policy. They stated that the incidence of NEC in preterm infants with birthweights above 1500 g, in whom there was no change in feeding policy, was increased during the trial period, but they did not provide any relevant figures.

In 1981 at Southmead General Hospital where it is our policy to feed very low birthweight infants enterally except in the presence of tachypnoea, respiratory support, or failure to tolerate such feeding, the incidence of NEC in such infants was only 3% (2 out of 64) which coincides with the ‘good year’ of Eyal et al. However, large variations in incidence of NEC have been observed here from year to year, as is common experience.

The duration of parenteral feeding recommended by Eyal et al. is based on the assumption that infants are particularly vulnerable for NEC in the first 2 or 3 weeks of life. In the UK surveillance programme for NEC (Communicable Disease Surveillance Centre, 1980, unpublished report) the mean age of onset in 56 infants of very low birthweight was 20 days, and 61% of them developed NEC after 14 days. In the series of Bunton et al.2 out of 5 infants of very low birthweight developing NEC did so after 21 days of age. Stoll et al.3 reported an inverse relationship between the age of onset of NEC and gestational age at birth with a mean age at diagnosis in the 26–30-week gestational age group of 20–2 days. Both our Southmead very low birthweight infants developing NEC in 1981 were more than 3 weeks old. It appears that Eyal et al.’s introduction of enteral feeding between 14 and 21 days for 1001–1500 g infants was during their period of maximum risk.

When assessing the introduction of such a major change in management, changes in other parameters of morbidity should be considered. However, Eyal et al. do not mention the incidence of problems in their patients on parenteral nutrition. The danger of bacteraemia may be reduced by using peripheral lines but serious biochemical and technical problems remain. We are concerned by the withholding of enteral nutrition at a time of possible critical brain growth, and the implications of such withholding on gut hormone production and gut growth are only now being discovered. Therefore it is very disappointing that although it was felt justifiable to subject a large number of infants prophylactically to prolonged parenteral nutrition, a randomised controlled trial was not carried out to evaluate the new policy. Without such evidence Eyal et al.’s experience is most likely explained by the well-known epidemic clustering of NEC.

References


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