A silent grunt for neonatal respiratory distress syndrome (or what's this EER?)

Sir,

Grunting in neonatal respiratory distress syndrome (RDS) appears to be a form of variable expiratory resistance. Simply inserting an endotracheal tube into an infant with acute RDS, and thus removing the grunt, may cause a marked deterioration in clinical condition.[1] Moomjian *et al.*[2] have completed the circle, replacing the grunt in their intubated babies, by an external expiratory resistance (EER). Perhaps extubation would have been simpler and equally satisfactory.

As well as questioning the rationale for the experiment, I would question some of the results. The authors state that the 'mean net work of breathing' was unaffected by EER. This is a fallacy, since the method usually used for calculating work of breathing does not take account of the shift in the pressure-volume loop, as lung volume is altered. When lung volume is increased by the application of EER, the increase is achieved by muscular effort on the part of the baby (and not, as in CPAP, by any external distending force). This extra effort, which may be considerable at high lung volumes, is not represented in the 'work of breathing' term. It is thus misleading to conclude that, because the 'work of breathing' was unaltered, the respiratory muscles were under no more stress. Indeed, the extra respiratory muscle load imposed by EER could merely hasten the onset of muscle fatigue in this susceptible group of infants.[3]

Clearly, without more appropriate measurements of lung mechanics, gas exchange, and respiratory muscle function, it would be premature to draw any conclusions concerning the clinical value of EER.

References

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Parenteral feeding and necrotising enterocolitis in very low birthweight infants

Sir,

Eyal *et al.*[1] believe that they have caused a reduction in the incidence of necrotising enterocolitis (NEC) in their hospital by the more extensive use of parenteral nutrition in very low birthweight infants. Unfortunately, the well-known tendency for cases of NEC to occur in clusters makes the 'historical' controls they have used particularly unsuitable for this kind of study.

A similar change in management of very low birthweight infants occurred here in 1980 and 1981. During the first year parenteral nutrition was not often used, only if enteral nutrition was impossible. Six cases of NEC among 132 very low birthweight infants occurred; in the second year parenteral nutrition was used much more extensively and the infants were not fed enterally while ventilated, suffering from major recurrent apnoea, or tachypnoea exceeding 60 breaths a minute. Thirteen cases of NEC occurred among 181 very low birthweight infants none of which happened while the infant was receiving parenteral feeding, but symptoms appeared within a day or two of reaching full volume enteral feeds of fresh human milk or 'humanised' formula. Onset of the disease occurred between 1 and 8 weeks after birth; thus, although withholding enteral feeding protected against early NEC, the effect was to delay rather than prevent the disease. A small increase in incidence was seen but this could have been owing to the sporadic nature of the disease.

In most cases NEC is undoubtedly dependent on the presence of milk in the gut, but the need or desirability for total avoidance of enteral feeding in early weeks in very low birthweight infants is still far from proved. Provided that a low osmolality, well absorbed feed is used its exact nature may not be important; the total volume and rate of increase of volume of feed may be more relevant. Concern that poor weight gain may have later consequences in terms of brain development prompts many neonatal units to overload the capacity of the gastrointestinal system of these tiny infants. A combination of enteral and parenteral feeding may allow the optimal solution by allowing a very slow increase in enteral load during the first 2 or 3 weeks with adequate calorie intake. In a large hospital the use of such a conservative feeding regimen has already been shown to reduce the incidence of NEC to insignificant levels without the problems of wholesale parenteral feeding.[2]

References

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Dr Eyal and co-workers comment:

The suggestion that the sharp reduction in the incidence of NEC in our very low birthweight infants after the

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)[1] 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.[2]

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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] 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.

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