antibody is associated with a favourable outcome.6

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Dr McIntosh and Professor Isaac comment: Our table 2 was in fact obtained from a 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 presence of 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 1 to 3 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 delivery.1 The 1991 Red Book has not yet recommended varicella immunoglobulin (VZIG) for maternal chickenpox at 5 to + 2 days, even though the JCVI has been persuaded to change to + 7 days on theoretical grounds.

There have been anecdotal reports of 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 before delivery (days)</th>
<th>Proportion of babies with detectable antibody*</th>
</tr>
</thead>
<tbody>
<tr>
<td>= 2</td>
<td>060</td>
</tr>
<tr>
<td>3–5</td>
<td>31/56</td>
</tr>
<tr>
<td>6</td>
<td>4/9</td>
</tr>
<tr>
<td>7</td>
<td>5/6</td>
</tr>
<tr>
<td>8–14</td>
<td>38/38</td>
</tr>
</tbody>
</table>

*1 arbitrary unit detected by enzyme linked immunosorbent assay.

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

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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.’ As the infants did not receive additional calcium and potassium during the first 24 hours with the serum electrolytes were likely 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.

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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 in 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 measurements and doppler ultrasound, that the flow velocities change merely reflect a change in vessel distortion with CSF taps rather than a true change in flow velocities. 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 cerebrospinal fluid drainage may 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 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 this study after 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 increased intracranial pressure depends on a number of factors, such as arterial blood pressure or the presence of a patent ductus arteriosus.

The popular dismissing Doppler ultrasound as a tool which could aid decision making in


Echocardiographic assessment of cardiac function in shocked very low birthweight infants.
J W Scanlon

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