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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. 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, 2 There are no directives from governing bodies for midwives to attend mandatory neonatal life support update meetings. Moreover, the national availability of specific neonatal resuscitation programmes for midwives is not known.

A standardised written and telephone questionnaire survey of all national maternity units (n = 245) was undertaken. The questionnaire primarily examined duration, structure, and assessment strategies of the resuscitation programmes for midwives.

All 245 maternity units were surveyed by written and telephone questionnaires; 196 responded (80%). Of these, 172 (88%) have some form of resuscitation programme available for midwives. The resuscitation programmes have been in existence for a mean (SD) of 3.7 (2.6) years (range 0.5–20). The programmes involve on average 1.9 main trainers (range 1–5), including senior midwives, paediatricians, and resuscitation training officers. There are pronounced structural differences between the available resuscitation programmes. Those in 100 (58%) units closely follow the Neonatal Life Support course guidelines (UK Resuscitation Council). The programmes in the remaining 72 (42%) units are variably incomplete in their evaluation of neonatal basic life support. Of the units currently not following standard guidelines, 61 (84%) expressed a desire to change. Of the units with resuscitation programmes, 116 (67%) have no standards of achievement set for resuscitation training. Standards were characterised by competence in basic life support, clinical scenarios, and theoretical knowledge of neonatal resuscitation. Resuscitation training was compulsory for midwives in 132 (72%) units. Managers agreed on average every 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%).

Overall, North West hospitals have high scores in the above three categories stated. 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 and is not publicly 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 skill retention has been shown to be lost within six months of a neonatal resuscitation programme. 3

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. 4 In addition, there is a collective call for consistent skills attainment, nationally and internationally. 5 The availability of adequate 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, the 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.

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**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 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 infants 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 were guessing the amount of 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 inpatient care would be provided by routinely weighing on day 3, 4, or 5 after birth (midwifery practice v consultant opinion, p = 0.006; χ 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. 2 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. 3

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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>Birthweight (kg)</th>
<th>Day next weighed</th>
<th>Weight loss (%)</th>
<th>Plasma sodium</th>
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<tr>
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<td>3.420</td>
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<td>13</td>
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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 ofcord 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 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 course, 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, that is, primarily because it is highly expressed by reticuloocytes and other immature erythroid cells, with or without iron deficiency.

The high sensitivity and specificity 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.

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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. 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 on 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 SPSS (statistical package for 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 a nucleated red cell in the fetus and neonate

EDITOR,—We are interested in the article by Hermansen on the causes of peripheral nucleated red cell 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 samples. In both families, there was potential for significant haemoglobin disorders suspected. The families concerned were Indian in origin and the marriage was 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 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/100 WBC. Other causes having been excluded, haemoglobin studies revealed only the existence of b-thalassaemia trait (codon 16bD) in the father. The boy is now anaemic, has thalassaemic bossing of the skull and sphenomegaly, 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 fluoxetine and two case reports with paroxetine. 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. 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 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.

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>
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<th>Age (days)</th>
<th>Hb (g/dl)</th>
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neonatal neonatal 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 symptomatological, poor neonatal adaptation secondary to “neonatal serotonin toxicity” or, less specifically, “neonatal withdrawal syndrome.” This may prompt the use of the term “neonatal withdrawal syndrome” in symptomatological, poor neonatal adaptation secondary to serotoninergic agents.

Authors’ response

EDITOR,—Isbister and colleagues point out the importance of clearly deciding how to define the syndrome we and others described.1 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 breast feeding. 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 subsided 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.1

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