Review article

The first feed of low birthweight infants

Changing attitudes in the twentieth century

D. P. DAVIES

From the Department of Child Health, Leicester Royal Infirmary

For low birthweight (LBW) infants there can surely be few more important events in life than the first feed after birth. Few paediatricians would now disagree that LBW infants need to begin feeding within the first few hours of delivery. The modern awareness of this practice dates from 1964 when Smallpeice and P. A. Davies published their important paper describing the short-term benefits of early feeding, and it is now well known that this study saw the end of an era when it was the custom to delay feeding, often for many days after birth. However, what is less well appreciated is that early in this century, when paediatricians were becoming increasingly involved in the care of LBW infants, it was believed essential for the survival of these infants that they should be fed soon after birth.

This article traces some of the important events in this rather remarkable cycle of changing opinion concerning the early feeding practices of LBW infants.

Early practices

At the beginning of this century early feeding was encouraged because of the belief that small infants could not tolerate starvation. Hess (1923), for example, recommended that the first milk feed should be given to premature* babies at about 12 hours of age. In 1923 he wrote: 'The necessity of an early supply of food cannot be over-emphasised, as even the better developed infants do not withstand starvation'. Hill in 1917 also believed that milk should be given to premature infants at the age of 12 hours, and Goodhart, writing in 1913, stated: 'These feeble infants must not be allowed to wait 2 or 3 days for regular feeding with the mothers' milk: the loss of weight and possible rise of temperature

which such waiting involves may be the last straw for some of these infants, who are fighting a feeble struggle for existence'.

The era of delayed feeding

Over the next 20 years or so the practice of early feeding was often challenged and in the 1940s an era dawned when delaying the first feed until the second or third day, or even longer in sick or very small infants, became the custom. It seems that this change in attitude was due to two factors. The first was to avoid the risk of pneumonia from aspirating milk into the respiratory passages (Clifford, 1947). The second was the growing belief that many of these infants were retaining an excessive amount of extracellular fluid in the early days of life so that early feeding was felt to be unnecessary and even stressful to the infant's kidneys (Clifford, 1947; Hansen and Smith, 1953). Severe loss of weight of as much as 20% was often reported, as well as haemoconcentration and hyperosmolality (Smith *et al.*, 1949; Hansen and Smith, 1953; Drillien, 1970). However, because these metabolic disturbances were quickly reversed when feeding was started it was felt that premature infants could tolerate fluid and calorie restriction without adverse affect (Hansen and Smith, 1953).

This practice of delayed feeding originated in the United States but it was soon to be adopted in the United Kingdom. Gaisford and Schofield in 1950 believed in a 3- to 4-day period of starvation (Gaisford and Schofield, 1950). Crosse, writing in 1954, even believed that the survival of premature infants had been greatly improved by giving nothing by mouth for several days after birth and suggested starving the infants from 12 hours to 4 days after birth (Crosse *et al.*, 1954).

The advantages of delayed feeding were not however accepted unanimously. In Finland in 1954 Ylppö

*The term 'premature' is used frequently in this paper. The various studies referred to were undertaken at a time when small infants were universally considered 'premature' irrespective of the aetiology of their small size.

questioned the delay in starting feeding and believed in fact that milk should be given as soon as possible after birth. In Germany, Gleiss in 1955 also resisted the fashion of starving premature infants, claiming a lower mortality (28%) in premature infants who were given their first feed between 12 and 24 hours, compared with infants who were fed initially at 36 hours (41%).

Questioning the wisdom of delayed feeding

In the early 1960s serious doubts began to be expressed over the wisdom of delaying the feeding of premature infants. Two areas of study seem to have been responsible for these changing attitudes. Firstly an increasing awareness from clinical studies that some types of cerebral palsy might be due to early starvation. Secondly there was accumulating evidence from experimental studies that early under-nutrition could permanently affect the growth of many organs, including particularly the brain.

Clinical studies. Since the nineteenth century it had been appreciated that a high proportion of children with cerebral palsy were prematurely born (Little, 1861; Freud, 1897; Ford, 1926) and that the commonest form of cerebral palsy among these infants was the syndrome of spastic diplegia (Brissaud, 1894; Childs and Evans, 1954; McDonald, 1963), a symmetrical spasticity affecting the lower limbs more than the upper limbs. However, while birth injury and asphyxia during delivery were important in causing other types of cerebral palsy, these events were not considered the most likely cause of the typical cerebral palsy of premature infants (McDonald, 1963; Churchill, 1963). Instead, damage occurring after birth was considered a more likely cause, possibly by interfering with the development of certain neurones which were maturing at this critical time (Polani, 1958, 1959).

That early starvation might be a factor in the aetiology of cerebral palsy in premature infants was suggested initially by Freedman in 1961. He showed that later neurological and intellectual impairment in infants of low birthweight was related to the amount of weight loss after birth. 2 years later Churchill also reported that infants with spastic diplegia who had weighed less than 2000 g at birth and who had been born in the years 1950 to 1959 had lost more weight and taken longer to regain birthweight than a control group of infants of similar birthweight who did not develop permanent neurological disability (Churchill, 1963). From these data it was suggested that neurological damage was possibly due to early under-nutrition since milk intake was markedly less in these infants than in those who developed normally.

Churchill did concede that the relationship between neonatal weight loss and spastic diplegia might have been due to infants who were already brain damaged at birth not feeding as well and therefore gaining weight more slowly, but in fact there was no indication that early weight loss was in any way associated with signs of central nervous system damage at the time.

Further evidence for the possible relationship between early undernutrition and cerebral palsy became available in 1964 from the Edinburgh studies of Drillien. Three-quarters of the infants weighing less than 1367 g at birth who were born in the years 1953 and 1954 had developed severe neurological handicap. On the other hand infants of similar birthweight who were born before this time (1948 to 1952) and afterwards (1955 to 1960) showed a much lower incidence of neurological handicap (about 30% in each period). Drillien believed that early feeding practices might have accounted for the very high incidence of handicap in the children born in 1953 and 1954 since these infants had not received any fluid until the third or fourth days and the introduction of milk was delayed until the fifth to the ninth days.

Until 1952 it was the custom to give a first feed of glucose water on the first or second days and seldom later than the third with milk beginning on the third or fourth days. In the years 1955 to 1960 the enthusiasm for delayed feeding was also less, with most infants starting on oral fluids on the third day and milk on the fifth or sixth days. The percentage weight loss was much greater in 1953 and 1954 than in the earlier and later periods. The 21% weight loss and 33 days to regain birthweight in the infants who were born in 1953 and 1954 suggested that the long delay in feeding these infants might somehow have been related to the very high incidence of cerebral palsy and mental retardation in these infants. Correlation coefficients between intelligence scores at the age of 5 years or later and the feeding and early weight changes in these infants (excluding those whose delay in feeding was due to oedema or respiratory distress) were also highly significant. Drillien concluded: 'Although I do not consider the case proven that delay in feeding has any serious after-effects the findings are at least suggestive. There would appear a case for routine early feeding, unless there are definite contra-indications, rather than routine delay'.

Experimental studies. Around the same time that cerebral palsy in premature infants was being attributed to early undernutrition, parallel evidence was emerging from experimental studies of under-nutrition in young animals which substantiated claims that early milk intake might be necessary for

normal growth and development. (Only those studies particularly relevant to LBW infants are considered here.)

In 1960, Widdowson and McCance showed that rats who were underfed during the suckling period did not reach their full adult size despite adequate feeding after the insult. The activity, inquisitiveness, and capacity to learn of these underfed rats were also permanently affected (Lát *et al.*, 1961). However, if the rats were underfed at a later stage of development there resulted only a temporary retardation of growth which was completely reversed with adequate later feeding (McCance and Widdowson, 1962). Particularly sensitive to early undernutrition was the brain with the incorporation of amino acids, cholesterol, phospholipids, and cerebroside being markedly reduced (Culley and Mertz, 1965). The long-term effects of early undernutrition on later growth and development were explained by the phase of cellular growth which was reached in the various organs and tissues at the time of undernutrition (Winick and Noble, 1965, 1966). Early growth was due mainly to cell proliferation and this was particularly vulnerable to undernutrition with a greater likelihood of a permanent distortion to the growth of the body and brain. Later growth resulted more from cell hypertrophy and this phase was less vulnerable to undernutrition with complete growth recovery being possible with adequate feeding after the nutritional insult.

The era of early feeding

The dangers to brain and body growth from early starvation which were suggested by these clinical and experimental studies provided a sound basis to question the wisdom of delaying feeding preterm infants. In 1962 Dr C. A. Smith, formerly a staunch advocate for delayed feeding in the United States in the 1940s and 1950s, presented an important critical appraisal of his neonatal feeding practices. He now admitted that the most important assumption which had guided his early thoughts to delay feeding of preterm infants—namely, the accumulation of extracellular fluid which had suggested overhydration—was wrong. It was now believed that much of this oedema was due to early tissue breakdown which could be prevented by early feeding (McCance and Widdowson, 1959; Usher, 1961). Smith concluded: 'If metabolic and fluid disturbances of sick premature and other newborn infants may stem from increased metabolism (including the catabolism of body tissue) . . . the therapeutic possibilities of earlier feeding become suddenly more attractive'.

Short-term effects of early feeding

The first detailed clinical study of the effects of feeding milk very early to premature infants was reported by Smallpeice and P. A. Davies in 1964. The infants studied weighed between 1000 and 2000 g at birth and were fed undiluted human breast milk from within 2 hours of birth in volumes of 60 ml/kg body weight given in the first day, increasing to 160 ml by the fourth day. This 'early fed' group was compared with two other groups of low birthweight infants. (i) Infants of comparable birthweight who were born during the study period but who were not fed until between 4 and 32 hours after delivery and given considerably less over the next week ('later fed' group). (ii) Infants weighing between 1000 and 2000 g who were born before the study period and who had been starved for at least 24 hours ('late fed' infants). The results were impressive. The 'early fed' infants regained their birthweight sooner and lost less weight than the other groups. Bilirubin levels were lower and there was also less symptomatic hypoglycaemia in the early fed infants. There was also no increase in the incidence of aspiration pneumonia. Smallpeice and Davies concluded: 'The arguments for early and adequate feeding of these small infants seemed to be forceful. It is fully justified if hyperbilirubinaemia can be reduced and symptomatic hypoglycaemia largely eradicated. Still more if shortening the time to regain birthweight reduced neurological sequelae'.

This study therefore showed that it was feasible to feed milk to LBW infants soon after birth. It is interesting, however, that it was not received enthusiastically. A leading article in the *Lancet* in 1965 commented that the advantages of early feeding were outweighed by the risks, particularly since many earlier studies had failed to show any real benefits. The article concluded: 'If the healthy mature baby chooses to fast for several hours after birth, we should bow to his superior knowledge, and profit from his example in our care of the premature, less able to tend for themselves. We may not always understand the ways of Nature, but if she has created the human infant to observe temperance and moderation over his first drink, then presumably she knows best'. It is interesting to record the reply to this statement by Smallpeice and Davies (1965): 'Finally, just where does your study of Nature lead us? We have watched many healthy mature babies who were seemingly unaware that Nature intended them to observe "temperance" and "moderation" over their first feed. We are surprised that you should quote her as our model for the care of the premature baby. Are we not correct in thinking that her neonatal mortality in this group in the animal kingdom is nearly 100%?'.

Other studies confirmed the benefits of early feeding in reducing early weight loss, raising blood glucose levels, and lowering unconjugated bilirubin in the serum (Beard *et al.*, 1966; Wu *et al.*, 1967). Among these was an important study by Wharton and Bower in 1965. Two groups of infants who weighed between 1000 g and 2250 g at birth were given liberal volumes of milk similar to those used by Smallpeice and Davies (starting within the first few hours of birth) or smaller volumes starting at 12 to 16 hours. The results confirmed the value of early milk feeding in reducing the incidence of hypoglycaemia and neonatal jaundice and amount of weight loss. There was, however, a higher mortality in the 'immediate' fed infants from aspiration of regurgitated milk compared with the 'later fed' infants and no explanation could be offered other than the difference in feeding methods since inhalation of vomit was a frequent necropsy finding in the 'immediate fed' infants. Wharton and Bower concluded that although there was very good reason for immediate feeding with undiluted human breast milk the practice was not without danger. They suggested that smaller volumes of milk should be given—only about one-third of the volumes suggested by Smallpeice and Davies and that the regimen might have to be modified in special circumstances.

Long-term advantages of early feeding

By preventing hypoglycaemia, hypernatraemia, and severe jaundice and with less interruption to perinatal growth it was likely that the long-term prognosis of LBW infants with regard to their neuropsychological development would also be improved. The first to show long-term benefits of early feeding were P. A. Davies and Russell in 1968. They showed that at a mean age of 2 years the intellectual and neurological development and physical growth of LBW infants who had been fed milk early showed a considerable improvement over those children who were born at a time when delayed feeding was practised.

Further support for the hypothesis that early feeding might improve the quality of later development came 2 years later. In a study from Hammersmith Hospital (Davies and Davis, 1970), later head growth of infants weighing less than 1500 g at birth in two consecutive 4-year periods (1961–1964 and 1965–1968) was evaluated and correlated with milk intake and temperature in the first weeks of life. Different feeding regimens were given in the two periods: in the first period (1961–1964) milk intake in the first week after birth was restricted while in the second period (1965–1968) the more liberal policy as described by Smallpeice and Davies was used.

When the infants were seen later in childhood the following interesting points emerged. The preterm infants who were born between 1961 and 1964 had smaller head circumferences than those born between 1965 and 1968. Height and weight distributions also paralleled changes in head circumference. Because of the close correlation between head circumference and brain weight (Fedrick, 1971) and cellular brain growth (Winick and Rosso, 1969) Davies and Davis suggested that relatively minor undernutrition in preterm infants during the time of very rapid brain growth could produce a deficit in the ultimate size of the brain.

Also stressed in this study was the role of the infants' thermal environment in the economy and use of food. In the years 1961 to 1964 the mean body temperature of all the low birthweight infants was 35.9°C, while between 1965 and 1968 it was 36.3°C. It was suggested that the improvement in the quality of growth and development of the infants born between 1965 and 1968 was due not only to the early and liberal provision of milk but also to the economic use of the food in promoting growth instead of being diverted to maintaining basal metabolic processes which might have been the case in the infants born in the years 1961 to 1964.

Conclusion

By the early 1970s the practice of early feeding LBW infants was well established. The wheel had now turned a complete circle since the beginning of the 20th century. The earlier preoccupation with survival alone had been gradually replaced by an increasing need to improve the quality of survival with normal intellectual function. To delay feeding could no longer be justified. It is indeed humbling to recall again the views which were expressed by Goodhart as long ago as 1913: 'These feeble infants must not be allowed to wait 2 or 3 days for regular feeding with the mothers' milk. The loss of weight and possible rise of temperature which such waiting involves may be the last straw for some of these infants, who are fighting a feeble struggle for existence.'

In 1977 it is interesting to look back at these events and to try to see them in their true perspective. To do this it is important to realise that the long-term prognosis of LBW infants depends upon many factors, adequate nutrition being only one. The 1950s and early 1960s were not only the years of early starvation; they were also years where 'modern neonatal iatrogenesis reached a peak when almost every major error in newborn care was widely practised, at least for a time by overextended paediatricians using inadequate equipment' (*Lancet*,

1974). Thus, as well as infants being frequently starved for 2 to 5 days, they were exposed in incubators and often allowed to become chilled; they were all too often resuscitated at birth by large doses of nikethamide or by intragastric oxygen and showed an increased risk of kernicterus from the excessive use of vitamin K and sulphonamides.

Recently it has also come to light that during these same years there was also an indiscriminate restriction in the use of adequate oxygen concentrations soon after birth for infants requiring resuscitation (Bolton and Cross, 1974). This is believed to have been a reaction to the newly discovered association in the early 1950s of high oxygen concentration in the blood and retrolental fibroplasia (Ashton *et al.*, 1953). Until about 1950 there was a steady decline in early neonatal mortality both in Britain and the United States, but between about 1950 and 1965 this decline was brought to a temporary halt, mainly because more LBW infants were dying on the first day of life from lack of oxygen. It is also likely that the LBW infants who survived the first few days of life during this time of oxygen restriction were more at risk of permanent brain damage from neonatal hypoxia.

In about 1965 neonatal mortality again began to fall and this trend has continued unabated into the 1970s. Many factors have been responsible which are embraced by the term neonatal intensive care. These include improved obstetric care; prompt treatment of birth asphyxia; the introduction of the oxygen electrode to accurately measure arterial oxygen tension; conservation of body temperature; a better understanding of neonatal pulmonary problems; and the introduction of phototherapy for the management of jaundice. Alongside this falling neonatal mortality there has also been a dramatic drop in the incidence of long-term handicap such as epilepsy, cerebral palsy, mental subnormality, blindness, and deafness (Davies and Stewart, 1975). The practice of early feeding LBW infants has coincided with all these other improvements in perinatal care. (It is also perhaps significant that none of the early experimental studies of undernutrition in fact really produced syndromes of cerebral palsy.) The improved long-term prognosis of LBW infants cannot therefore be attributed solely to early feeding. Attention to adequate feeding, which has been made possible by improvements in nursing skills, has undoubtedly played an important part, but it must be considered as only one of many improvements of perinatal care which have made possible the much better outlook for LBW infants.

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Correspondence to Dr D. P. Davies, Department of Child Health, Leicester Royal Infirmary, Leicester LE1 5WW.