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**LETTERS TO THE EDITOR**

**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. This may range from tactile stimulation to bag and mask ventilation to endotracheal intubation. Midwives in the United Kingdom are currently not following the Neonatal Life Support Core Curriculum 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 skills retention has been shown to be lost within six months of a neonatal resuscitation programme. There are variably incomplete in their evaluation of neonatal life support, clinical scenarios, and theoretical knowledge of neonatal resuscitation. Resuscitation training was compulsory for midwives in 132 (67%) units that responded that found their training had no form of resuscitation programme available for midwives. However, the programme in 42% of these units does not directly follow the Neonatal Life Support Core Curriculum 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 skills 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 not been objectively evaluated, in contrast with the United States and Canada. In addition, there is a collective call for consistent skills attainment, nationally and internationally. The availability of resources and personnel may contribute to regional differences in resuscitation programmes. Continued structural differences in neonatal resuscitation programmes will further exaggerate differences in local and national practices. Midwives need to establish uniform standards in neonatal resuscitation and for mandatory hospital trust support not only in organising suitable resuscitation programmes, but also in ensuring 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.

**M G GNANALINGHAM C ROBINSON
Neonatal Department, Warrington General Hospital, Lovely Lane, Warrington WA2 1QG, UK moolingham@hotmail.com**

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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 weighing</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>3.120</td>
<td>7</td>
<td>25</td>
<td>172</td>
<td>34.8</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>3.789</td>
<td>6</td>
<td>25</td>
<td>158</td>
<td>48.3</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>3.630</td>
<td>7</td>
<td>20</td>
<td>150</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>3.240</td>
<td>6</td>
<td>20</td>
<td>157</td>
<td>61</td>
</tr>
</tbody>
</table>


**hypernatraemic dehydration**

**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 hypernatraemic dehydration; mandatory 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 hydration and hydration had raised no concern. Four of five of these mothers were primiparous and four of these infants breast fed 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 on the 7th day or after (midwifery practice v consultant opinion, p = 0.006; χ² 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 infant hydration. Identification of excessive weight loss can swiftly identify breast feeding problems, enabling appropriate lactation support to be given and prevention of hospital admission.

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.

**DAVID HARDING**

**PAMELA CARNS**

*Peter Dunn Neonatal Unit St Michael's Hospital Southwell St Bristol BS2 8EG, UK*

**SANJAY GUPTA**

*FIONA COWAN Southmead Hospital Westbury-on-Trym Bristol BS10 5NB, UK*
Placement of neonatal central venous catheter tips in the right atrium: a practice to be avoided?

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 our experience. Our previous policy was to accept right atrial placement of percutaneous central venous catheter tips. This was in line with published recommendations and is still considered acceptable practice in some units in the United Kingdom, in contrast with practice in the United States. Between 1996 and 1997, we had five cases of neonatal pericardial tamponade, three of which resulted in death. All were associated with right atrial tip placement, caused by angulation, curvature, or looping of the line. We have now changed our unit policy to avoid placement of catheter tips in the right atrium, and instead place them in the superior or inferior vena cava. In addition, to allow for the possibility of catheter migration, we recommend that catheter tips should lie at least 0.5 cm outside the cava. In addition, to allow for the possibility of right atrial placement of percutaneous neonatal central venous catheter tips, we wish to alert readers to the need for a wider debate about current practice.

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 7TJ, UK

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 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 TFR 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 correlates 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. Usage of 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. Differences in serum ferritin, serum ferritin may not accurately represent tissue iron stores.

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 7TJ, UK

The reciprocal regulation of the transferrin receptor and ferritin has been reviewed by Hentze and Kuhn.1 We agree that serum ferritin is increased in response to inflammation but the infants that we studied were born at term following normal pregnancies. All the babies were healthy and did not require neonatal care. We think that it is unlikely that inflammation or other stimuli affected our serum ferritin values. Furthermore, in this study1 and in our previous study of preterm infants2 we found no gender differences in contrast to the results published by Tamura et al.3 Our figure for cord ferritin levels at term (listed first as mean ± SD) in female infants is almost identical to that described by Tamura et al (164 + 106 µg/l v 166 + 110 µg/l, but our value for male infants is higher (160 + 97 µg/l v 123 + 71 µg/l). We doubt if there are real gender differences in fetal ferritin levels. Therefore, we are still of the opinion that TFR-F index is a measure of iron requirements in relation to iron availability in the fetus and newborn as in adults and children.

H I Halliday
Department of Child Health
The Queen’s University of Belfast
Belfast, Northern Ireland, UK

T R J Lappin
Department of Haematology

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 7TJ, UK

Changes in plasma creatinine in first 72 hours of life

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

Neonates with congenital anomalies, perinatal 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, 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 reported down and were analysed using the SPSS Windows statistical programme. 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 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 erythroblastosis were excluded. Haemoglobin analyses on the parents showed that the mother was heterozygous for Indian inversion/deletion d-b-thalassaemia, and the father was a compound heterozygote for d-b-thalassaemia and Haemoglobin Headington.1 This child and two other children are homozygous for d-b-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 16bO) in the father. The boy is now anaemic, has thalassaemia bencejohannis and spleenomegaly, 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 5AH, 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,2 two similar case reports with fluoxetine3 and two case reports with paroxetine.4 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.5 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.6 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 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.7

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.8 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 (male)</td>
<td>09/11/96</td>
<td>1</td>
<td>13.3</td>
<td>9.0</td>
<td>0</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.

Geoffrey 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. Their argument is that the described syndrome is due to a hyper serotoninergic state, rather than a lack of serotonin 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 breast feeding. At the same time, we are unsure whether we have sufficient data to declare that this is a hyper serotoninergic 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 weeks. 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 patients 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.

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

JONATHAN C DARLING, SIMON J NEWELL and PETER R F DEAR

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