A manual of neonatal intensive care, 4th edition

J M Rennie, N R C Roberton. London: Edward Arnold, 2001, £19.99, pp. 566. ISBN 0304720107. 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 typeset 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 philosophy, reading 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 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: no OI. Or ECMO, as I am sure many other SHOs would have called it.

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 embalmed SHO against the claims of the 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 authors deserve success with this winner.

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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 neuronal tissue 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...
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 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. Instant 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 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 nutritious because of the reduced availability of essential elements.1 Two recent reviews found no evidence to support the practice of feed thickening in infants with GOR.2,3

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 feeds thickened with dense milk. Bacterial overgrowth is plausible because feed thickeners have been shown to significantly increase microbial population and enzyme activities in the weaning rat cecum.4 Enterocolitis has previously been reported in an infant secondary to feeds thickened with pectin and cellulose,5 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 this practice may not be free 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 that 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 eruption, 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 ari-epiglottic fold. He was extubated on day 58 in the middle of a three week course of prednisolone. After extubation, stridor gradually subsided in a couple of weeks.

The diagnosis of linear IgA bullous dermatosis was made by skin biopsy on a bulla. Histological sections showed splitting of the skin at the dermo-epidermal junction with predominant polymorph infiltrate. Immunofluorescence showed a linear deposit of IgA at the dermo-epidermal junction. Staining for IgG and C3 was also positive.

Linear IgA bullous dermatosis commonly occurs in childhood with onset from 6 months to 10 years.5 It classically runs a relapsing course with complete remission attained after puberty. The overall incidence of involvement of mucous membranes of the oral cavity, eyes, and external genitalia is 57%, 40%, and 72% respectively.6 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, oesophageal dysmotility with choking during feeding, and blindness as a result of conjunctival scarring.7 In both these neonatal cases, complete remission was attained after the unsettled neonatal period. Hence, linear IgA bullous disease with onset in the neonatal period contrasts 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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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, resistant to ampicillin but sensitive to gentamicin, cephalosporins, and ciprofloxacin, was isolated from neonatal blood cultures taken on admission. Gram negative rods were seen on microscopy of cerebrospinal 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. Ampicillin and gentamicin were discontinued, and ciprofloxacin and cefotaxime started for a three week course. Serial cranial ultrasound and computed tomography scans showed no evidence of intracranial abscess or ventriculitis. 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.8 However, the only previous report of vertical transmission of C freundii describes a 32 day infant in whom the organism was identified from maternal high vaginal swab and infant gastric aspirate, but not from blood cultures.9 Neonatal septicaemia with meningitis, as in our patient, has not been previously reported. C 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 in intensive care. We recently had to abandon recruitment to research project on neonatal unit (NNU) over 12 month period. Figure 1 Recruitment to research project on neonatal unit (NNU) over 12 month period.

With additional local research ethics committee 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, 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, humane, and realistic framework that is acceptable to both parents and clinical researchers alike.

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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 inexact days—for example, 26+3 (GA = 26 completed weeks and 3 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\(^{\text{th}}\) to 26\(^{\text{th}}\) or 26\(^{\text{th}}\) to 26\(^{\text{th}}\). 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, prospective study that uses the ambiguous description of GA.\(^3\) 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.\(^4\) 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.\(^5\) The significance of such a change by 12% on either side of 26% is that GA can be of immense help.\(^6\) 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.

Fever in the neonatal period

This is in reference to the recent article by Maayan-Metzger et al.\(^1\) The clinical implication of the study is questionable. It is difficult to make a prospective decision on retrospective data. What should a clinician do if a baby of 26 weeks and 3 days old baby has a fever of 37.9°C? There is no problem in labelling the infant as having non-specific fever of 37.9°C? There is no problem in a healthy asymptomatic 3 day old baby has a fever of 37.9°C? There is no problem in making a prospective decision on retrospective data. What should a clinician do if a healthy asymptomatic baby has a fever of 37.9°C? There is no problem in making a decision on the presence of fever in neonates.

Having said all this, I appreciate the methodology of the study and the authors' endeavour to look further into the issue of fever in neonates. I hope my suggestion can generate intense discussion and not just be taken as a critical review of the paper. Lastly, in my view after reviewing the above paper in detail, dehydration still remains a diagnosis of exclusion, just as we take transient tachypnoea of the newborn as a diagnosis of exclusion in cases of respiratory distress in neonates.

References


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Reference


Home phototherapy in the United Kingdom

Although successful home treatment of neonatal jaundice using fibre-optic phototherapy units has been reported elsewhere,\(^2\) we are not aware of any such provision in the United Kingdom. We have introduced a regional home phototherapy programme in Tayside, Scotland and wonder if our initial experience can be wise and safe? These are the questions we should be struggling to answer.


