Short reports

Continuous measurement of subarachnoid pressure in the severely asphyxiated newborn

M I LEVENE AND D H EVANS

Department of Child Health, Leicester University Medical School and Department of Medical Physics, Leicester Royal Infirmary

SUMMARY We describe a method of monitoring continuously subarachnoid pressure in the severely asphyxiated newborn by a percutaneously placed catheter. Five infants were studied for a total of 180 hours and although opening pressures were not appreciably raised in any, maximum pressures were above 20 mmHg in four infants and reached a peak of 48 mmHg in one.

Although attempts have been made to measure intracranial pressure (ICP) non-invasively in the newborn by devices attached to the fontanelle,¹ severe methodological difficulties exist. In particular, changes in the application force of the transducer alters the perceived ICP² and some infants, particularly those born at term, are unsuitable for transfontanelle measurement. We report a simple method of monitoring ICP continuously by a catheter placed percutaneously into the subarachnoid space.

Patients and methods

Placement was attempted in five live born infants. Before attempting this procedure in living children, however, and to ensure that the catheter could be inserted without injury to the brain, 10 attempts had been made in 6 term neonates, who had died shortly after birth and were to undergo necropsy. Approval by the hospital ethical committee was obtained and informed and signed parental consent was sought before placement in living infants. The indications for performing this procedure were failure of the infant to develop regular respiration within 20 minutes of birth, together with convulsions and the need for mechanical ventilation.

The infant's head was completely shaved and a 16 G Medicut (Sherwood Medical Industries) was inserted through the lateral margin of the anterior fontanelle, on the right side with the needle angled

forwards and just under the table of the frontal bone, and aimed at a point 1 cm above the infant's right eye. When the characteristic 'give' of the needle was felt on penetration of the galea the needle was removed and the plastic cannula advanced slowly until cerebrospinal fluid (CSF) appeared. A round ended polyethylene 16 G Portex epidural catheter with three elliptical side holes was then inserted through the cannula to lie over the right frontal lobe, and the cannula was then removed. The catheter had been primed previously with normal saline and calibration marks ensured that it was inserted to no more than 3 cm from the scalp puncture. The catheter was secured to the scalp by tape or a suture. The hub of the catheter was connected to an Elcomatic EM 750A pressure transducer via low compliance manometer tubing, and the ICP trace was displayed on a chart recorder. The position of the transducer was zeroed with the midpoint of the baby's head at the beginning of monitoring and was zeroed again every time the baby was repositioned. The Table gives details of the infants studied.

Results

The five infants were monitored for a total of 180 hours and good pressure waveforms were obtained at the first attempt in all cases. The transmitted pulse and respiratory waveforms were superimposed on the ICP trace in all cases (Figure) and were clearly seen throughout monitoring. The opening pressure was 15 mmHg or below, but all infants were being mechanically ventilated to maintain an arterial P_{CO_2} of between 3 and 4 kPa (22.5-30 mmHg). Pressures rose in all babies up to a maximum of 48 mmHg (see Table). No infant showed any evidence of infection either locally or in the CSF during or after monitoring. Three infants died but we believe this was caused by the severity of the asphyxia rather than any complication associated with the catheter. The frequency characteristics of

Table Details of the five infants in whom continuous subarachnoid pressure monitoring was attempted

Infant No	Gestational age (wks)	Birthweight (kg)	Apgar score			Age at catheter insertion (hrs)	Duration catheter in situ (hrs)	Opening pressure (mm Hg)	Maximum pressure (mm Hg)	Outcome
			1 min	5 min	10 min					
1	40	3.10	0	1	4	12	38	9	30	Died
2	40	3.00	0	1	3	28	76	10	38	Died
3	35	2.52	0	2	6	4	50	6	48	Died
4	40	2.90	1	4	5	12	4	15	21	Alive
5	37	2.36	1	1	1	6	12	4	10	Alive

Figure Traces from infants 1 and 2 showing cardiac and respiratory waveforms on the baseline intracranial pressure trace. Intracranial pressure (ICP) scale on the left is 0–25 mmHg.

the catheter and interconnecting manometer tubing were studied in vitro. The amplitude response varied by only 20% over the frequency range of 0–14 Hz, and was not considered to distort the pressure waveform to an appreciable extent.

Discussion

Severe perinatal asphyxia with failure to breathe spontaneously by 20 minutes is associated with a mortality rate greater than 50%^{3,4} and a risk of severe neurological abnormality of up to 50% in survivors.³ Convulsions in these infants also correlated with adverse outcome.⁴ Irrespective of the severity of the initial asphyxial cerebral episode, further damage may occur as a result of cerebral oedema and intracranial hypertension. Monitoring and adequate treatment of raised ICP may prevent subsequent damage and although insertion of a

subarachnoid catheter is an invasive procedure, we considered that the prognosis for such severely asphyxiated infants justified the risk. Placement was relatively simple and prolonged monitoring feasible. We would, however, strongly recommend attempting this technique on cadavers before placing the catheter in a living baby.

Measurement of ICP in adults and children has a well accepted role in the management of many neurological conditions associated with the likelihood of intracranial hypertension. Mayer and Walker⁵ report a minor complication rate of 5.3% for subarachnoid ICP monitoring in children, with no serious complications noted. A recent report from Israel⁶ describes the percutaneous placement of a subdural cannula in small children through the open fontanelle, but in only one case was it performed on a neonate asphyxiated at birth. We suggest that subarachnoid catheters may be better suited to long term monitoring.

In the five infants described here, all of whom had severe intrapartum asphyxia, considerable and unpredictable variation in the severity of intracranial hypertension was found. Although measurement of ICP is only one facet in the overall management of severe asphyxia, and stabilisation of blood pressure, control of convulsions, and respiratory support are all important, we suggest that it may be a useful adjunct.

We thank paediatric colleagues for referral of infants, the nursing staff of the Leicester Royal Infirmary Neonatal Unit, and the Department of Medical Illustration.

References

- 1 Vidyasagar D, Raju TNK. A simple non-invasive technique of measuring intracranial pressure in the newborn. *Pediatrics* 1977;**59**:957–61.
- 2 Horbar JD, Yeager S, Philip AGS, Lucey JF. Effect of application force on non-invasive measurements of intracranial pressure. *Pediatrics* 1980;**66**:455–7.
- 3 Nelson KB, Ellenberg JH. Apgar scores as predictors of chronic neurologic disability. *Pediatrics* 1981;**68**:36–44.
- 4 Scott H. Outcome of very severe birth asphyxia. *Arch Dis Child* 1976;**51**:712–6.
- 5 Mayer T, Walker ML. Emergency intracranial pressure monitoring in pediatrics. Management of the acute coma of brain insult. *Clin Pediatr (Phila)* 1982;**21**:391–6.

⁶ Goitein KJ, Amit Y, Mussaffi H. Intracranial pressure in central nervous system infections and cerebral ischaemia of infancy. *Arch Dis Child* 1983;58:184-6.

Correspondence to Dr M I Levene, Department of Child Health, Clinical Sciences Building, Leicester Royal Infirmary, PO Box 65, Leicester LE2 7LX.

Addendum

To date this procedure has been successfully performed in 9 infants with no recognised complications. Neither necropsy nor computed tomography in survivors have shown trauma related to the catheter.

Intrauterine hydrops caused by premature closure of the foramen ovale

E PESONEN, H HAAVISTO, P ÄMMÄLÄ, AND K TERAMO

Children's Hospital and the Departments I and II of Obstetrics and Gynecology, University of Helsinki, Finland

SUMMARY Intrauterine hydrops was diagnosed by two dimensional echocardiography. The fetus had a pericardial effusion and a thick interatrial septum without a foramen ovale flap. The condition was treated by giving the mother digitalis. Postnatally, the effusion had disappeared and a parachute mitral valve was found.

Fetal hydrops may be reliably diagnosed by ultrasonography. It is often serious and is associated with a high perinatal mortality rate,¹ but fetal death rarely occurs before the 28th gestational week. Cardiac causes of fetal hydrops include congenital heart diseases and rhythm disturbances. With two dimensional ultrasonography the anatomy of the heart may be visualised² and some idea of the cardiac rhythm obtained. We describe a fetus in whom the foramen ovale was found to be closed on antenatal ultrasound examination: the condition was associated with severe fetal hydrops and an irregular heart rhythm.

Case report

The mother was a healthy gravida I, para 0 with no family history of cardiac disease. Polyhydramnios was diagnosed by ultrasound on week 31 of gestation. The fetus was hydropic and had ascites. When fetal cardiac ultrasonography was performed on week 32 of gestation, the heart was enlarged and there was fluid in the pericardial cavity (Fig. 1), the interatrial septum was thick and bulged towards the right atrium, the foramen ovale and its valve were not seen and the movement of the atrioventricular valves was irregular. Fetal heart rate monitoring by cardiotocography was attempted unsuccessfully several times and the failure was considered to be caused by irregular heart rate. The fetus was thought to be in cardiac failure and the mother was given digitalis 7 µg/kg/day.

Fig. 1 2-D antenatal echocardiography showing fluid in the pericardial cavity.

EF = effusion, LV = left ventricle, LA = left atrium, RV = right ventricle, RA = right atrium.

A boy was born by caesarean section on week 34 of gestation. His Apgar score was 6 at 1 and 5 minutes and he was intubated immediately. His face, neck, abdomen, and scrotum were hydropic and the liver edge was palpable 1.5 cm below the costal margin. The femoral and brachial arterial pulses were palpable and irregular and on auscultation the second heart sound was not split. A 3/6 systolic heart murmur was heard at the middle left sternal border radiating to the axilla. His electrocardiogram showed three distinct rhythmical morphologies with regular repetition, and the T waves were flat in the left precordial and limb leads suggesting pericardial irritation. Postnatal echocardiography showed no pericardial fluid, the atrial septum was thick and bulging to the right, and there was no evidence of the foramen ovale and its flap in the atrial septum. Mitral valve movement was limited and the chordae tendinae were short and attached to a single large