LETTERS TO THE EDITOR

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A national review of neonatal resuscitation programmes for midwives

Editor,—A considerable number of babies require help in establishing respiration at birth. Resuscitation training was compulsory for midwives in 132 (72%) units. Mann-Whitney U test was used to compare average (range 9.2–5.8 months) (range 6–24), with 148 (86%) units holding a logbook of attendance. There are regional differences in the availability of resuscitation programmes (range 77–100%), existence of standards of achievement (range 1–50%), and existence of compulsory resuscitation programmes (range 50–92%).

Currently, no individual region has the highest scores for all the categories stated.

This is the first national survey examining neonatal resuscitation programmes for midwives. Most (88%) of the 196 maternity units that responded have some form of resuscitation programme available for midwives. However, the programme in 42% of these units does not directly follow the Neonatal Life Support Course. Recommendations for continuous education are provided by the Neonatal Life Support Group and the Neonatal Resuscitation Project of the American Academy of Pediatrics and others. We recommend that midwives be trained in neonatal resuscitation, both in theory and practice. Encouragingly, 84% of units currently not following the UK Resuscitation Council guidelines expressed a desire to change accordingly.

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Table 1 Clinical details of five neonates presenting with hypernatraemic dehydration

<table>
<thead>
<tr>
<th>Infant</th>
<th>Maternal parity</th>
<th>Birth weight (kg)</th>
<th>Day next weighed (kg)</th>
<th>Weight loss (%)</th>
<th>Plasma sodium</th>
<th>Plasma area</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>3.430</td>
<td>8</td>
<td>25</td>
<td>168</td>
<td>25</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>2.120</td>
<td>7</td>
<td>24</td>
<td>172</td>
<td>34.8</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>3.599</td>
<td>6</td>
<td>19</td>
<td>158</td>
<td>46.3</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>3.630</td>
<td>6</td>
<td>20</td>
<td>150</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>2.420</td>
<td>4</td>
<td>13</td>
<td>157</td>
<td>6.1</td>
</tr>
</tbody>
</table>


Hypernatraemia: why bother weighing breast-fed babies?

Editor,—Hypernatraemic dehydration is associated with cerebral oedema, intracranial haemorrhage, hydrocephalus, gangrene, and death,1 but is notoriously difficult to detect clinically. It is accepted in paediatric practice that weighing is an essential part of the assessment of an infant’s hydration.

In Bristol, one neonate a month is admitted with hypernatraemic dehydration pending to delayed recognition of inadequate lactation. We believe that this is a result of the reluctance of midwives to weigh breast-fed infants.

Five neonates admitted after they became unwell from dehydration secondary to lactation failure are described (table 1). Assessment of lactation and hydration had raised no concern. Four of five of these mothers were primiparous and four of five of these breast fed infants during labour. We believe this is a result of the reluctance of midwives to weigh breast-fed infants.

In common with the American Academy of Pediatrics and others, we recommend that breast fed infants should be weighed between 72 and 96 hours after birth when normal weight loss is at its maximum.

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EDITOR,—Following the recent media interest in pericardial tamponade complicating the use of percutaneous central venous catheters in neonatal patients, we wish to alert readers to the possible risk of right atrial placement of percutaneous central venous catheters in neonatal patients, we wish to alert readers to the possible risk of right atrial placement of percutaneous central venous catheters.

Although the caval position carries a small risk of thrombosis or hydrothorax, these complications are more benign than pericardial tamponade, which has a mortality of 65%. We recommend that placement of a percutaneous central venous catheter tip in the right atrium should now be avoided. In addition, we suggest that catheters that display angulation, curvature, or looping of the tip should be avoided.

placement of neonatal central venous catheter tips in the right atrium: a practice to be avoided?

Newborns have unique confounding factors regarding the TIR-F ratio

EDITOR,—Sweet et al investigated the serum transferrin receptor (STIR) and, for the first time in neonates, transferrin receptor-log ferritin (TIR-F) ratio in a prospective series of cord blood taken from term infants and their mothers. They are to be congratulated on completing another piece of the complex jigsaw that is fetal and neonatal iron metabolism.

STIR and TIR-F were increased in iron deficient mothers, but not in their infants. The authors discuss at some length the translational (not transcriptional as stated in the discussion) control of intracellular ferritin synthesis. They measured serum ferritin, which is a glycosylated form of L-ferritin, and has been shown to correlate with intracellular iron in the absence of confounding factors. However, serum ferritin is secreted in response to a wide variety of stimuli, including, for example, inflammation and shows gender differences in newborns. Of these confounding factors, serum ferritin may not accurately represent tissue iron stores.

It has already been reported that STIR does not correlate with other measures of iron metabolism in the newborn. Therefore, it is highly expressed by reticuloocytes and other immature erythroid cells, with or without iron deficiency. The high specificity and sensitivity of the TIR-F ratio in adults is based upon their relationship in iron deficiency in the absence of factors that might otherwise elevate STIR levels. With both variables subject to these confounding factors in the neonate, I do not agree with the author's assertion that the TIR-F index “gives a measure of iron requirements in relation to iron availability” in this unique population.

Changes in plasma creatinine in first 72 hours of life

EDITOR,—Recently, Miall et al have reported a rapid rise in serum creatinine in the first 48 hours of life in neonates. We have noticed in our clinical daily to day practice that this rise is transient and may not be clinically significant. To confirm this, we looked at our initial serum creatinine levels in a stable group of term neonates admitted to the neonatal intensive care of King Fahd Hospital of the University, Al-Khobar, Saudi Arabia.

Neonates with congenital anomalies, peri- natal asphyxia, and those requiring ventilatory support were excluded. The serum creatinine levels were measured together with electrolytes by using an automatic analyser (Dimension, Delaware, USA), which were relayed by the reporting computer system (Ulti-view, Los Angeles, USA). Serum creatinine was available for the first 72 hours on a limited number of neonates as most of the selected babies were stable within the first 48 hours of life and there was no need for extra serum electrolyte and creatinine measurements. Out of all the newborns admitted during the three month study period who fulfilled the inclusion criteria, 13 neonates had serum creatinine measurements available for the first 72 hours. These readings were noted down and were analysed using the SPS statistical package to calculate the mean, standard deviation and statistical significance.

Out of thirteen neonates, seven (53.8%) had an increase in their plasma creatinine on the second day while four (30.7%) had a
Haemoglobinopathy as a cause of nucleated red cells in the fetus and neonate

EDITOR,—We are interested in the article by Hermansen on the causes of peripheral nucleated red blood cells in newborn children and would add another differential diagnosis to this finding.

In the last decade, we have discovered two families affected by haemoglobin disorders where the diagnosis was suspected by the presence of high numbers of nucleated red cells in neonatal blood tests. In neither family was the potential for significant haemoglobin disorders suspected. The families concerned were Indian Asian in origin and the marriages were consanguineous. The children now present with thalassaemia intermedia, but because of the difficulty in predicting the clinical course of these disorders, it is not yet clear whether they will become transfusion dependant, although this is highly likely for two individuals, one in each family.

The first recognised child in Family I was born in 1991. A blood test performed because of jaundice on the third day of life showed 160NRBC/100WBC. Other causes of erythroblastosis were excluded. Haemoglobin analyses on the parents showed that the mother was heterozygous for Indian inversion/ deletion dh-thalassaemia and the father was a compound heterozygote for dh-thalassaemia and Haemoglobin Headington. This child and two other children are homozygous for dh-thalassaemia. The eldest child seems more severely affected and has been transfused twice, following infections.

The second family presented in 1996 when their first son was found at birth to have 2000NRBC/100 WBC. Other causes having been excluded, haemoglobin studies revealed only the existence of b-thalassaemia trait (codon 1660) in the father. The boy is now anaemic, has thalassaemia bissing of the skull and splenomegaly, and looks as if he will need a transfusion programme. A brother, born in 1999, had 983NRBC/100WBC in his initial blood test, and has also inherited his father’s haemoglobin pattern. It is likely that this family is showing dominant b-thalassaemia, although recent studies suggest there may be a co-inherited aldolase deficiency, akin to aldolase, from the mother. (J Porter, personal communication).

We hope this report may help in the investigation of other families.

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Neonatal paroxetine withdrawal syndrome or actually serotonin syndrome?

EDITOR,—We would like to comment on the article “Neonatal paroxetine withdrawal syndrome” in the March 2001 issue of the journal. The authors describe what they have called “neonatal paroxetine withdrawal syndrome”. However the syndrome reported in the 4 neonates appears to be more consistent with serotonin toxicity, rather than withdrawal of paroxetine.

The literature to date contains one large series, two similar case reports with fluoxetine1 and two case reports with paroxetine.2 In the fluoxetine cases the syndrome was not described as a withdrawal phenomenon. In the first, a neonate born to a mother on fluoxetine had jitteriness, irritability, tachypnoea, temperature instability, tremors, increased muscle tone, and a hyperactive Moro reflex.3 All except the last of these are clinical features seen in serotonin toxicity in adults using selective serotonin uptake inhibitors (SSRIs) therapeutically or in overdose.4 The neonate in this case had fluoxetine levels that were measurable initially and which fell as symptoms resolved.

In the two case reports with paroxetine, the syndrome is referred to as a withdrawal phenomenon. However the time course and symptoms were similarly typical of serotonin excess.

In the cases reported by Stiskal et al, the neonates developed the features soon after birth and they resolved over a period of days. In case 2 an increased serum paroxetine level was reported in the infant. The level was too low to detect by day 15, supporting a toxicity phenomenon. Similarly, in case 4 there was a raised serum paroxetine level at the time of the adverse effects. Serum paroxetine levels have been positively related to serotonin toxicity in adults.

The features of case 4 may also have been exacerbated by the use of opiates in the delivery room. Pethidine is a well recognised cause of serotonin toxicity in conjunction with a serotonergic agent.5 By March 2001, there were 13 reports to the Australian Drug Reaction Advisory Committee classified as “withdrawal syndrome

Table 1

<table>
<thead>
<tr>
<th>Child</th>
<th>Date of birth</th>
<th>Age (days)</th>
<th>Hb (g/dl)</th>
<th>WBC (corrected)</th>
<th>NRBC/100WBC</th>
</tr>
</thead>
<tbody>
<tr>
<td>ZR (mAle)</td>
<td>20/07/90</td>
<td>3</td>
<td>11.1</td>
<td>13.0</td>
<td>22</td>
</tr>
<tr>
<td>MR (femalE)</td>
<td>16/08/91</td>
<td>5</td>
<td>12.7</td>
<td>24.0</td>
<td>160</td>
</tr>
<tr>
<td>ZR (female)</td>
<td>09/10/96</td>
<td>9</td>
<td>13.3</td>
<td>9.0</td>
<td></td>
</tr>
<tr>
<td>HA (male)</td>
<td>16/07/96</td>
<td>1</td>
<td>13.7</td>
<td>11.7</td>
<td>2000</td>
</tr>
<tr>
<td>AA (male)</td>
<td>02/07/99</td>
<td>1</td>
<td>14.2</td>
<td>13.9</td>
<td>983</td>
</tr>
</tbody>
</table>

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neonatal" in conjunction with maternal use of an SSRI. However, on perusal, many appear to describe serotonin toxicity. We have also been involved with the management of a neonate, born to a mother following a sertraline overdose, who exhibited features of serotonin toxicity. In this case there was a single maternal ingestion 1 hour before delivery and therefore no earlier foetal exposure to cause withdrawal.

We are concerned about the increasing use of the term “neonatal withdrawal syndrome” in symptomatic neonates being born to mothers on SSRIs. This may prompt the use of SSRIs themselves to treat the condition with the potential to increase toxicity. The condition should be correctly referred to as “neonatal serotonin toxicity” or, less specifically, poor neonatal adaptation secondary to serotonergic agents.

Authors’ response

E:—Isbister and colleagues point out important issues in defining the syndrome we and others described.1 2 Their argument is that the described syndrome is due to a hyper serotonergic state, rather than a lack of serotonin effect, as there is considerable overlap between the two entities. The cause of the discontinuation syndrome in adults also remains incompletely understood.3

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