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 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. There are no directives from governing bodies for midwives to attend mandatory neonatal life support updates. 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 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 nurses. 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, 16 (67%) have no standards of achievement set for resuscitation training. Standards are 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. Mandatory training is assessed 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. Standards are 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.

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

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 recommendations1 and is still considered acceptable practice in some units in the United Kingdom, in contrast with practice in the United States.2 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 positions. The catheter tips were angulated, curved, 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,3 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 an atrial position carries a small risk of thrombosis or hydrothorax,4 these complications are more benign than pericardial tamponade, which has a mortality of 65%.5,6 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 positions, we believe there is a need for a wider debate about current practice in the United Kingdom.

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Newborns have unique confounding factors regarding the TFR-F ratio

EDITOR,—Sweet et al investigated the serum transferrin receptor (STFR) and, for the first time in neonates, transferrin receptor-to-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. STFR 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 intracelluar ferritin synthesis.7 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.8 However, serum ferritin is secreted in response to a wide variety of stimuli, including, for example, inflammation and shows gender differences in newborns.9,10 In these circumstances, serum ferritin may not accurately represent tissue iron stores. It has already been reported that STFR does not correlate with other measures of iron metabolism in the newborn infant, mainly because it is highly expressed by reticuloocytes and other immature erythroid cells, with or without iron deficiency.11

The high sensitivity and specificity of the TFR-F ratio in adults is based upon their relationship in iron deficiency in the absence of factors that might otherwise elevate STFR levels.12 With both variables subject to these confounding factors in the neonate, I do not agree with the author’s assertion that the TFR-F “gives a measure of iron requirements in relation to iron availability” in this unique population.

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Reply

EDITOR,—We thank Peter Reynolds, but feel that our use of the term post-transcriptional is incorrect. The regulation of transferrin receptor mRNA expression is controlled at the transcriptional level by the ironresponsive element (IRE). IREs are stem loop structures of some key messenger RNA (mRNA) encoding proteins of iron metabolism. IREs can be located in the 5′ region—for example, ferritin, or 3′ region—for example, transferrin receptor, of the untranslated region of the mRNA. In relative iron deficiency, the interaction of the IREs with iron responsive proteins, transferrin uptake increases because the transferrin receptor mRNA is stabilised, whereas ferritin storage of iron decreases because translation of ferritin mRNA is blocked. These are clearly post-transcriptional events. The reciprocal regulation of the transferrin receptor and ferritin have recently been expertly 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 well 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 we found no gender differences in contrast to the results published by Tamura et al.2 Our figure for cord ferritin levels at term (listed first as mean + SD) in female infants is almost identical to that of Tamura et al (164 + 106 µg/l vs 166 + 110 µg/l, but our value for male infants is higher (160 + 97 µg/l vs 123 + 71 µg/l). We doubt if there are real gender differences in 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.

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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 daily to day practice that this rise is transient and may not be clinically significant. To confirm this, we assessed 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.

Newborns 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 version 10.0 statistical package with 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
was statistically significant, p = 0.04.

The drop to 0.44, as compared with day 1 of disease.

necessarily indicate renal failure or kidney
atinnate level in early newborn period does not
in interpretation of serum creatinine levels in
validity to our preliminary results.

larger study will provide more reliability and
the purpose of study. The results with 12 out
of most of the creatinine levels (92%) had
48 hours was transient and by the third day
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

In conclusion, caution should be exercised
in comparison to peripheral 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 erythroxanthoblastosis were excluded. Haemoglobin analyses on the parents showed that the mother was heterozygous for Indian inversion/ deletion db-thalassaemia (codon 16bO) in the father. The boy is now 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 have 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 sphenomegaly, and looks as if he will need a transfusion programme. A brother, born in 1999, had 983NRBC/100/WBC 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).
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 the term “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 and Laidlaw3 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.1

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

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