antibody is associated with a favourable outcome.6

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Dr McIntosh and Professor Isaacs comment: Our table 2 was in fact obtained from an earlier article published by the same authors in the Communicable Disease Report 4th April 1986 (CDR 86/14), which cannot be used as a reference. We had overlooked the increased numbers of children studied in Dr Miller’s 1989 Lancet article,1 and regret this. We do not necessarily agree, however, with her subsequent conclusions.

The neonatal chickenpox depends on the dose of virus as well as the presence or absence of maternal antibody. Epidemiologically most of the severe and fatal cases have been babies whose mother developed chickenpox in the first few days before delivery. In the paper of Miller et al.16 of 19 ‘severe’ neonatal chickenpox cases were babies whose mother’s rash was 4 days before to 2 days after birth. The 1991 Red Book has the same recommendation VZIG for maternal chickenpox at 5–2 days,2 even though the JCVI has been persuaded to change to 7 days on theoretical grounds.

There have been anecdotal reports of fatal postnatally acquired chickenpox, but there are also fatal cases in apparently immune competent children and adults. It is not known whether the incidence of fatal disease is higher in postnatally infected neonates than later in life, and the Red Book does not recommend VZIG for these babies.


Proportion of neonates with antibodies to varicella zoster at birth according to onset of maternal rash*

<table>
<thead>
<tr>
<th>Onset of maternal rash</th>
<th>Proportion of babies with detectable antibody*</th>
</tr>
</thead>
<tbody>
<tr>
<td>before delivery (days)</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>0.60</td>
</tr>
<tr>
<td>1–2</td>
<td>3.15%</td>
</tr>
<tr>
<td>3–5</td>
<td>4.0%</td>
</tr>
<tr>
<td>6</td>
<td>7.6%</td>
</tr>
<tr>
<td>7–14</td>
<td>38.3%</td>
</tr>
</tbody>
</table>

*1 arbitrary unit detected by enzyme linked immunosorbent assay.

Echocardiographic assessment of cardiac function in shocked very low birthweight infants

EDITOR,—In their excellent article Gill and Weindling state that neonatal serum electrolyte values are not routinely measured immediately after birth because they, ‘invariably reflect maternal levels’. While this statement may be true, and is a frequent excuse for not determining newborn serum electrolyte concentrations early on, obstetricians regularly provide appropriate maternal results. Neonatologists suffer from this lack of information and are frequently surprised at serum electrolyte values eight or 12 hours after birth.

Electrolyte concentrations hold considerable interest for caretakers of sick newborns. Furthermore, electrolyte levels influence whether maternal antibodies reach the neonates’ circulation at parturition or the neonatologist should measure the baby’s concentration shortly after birth. To wait eight or 12 hours because values reflect maternal levels, yet not to know maternal or newborn levels, is a cop out.

For Gill and Weindling not to have presented such information significantly tarnishes an otherwise excellent, informative report.

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Dr Gill and Weindling comment: Unfortunately Dr Scanlon has failed to quote exactly as written in the paper regarding serum electrolyte levels in the first few hours after birth. In our discussion we stated ‘Serum calcium and potassium concentrations were not routinely measured in the first few hours after birth as they invariably reflect maternal levels’. Subsequent measurements of calcium and potassium after 12 hours of age did not show any difference between the groups.3 As the infants did not receive additional calcium and potassium during the first 24 hours while the serum electrolytes were constant, it is unlikely to be abnormal at the time of the echocardiogram. To our knowledge, we have not seen any research evidence to suggest that electrolytes should be measured routinely in the first few hours after birth in all preterm infants.

Changes in cerebral artery blood flow velocity after intermittent cerebrospinal fluid drainage

EDITOR,—We agree with the findings of Kempley and Gamsu1 but dispute their suggestion that these may be used to establish the presence of cerebrospinal fluid (CSF) drainage in posthaemorrhagic hydrocephalus (PHH).

In a similar study of PHH and ventricular dilatation, we showed a reduction in the pulsatility index and increase in mean flow velocity after CSF taps, but because of the very wide range of normal values, we were unable to construct a ‘across the line’ to indicate the need for intervention.2 In addition, a number of factors unrelated to intracerebral haemodynamics (such as hypoacapnia or the presence of a patent ductus arteriosis) may influence the pulsatility index and cerebral blood flow velocity in the ill newborn with PHH. This is not the case in older children with hydrocephalus in which a clear clinical role for Doppler ultrasound exists.3

We believe that the changes observed by Kempley and Gamsu are acute changes and are of interest so far as they may reflect the process of autoregulation but that they are unlikely to guide the timing of intervention. Furthermore, it is possible, in the absence of accurate measurement of the angle of insonation, that the flow velocity changes merely reflect a change in vessel distortion with CSF taps rather than a true change in flow velocity.

Intervention should continue to be guided by the rate of head growth and symptoms of raised intracranial pressure4 rather than changes in Doppler parameters.

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Dr Kempley and Dr Gamsu comment: Dr Quinn and Professor Levene suggest that the changes in blood flow velocity (BFV) we observed after intermittent ventriculostomy drainage be due to alterations in the angle of insonation from changes in vessel distortion. This is not the case, as in all measurements we visualised the artery and corrected for the angle of insonation.

We agree that at present, intervention in posthaemorrhagic hydrocephalus should be guided by the rate of head growth and by symptoms and signs of raised intracranial pressure. However, both of our studies demonstrated considerable variations in cerebral artery BFV with this form of management.1,2 Compromise of the cerebral circulation by raised intracranial pressure in combination with other circulatory factors, could cause further cerebral damage in some infants.

The lack of a consistent reduction in cerebral artery BFV in the face of the onset of ventricular dilatation (ventricular index > 2 SD from the mean) may have been because intracranial pressure was not increased at this early stage of ventricular dilatation.

Neither study found a significant correlation between CSF pressure and BFV, perhaps because an infant’s ability to maintain cerebral blood flow in the face of raised intracranial pressure depends on a number of factors, such as arterial blood pressure or the presence of a patent ductus arteriosis.

Before dismissing Doppler ultrasound as a tool which could aid decision making in...