

Short reports

Neonatal and postneonatal mortality in very low birthweight infants

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SUMMARY We reviewed 388 very low birthweight infants admitted to this neonatal intensive care unit over a four year period to determine the pattern of neonatal and postneonatal deaths up to age 2 years. Neonatal mortality is no longer an adequate indicator of outcome because deaths arising from perinatal events occur after the first month of life.

Advances in neonatal intensive care have been associated with improved overall survival and quality of survival among critically ill or preterm infants. It has been reported, however, that among infants admitted to a neonatal intensive care unit, postneonatal infant mortality is 10 times and infant mortality after hospital discharge is seven times that of the general population.^{1,2} Both neonatal and postneonatal infant mortality are inversely related to birthweight in the general population.³⁻⁶ A trend in postponement of neonatal deaths to the postneonatal period has also been observed in very low birthweight (less than 1500 g) infants.^{7,8} This review was conducted to determine the pattern of neonatal and postneonatal deaths up to 2 years of age among very low birthweight infants admitted to this neonatal intensive care unit.

Patients and methods

Three hundred and eighty eight very low birthweight infants were admitted to Queen Victoria Medical Centre between 1 January 1977 and 31 December 1980. A total of 108 infants weighed 501 to 1000 g and 280 weighed 1001 to 1500 g at birth. Seventy six (24%) infants were outborn and transferred via the Newborn Emergency Transport Service and 312 were inborn. Neonatal intensive care was not initiated in 16 infants in whom a major congenital malformation was evident on admission.

After discharge home all very low birthweight survivors were enrolled for follow up at the Growth and Development Clinic to allow for documentation of their long term outcome. Deaths which occurred

within the first 2 postnatal years were recorded. The cause and age of death were determined, the latter being categorised into early neonatal (age less than seven days), late neonatal (seven to 27 days), and postneonatal (28 or more days) periods. Data were analysed using χ^2 tests with Yates's correction.

Results

One hundred and two children (26%) died before their second birthday; 67 (66%) of the deaths occurred in the early neonatal period, 20 (20%) in the late neonatal period, and 15 (15%) in the postneonatal period. The percentage of deaths in the postneonatal period was significantly higher in infants who weighed 1001 to 1500 g at birth than in those who weighed 501 to 1000 g at birth (23% v 6%, $P < 0.05$). Table 1 shows the survival rates of infants over the three mortality periods.

Six (40%) of the 15 postneonatal deaths occurred during the initial hospital admission (Table 2). All died from conditions directly related to complications of prematurity such as bronchopulmonary dysplasia, necrotising enterocolitis, and fulminating sepsis.

Of the remaining nine postneonatal deaths which occurred after discharge following the first hospital admission the most common cause of death was

Table 1 Survival in very low birthweight infants admitted to this neonatal intensive care unit between 1977 and 1980

| Survival | Birthweight (g) | | Total (n=388) No (%) |
|--|-------------------------------|--------------------------------|----------------------------|
| | 501-1000 (n=108) No (%) | 1001-1500 (n=280) No (%) | |
| To the end of the 6th day | 73 (68) | 248 (89) | 321 (83) |
| To the end of the 27th day | 62 (57) | 239 (85) | 301 (78) |
| To the end of the 1st year | 59 (55) | 228 (81) | 287 (74) |
| To the end of the 2nd year | 59 (55) | 227 (81) | 286 (74) |
| % Of early neonatal survivors attaining 2 years of age | 81* | 92* | 89 |

* $\chi^2 = 15.86$, $P < 0.0005$.

Table 2 *Causes of postneonatal deaths among very low birthweight infants admitted to this neonatal intensive care unit between 1977 and 1980*

| Case no | Birthweight (g) | Gestation (wks) | Cause of death | Age died (wks) |
|--|-----------------|-----------------|------------------------------|----------------|
| <i>Death during initial hospital admission</i> | | | | |
| 1 | 560 | 30 | Necrotising enterocolitis | 4 |
| 2 | 860 | 26 | Bronchopulmonary dysplasia | 7 |
| 3 | 1090 | 30 | Bronchopulmonary dysplasia | 8 |
| 4 | 1170 | 28 | Septicaemia, meningitis | 8 |
| 5 | 1190 | 27 | Bronchopulmonary dysplasia | 9 |
| 6 | 1160 | 26 | Bronchopulmonary dysplasia | 22 |
| <i>Death after hospital discharge</i> | | | | |
| 7 | 1500 | 29 | Congenital heart disease | 7 |
| 8 | 1180 | 32 | Sudden infant death syndrome | 18 |
| 9 | 1370 | 30 | Sudden infant death syndrome | 23 |
| 10 | 1230 | 30 | Sudden infant death syndrome | 31 |
| 11 | 1130 | 27 | Pneumonia | 33 |
| 12 | 1000 | 27 | Cerebral haemorrhage | 39 |
| 13 | 1390 | 37 | Pneumonia | 41 |
| 14 | 1300 | 28 | Septicaemia, gastroenteritis | 52 |
| 15 | 1290 | 27 | Aspiration pneumonia | 90 |

sudden infant death syndrome (SIDS). Only one (two per cent) of the deaths after discharge occurred in infants who weighed 501 to 1000 g compared with eight (16%) in infants who weighed 1001 to 1500 g ($P < 0.05$).

Discussion

The period during which the very low birthweight infant remains at risk of death has extended beyond the first 28 days. Neonatal mortality data will exclude six per cent of infants who died in the postneonatal period from the complications directly related to prematurity such as sepsis, necrotising enterocolitis, and complications of intensive treatment such as bronchopulmonary dysplasia. It has been reported that 50% of the postneonatal deaths which occurred during the initial hospital admission were caused by bronchopulmonary dysplasia.⁸ In the present study this disorder was responsible for four (67%) of the six postneonatal hospital deaths. The duration of the initial hospital stay seems to be a more appropriate parameter for reporting very low birthweight survival data in the neonatal period, as most deaths from neonatal causes occur before hospital discharge.

Fifteen per cent of the deaths in very low birthweight infants in the present series occurred in the postneonatal period, similar to the 14% incidence previously reported.⁸ Fourteen infants died between 28 days and 1 year of age, yielding a postneonatal infant mortality of 47 in 1000 infants surviving the neonatal period. This is approximately 15 times that found in the general population during the study period.⁹ None of the postneonatal deaths reported, with the exception of the death from

cerebral haemorrhage after head injury, seem currently to be preventable. SIDS and infection are the two predominant causes of death after hospital discharge. SIDS was responsible for three deaths, yielding a mortality of 10 per 1000 infants surviving the neonatal period. This is approximately five times that in the general population during the study period⁹ and is comparable to the three to eight times increased risk of SIDS which has been reported in infants of all birthweights discharged from a neonatal intensive care unit.^{1 2 10} This high postneonatal infant mortality, particularly from SIDS, is not yet completely understood. A major reduction in late mortality will only result from elucidation and prevention of the causes of SIDS.

Both postneonatal deaths and those occurring after hospital discharge are significantly more common in the larger very low birthweight survivors (1001 to 1500 g) than in those who weighed 501 to 1000 g at birth. All three SIDS deaths occurred in the heavier birthweight group. It is encouraging to note that the current improved survival of those below 1000 g has not resulted in their increased vulnerability to late mortality to the same or a greater extent than the larger very low birthweight survivor. Nevertheless, the overall increased postneonatal infant mortality in these infants indicates that an in depth pathological and psychosocial study¹¹ in this high risk group is required to determine the underlying causes of inadequate perinatal and postperinatal health care.

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Functional palatal incompetence in the fetal anticonvulsant syndrome

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SUMMARY We report two children with mild mental retardation and appreciable articulation difficulties whose mothers took phenobarbitone and phenytoin throughout their pregnancies. The speech difficulties in both children were due to malfunction of an anatomically normal palate.

The risk of cleft lip with or without cleft palate is up to eight times greater in the children of epileptic mothers.¹ We describe two children of epileptic mothers with incompetence of the soft palate leading to nasal escape and poor speech intelligibility.

Case reports

Case 1. This boy was born by emergency caesarean section at 36 weeks' gestation; he weighed 2.5 kg. His mother, a long standing epileptic, took phenobarbitone and phenytoin throughout pregnancy. She had a single brief grand mal convulsion just before induction of anaesthesia for caesarean section. The mother also smoked heavily throughout the pregnancy, was known to abuse alcohol, and had a history of poor drug compliance. The baby seemed normal at birth and was discharged home with the mother.

He was next seen at age 3½ years when he presented with speech delay. History showed that the gross motor milestones had been delayed—he first sat unsupported at 18 months and walked

independently at 2 years. Mild generalised hypotonia but no other neurological abnormality was found on examination. With time the hypotonia has improved and is no longer a feature. His developmental quotient on the Griffiths scales was between 70 and 80 on several occasions. His language development was delayed both in comprehension (-2 SD) and expression (-1.1 SD) when assessed on the Reynell scales at 4.5 years. His speech was unintelligible because of considerable hypernasality. His height was on the third centile for his age, his weight below the third centile, and his head circumference on the 50th centile. Growth since then has continued along these parameters. He has poor dentition with deficient enamel. His vision and hearing are normal.

Investigations including electroencephalogram, electromyogram, thyroid function tests, and nerve conduction studies were normal. Examinations by an ear, nose, and throat surgeon and a plastic surgeon showed that the palate was anatomically normal with no submucous cleft; however, a cinematogram at age 4½ years confirmed abnormalities of movement of the soft palate. Normally, when swallowing or producing most speech sounds the soft palate closes against the nasopharynx. In this child the soft palate moved normally in swallowing but during speech closure was mostly inadequate allowing considerable air escape.

Case 2. This boy was born by normal vertex delivery at 38 weeks' gestation and weighed 2.75 kg. His mother has been well with no fits during pregnancy