LETTERS TO THE EDITOR

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A national review of neonatal resuscitation programmes for midwives

**EDITOR**—A considerable number of babies with no obstetric or neonatal at risk factors require help in establishing respiration at birth. Newborn babies in delivery units and at home. Primary involvement in the initial resuscitation of newborn babies in delivery units and at home. 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, North West hospitals have high scores in the above three categories stated. Overall, there is no individual region that 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. The programme is recommended by the UK Resuscitation Council. Moreover, 67% of programmes have no established standards. The average period of reassessment in these units is nine months. This interval may be too long because the knowledge and retention has been shown to be lost within six months of a neonatal resuscitation programme.

The specific needs of UK midwives to provide basic neonatal life support have been objectively evaluated, in contrast with the United States and Canada. There is a collective call for consistent skills attainment, nationally and internationally. The availability of competent and skilled personnel may contribute to regional differences in resuscitation programmes. Continued structural differences in neonatal resuscitation programmes will further exaggerate differences in local and national practice. Hence, we recommend to establish uniform standards in neonatal resuscitation and for mandatory hospital trust support not only in organising suitable resuscitation programmes, but also in compulsory attendance by midwives at these essential training sessions. 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 parities</th>
<th>Birth weight (kg)</th>
<th>Day next weighed</th>
<th>Weight loss (%)</th>
<th>Plasma sodium</th>
<th>Plasma area</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 0</td>
<td>0</td>
<td>3.430</td>
<td>8</td>
<td>25</td>
<td>168</td>
<td>25</td>
</tr>
<tr>
<td>2 7</td>
<td>1</td>
<td>2.120</td>
<td>12</td>
<td>24</td>
<td>172</td>
<td>34.8</td>
</tr>
<tr>
<td>3 6</td>
<td>2</td>
<td>3.599</td>
<td>19</td>
<td>158</td>
<td>46.3</td>
<td>43.8</td>
</tr>
<tr>
<td>4 0</td>
<td>3</td>
<td>3.630</td>
<td>20</td>
<td>150</td>
<td>43.0</td>
<td>33</td>
</tr>
<tr>
<td>5 5</td>
<td>4</td>
<td>2.420</td>
<td>13</td>
<td>157</td>
<td>6.1</td>
<td>33</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, 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 hypertonic dehydration secondary to delayed recognition of inadequate lactation. We believe that this is a result of the reluctance of midwives to weigh breast fed infants.

Five mothers 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 primiparae. The hypertonic dehydration as the result of failure to establish lactation is well described, although not recently in this country. Associated factors include first time motherhood, poor support of lactation, and failure to monitor early weight loss.

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Newborns have unique confounding factors regarding the TIR-F ratio

**Editor—** Sweet et al investigated the 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 some length the translational (not transcriptional as stated in the discussion) control of intracelluar 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 other stimuli, including, for example, inflammation and shows gender differences in newborns. Under these circumstances, 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. Under these circumstances, serum ferritin may not accurately represent tissue iron stores. Under these circumstances, serum ferritin may not accurately represent tissue iron stores. Under these circumstances, serum ferritin may not accurately represent tissue iron stores. Under these circumstances, serum ferritin may not accurately represent tissue iron stores. Under these circumstances, serum ferritin may not accurately represent tissue iron stores.
The drop to 0.44, as compared with day 1 of disease.

Activity level in early newborn period does not add validity to our preliminary results.

A larger study will provide more reliability and be wide variation in the results. However, a

The purpose of study. The results with 12 out of 13 (92%) had restricted as no extra blood was extracted for most of the creatinine levels (92%) had

pared with preterm infants.

levels in term and near term infants as com-

ceptional age, reflected by lower creatinine GFR has shown to be a function of postcon-

serum creatinine level by 72 hours. This

cohort had dropped to a significant level, of life, 12 out of 13 (92%) of the cases had a

reduction. The remaining two (15.3%) had no change in their creatinine. After 72 hours of life, 12 out of 13 (92%) of the cases had a reduction in the creatinine (fig 1). By the third day, the mean serum creatinine of the cohort had dropped to a significant level, 0.64 mg/dl to 0.44 mg/dl, p = 0.04 (table 1).

The study demonstrated a trend of fall in serum creatinine level by 72 hours. This reflected the improvement in the renal function and GFR. The improvement in the GFR has shown to be a function of postcon-

ceputational age, reflected by lower creatinine levels in term and near term infants as com-

pared with preterm infants.

We noted that the rise in creatinine in first 72 hours was consistent and by the third day most of the creatinine levels (92%) had dropped down. One could argue about the sample size of our study. The sample size was restricted as no extra blood was extracted for the purpose of study. The results with 12 out of 13 cases (92%) suggest that there may not be wide variation in the results. However, a larger study will provide more reliability and validity to our preliminary results.

In conclusion, caution should be exercised in interpretation of serum creatinine levels in the first 72 hours of life. A raised serum creatinine level in early newborn period does not necessarily indicate renal failure or kidney disease.

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We hope this report may help in the inves-
tigation of other families.

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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 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 1 was born in 1991. A blood test performed because of jaundice on the third day of life showed 160NRBC/100WBC. Other causes of erythroid blastosis were excluded. Haemoglobin analyses on the parents showed that the mother was heterozygous for Indian inversion/ deletion db-thalassaemia, while the father was a compound heterozygote for db-thalassaemia and Haemoglobin Headington.1 This child and two other children are homozygous for db-thalassaemia. The eldest child seems more 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 16bO) in the father. The boy is now anaemic, has thalassaemic bossing 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).

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>2</td>
<td>11.1</td>
<td>13.0</td>
<td>22</td>
</tr>
<tr>
<td>MR (female)</td>
<td>16/08/91</td>
<td>3</td>
<td>12.7</td>
<td>24.0</td>
<td>160</td>
</tr>
<tr>
<td>ZR (female)</td>
<td>09/01/96</td>
<td>9</td>
<td>13.3</td>
<td>9.0</td>
<td>950</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>

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.2 The authors describe what they have called “neonatal paroxetine withdrawal syn-

drome”. 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,3 two similar case reports with fluoxetine4 and two case reports with paroxetine.5

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, tachy-noea, temperature instability, tremors, in-
creased muscle tone, and a hyperactive Moro reflex.6 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.7 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 similar to typical of serotonin excess.

In the cases reported by Stiskal et al7 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 syndrome, rather than a withdrawal 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 deliver-
y room. Pethidine is a well recognised cause of serotonin toxicity in conjunction with a serotonergic agent.8

By March 2001, there were 13 reports to the Australian Drug Reaction Advisory Committee classified as “withdrawal syndrome


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neonatal 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.

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Authors’ response

EDITOR,—Isbister and colleagues point out and others described. Their argument is that the described syndrome is due to a hyper serotonergic state, rather than a lack of serotonergic effect, as the term “withdrawal” suggests. We agree that this issue must be clearly solved because of the significant implications in the clinical management of some of the patients, especially concerning the role of continued breastfeeding. At the same time, we are unsure whether we have sufficient data to declare that this is a hyper serotonergic condition. When we started summarising our experience as a report, we debated what terminology should be used to describe our patients. The term “SSRI discontinuation syndrome” was considered as it simply describes the temporal relationship between the dose and the syndrome. However, we opted for “withdrawal” because of its common use in similar cases in the literature. For example, a report by Kent and Laidlaw describes a full term healthy boy born to a mother on sertraline who was breast fed for three days. A day after weaning he developed agitation, poor feeding, constant crying, insomnia, and an enhanced startle reaction. These effects intensified over 48 hours then subsided. The time course in this case strongly suggests a withdrawal reaction. Our 2 cases had therapeutic serum concentrations of the drug. However, we do not know the concentrations prior to the presentation, hence the interpretation of the data is not as simple as Isbister and the colleagues indicate.

We think that the conditions we described resulted from a hypo-serotonergic state due to withdrawal. However, the possibility of functional excess of serotonin cannot be ruled out from the clinical assessment alone as there is considerable overlap between the two entities. The cause of the discontinuation syndrome in adults also remains incompletely understood.

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