[3] Cook R C M, Rickham P P. The liver and biliary tract. In: Rickham P P, Lister J, Irving I M, eds. *Neonatal surgery*, second edition. London: Butterworth, 1978: 483.
[4] Reid I S. Biliary tract abnormalities associated with duodenal atresia. *Arch Dis Child* 1973; **48**: 952–7.

Correspondence to Professor L Spitz, Institute of Child Health, 30 Guilford Street, London WC1N 1EH.

Received 17 January 1983

Hyponatraemia caused by repeated cerebrospinal fluid drainage in post haemorrhagic hydrocephalus

P MACMAHON AND R W I COOKE

Regional Neonatal Intensive Care Unit, Department of Child Health, Liverpool Maternity Hospital

SUMMARY Three hyponatraemic infants had post-haemorrhagic hydrocephalus which was treated by repeated drainage of cerebrospinal fluid. Each required oral sodium supplements. The concentration of sodium in the cerebrospinal fluid was such that as much as 3 mmol of sodium could have been removed with each ventricular tap. Serum sodium concentrations should be monitored closely in any infant requiring regular drainage of cerebrospinal fluid.

Periventricular haemorrhage is common in infants of very low birthweight.[1] Hydrocephalus is a serious complication in many survivors.[2] It is now possible to diagnose both these conditions early by the use of real-time ultrasound scanning via the anterior fontanelle.[3] We report 3 patients whose post-haemorrhagic hydrocephalus was treated initially by repeated drainage of cerebrospinal fluid. Each developed hyponatraemia which was treated with oral sodium supplements.

Patients

Case 1. A boy weighing 1·25 kg was delivered at 28 weeks' gestation. He developed severe hyaline membrane disease and ventilatory support was required for the first 8 days of life. During this time the use of real-time ultrasound scanning enabled bilateral intraventricular haemorrhages to be detected. Progressive communicating hydrocephalus developed and after a total of 10 therapeutic lumbar punctures had been performed a ventriculoperitoneal shunt was inserted on day 37.

Between days 19 and 29, nine therapeutic lumbar punctures were performed (mean volume 22 ml). During the same period the serum sodium concentration declined from 133 to 122 mmol/l. Throughout this time the patient was fully enterally fed on expressed breast milk. There was no gastrointestinal upset and no diuretics were administered. During the next 4 days 3·5 mmol/kg a day of supplementary oral sodium were given and the serum sodium concentration rose to 140 mmol/l.

Case 2. A boy weighing 1·070 kg was delivered at 27 weeks' gestation. He developed signs of persistent fetal circulation and required ventilation for the first 3 days of life. Bilateral intraventricular haemorrhages were detected using real-time ultrasound scanning. Progressive non-communicating hydrocephalus developed and after a total of 16 therapeutic ventricular taps a ventriculoperitoneal shunt was inserted on day 36.

Before his first ventricular tap the patient was on full intravenous feeding. His serum sodium concentration was normal on a sodium intake of 4 mmol/kg a day. Four ventricular taps were performed during the next 5 days (mean volume 15·5 ml). His serum sodium concentration fell to 120 mmol/l, despite increasing his sodium intake to 8 mmol/kg a day. During the next 5 days daily ventricular taps (mean volume 18 ml) continued and the patient was fully enterally fed on expressed breast milk, with the addition of 23 mmol/kg a day of oral sodium supplements. The serum sodium concentration rose to 153 mmol/l. Sodium supplements were therefore stopped. Daily ventricular taps continued and 4 days later the serum sodium value had fallen progressively to 126 mmol/l. Throughout this time there was no gastrointestinal upset and the patient was not on diuretics.

Case 3. A boy weighing 1·48 kg was delivered at 30 weeks' gestation. Severe hyaline membrane disease developed and ventilatory support was required for the first 8 days of life. A unilateral intraventricular haemorrhage was detected using real-time ultrasound scanning. Progressive communicating hydrocephalus developed which was initially treated by lumbar puncture and later by the insertion of a ventriculoperitoneal shunt.

While being treated by lumbar puncture the patient was enterally fed and suffered no gastrointestinal upset. After 7 taps (mean volume 19 ml) the serum sodium concentration fell from 131 to 114 mmol/l. Oral sodium supplements of 7·5 mmol/kg a day were given and the serum sodium level rose gradually to 130 mmol/l over 7 days. Sodium supplements were continued until regular drainage of the cerebrospinal fluid was no longer required.

Discussion

Hydrocephalus can cause cerebral damage, either by direct compression[4] or by vascular compromise.[5] Attempts have therefore been made to arrest the progress of ventricular dilation by repeat cerebrospinal drainage.[6] No adverse effects on serial electrolyte concentrations were found in this series. However, cerebrospinal fluid contains an appreciable concentration of sodium. In Case 2 we measured the sodium concentration on 8 occasions. The levels ranged from 96 to 149 mmol/l. A typical ventricular tap of 20 ml in a 1 kg infant may therefore contain as much as 3 mmol of sodium. This is nearly equal to the total daily requirements.

Serum electrolyte concentrations are not routinely performed in fully enterally fed infants. The symptoms of hyponatraemia are non-specific and may be attributed to underlying hydrocephalus. We recommend that the serum sodium concentration is regularly measured in all infants who require frequent drainage of the cerebrospinal fluid.

References

1 Papile L A, Burstein J, Burstein R, Koffler H. Incidence and evolution of subependymal and intraventricular hemorrhage. A study of infants with birthweights less than 1·500 gm. *J Pediatr* 1978: **92:** 529–34.

2 Deonna T, Payot M, Probst A, Prod'hom L-S. Neonatal intracranial hemorrhage in premature infants. *Pediatrics* 1975; **56:** 1056–64.

3 Pape K E, Blackwell R J, Cusick G, *et al.* Ultrasound detection of brain damage in preterm infants. *Lancet* 1979; **i:** 1261–4.

4 Weller R O, Schulman K. Infantile hydrocephalus: clinical, histological, and ultrastructural study of brain damage. *J Neurosurg* 1972; **36:** 255–65.

5 Wozniak M, McLone D G, Raimondi A J. Micro and macrovascular changes as the direct cause of parenchymal destruction in congenital murine hydrocephalus. *J Neurosurg* 1975; **43:** 535–45.

6 Papile L A, Burstein J, Burstein R, Koffler H, Koops B L, Johnson J D. Post-hemorrhagic hydrocephalus in low birthweight infants: treatment by serial lumbar punctures. *J Pediatr* 1980; **97:** 273–7.

Correspondence to Dr P MacMahon, Charing Cross Hospital, Fulham Palace Road, London W6 8RF.

Received 10 February 1983