LETTERS TO THE EDITOR

Rapid responses

If you have a burning desire to respond to a paper published in *ADC* or *FeN*, why not make use of our "rapid response" option?

Log on to our website (www.archdischild.com), find the paper that interests you, click on "full text" and send your response by email by clicking on "submit a response".

Providing it isn't libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on "read eLetters" on our homepage.

The editors will decide, as before, whether to also publish it in a future paper issue.

A national review of neonatal resuscitation programmes for midwives

**Editor,**—A considerable number of babies with no obstetric or neonatal risk factors require help in establishing respiration at birth. This may range from tactile stimulation to bag and mask ventilation to endotracheal intubation and ventilators. Midwives in the United Kingdom are primarily involved in the initial resuscitation of newborn babies in delivery units and at home. There is a national lack of neonatal resuscitation training in the United Kingdom, with inadequate provision of neonatal life support skills remaining an acknowledged contributory factor to perinatal death.1 There are no directives from governing bodies for midwives to weigh breast fed infants. There are pronounced structural differences between the available resuscitation programmes. Those in 100 (58%) midwifery units holding a log book of 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.

M G GNANALINGHAM
C ROBINSON
N A MIR
Neonatal Department, Warrington General Hospital, Lovely Lane, Warrington WA2 1QG, UK
molingham@hotmail.com


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</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>0</td>
<td>3.756</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>70</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>2.420</td>
<td>4</td>
<td>13</td>
<td>157</td>
<td>61</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,2 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 as a direct cause of 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 primigravidas and four of five gave up breast feeding despite encouragement to continue. The weighing practices of midwifery teams and the opinion of neonatologists throughout the South West Region were compared by telephone survey. Four of 13 community midwifery units always or often weighed babies on day 3, 4, or 5, and the other nine routinely weighed for the first time since birth on day 7 or 10. Twelve of 14 consultant neonatologists thought that if care was to be provided by routinely weighing on day 3, 4, or 5 after birth (midwifery practice v consultant opinion, p = 0.006; χ square test).

Hypernatraemic 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.3 Five neonates admitted after they became unwell from dehydration secondary to lactation failure are described (table 1). There are no directives from governing bodies for midwives to weigh breast fed infants. There are pronounced structural differences between the available resuscitation programmes. Those in 100 (58%) midwifery units holding a log book of 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.

M G GNANALINGHAM
C ROBINSON
N A MIR
Neonatal Department, Warrington General Hospital, Lovely Lane, Warrington WA2 1QG, UK
molingham@hotmail.com

2 Edmondson MB, Stoddart JS, Leal HM. Hospital admission with feeding-related problems after early postpartum discharge of normal newborns. *JAMA* 1997;277:589–95.

www.archdischild.com

2 Edmondson MB, Stoddart JS, Leal HM. Hospital admission with feeding-related problems after early postpartum discharge of normal newborns. *JAMA* 1997;277:589–95.
Newborns have unique confounding factors regarding the TFR-F ratio

Editor,—Sweet et al investigated the serum transferrin receptor (STIR) and, for the first time in neonates, transferrin receptor-log ferritin (TFR-F) ratio in a prospective series ofcord blood 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 TFR-F were increased in iron deficient mothers, but not in their infants. The authors discuss at some length the translational (post transcriptional, not post translational) relationship in iron deficiency in the absence of confounding factors.1 However, serum ferritin is secreted in response to a wide variety of other stimuli, including, for example, inflammation and shows gender differences in newborns.2 Although these confounders, 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.3 However, we recommend that catheter tips lie at least 0.5 cm outside the cava. In addition, to allow for the possibility of catheter migration,4 we recommend that catheter tips should lie at least 0.5 cm outside the cava. Although this issue has been the subject of correspondence in the RCPCH email discussion list, there is a need for a wider debate about current practice in the United Kingdom.

Jonathan C Darling
Simon J Newell
Peter R F Dear
Department of Paediatrics and Child Health
Clinical Sciences Building
St James’s University Hospital
Leeds LS9 7TL UK


Letters

Serum creatinine level by 72 hours. This cohort had dropped to a significant level, of 0.44 *(0.13) (0.3–0.7)

Figure 1 Graphic representation of changes in serum creatinine in first 72 hours of life. *mean creatinine values; **p = 0.04, compared with day 1. To convert to nmol/l multiply by 88.4.

Table 1 Differences in the mean creatinine in first 72 hours

<table>
<thead>
<tr>
<th>Number of samples</th>
<th>Time</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>Day 1</td>
<td>0.64 (0.18)</td>
<td>(0.3–0.8)</td>
</tr>
<tr>
<td>13</td>
<td>Day 2</td>
<td>0.68 (0.20)</td>
<td>(0.3–1.0)</td>
</tr>
<tr>
<td>13</td>
<td>Day 3</td>
<td>0.44 *(0.13)</td>
<td>(0.3–0.7)</td>
</tr>
</tbody>
</table>

*The drop to 0.44, as compared with day 1 of 0.68, was statistically significant, p = 0.04.

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 postconceptional age, reflected by lower creatinine levels in term and near term infants as compared with preterm infants.

We noted that the rise in creatinine in 48 hours was significant 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.

HABIB MANZAR
KHALID AL-UMRAN
BASSAM H AL-AWARY
ABDULLATIF AL-FARADY
Division of Neonatology, Department of Pediatrics, King Faisal University and King Fahd Hospital of the University, PO Box 40211, Al-Khobar 31952, Saudi Arabia shahbman@hotmail.com


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 plasma creatinine 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 erythroblastic exclusion 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.

This child and two other children are homozygous for db-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/100WBC. Other causes having excluded, haemoglobin studies revealed only the existence of b-thalassaemia trait (codon 166B) in the father. The boy is now anaemic, has thalassaemia b-skin 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.

CAROL BARTON
MELANIE POLLITZER
Royal Berkshire Hospital, Reading RG1 5AN, UK
m.pollitzer@doctors.org.uk

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.

1 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 fluoxetine and two case reports with paroxetine. 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. 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 syndrome. 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. 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. The neonate in this case had fluoxetine levels that were measurable initially and which fell as symptoms resolved.

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.

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>09/11/90</td>
<td>2</td>
<td>13.3</td>
<td>9.0</td>
<td>56</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>

www.archdischild.com
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.

GEORGE K ISBISTER
ANDREW DAWSON
IAN M WHYTE
Department of Clinical Toxicology and Pharmacology, Newcastle Mater Hospital, Newcastle, Australia
gbite@bigpond.com
FELICITY H PRIOR
CHRISTINE CLANCY
Hunter Drug Information Service
Newcastle, Australia
ANTHONY J SMITH
Discipline of Clinical Pharmacology, University of Newcastle, Australia


Authors’ response

EDITOR,—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 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.3

JOSEPH A STISKAL
Division of Neonatology, Morristown Memorial Hospital
Morristown, NJ 07960, USA
SHINYA ITO
Division of Clinical Pharmacology and Therapeutics
Hospital for Sick Children
Toronto, Ontario, Canada M5G 1X8

Neonatal paroxetine withdrawal syndrome or actually serotonin syndrome?

GEOFFREY K ISBISTER, ANDREW DAWSON, IAN M WHYTE, FELICITY H PRIOR, CHRISTINE CLANCY and ANTHONY J SMITH

Arch Dis Child Fetal Neonatal Ed 2001 85: F145
doi: 10.1136/fn.85.2.F145g

Updated information and services can be found at:
http://fn.bmj.com/content/85/2/F145.7

These include:

References
This article cites 7 articles, 4 of which you can access for free at:
http://fn.bmj.com/content/85/2/F145.7#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/