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Nutrition and bronchopulmonary dysplasia

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Abstract

Twenty two babies who developed bronchopulmonary dysplasia were compared with 22 babies matched for gestational age who did not. Those with bronchopulmonary dysplasia weighed less at birth and had lower energy intakes from day 7 to day 56. Undernutrition before and after birth is a major problem in babies who develop bronchopulmonary dysplasia.

Advances in neonatal intensive care such as surfactant replacement have led to increased survival rates among very small preterm babies with respiratory distress syndrome, but many of these babies develop bronchopulmonary dysplasia.[1] One of the reasons for this may be that prolonged mechanical ventilation is required because these babies are very small and may be undernourished. We therefore decided to compare energy intakes over the first two months of life in a group of babies who developed bronchopulmonary dysplasia with a group of babies matched for gestational age who did not.

Patients and methods

From January 1987 to December 1988, 129 very low birthweight (VLBW) infants were born in, or transferred on the first postnatal day to, this hospital. Thirty two babies were either transferred back to their referral unit before the age of 30 days or died. Clinical details, serial measurements of energy intake, and outcome data were obtained for the remaining 97 infants. Energy intakes were recorded from medical and nursing records, and are actual amounts taken rather than those prescribed. Statistical analysis was by the Mann-Whitney U test and the χ^2 test as appropriate.

Bronchopulmonary dysplasia was diagnosed if the babies were dependent on oxygen for 28 days or longer and on radiological findings.[2] Twenty two infants who developed bronchopulmonary dysplasia were compared with 22 infants matched for gestational age who required mechanical ventilation for more than three days for respiratory distress syndrome but who did not develop bronchopulmonary dysplasia.

Results

Table 1 shows the characteristics of the two groups of infants. Those who developed bronchopulmonary dysplasia weighed significantly less at birth, indicating pre-existing intrauterine undernutrition. There were no significant differences in the incidences of patent ductus arteriosus or pulmonary air leaks (pulmonary interstitial emphysema or pneumothorax).

From 10 days onwards the energy intake of the babies with bronchopulmonary dysplasia was significantly lower than that of the control babies (table 2). Infants who developed bronchopulmonary dysplasia started enteral feeds significantly later than the control babies (median (range) times of 19 (6–71) and 9 (3–39) days, respectively). They required a median of 88 (37–130) days to reach and maintain full enteral feeds, compared with 47 (20–120) days for the control babies. Intravenous feeding was required for a median of 75 (29–126) days compared with 39 (14–111) days, and lipid emulsion was given for a median of 44 (0–123) days compared with 22 (3–95), respectively. These values are all significant ($p<0.05$).

Table 2 Mean (SD) energy intake (kJ/kg/day)

Day	*Babies with bronchopulmonary dysplasia (n=22)*	*Babies without bronchopulmonary dysplasia (n=22)*
7	278·8 (72·7)	322·3 (94·9)
10	270·9 (65·2)**	357·4 (66·9)
14	321·9 (61·0)*	367·8 (60·6)
28	357·0 (53·9)	387·5 (81·1)
56	364·5 (66·0)**	455·6 (63·5)

*p<0·05, **p<0·001.

Table 1 Clinical characteristics and outcome of babies requiring mechanical ventilation for respiratory distress syndrome

	Babies with bronchopulmonary dysplasia (n=22)	*Babies without bronchopulmonary dysplasia (n=22)*
Mean (SD) gestational age (weeks)	26·9 (1·7)	27·4 (1·7)
Mean (SD) birth weight (g)	839 (229)*	1015 (170)
No (%) of babies small for gestational age	8 (36)	5 (23)
No (%) of boys	12 (55)	9 (41)
Median (range) duration of supplementary oxygen (days)	109 (33–268)**	37 (9–97)
Median (range) duration of assisted ventilation (days)	68 (19–206)**	19 (4–86)
No (%) who developed patent ductus arteriosus	10 (45)	15 (68)
No (%) who died	5 (23)	0

*p<0·05, **p<0·001.

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Discussion

Undernutrition is a serious problem in VLBW infants who develop bronchopulmonary dysplasia.[3] We have found that this may begin before birth and have a significant effect on the energy reserves of the very preterm infant. The appropriately grown infant weighing 1000 g at birth has a body composition containing less than 2% fat, compared with 16% at full term.[3] Intrauterine undernutrition, which occurred in the babies who developed bronchopulmonary dysplasia, lowers these energy reserves still further.

Poor energy reserves will be exacerbated by poor energy intake after birth. The American Academy of Pediatrics has recommended that a heathly preterm infant should receive an energy intake of 501·6 kJ (120 kcal)/kg/day.[4] Why have we had such difficulty in reaching this figure? After analysis of our data we think that there are several reasons for this. There is often a need to restrict fluids in critically ill babies, in those with patent ductus arteriosus, and in babies with established bronchopulmonary dysplasia. These infants also commonly will not tolerate standard dextrose infusions. Furthermore, lipid infusions are often delayed and there may be frequent periods of lipid free feeding because of concerns about respiratory function, hyperbilirubinaemia, and sepsis. Our policy is to delay the start of enteral feeding until the infant's vital signs are stable, ventilator settings are being reduced, and umbilical arterial catheters have been removed. Delay in achieving full enteral feeds often occurs in VLBW infants because they have abdominal distension or features suggestive of necrotising enterocolitis. Finally, there may be technical problems such as the length of 'down time' between replacement of intravenous cannulas.

It is also probable that there are increased metabolic demands in sick preterm infants that require mechanical ventilation, although these have not been measured. Studies in oxygen dependent infants with bronchopulmonary dysplasia, however, have shown that they have an increase in energy expenditure of 25%.[5]

Recently, Frank hypothesised that the early provision of adequate nutrition may be a key prophylactic measure for limiting the risk of development of bronchopulmonary dysplasia.[3] Factors thought to be important in the pathogenesis of bronchopulmonary dysplasia—hyperoxia, barotrauma, infection, and pulmonary immaturity—are all adversely affected by undernutrition. If inspiratory muscles are deprived of substrate the resultant fatigue will lead to failure of the respiratory muscle pump. Undernutrition has been shown to alter ventilatory muscle function in animals and man.[6] Improving nutritional state allows more rapid weaning of seriously ill adult patients from mechanical ventilation but similar studies have not been carried out in babies.

Our study shows that in both the acute phase when bronchopulmonary dysplasia is developing and in the chronic phase when infants with bronchopulmonary dysplasia remain dependent on the ventilator there is evidence of undernutrition. Intrauterine growth retardation, poor energy reserves, and increased metabolic demands are also important. A prospective randomised trial is needed to find out if improvement in nutritional state will reduce the incidence and severity of bronchopulmonary dysplasia.

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