

LETTERS TO THE EDITOR

A survey of neonatal resuscitation training provided to general professional trainees at neonatal units in England and Wales

EDITOR.—The Royal College of Paediatrics and Child Health produced recommendations on teaching and training junior doctors involved in neonatal resuscitation.¹ These stated that all new staff members should receive an induction programme on neonatal resuscitation that includes practice with mannequins. All should also be familiarised with resuscitation equipment on labour wards and be appraised before being permitted to operate alone. Regular updates on neonatal resuscitation training are also recommended and ideally all junior staff's participation in resuscitation procedures should be formalised and documented.

We attempted to assess by telephone questionnaire the current position of neonatal resuscitation training in England and Wales, using a questionnaire of general professional trainees (GPTs). The questionnaire covered key areas within the college recommendations. An attempt within the questionnaire was made to assess the previous neonatal experience of the GPT respondents. A single GPT in each unit was interviewed by telephone. The survey was conducted from October 1999 to January 2000 (the latter part of most attachments). Two hundred and nine units were identified as units with paediatric Senior House Officer cover. One hundred and seventy seven GPTs successfully completed the questionnaire (85%). None contacted were unwilling to participate (some were busy while on duty and others did not answer within three attempts).

Most units (94%) provide a formal theoretical session on neonatal resuscitation during induction (table 1). 86% of units provided practical skills training at induction including endotracheal intubation. Training on practical skills of attaining vascular access in a compromised neonate was undertaken in just 44% of units although 90% of the GPTs were taught on resuscitative function and set up. Not all units provided 24 hour registrar cover on site with 12% of respondents attending deliveries with consultant cover. Only one third (31%) of GPTs interviewed were accompanied by a consultant or

registrar until they achieved their first successful intubation (although it was widely accepted that this was difficult to achieve, especially in some units). Three quarters (75%) did not receive a formal in-post appraisal or test of skills before they were left alone. Post induction training was provided to less than half of respondents (45%) although across the experience range 71% of trainees had already attended (or been encouraged to attend) a course on neonatal resuscitation. Two thirds (69%) of the trainees had little previous neonatal experience in the UK.

When regional units were compared with district general units, training in attaining vascular access appeared to be common in regional units (84% v 39%) along with higher chance of formal appraisal before being left alone for resuscitation (47% v 22%). District general hospitals provided more encouragement to attend courses (73% v 53%) but had fewer trainees with previous experience.

Resuscitation training includes induction with didactic teaching, practical skills training, and ongoing assessment of performance. In an ever changing situation of GPT education, our results need to be interpreted with caution but nevertheless, we believe, reflect current concerns about experience and education offered to neonatal trainees, which will hopefully to be addressed by the Neonatal Life Support Course.

This study indicates that some, but not all, units meet the standards encompassed within the Royal College guidelines. In 1993 Barrie also recorded considerable variation in the delivery of such training in a Northwest area.² This study reveals an identical problem right across England and Wales.

We hope the new neonatal life support will address many of the issues raised within the area of training of GPTs, but universal and mandatory attendance of this is necessary if standards of newborn resuscitation are not to vary unacceptably across England and Wales. We believe this aspect of care should be the subject of quality assurance when assessing the capacity of individual units to provide care in the future.

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1 Royal College of Paediatrics and Child Health and Royal College of Obstetricians and Gynaecologists. *Resuscitation of Babies at Birth*. London: BMJ Publishing Group, 1997:42.

2 Barrie J R, Greenhalgh D L. Training in neonatal resuscitation: the views of junior paediatricians. *J R Coll Physicians Lond* 1993;27:151-3.

A rare cause of respiratory distress

EDITOR.—We would like to describe a case of respiratory distress associated with haemodynamic compromise. This is a common clinical scenario, and the underlying cause is usually evident. Occasionally the cause is rare, and we report a case of respiratory distress caused by bilateral adrenal agenesis.

A baby girl was born at term to a diabetic mother. She remained well for the first six days, but on day seven she was transferred to our unit because of severe respiratory distress requiring intubation and ventilation. A chest radiograph showed a small pneumothorax and changes consistent with surfactant deficiency. Serum sodium concentration was 123 mmol/l, and serum potassium concentration was 7.0 mmol/l. A septic screen and cardiac echocardiography were normal. Because of persistent electrolyte abnormalities and refractory hypotension, a random cortisol measurement was taken and found to be 3 mmol/l (normal range 30-660 mmol/l). Adrenocorticotropic hormone concentration was significantly raised at 341 pmol/l (normal range 2-13 pmol/l). Following intravenous hydrocortisone, her clinical condition improved dramatically allowing withdrawal of inotropic support and extubation within 48 hours. An abdominal ultrasound scan suggested absence of the adrenal glands which was later confirmed by MRI scanning. She was started on regular exogenous steroid replacement, and, on follow up aged 6 months, is thriving and developing normally.

Congenital adrenal agenesis is rare with only two reported cases in the literature.^{1,2} Two transcription genes, DAX-1 and SF-1, are known to be involved in the development of the adrenal glands.³ Analysis of the SF-1 gene showed it to be normal in our patient, and a DAX-1 abnormality was excluded on clinical grounds.

Infants of diabetic mothers are known to be at increased risk of surfactant deficiency. Antenatal and postnatal surfactant production is inhibited by glucocorticoid deficiency.⁴ The combination of both fetal hyperinsulinaemia and glucocorticoid deficiency would considerably impair surfactant production and may explain the severity of our patient's respiratory distress.

Both respiratory distress and electrolyte imbalances are common in the neonatal unit. Therefore when the diagnosis is not obvious, the search for causative factors must be broadened to ensure that rare but clinically important diagnoses are not missed.

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Table 1 Results of the telephone survey of General Professional Trainees in England and Wales on neonatal resuscitation training provided to them in their neonatal units

Questions	Number (%) of respondents who received training				
	Total units (n=177)	Regional units (n=19)	DGH units (n=158)	Previously experienced (n=55)	Previously inexperienced (n=122)
Formal induction	167 (94)	18 (95)	149 (94)	50 (91)	117 (96)
Intubation skills	153 (86)	18 (95)	135 (85)	46 (84)	107 (88)
UVC insertion	77 (44)	16 (84)	61 (39)	26 (47)	51 (42)
Resuscitative	159 (90)	16 (84)	143 (91)	48 (87)	111 (91)
24 hr registrar cover	155 (88)	19 (100)	136 (86)	43 (78)	112 (92)
Accompanied until 1st successful intubation	54 (31)	6 (32)	45 (29)	21 (38)	33 (27)
Formal appraisal before left unaccompanied	44 (25)	9 (47)	35 (22)	16 (29)	28 (23)
Ongoing training	80 (45)	11 (58)	69 (44)	29 (53)	51 (42)
Courses attended/encouraged	126 (71)	10 (53)	116 (73)	46 (84)	80 (66)

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Disseminated candidiasis after steroid treatment for early neonatal hypotension

EDITOR.—We are concerned that intravenous steroids are increasingly being used for the treatment of early neonatal hypotension without proper evaluation. We recently treated three newborn premature infants who had received a six day reducing course of intravenous hydrocortisone (starting at 12.5 mg/kg/day in divided doses) from day 1 of age for the treatment of hypotension. They were otherwise stable, apart from one infant who had a patent ductus arteriosus. All developed severe systemic candidiasis and necrotising enterocolitis by 14 days of age, with only one infant surviving.

Reported adverse effects of steroid treatment for neonatal hypotension include fungal infection,¹ hyperglycaemia, septal and ventricular hypertrophy,² and hypertension.³ There are also well described short term complications of early steroid use for chronic lung disease including gastrointestinal haemorrhage and intestinal perforation.⁴ Long term adverse effects in these neonates include an increased risk of cerebral palsy and developmental delay.⁵ Any benefit of treating neonatal hypotension with intravenous steroids would have to be substantial in order to outweigh these risks.

It is not clear that steroids confer any advantage over appropriate inotropic treatment of neonatal hypotension. A number of small studies have reported increases in blood pressure when steroids are used. The largest study of 40 infants randomised to receive inotropes or hydrocortisone³ showed 81% success when hypotension was treated with hydrocortisone compared with 100% success with dopamine treatment (at rates of 5–20 µg/kg/min). Inotropes allow more accurate titration of drug dose to response and are known to be effective in maintaining blood pressure. If dopamine is insufficient, adrenaline (epinephrine) or noradrenaline (norepinephrine) should be added for further inotropic support.

Further studies are needed to understand the role of steroids in the newborn premature infant before their use for the treatment of hypotension becomes universal, and at present any advantages of steroid treatment over escalation of inotropic support are outweighed by adverse effects. This is of particular importance in the light of the increasing evidence⁵ that the benefits of steroid treatment of chronic lung disease in newborn premature infants may not outweigh the adverse effects. Consideration must be given to limiting the use of steroids for neonatal hypotension to situations where other proven methods of cardiovascular support have failed.

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- 4 Soll RF, for the Vermont Oxford Network Steroid Study Group. Early postnatal dexamethasone therapy for the prevention of chronic lung disease [abstract]. *Pediatr Res* 1999;42:123A.
- 5 Halliday HL, Ehrenkranz RA. Early postnatal (<96 hours) corticosteroids for preventing chronic lung disease in preterm infants. *Cochrane Database Syst Rev* 2000;2:CD001146.

Serum ferritin level in neonatal fulminant liver failure

EDITOR.—Elevated serum ferritin is a non-specific indicator of severe hepatocyte injury.^{1,2} Neonatal haemochromatosis (NH) is an uncommon disorder of neonatal iron storage, which often requires liver transplantation.^{1,3} Serum ferritin levels are considerably elevated in NH,^{1,4} but differentiating NH from other forms of neonatal fulminant hepatic failure (FHF), in which liver transplantation is contraindicated, can be very misleading.¹

We reviewed the case records of all neonatal FHF (onset of liver failure less than 28 days of age) seen in this unit from 1990 to 1999 to determine whether other causes of neonatal FHF can lead to an elevated serum ferritin level. Sixteen cases of neonatal FHF were seen, with a median onset of symptoms at 9 days (range 1–18). Eight had NH, confirmed either by histology showing parenchymal haemosiderin deposition in liver or buccal salivary tissue, and/or a positive family history.¹ The remaining eight cases had the following causes: mitochondrial disorders (two); galactosaemia, *Escherichia coli* sepsis, *Herpes simplex virus* type 1 and 2 hepatitis (one each); undetermined (two). All had raised serum ferritin levels (range 1000–217 000 µg/l). The median serum ferritin level for the patients with NH and the miscellaneous group were 15 000 µg/l (range 1650–217 000) and 2000 µg/l (range 1000–19 530) respectively. There was no significant difference between the mean serum ferritin level of the NH group and that of the miscellaneous group (40 916 *v* 5409 µg/l, *p* = 0.08).

This observation confirms that the serum ferritin level is raised as the result of severe hepatocellular injury in newborn infants, irrespective of the cause.¹ In neonatal liver failure, other diagnostic methods, such as demonstration of extrahepatic iron—for example, in lip salivary glands—or magnetic resonance imaging should be used to confirm or refute the diagnosis of NH.⁵

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CD ROM REVIEW

Practical Echocardiography for the Neonatologist. Nick Evans and Girvan Malcolm (CD ROM; A\$250.00). Australia: RPA.

The heart is a deceptively simple structure. Its main function is to power the circulation and it achieves this with only two pumps and only four valves. Most diagrammatic representations of the heart underestimate its true anatomical complexity so that the first contact of the novice with the complex three dimensional anatomy is intimidating. Even the great arteries, which are easy to depict diagrammatically, have a complex changing relationship with each other as they curve through the upper mediastinum. All these problems are compounded by our terminology because the “right” ventricle is in fact anterior to the “left” ventricle and the “left” atrium is posterior.

Neonatologists are interested in echocardiography because it allows assessment of the ductus in prematurity, it provides functional haemodynamic assessment, and it offers recognition or exclusion of structural heart disease although exclusion with confidence often requires expert assessment. The foundation of expertise in echocardiography is a clear understanding of the three dimensional anatomy. This is difficult to teach and time consuming to learn.

This CD provides the neonatologist with a superb introduction to three dimensional cardiovascular anatomy. It then links the anatomical arrangement to the standard echocardiographic views and illustrates the basics of Doppler and colour Doppler assessment of blood flow. The video images are clear and are provided side by side with anatomical diagrams illustrating the plane of the cross sectional image or the orientation of the Doppler probe. This CD will be of interest and of value to all those first learning echocardiography, including technicians and trainee paediatric cardiologists, and not just neonatologists. Obviously, recognition of normality requires familiarity with abnormality and this is due to be presented in a later CD. If that matches the quality of this first CD then between them they will provide a valuable introduction to echocardiographic assessment of the neonate.

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