# PostScript

## BOOK REVIEWS

### A manual of neonatal intensive care, 4th edition

J M Rennie, N R C Roberton. London: Edward Arnold, 2001, £19.99, pp 565. ISBN 0340720107

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 physiology, 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 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. By 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 display 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 $P_{CO_2}$ 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, which comes to the same thing. And 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.

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### Neonatology & laboratory medicine

A Green, I Morgan, K Gray. Guildford: ACB Venture Publications, 2003, £30.00, pp 312. ISBN 0902429418

*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 finishes with appendices including normal reference ranges and a useful glossary.

The expenditure of £30 rewards the reader with more than 300 pages which are clear and well arranged. Tables and flow diagrams are easy to dip into. More senior readers may be frustrated that the book is not referenced, but recommended reading is provided at the end of each chapter.

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 vecuronium, insulin, surfactant, salbutamol, 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 polycythaemia is said to be carried out in 10 ml aliquots, and does not recommend smaller volumes for the infant 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 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 predatory instincts 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

Edited by D K Stevenson, W E Benitz, P Sunshine. Cambridge: Cambridge University Press, £140.00, pp 926. ISBN ISBN 0521806917

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 neuroscientific 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

long term outcome. A section on medico-legal issues makes interesting reading, although 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 journals.

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 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 nutritive because of decreased bioavailability 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 by calorific 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.[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 the practice may not be free from serious adverse effects and should not become widely adopted without a formal randomised trial.

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## 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 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 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 disease commonly occurs in childhood with onset from 6 months to 10 years.[1] 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.[1] However, the mucosal involvement is not life threatening.

The other neonatal case of linear IgA bulbous 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.[2] 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 midstream 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.[1] 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.[2] Neonatal septicaemia with meningitis, as in our patient, has not been previously described.

*C freundii* differs from other organisms causing neonatal meningitis by being able to

replicate within brain capillary epithelium, perhaps accounting for the propensity of this organism for causing cerebral abscesses.[3] However, including this case, this complication 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 concentrations of gentamicin may only be marginally above the minimum bactericidal concentration of Gram negative organisms.[4] Ciprofloxacin has been shown to be effective in Gram negative meningitis, and should be considered in the treatment of this condition.[5]

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## 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 an observational, non-invasive study because of practical difficulties arising from the new Research Governance Framework,[1] 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 $\leqslant$ 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[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.

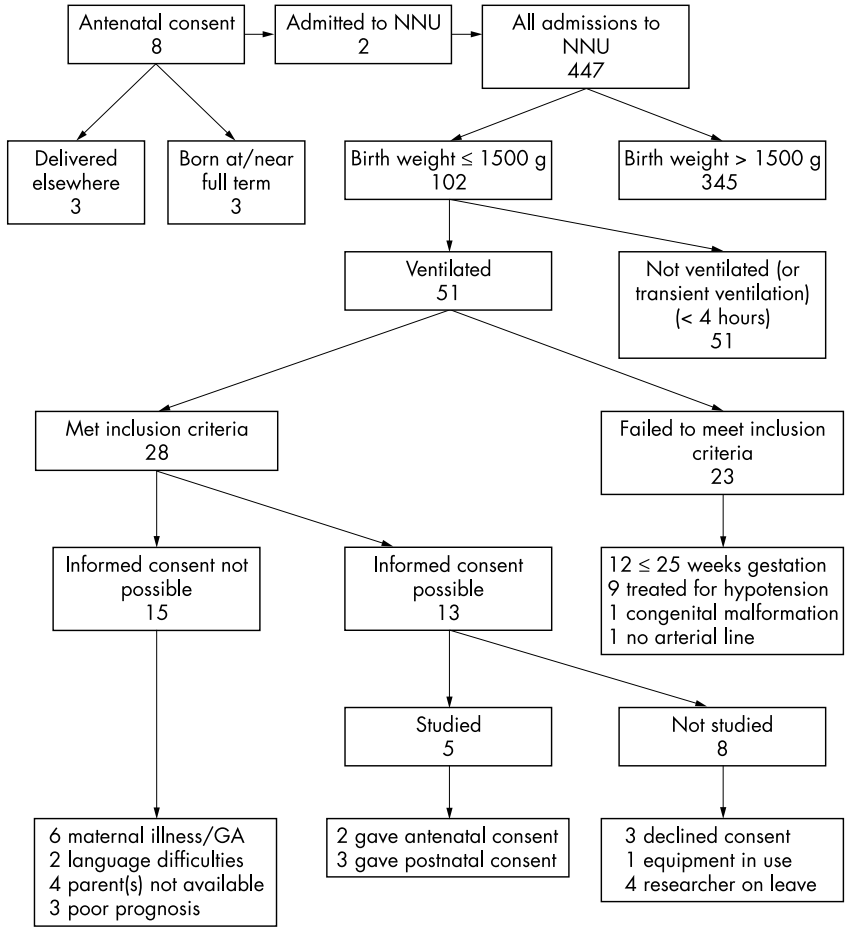


**Figure 1** Recruitment to research project on neonatal unit (NNU) over 12 month period.

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,[3] 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.[3] 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 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

26%.[1] This description of GA is open to interpretation. It could mean $25^{+1}$ to $26^{+1}$ or $26^{+1}$ to $26^{+6}$. 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, important 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 3] 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$^{+\text{extra days}}$.

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## 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 healthy asymptomatic 3 day old baby has a fever of 37.9°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, 88%) were 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, one receiving antibiotics (treatment group) and the other not (observation group) carried out in an ethical way, would be more informative for 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.

I 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. With our present knowledge, any febrile neonate with 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,[1 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 use (Biliblanket, Ohmeda), and the assessment of parental competence. The protocol conditions were: a daily capillary serum bilirubin (SBR), discussing all results with a paediatrician; basing treatment on SBR and age of the infant[3] and an SBR measured after discontinuing phototherapy. Parents underwent a one hour "training" session (equipment use and advice on feeding, skin care, and temperature control) and were given written advice. Tayside Committee on Medical Research Ethics advised that ethical approval for the programme and written consent were not required, as the treatment being offered was not novel.

Between February and August 2002, 28 families were offered home phototherapy in Tayside: six refused (difficulties with feeding, distance from home to hospital, and parental choice). The mean birth weight was 3245 g (range 2240–4220), with a median gestation of 38 weeks (range 35–41). Mean maternal age was 30 years (range 17–41). Twenty (91%) infants were breast fed. Ten were first born. Seven families lived in affluent areas and two in areas of high deprivation.[4] Phototherapy started at a median age of 5.5 days (range 1–13). Eight infants received all their phototherapy at home. Mean treatment duration was 47.3 hours (range 17.5–97.0) with a median decrease in SBR of 16.6 μmol/l per day (ranging from a fall of 50 μmol/l to a rise of 53 μmol/l in one case). Community midwives spent about 60 minutes on the first home visit. Subsequent visits were shorter. Poor compliance, without compromise to either infant, was identified in two families and rectified quickly. No other adverse incidents were reported, and there was no equipment failure. All parents preferred home phototherapy to inpatient treatment. Community midwives have been happy 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.

We are grateful to the rest of the Tayside Home Phototherapy Project Team (J Dalzell, A Jarvis, M Meldrum, V Samson) and the community midwives who contributed to the success of the project. This project was supported by a grant from the Scottish Executive Health Department – Innovative Fund for Children's Services.

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