Correspondence

Hyponatraemia and CSF drainage in posthaemorrhagic hydrocephalus

Sir,

MacMahon and Cooke1 have shown that repeated cerebrospinal fluid drainage in low birthweight babies can create a negative sodium balance due to extra loss of sodium. They do not, however, exclude excessive renal sodium leak or syndrome of inappropriate antidiuretic hormone secretion (SIADHS). SIADHS has been well documented in neonates2 3 and intracranial haemorrhage is a known predisposing factor. Sodium content of tapped cerebrospinal fluid can be measured easily and accurate replacement done. As the tapping continues occasional serum sodium estimation could be done to exclude hyponatraemia, which, if present, would be due to causes other than sodium loss. Before giving the massive sodium supplements of the magnitude of 23 mmol/kg/day that were needed in MacMahon and Cooke’s second case, one must exclude SIADHS. Differentiation of these two conditions is crucial, as treatment of each is diametrically opposite. Simultaneous measurements of serum and urinary osmolality would resolve the question in most patients.

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Drs MacMahon and Cooke comment:

All the infants we reported were at least 20 days old when sodium supplements were commenced. A particularly large renal sodium leak is atypical in well infants at this postnatal age. Although the syndrome of inappropriate antidiuretic hormone secretion (SIADHS) is associated with intraventricular haemorrhage this usually occurs at the time of the haemorrhage and not later on when obstructive hydrocephalus has developed. We agree, however, that it would be prudent to investigate for renal sodium loss and SIADHS should the electrolyte disturbances we described recur despite sodium replacement equal to the sodium loss in the drainage cerebrospinal fluid (CSF). The purpose of our report was to draw attention to the occurrence of electrolyte disturbances during repeated CSF drainage. Previous reports have specifically stated that this was not a problem.

References


Perinatal listeriosis and hospital cross infection

Sir,

Listeria monocytogenes is associated with severe disease in the fetus and the newborn infant and in the past has been associated with high morbidity and mortality.1 Even 50 years after its original description, the epidemiology and pathogenesis of perinatal listeriosis is not fully understood. The development of a phage typing system,2 with its ability to differentiate between strains of different origin raises hopes of obtaining useful epidemiological information on clusters of cases and in instances of hospital cross infection. We have already reported its use in the study of outbreaks involving adults and neonates3 4 and here report two more cases where phage typing of isolates from neonates supports the view that cross infection occurred.

Both infants were born at term, the mothers came from different geographical areas of Leicester, and there was no history of miscarriage or abortion. They did not note any episodes of diarrhoea during pregnancy but the mother of the first baby suffered an influenza like illness two months before delivery. Both mothers had an uneventful puerperium and listeria was not isolated from high vaginal swabs.

Case 1

A boy of 38 weeks’ gestation, weighing 2400 g, had an early onset disease, and presented with respiratory distress at birth. Liquor was meconium stained. Surface swabs showed heavy growth of L. monocytogenes but blood and cerebrospinal fluid were sterile, although the latter had an increased number of leucocytes, mainly polymorphonuclears. He made satisfactory progress after treatment with ampicillin and gentamicin.

Case 2

A girl born at 39 weeks’ gestation, weighing 3260 g, was nursed with the mother until day 8, when she became ill and septicaemia and meningitis were diagnosed.
Surface swabs were all negative but *L. monocytogenes* was grown from blood cultures and CSF. She made a complete recovery after treatment with ampicillin, gentamicin, and chloramphenicol. Her follow up assessments did not show any sequelae.

The strains of *L. monocytogenes* from the two infants were both of serovar 4, and on phage typing were indistinguishable giving the same octal code of 000 520 000 suggesting either a common source outbreak or cross infection. The fact that the two babies were born by caesarean section in the same obstetric theatre within a 12 hour period, and that they were attended by the same paediatrician on the same resuscitaire makes, in our view, the latter the more likely probability.

This report strongly supports the plea that all future *L. monocytogenes* isolates from cluster or outbreaks of listeriosis should be phage typed and detailed epidemiological investigations made in an effort to elucidate the mechanisms involved. The survival of both babies supports the view that the high mortality rate must no longer hold for today’s infants receiving swift adequate treatment and supportive management.1

References


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Cleft palate and oesophageal atresia

Sir,

In a most useful study, paediatric surgeons in Glasgow noted that 6 of 114 (5.3%) patients with oesophageal atresia also had cleft palate.1 In fact this is a pretty high incidence. The authors quoted an American series that reported the association in 3.1% cases. However, in a series of 345 cases in the South West of England, only 4 had a cleft palate (with cleft lip in one), an incidence of 1.2%.2 The difference between the Scottish and English figures is statistically significant at the 2.5% level.

It seems likely that oesophageal atresia is aetologically heterogeneous, and a rather non-specific consequence of several teratological processes.3 The rather high incidence of cleft palate in the Glasgow series suggests the local operation of some different aetiological factors. Oesophageal atresia is a common defect (incidence 0.3–4/1000 births),3 yet has attracted remarkably little epidemiological research—surprising in view of Knox’s albeit unconfirmed report4 of clustering of cases. Similar studies from other paediatric surgeons could be most productive.

References


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Caseating regional lymphadenitis complicating BCG vaccination

Sir,

We strongly disagree with Tam et al.1 in their management of axillary adenitis after BCG immunisations. They describe 6 cases treated by surgical dissection and excision of the axillary nodes followed by multiple antituberculous chemotherapy for 6 months.

We believe that such treatment is unjustified and will eventually adversely affect the acceptability of their immunisation policy in infants. We have seen some 16 cases in Dominica. Initially we began treatment with isoniazid alone, but later we have given no treatment. The adenitis has slowly resolved over many weeks in all cases. One patient required surgical incision because of abscess formation and in two the abscesses drained spontaneously. This policy of no treatment is supported