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. 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. There are no directives from governing bodies for midwives to attend mandatory neonatal life support updated. 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 midwives. There are pronounced structural differences between the available resuscitation programmes. Those in 100 (58%) units currently not following the UK Resuscitation Council guidelines expressed a desire to change accordingly.

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Hyponatraemia: why bother weighing breast fed babies?

Editor—Hyponatraemia 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 hyponatraemic 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 primiparas. The case histories showed that the infants were deprived of breastfeeding 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 additional care would be provided by routinely weighing on day 3, 4, or 5 after birth (midwifery practice v consultant opinion, p = 0.006; χ² test).

Hyponatraemia 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 after the early postpartum discharge. 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 Academy of Paediatrics 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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Table 1 Clinical details of five neonates presenting with hyponatraemic 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>
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<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>3.799</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>
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<tr>
<td>5</td>
<td>0</td>
<td>2.420</td>
<td>4</td>
<td>13</td>
<td>157</td>
<td>6.1</td>
</tr>
</tbody>
</table>

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 1991 and 1997, we had five cases of neonatal pericardial tamponade, three of which resulted in death. All were associated with right atrial tip placement as determined by auscultation, percussion, 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 cardiac outline on chest radiograph in small infants, or 1.0 cm outside in larger infants. Although left atrial 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 no longer be accepted. In addition, we suggest that catheters that display angulation, curvature, or looping within the right atrium carry a particularly high risk of pericardial tamponade and demand urgent action. Although this issue has been the subject of correspondence in the RCPCH email discussion list, where the consensus was to avoid right atrial tip position, we believe there is a pressing need for a wider debate about current practice in the United Kingdom.

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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 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. Uterine differences, 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, mainly 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, Miiall 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 have looked at the initial serum creatinine levels on a stable group of term neonates admitted to the neonatal intensive care unit of King Fahd Hospital of the University, Al-Khobar, Saudi Arabia. We compared 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, 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 10.0 statistical package. 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
The drop to 0.44, as compared with day 1 of disease, necessarily indicate renal failure or kidney function. The serum creatinine level in early newborn period does not affect the interpretation of serum creatinine levels in validity to our preliminary results. A larger study will provide more reliability and show wide variation in the results. However, a most of the creatinine levels (92%) 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 first 48 hours was found 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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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 like to 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 db-thalassaemia. The father was a compound heterozygote for db-thalassaemia and Haemoglobin Headington. This child and two other children are homozygous for db-thalassaemia. The oldest child seems more severely affected and has been transfused twice, following infections.

The second family presented in 1996 when their first son was found 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 thalassaemic bosing of the skull and spondomegaly, 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,

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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/100 WBC</th>
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<tr>
<td>ZR (male)</td>
<td>20/07/90</td>
<td>20</td>
<td>11.1</td>
<td>13.0</td>
<td>22</td>
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<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>09/11/96</td>
<td>950</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>14.2</td>
<td>13.9</td>
<td>983</td>
<td></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. This may prompt the use of SSRIs themselves to treat the condition. 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 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 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 et al 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.3 4

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