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 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 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 will have been called away to the next consultant ward round. Every neonatal unit should purchase a copy. I predict that it will extend its scope to include other laboratory disciplines such as genetics and electrophysiology. The three authors deserve success with this winner.

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 vasoconstrictor, insulin, surfactant, salbutamol, 5-fluorocytosine, and steroids. Even those lucky 11 have curious omissions—for example, the oliguria and fluid retention associated with indomethacin.

• 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 cortical neurones across an area of periventricular leucomalacia.

The strength of a textbook such as this is to present the oliguria and fluid retention associated with indomethacin infant—for example, dilutional exchange for polycythaemia 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.

Nevertheless this is a volume that is informative and attractive, from the cartoon of a neonate’s head (front cover) to the photographs 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 emboldened SHO against the embattled consultant 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 three 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 axons from intact cortical 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
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. 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 levels of 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 marked 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 dense milk. Bacterial overgrowth is plausible because feed thickeners have been shown to significantly increase microbial population and enzyme activities in the weanling rat caecum. Enterocolitis has been previously reported in an infant secondary to feeds thickened with pectin and cellulose, 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 the practice may not be free from serious adverse effects and should not become widely adopted without a formal randomised trial.

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 ary-epiglottic fold. He was extubated on day 38 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 derma- tosis 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 disease commonly occurs in childhood with onset from 6 months to 10 years. 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. 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.

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 midstream 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 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 septicemia, 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 C 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. Neonatal septicemia with meningitis, as in our patient, has not previously been 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 difficulty 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, 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 compromise. The last two requirements meant that, in reality, babies had to be recruited within the first 12 hours. A non-invasive measurement of peripheral oxygen consumption, the first 12 hours. A non-invasive measure in reality, babies had to be recruited within the first 48 postnatal hours and no weight, newborns had to be ventilated, with an arterial line, and no clinical condition. Starting the study was often missed because their decision meant that the opportunity for the treatment of this condition.

The combination of cefotaxime and an aminoglycoside is recommended for neonatal Gram negative meningitis, but CSF concentrations of gentamicin may only be marginally above the minimum bactericidal concentration of Gram negative organisms. Ciprofloxacin has been shown to be effective in Gram negative meningitis, and should be considered in the treatment of this condition.

References


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26. This description of GA is open to interpretation. It could mean 25\textsuperscript{1} to 26\textsuperscript{0} or 26\textsuperscript{1} to 26\textsuperscript{2}.\textsuperscript{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 landmark full study that uses the ambiguous description of GA.\textsuperscript{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.\textsuperscript{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 extra days.

References


Fever in the neonatal period

This is in reference to the recent article by Maayan-Metzger et al.\textsuperscript{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 3 day old baby has a fever of 37.9\textdegree C? There is no problem in labelling the infant as having non-specific fever, which may be due to dehydration. The problem is to decide on the treatment. Unfortunately, the study in question not only lacks that information but also supports treatment with antibiotics. This inference is drawn from the results of the study, stating that 108 of 122 healthy asymptomatic babies (that is, fever < 37.9\textdegree C) were not treated with antibiotics. In five years (January 1997 to December 2001), 122 cases were identified with fever giving a rough figure of 25 febrile cases in one year—that is, about two cases a month. A prospective follow up of these febrile neonates after separating them into two groups, a) receiving antibiotics and b) not receiving antibiotics, would be more informative in clinical decision making. Merely adding the risk factors in the list of possible causes for fever in neonates without solution or how one should deal with it is of very little clinical worth. It would be very brave of a paediatrician not to treat neonatal fever with antibiotics on the basis of the inference drawn from this study, but would it be wise and safe? These are the questions we should be struggling to answer.

1 have reservations about the authors’ “standard work up protocol”. A cerebrospinal fluid analysis on asymptomatic, otherwise healthy neonates with fever is probably unwarranted. I think it is unwise to perform a spinal tap on a baby with suspicion of dehydration fever. In other words, if one suspects meningitis in a neonate, it is not fair to withhold antibiotics. About the treatment protocol, the authors treated 107 infants with antibiotics unnecessarily; only one had a positive culture. This approach of empiric antibiotic use needs critical appraisal in the protocol of the institution.

Fever without symptoms is not uncommon in healthy, full term babies in the postnatal ward. To carry out a prospective study on these babies would be feasible. There are two issues that need clarification, how to investigate and how to treat. I do not think that there is much controversy about investigating a febrile neonate. Will the present knowledge, any febrile neonate with a fever, irrespective of symptoms, should be investigated appropriately with full blood count and blood and urine cultures. It is the treatment that is the root of the controversy and needs further evaluation. However, in view of the present study, in spite of a promising conclusion, fever in healthy neonates should not be treated as something benign and dealt with casually.

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 will 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.

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Home phototherapy in the United Kingdom

Although successful home treatment of neonatal jaundice using fibre-optic phototherapy units has been reported elsewhere,\textsuperscript{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 would be of interest to others.

Before introducing the service, hospital and community midwives undertook training covering inclusion criteria (physiological jaundice in well, term infants), the treatment protocol, equipment, and advice on discharge. We have not had any equipment failure. All parents and staff have been satisfied with the service and would like to continue the programme.

We believe this is the first report of a home phototherapy programme in the United Kingdom. With appropriate training and enthusiastic community support, it appears to be feasible, safe, and well accepted by families and staff. We would encourage others to consider establishing such programmes.

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