Early prognosis of low birthweight infants treated for progressive posthaemorrhagic hydrocephalus

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SUMMARY Twelve low birthweight preterm infants were treated for progressive posthaemorrhagic hydrocephalus by ventriculoperitoneal diversion of cerebrospinal fluid. At a median postnatal age of 20 months, 6 were moderately or severely retarded. Convulsions in the first week of life were associated with a poorer prognosis and frequent taps to remove cerebrospinal fluid with a better outcome.

Although survival rates of low birthweight infants with severe respiratory problems have improved, intraventricular haemorrhage has become a common complication affecting the quality of survival. An appreciable proportion of survivors develop enlargement of the cerebral ventricles or clinically evident progressive hydrocephalus.¹ In many ventricular enlargement stops spontaneously or after intermittent drainage of cerebrospinal fluid through a lumbar or ventricular needle tap.² The rest of these infants need surgical placement of ventriculoatrial or ventriculoperitoneal shunts if gross hydrocephaly and eventual death is to be prevented. Over 2½ years a number of variables related to early prognosis in a group of low birthweight infants treated by ventricular shunt have been examined.

Patients and methods

Between July 1979 and December 1981 14 low birthweight infants referred to the regional neonatal intensive care unit at Liverpool Maternity Hospital developed progressive hydrocephalus after intraventricular haemorrhage. During the same period 808 low birthweight infants were admitted to the unit. Of those infants developing intraventricular haemorrhage, 91 died during the neonatal period or before discharge. All but 1 hydrocephalic infant, who developed hydrocephalus at a late stage, were treated at first by intermittent lumbar or ventricular needle drainage of cerebrospinal fluid. Two also received isosorbide.³ After discussion between staff and parents, 2 infants were not treated further by insertion of a shunt because of gross neurological deficit. Both died within a few days. The 12 remaining infants form the study group.

Intraventricular haemorrhage was diagnosed and

the development of hydrocephalus monitored by echoencephalography via the anterior fontanelle.⁴ The decision on timing of the shunt insertion was made by 1 of 2 paediatric surgeons. All infants had clinically evident hydrocephalus at the time of operation and ultrasound scan appearances were never the sole criterion for operation. No infant was dependent on a ventilator immediately before surgery, although several required short periods of respiratory support afterwards. One infant suffered postoperative cardiorespiratory arrest and recovered only after prolonged resuscitation. This infant was the most severely handicapped at follow up. Other postoperative complications were minor.

The first follow up after discharge from hospital included a full neurological examination, Denver developmental screening test, and Stycar tests of hearing and vision, as appropriate. As disabilities became evident specialist ophthalmic, physiotherapy, and developmental referrals were made. The assessment of outcome used here is a combination of all assessments made of an individual child's progress. The median length of follow up to date is 20.5 months (range 15–42 months). So that the relation of antecedent factors to early outcome of treatment could be examined, the infants were assigned to 1 of 2 groups on the basis of neurological sequelae and developmental delay. The 'better outcome' group (group 1) comprised 6 infants with failures in 1 or no sections of the Denver developmental screening test and with either physical disabilities that did not appear to interfere seriously with normal function or normal findings on neurological examinations. The 'poorer outcome' group (group 2) all had moderate to severe developmental delay in 2 or more sections of the Denver developmental screening test and major

Fig. 1 Coronal section ultrasound scans of brain through anterior fontanelle: (a) posthaemorrhagic hydrocephalus; (b) same infant immediately after shunt insertion; (c) same infant 3 months after shunt insertion showing complete resolution of hydrocephalus.

physical disabilities. The composition of the groups is shown in the Table. Because of the small numbers a further breakdown of outcome was not practicable. Factors possibly related to outcome were analysed by the Fisher exact test or Mann-Whitney U test, as appropriate.

Results

Ten boys and 2 girls were treated. Both girls were in group 2. The median birthweight was 1295 g (range 1150–2070 g) and median gestational age was 29.5 weeks (range 27–33 weeks). No infant was small for

gestational age. All infants received respiratory support (median 9.5 days, range 2–28 days) and most suffered severe acidosis (median lowest pH 6.90, range 6.75–7.30). Nine infants had intraventricular haemorrhage with parenchymal extension, 2 intraventricular bleeding only, and 1 subependymal haemorrhage only. The median time of onset of clinically evident hydrocephalus was 18 days (range 15–70 days). The shunts were inserted at median of 61.5 days (32–154 days). None of these variables was statistically significant when related to outcome.

Fig. 2 Post shunt insertion follow up scans: (a) cerebral atrophy in infant with spastic diplegia; (b) porencephalic cyst in normally developing infant; (c) porencephalic cyst in infant with hemiplegia.

Resolution of hydrocephalus, clinically and on ultrasound examination, followed shunt insertion in all cases. In 2 infants the size of the lateral ventricles did not return to normal after shunt insertion and this may have been caused by some periventricular or cerebral atrophy. Four infants required 1 or more replacements of their shunt because of malfunction. In 8 infants porencephalic cysts remained at the site of extraventricular extension of intraventricular haemorrhage.

All infants in group 2 had convulsions in the first week of life. One infant in group 1 had a short fit on

the second day. This difference was statistically significant ($P < 0.025$). Eleven infants had lumbar or ventricular taps to relieve symptoms of acute hydrocephalus such as recurrent apnoea, vomiting, or fits. The number of taps done was not related to the severity of the condition, as those infants in group 2 had significantly fewer taps (median = 3, range 1–15; $P = 0.026$) than those in group 1 (median 14, range 3–20).

Discussion

The prognosis of low birthweight infants suffering

Table Clinical data and outcome of infants treated for posthaemorrhagic hydrocephalus

Case details No, sex	Birth- weight (g)	Gestational age (wks)	Minimum pH value	Fits in week 1	Duration of respiratory support (dys)	Grade of periven- tricular haemorrhage	Onset of hydrocephalus (dys)	Taps	Shunt inserted (dys)	Onset to shunt (dys)	OFC centile at operation	Follow up ultra- sound or CAT	Re- visions	Outcome
Group 1														
1, M	1275	30	7.10	No	11	PAR H (R) only	15	13 + iso- sorbide	66	51	75	Porenc- cephalic cyst (R)	1	42 mths moderate retinopathy, clumsy, good language development
2, M	2070	33	6.90	No	7	IVH (bilateral)	17	15	43	26	97	Normal	1	36 mths normal development
3, M	1280	29	7.13	No	20	IVH (bilateral)	24	3	57	33	97	Cerebral atrophy	0	15 mths mild motor delay
4, M	1480	30	6.70	Yes	3	PAR H (R) IVH (L)	15	20	81	66	90	Porenc- cephalic cyst (R)	0	22 mths mild (L) hemiplegia, strabismus
5, M	1250	28	7.20	No	8	PAR H (L) SEH (R)	15	14	32	18	50	Porenc- cephalic cyst (L)	0	17 mths normal development
6, M	2050	30	7.30	No	2	SEH (R)	70	—	150	80	97	Normal	0	17 mths mild (L) hemiplegia
Group 2														
7, F	1240	28	6.80	Yes	23	PAR H (L) only	27	8	154	127	97	Porenc- cephalic cyst (L)	0	30 mths spastic quad- raplegia, (R) hemiplegia, severe developmental delay
8, M	2100	33	6.80	Yes	13	PAR H (L) IVH (R)	28	1	42	14	97	Porenc- cephalic cyst (L)	0	28 mths (R) hemi- plegia, moderate developmental delay
9, M	1950	30	6.80	Yes	28	PAR H (L) IVH (R)	15	3	35	20	97	Residual dilated ventricles	2	36 mths (R) hemi- plegia, moderate developmental delay
10, F	1310	28	6.95	Yes	9	PAR H (L) IVH (R)	19	2 + iso- sorbide	37	18	97	Cerebral atrophy	1	15 mths spastic diplegia, hearing loss, moderate developmental delay
11, M	1250	28	6.9	Yes	10	PAR H (R) IVH (L)	17	15	69	52	50	Porenc- cephalic cyst (R)	1	17 mths (L) hemi- plegia, hypotonia, marked develop- mental delay
12, M	1150	27	7.18	Yes	4	PAR H (R) IVH (L)	20	3	129	109	10	Porenc- cephalic cyst (R)	0	16 mths spastic diplegia, hearing loss, severe developmental delay

PAR H = parenchymal haemorrhage, IVH = intraventricular haemorrhage, SEH = subependymal haemorrhage.

from posthaemorrhagic hydrocephalus is probably related to the extent of the first insult and haemorrhage and the rate and extent of the development of subsequent hydrocephalus. Infants in group 2 were ventilated for longer, suffered more severe acidosis, and more extensive haemorrhage than those in group 1, but these differences were not statistically significant. Early convulsions have been widely recognised as a risk factor for later disability and probably relate to the extent of acute neurological insult in the first few days.⁵ Greater attention to neurological examination at this time could provide more subtle indicators of prognosis.⁶

Although the time of onset of clinical hydrocephalus was fairly uniform, the rate of progression was variable and accounted for the wide differences in the timing of the operation. It has been suggested that hydrocephalus produces further impairment by causing ischaemia of periventricular areas and that lowering intraventricular pressure by intermittent drainage may reduce this.⁷ The 12 infants described here had taps for a variety of reasons, but it is interesting that more frequent taps were associated with a better outcome. Even if ventriculoperitoneal shunts cannot be avoided by repeated tapping, earlier reduction of hydrocephalus warrants further investigation as a means of improving prognosis.

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