A manual of neonatal intensive care, 4th edition


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 Robertson Manual was the book to have!

Nearly 20 years on, where has the 4th edition taken us? Bigger, certainly: a beehive of a “small” manual with 550 pages. Not much taller or wider than its predecessors, but much thicker, the rather thin and closely typeset pages distantly 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 Robertson 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 coming first. How much physiology do you want or need in a practical manual? Not this 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 it, 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 and minute volume, and play 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 Robertson 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 when you find them, they are often not very clear.

Thirdly the section on viral disease and transmission should be more detailed. “Low risk” is not quantitated, and CMV is described variously as “largely inactivated by freezing” and (one page later) “does not survive freezing”—an inconsistency that leaves the reader feeling insecure about such an important safety issue.

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 emboldened consultant ward round. Every neonatal unit should purchase a copy. I predict that these valuable pages will be well thumbed within a month. I look forward to a further edition, and hope that it will extend its scope to include other laboratory disciplines such as genetics and electrophysiology. The three authors deserve success with this winner.

Neonatology & laboratory medicine


Neonatology & laboratory medicine is a novel concept and a valuable addition to our literature. The book brings together a clinical biochemist, a neonatologist, and a medical microbiologist as authors in a successful attempt to describe appropriate laboratory investigation and clinical management of the neonate. This paperback aims to provide junior doctors, laboratory scientists, and neonatal nurses with background information that will help solve common neonatal problems. The chapters deal systematically with common biochemical and infective problems that may befall neonates. There are also sections on breast feeding, parenteral nutrition, and therapeutics. Best of all 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 when you find them, they are often not very clear.

Three small criticisms and suggestions for the next edition.

- The chapter entitled “Drugs and the neonate” is too short. The figure referring to biochemical and haematological monitoring cites only 11 drugs, ignoring commonly used drugs such as vucuroin, insulin, surfactant, salbutalol, 5-flucytosine, and steroids. Even those lucky 11 have curious omissions—for example, the oliguria and fluid retention associated with indomethacin.
- Secondly the book recurrently ignores the unusual demands of the extreme preterm infant—for example, dilutional exchange for polycythemia is said to be carried out in 10 ml aliquots, and does not recommend smaller volumes of 500 g whose total blood volume may be little more than 40 ml.
- Thirdly the section on viral disease and transmission should be more detailed. “Low risk” is not quantitated, and CMV is described variously as “largely inactivated by freezing” and (one page later) “does not survive freezing”—an inconsistency that leaves the reader feeling insecure about such an important safety issue.

Fetal and neonatal brain injury: mechanisms, management and the risks of practice, 3rd edition


Brain injury remains a common theme in a large proportion of survivors of extreme prematurity and/or neonatal encephalopathy. The headline rates of significant disability have been largely unchanged despite the enormous advances in neonatal intensive care of the post-surfactant era, and more subtle educational difficulties are later declared in many others. It is essential that clinicians continue to strive for a deeper understanding of the mechanisms of brain injury to not only guide conventional management, but also look ahead to the future strategies in which neuroscience advances may translate into plausible clinical strategies—for example, promoting the regrowth of damaged axons from intact neurones across an area of periventricular leucomalacia.

The strength of a textbook such as this is to give an in depth overview of many aspects of brain injury. This is accomplished well by a distinguished list of mostly United States based contributors, who consider the many aspects of neonatal brain injury in terms of aetiology, epidemiology, diagnosis, management, and...
long term outcome. A section on medico-legal issues makes interesting reading, although it is not directly applicable to the British judicial system. Surprisingly little mention is made of the controversies surrounding the use of postnatal corticosteroids to treat chronic lung disease and the risk of cerebral palsy, but otherwise the range of topics is exhaustive. Particular care is also taken to relate the bedside management to the background neuroscience—for example, the neuroprotective effect of brain cooling. Readers will be encouraged to catch up with subsequent developments as they emerge in the journal.

Weaknesses are few. The section on imaging of brain injury is thorough, and as expected well illustrated. However, it leaves the reader wishing for more information on the prognostic value of MRI in particular.

Other sections would have been enhanced by greater use of illustrations—for example, I was disappointed that a section on congenital malformations fails to include a single illustrative image.

In summary, this is a comprehensive account of an area of vital importance to obstetricians, neonatologists, and paediatric neurologists. It should prove to be a useful reference for specialists in these fields.

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LETTERS

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 Carobel.

An 820 g boy and a 752 g girl, both of 23 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 Carobel (Cow & Gate) was started on postnatal day 12 and 24. Onset of NEC was day 26 and 30, with death one day later.

Carobel is unlicensed in the United Kingdom. The manufacturer advises that two to three level scoops may be added per 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 doxical increase in the occurrence of GOR has been described. Moreover, milk thickened with carob bean gum is less nutritive because of the carbohydrate, availability of calcium, iron, and zinc in vitro. J Pediatr Gastroenterol Nutr 2000;30:373-8.


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 on day 3 of life, which evolved into bullae on day 13. New bullae continued to erupt on day 13. New bullae continued to erupt on day 13. New bullae continued to erupt on day 13. New bullae continued to erupt on day 13. New bullae continued to erupt on day 13. New bullae continued to erupt on day 13.

Linear IgA bullous dermatosis commonly affects oral cavity, eyes, and external genitalia. It will be 57%, and 72% respectively. However, the mucosal involvement is not life threatening.

The other neonatal case of linear IgA bullous disease reported in the literature also showed serious mucosal involvement. It manifested as respiratory failure requiring treatment by extracorporeal membrane oxygenation.

References

Vertical transmission of Citrobacter freundii

An infant developed early respiratory distress after delivery at 34 weeks gestation after prolonged rupture of membranes. Citrobacter freundii was cultured from a maternal mid-stream urine sample at delivery. C freundii is resistant to ampicillin but sensitive to gentamicin, cephalosporins, and ciprofloxacin, which was isolated from neonatal blood cultures taken on admission. Gram negative rods were seen on microscopy of cerebral spinal fluid (CSF), with no white cells and 730 red cells per high power field. CSF protein was 1.26 g/l and glucose 3.0 mmol/l, with blood glucose of 4.9 mmol/l. No organisms grew on CSF culture.

Linear IgA bullous dermatosis reported in the literature contrasted sharply with the classical presentation of the childhood disease in having serious mucosal involvement and a non-relapsing course.

We hope that our report serves as a reference for neonatologists and dermatologists who may encounter similar cases in the future.

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replicate within brain capillary epithelium, per-
haps accounting for the propensity of this organ-
ism for causing cerebral abscesses.1 However, includ-
ing this case, this complica-
tion appears to be confined to late onset disease, with possible explanations being the early use of antibiotics, and absence of a putative virulence factor.1

The combination of cefotaxime and an aminoglycoside is recommended for neonatal Gram negative meningitis, but CSF concentra-
tions of gentamicin may only be mar-
ginally above the minimum bactericidal concentration of Gram negative organisms.1 Ciprofloxacin has been shown to be effective in Gram negative meningitis, and should be con-
sidered in the treatment of this condition.3

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3 Badger JL, Stirm MF, Kim KS. Citrobacter freundii invades and replicates in human brain micro-

Recruitment failure in early neonatal research
Rates of neurodevelopmental handicap are high among extremely low birthweight sur-
vivors, 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 dif-
ficulty in coming to terms with a small baby in intensive care. We recently had to abandon an observational, non-invasive study because of practical difficulties arising from the new Research Governance Framework,4 and we would like to share this experience, and its implications, with the research community.

We needed parental consent for the study, which had local research ethics committee approval. Babies had to be < 1500 g birth weight, > 25 weeks gestation, < 48 hours old, ventilated, with an arterial line, and no prior intervention for circulatory compro-
mise. The last two requirements meant that, in reality, babies had to be recruited within the first 12 hours. A non-invasive measure-
ment of peripheral oxygen consumption \(\text{PO}_{2}\) was to be made regularly over 24 hours. We aimed to recruit 50 babies over two years.

When an eligible baby was admitted, the parent(s) were given further information before consent was sought a minimum of four hours later. Postnatal recruitment proved difficult.

The need to give parents time to consider their decision meant that the opportunity for starting the study was often missed because of changes in the baby’s clinical condition.

With additional local research ethics com-
mittee permission, we tried to recruit women at high risk of delivering before term from 25 weeks gestation. The consent process was more complex in this group, as the explanation had to include information about standard neonatal care and procedures. Parents in this group were given 24 hours to come to a decision. Figure 1 shows that, of 28 eligible babies, only five were recruited. Eight out of nine mothers approached antenatally gave consent, but only two of their babies were studied, as three did not meet the entry criteria and the other three were born elsewhere.

What went wrong? Since the Griffiths report,5 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 criti-
cised.1 Consequently researchers are reluctant to approach parents who are in any way distressed, because of the difficulty in ensur-
ing valid consent. If it is important for early neonatal research to continue, we urgently need agreement on a sensitive, humane, and realistic framework that is acceptable to both parents and clinical researchers alike.

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Figure 1 Recruitment to research project on neonatal unit (NNU) over 12 month period.

References

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
This description of GA is open to interpretation. It could mean 25° 1 to 26° 0 or 26° 1 to 26° 2. Every extra day improves the survival of the premature baby by 2%. Therefore, for the above GA, survival could change by 12% on either side of 26°. This could have a large effect not only on survival but also on long term morbidity.

Many large studies 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 large, unplanned study that uses the ambiguous description GA.1 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.2 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.