Infant to staff ratios and risk of mortality in very low birth weight infants

We were very interested to read the article by Callaghan and colleagues. They report a decline in mortality with less nurses caring for high risk infants over the first three days of life. This is a surprising finding, which is counter-intuitive to established neonatal wisdom. Indeed the authors quote a smaller study by Hamilton and colleagues, which found an increase in mortality with a reduced ratio of nurses to infants. Callaghan and colleagues’ findings may be true, with the most likely explanation of the deaths being excessive handling. Clearly, if this finding is replicated, then establishing the optimum number of nurses could lead to improved outcomes for high risk infants. It certainly warrants further study within the NHS and the United Kingdom.

It is widely assumed that increased numbers of nurses in the UK will improve the outcome of very low birth weight infants. Currently, in the National Health Service there is difficulty in maintaining adequate numbers of neonatal nurses, with many units having nurse staffing levels substantially below those recommended by the British Association of Perinatal Medicine. Unfortunately, this recommendation for more staff is not based on a great deal of evidence, and the authors are to be praised for studying this topic.

Their results should, however be interpreted with caution. The health systems of the UK and Australia are different, most particularly in the proportion of centralised care and the ratio of nurses to infants. Callaghan and colleagues make the point that the UK has a ratio of two very low birth weight (VLBW) infants to one nurse, whereas in Australia the ratio is approximately one to one. The UK Neonatal Staffing Study has recently looked at 15,500 infants from 54 randomly selected units throughout the UK. This study did not show a clear relation between staff establishment and outcome, although it did show a linear relation between mortality and occupancy rates and a trend to increased risk of mortality with a lower nurse:infant ratio.

Callaghan et al discuss some of the weaknesses of their own study. There are also two factors that we wish to highlight. The first is that the authors have not looked in detail at the quality and abilities of the nursing staff. There is a wide variation in the abilities of staff, particularly when nursing agencies are used to provide nurses. As these staff may not work full-time or have much experience of the individual unit, they may be less efficient or able when compared to those full-time staff based on the unit.

The second factor is the method of determining staff workload. Measuring the ratio of babies to staff is not an accurate measure of nurse activity; a large number of well babies often need less care than a small number of sicker babies. It is not clear from the paper how the authors dealt with the term infants, and whether these are included in calculating the ratio. Did the authors use the number of nurses per VLBW infant or per all babies in the unit? In addition, large babies can also generate a substantial workload if they are very unwell (for example, babies with persistent pulmonary hypertension of the newborn or congenital diaphragmatic hernia). Further studies measuring the true overall workload may give a better indication of the relation with outcomes.

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The use of sodium resonium in pseudohypoaldosteronism

We describe the use of sodium resonium in a patient with pseudohypoaldosteronism (PHA). PHA is a rare autosomal recessive disorder characterised by raised plasma aldosterone, but mineralocorticoid resistance causing hyperkalaemia and hypotension. Severe recessive type 1 PHA is due to defective epithelial sodium channel (ENaC) activity, which warrants further study within the NHS and the United Kingdom.

Our patient presented aged 14 days with hyponatraemia (130 mmol/l) and hyperkalaemia (9.4 mmol/l). He made no response to hydrocortisone or fluid restriction. Plasma aldosterone level during crisis was extremely high (3820 pmol/l), confirming a diagnosis of PHA. Our patient’s sibling died neonatally with a presumptive diagnosis of PHA, suggesting autosomal recessive inheritance compatible with an ENaC defect.

Our patient was managed on intermittent rectal calcium resonium when hyperkalaemia, and daily solution G (a preparation containing high levels of sodium (1.3 mmol/ml)). The sodium requirement was 45 mmol/kg/day. Due to its unpalatability, solution G was given via gastrostomy.

After 18 months we changed his treatment to sodium resonium 0.25 g/kg twice daily by gastrostomy on advice from Professor Dillon (Great Ormond Street Hospital). Our patient...
improved and the electrolytes became tightly controlled (see fig 1). Our patient was so much better that after two years inpatient stay, we managed to discharge him home on treatment. The sodium requirement is 19 mmol/kg/day, and he has had no further electrolyte decompensation.

We have treated six patients with recessive PHA in the past 10 years, previously treating them with calcium resonium rectally at times of hyperkalaemic crisis. This treatment has not controlled electrolytes, and two of our patients died. Our experience with recessive PHA is that the defect does not improve with age; one of our patients died at the age of 21 years, and the survivors remain on vast daily intakes of sodium. Sodium resonium has improved our current patient’s quality of life and allowed his discharge. Although use of sodium resonium in PHA was first described in 1984, it is not widespread. This case should prompt greater use of sodium resonium in PHA.

We must acknowledge the huge number of nurses and doctors that contributed to a successful outcome in this case.

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Once upon a time ...

I very much enjoyed reading Storr and Rudolf’s review of literary perspectives on Oliver Twist.

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BOOK REVIEW

Child and adolescent psychiatry, 4th edition


It’s bigger, but is it better?

For me and my colleagues the answer is certainly, ‘Yes’.

Our Child and Adolescent Mental Health Service (CAMHS) now has all four editions of Child and Adolescent Psychiatry, “the child psychiatrist’s bible,” from the first edition, 1977 (Oxford University Press), to the second edition, 1985 (phone directory size); the paperback third edition, 1995 (Yellow Pages size, plus); and now this, the fourth edition, 2002, which is almost Data Sheet edition size. These awesome dimensions and the task of reviewing it (“you’ll have to read it, you fool” said a colleague) was somewhat off putting, but now that we’ve opened it and used it we do not want to be without it. This book is the bible of the child and adolescent psychiatry textbook and a must have for any district hospital or postgraduate library. The sort of emotional behavioural and developmental problems encountered by paediatricians in hospital and in the community, in the interface between paediatrics and psychiatry, are covered in sufficient detail to be of real use to the clinician, whether he is going it alone or has the luxury of cross referral.

The authors are experts in their fields, mainly eminent child and adolescent psychiatrists or psychologists from both sides of the Atlantic, but there are contributions from paediatricians in the chapter on soiling, for example.

Perhaps there is too much detail for the exam driven paediatrician in training, but then some only seem to grasp the value of developing some psychological frameworks later in their careers. Neither is this a quick fix alternative to the psychological component of the DCH and here again I suspect the size factor is likely to intimidate. Even SHOs in adult psychiatry who are now required by the Royal College to spend six months in child and adolescent psychiatry may be deterred and reach for a more MCQ orientated text in order to pass their exams.

For a hard pressed CAMHS team this book provides both the academic substrate and the clinical detail to be of real value in many everyday clinical scenarios. The reference lists are excellent and up to date and the index works, so that we have already checked out a number of clinical features and papers we have been meaning to chase but never got round to. The new and esoteric stuff seems to be there as well as the genuine advances we have heard about on CPD days, such as the guidelines for assessment and treatment of childhood depression, clinically useful rating scales, and developments in the management of ADHD and autistic spectrum disorders, using an evidence based approach wherever feasible. What I have read have found fascinating and so it seems that, in this electronic age, there is still a place for a comprehensive reference book on the department shelf.

Much of all this is new and just not in the 1995 edition, which is why the book is so big and why your library should buy it.

M A Griffiths