As an SHO, I bought the first edition of the Manual in 1982. It was a survival guide which provided safe certainties in the small hours of the night. It was small, light, and compact. There was no competition: the Roberton Manual was the book to have.

Nearly 20 years on, where has the 4th edition taken us? Bigger, certainly: a behemoth of a "small" manual with 550 pages. Not much taller or wider than its predecessors, but much thicker, the rather thin and closely typed pages distinctly reminiscent of a Bible. Thirty four chapters and eight appendices. There’s an awful lot of information in here.

Road testing a book like this is quite a challenge. Clearly one should not ask it to perform in a manner for which it was not designed, and the authors helpfully explain in the preface that their aim "is to provide a guide for the management of the acute medical and surgical problems a resident is likely to encounter on a modern neonatal intensive care unit." So I went for chapter 1, expecting it to plunge in where every resident is most nervous: resuscitation of the newborn.

Instead, I got "Organization of neonatal care". Admittedly it is only six pages, but does a resident really need this in a practical manual? Especially since the big Roberton textbook is likely to be on hand in most neonatal units to provide this and much more detail on this subject. In the Manual, you have to wait until chapter 6 to get "Resuscitation", with "Temperature control", "Fluid & electrolytes", "Enteral nutrition and parenteral nutrition", all packed with science and pathology for the first. How much physiology do you want or need in a practical manual? Not much, I think.

So I tried again with the oxygenation index (OI). There must be many units where the OI is used as a pragmatic threshold for giving nitric oxide or high frequency oscillation, and of course for referring for extracorporeal membrane oxygenation (ECMO). The resident will want to find the page with the formula for calculating OI, and how to deal with mm Hg versus kPa for the oxygen tension. To the index then—but no entry for oxygenation index. To the glossary of abbreviations at the front: there, sure enough, is OI. But where is it in the text? I could not find it under PPHN, or RDS, or ventilation. Eventually, by close reading, I found it mentioned under Meconium aspiration, and also under ECMO, but nowhere could I find the formula for calculating it. From this time, the luckless resident will have been called away to the next problem, and if the formula is indeed there, he/she will have lost interest in finding it.

Residents are increasingly likely to be faced with ventilators that read out the tidal volume, minute volume, and plateau-pressure-volume curves. They want to know how to use this information. They want to know what to do when babies on trigger ventilation drop their Pco2 to embarrassingly low levels. They want the formula for calculating the fractional excretion of sodium. They need to know that separate chest and abdomen radiographs give much better radiological information than "babygram" pictures. Sadly, they will be disappointed if they try to find such information in this book.

The 4th edition of the Manual seems to have lost the values of its roots. It feels like a pared down version of the big Roberton book, repackaged between smaller covers. It contains a level of detail that is unnecessary given the alternative sources of the material. It can be hard to find in a hurry the things you need, and some of the things you want are not there at all—or at any rate, I couldn’t find them. And if the formula is indeed there, the index is terrible. On the other hand, if you want a comprehensive introduction to the subject of neonatal intensive care medicine for under £20, look no further. This is your book.

For all professional staff there are 300 pages of clear descriptions containing information that will prove useful in organising investigations in the neonatal unit. There are also modern data which can be used to defend the embattled SHO against the pathologically cheerful authors at the end. For all professional staff there are 300 pages of clear descriptions containing information that will prove useful in organising investigations in the neonatal unit. There are also modern data which can be used to defend the embattled SHO against the pathologically cheerful authors at the end.

Nevertheless this is a volume that is informative and attractive, from the cartoon of a neonate’s head (front cover) to the photograph of the three distinguished and pathologically cheerful authors at the end. For all professional staff there are 300 pages of clear descriptions containing information that will prove useful in organising investigations in the neonatal unit. There are also modern data which can be used to defend the embattled SHO against the pathologically cheerful authors at the end. For all professional staff there are 300 pages of clear descriptions containing information that will prove useful in organising investigations in the neonatal unit. There are also modern data which can be used to defend the embattled SHO against the pathologically cheerful authors at the end.

PostScript
Thickening milk feeds may cause necrotising enterocolitis

Extremely low birthweight infants have the highest risk of developing necrotising enterocolitis (NEC). We report on two infants who developed fatal NEC while established on enteral feeds. A common antecedent was recent treatment with Carobol. An 820 g boy and a 752 g girl, both of 25 weeks gestation, were fully established on enteral feeds with expressed breast milk by day 12 and 18 respectively. Non-specific symptoms were attributed to gastro-oesophageal reflux (GOR), which was empirically managed by thickening milk feeds. Infant Carobol (Cov & Gate) was started on postnatal day 12 and 24. Onset of NEC was day 26 and 30, with death one day later.

Carobol is unlicensed in the United Kingdom. The manufacturer advises that two to three level spoons may be added per 60–90 ml milk, but mentions no precautions or contraindications for preterm infants. Its use in preterm infants may have crept in since the withdrawal of cisapride in July 2000. Although feed thickening may reduce the frequency and volume of regurgitation, acid reflux remains unaffected, and a paradoxical increase in the occurrence of GOR has been described. Moreover, milk thickened with carob bean gum is less nutritive because of reduced availability of essential elements. Two recent reviews found no evidence to support the practice of feed thickening in infants with GOR.

We are concerned that carob thickened milk may have played a role in the demise of these infants. The exact pathophysiology could not be further investigated because neither infant underwent postmortem examination. Thickened feeds may have led to NEC as a result of bowel obstruction with subsequent bacterial overgrowth or following direct mucosal injury from thickened milk. Bacterial overgrowth is plausible because feed thickeners have been shown to significantly increase microbial population and enzyme activities in the weaning rat cecum. Enterocolitis has previously been reported in an infant on enteral feed thickened with pectin and cellulase, as has neonatal intestinal obstruction and gastric lactobezoar.

Thickening feeds with carob bean gum is of unproven value in GOR. We feel that in preterm infants, NEC may not be fully explained from serious adverse effects and should not become widely adopted without a formal randomised trial.

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References

Linear IgA bullous dermatosis in a neonate

We encountered a neonatal case of linear IgA bullous dermatosis. Only one other case of the disease diagnosed in the neonatal period has been reported, so we felt it was important to describe this case.

Small vesicles first appeared on the face, hands, and legs of a Chinese full term baby boy on day 3 of life, which evolved into bullae on day 13. New bullae continued to erupt until day 18. By day 25, all the skin lesions had crusted, and skin healing was complete without scar formation. Besides skin erosion, the most overwhelming feature of the course was mucosal involvement. The infant presented with stridor on day 10 and went into respiratory failure requiring intubation. On day 30, bronchoscopy revealed a swollen larynx and a vesicle on the left aryepiglottic fold. He was extubated on day 38 in the neonatal intensive care unit. Cranial ultrasound and computed tomography scans showed no evidence of intracranial abscess or meningoencephalitis. At 1 year of age the infant is neurodevelopmentally normal.

Neonatal infection with Citrobacter species is usually acquired in a nosocomial fashion, and causes septicaemia, meningitis, and brain abscesses associated with a high morbidity and mortality. Eleven cases of vertically acquired Citrobacter koseri infection have been reported. However, the only previous report of vertical transmission of Citrobacter freundii describes a 32 week infant in whom the organism was identified from maternal high vaginal swab and infant gastric aspirate, but not from blood cultures. Neontal septicaemia with meningitis, as in our patient, has not been reported. Citrobacter freundii differs from other organisms causing neonatal meningitis by being able to...
Recruitment failure in early neonatal research

Rates of neurodevelopmental handicap are high among extremely low birthweight survivors, and the first 48 postnatal hours probably give the greatest opportunity for preventing damage. However, at this time, families are in turmoil and may have difficulty in coming to terms with a small baby starting the study was often missed because of changes in the baby’s clinical condition.

What went wrong? Since the Griffiths report, the emphasis has been on obtaining fully informed parental consent, and the research team has to ensure that the parents thoroughly understand the research and its implications. Research where parents signed consent forms, but later claimed that they did not understand the research, was heavily criticised. Consequently researchers are reluctant to approach parents who are in any way distressed, because of the difficulty in ensuring valid consent. If it is important for early neonatal research to continue, we urgently need agreement on a sensitive, humanistic yet realistic framework that is acceptable to both parents and clinical researchers alike.

Gestational age in the literature

In neonatology, the correct gestational age (GA) is extremely important, as the viability and survival of the premature baby depend on it. A difference of a few hours or a day can have a substantial impact on the survival and long term morbidity of premature babies.

Doctors are trained to report the GA of a premature baby in exact days—for example, 26+4 (GA = 26 completed weeks and 4 days). Reporting the GA in this format helps in understanding and assessing the postnatal and maturational age of premature babies. One would therefore expect GA to be reported exactly in the literature, especially in articles, studies, and trials dealing with survival and morbidity in premature babies. In fact, descriptions of GA are extremely ambiguous in most articles. An example of this ambiguity is survival at 26 weeks GA is
interpretation. It could mean 25\% + (that is, 88\%) were treated with antibiotics. Unfortunately, the study in question not only problem is to decide on the treatment. This ambiguous description of GA. The EPICure study is a good example of a language that uses the ambiguous description of GA. Such large studies have a major impact on doctors and parents, as the results and interpretation are used by neonatologist for counselling, teaching, and research. For those dealing with ethical issues, especially resuscitation in extremely premature babies, exact GA can be of immense help. As the limits of viability and survival are stretched, doctors need to be very clear in their minds about the exact age of the premature baby.

In view of the above, we propose that the reporting of GA in the literature should be uniform. It should be described in exact days—that is, weeks, and not approximate as is the current usage. The EPICure study obtained a result of 26\% change by 12\% on either side of 26\%. This would have a large effect not only on survival but also on long-term morbidity.

Many large, randomised controlled trials and articles published on survival, viability, and ethical issues of resuscitation in extremely premature babies have used this ambiguous description of GA. The EPICure study is a good example of a language that uses the ambiguous description of GA. Such large studies have a major impact on doctors and parents, as the results and interpretation are used by neonatologist for counselling, teaching, and research. For those dealing with ethical issues, especially resuscitation in extremely premature babies, exact GA can be of immense help. As the limits of viability and survival are stretched, doctors need to be very clear in their minds about the exact age of the premature baby.

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