PostScript

LETTERS

Clinical assessment of neonatal hyperbilirubinaemia

The study by Keren and colleagues[1] is a retrospective study, using infants in whom pre- and post-discharge TSB has been carried out, hence causing an inherent bias towards the same group.

The data for clinical risk have been collected from documents such as admission, intrapartum, and discharge forms. This retrospective collection can result in missing or ambiguous data, as has been accepted by the authors. Ideally, a study should be prospective using both methods on all neonates in a study group, and then the sensitivity and specificity (that is, false positives and false negatives) should be compared using actual data on follow up.

The clinical risk factor score includes factors that are interrelated such as vacuum and cephalhematoma. In cases where the cephalhematoma is caused by the use of vacuum the neonate gets a double rating. Obviously, authors have not found clinical risk factors more specific than pre-discharge TSB.

Contrary to this study, the AAP guidelines promote and support breast feeding and state that effective breast feeding can reduce substantially the risk for hyperbilirubinaemia.[2] It is known that inadequate feeds increase the level of neonatal jaundice; hence the emphasis on "effective" breast feeds. The study subjects date from 1993–97 and the feeding habits (that is, breast/bottle/combination feeds) have been given a considerable amount of significance, which contradicts the AAP guidelines by the subcommittee on hyperbilirubinaemia.[2]

Newman *et al* state that, compared to early TSB levels (<48 hours of life), clinical risk factors combined with TSB significantly improve prediction of subsequent hyperbilirubinaemia.[3]

Suresh *et al* have studied the cost effectiveness of strategies to prevent kernicterus, and concluded that to prevent one case of kernicterus, the cost was $10 321 463 for universal follow up of early newborn discharge, $5 743 905 for routine predischarge TSB, and $9 191 352 routine predischarge transcutaneous bilirubin with selective follow up.[4] They concluded that widespread implementation of these strategies would result in significantly increased healthcare costs with uncertain benefits.[4]

The AAP guidelines also focus on the rarity of kernicterus and aim to reduce the incidence of kernicterus, while minimising the risks of unintended harm such as maternal anxiety, decreased breast feeding, and unnecessary costs or treatments.[2]

They recommend a systematic clinical assessment before discharge and an early and focused follow up based on the risk assessment.[3] Finally we must remember that we are all clinicians and we should use the lab report as an adjunct to our clinical knowledge.

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References

1 **Keren R**, Bhutani VK, Luan X, *et al*. Identifying newborns at risk of significant hyperbilirubinaemia: a comparison of two recommended approaches. *Arch Dis Child* 2005;**90**:415–21.
2 **AAP**. Management of hyperbilirubinaemia in the newborn infant 35 or more weeks of gestation. Subcommittee on hyperbilirubinaemia. *Paediatrics* 2004;**114**:297–316.
3 **Newman TB**, Liljestraud P, Escobar GJ. Combining clinical risk factors with serum bilirubin levels to predict hyperbilirubinaemia in newborns. *Arch Pediatr Adolesc Med* 2005;**159**:113–19.
4 **Suresh GK**, Robin E Clark. Cost effectiveness of strategies that are intended to prevent kernicterus in new born infants. *Pediatrics* 2004;**114**:917–24.

Author's reply

Drs Kanjilal and Prasad make some important observations but are mistaken in several of their assertions. First, they suggest that because we limited our study sample to infants for whom pre- and post-discharge TSBs were performed, our results are affected by some form of selection bias. The bias they are referring to is verification bias, in which only patients with "positive" or more concerning test results have a follow up test to verify the original results. By decreasing the number of patients with "negative" test results, this bias has the effect of overestimating test sensitivity and underestimating specificity. However, as we point out in our manuscript, we studied infants enrolled in an early discharge follow up programme and minimised the potential for verification bias by restricting our sampling frame to months during which >75% of enrolled infants had post-discharge TSB measurements performed. In fact, for the majority of these months, >90% of enrolled infants had post-discharge TSBs measured.

The second point on which Drs Kanjilal and Prasad are mistaken concerns the inclusion of "interrelated" factors "like vacuum and cephalhaematoma" in our clinical risk factor scoring system. As summarised in table 2, vacuum extraction is included in the scoring system but cephalhaematoma is not. In fact, contrary to our expectation, cephalhaematoma was not associated with development of post-discharge TSB >95th centile. This simply may be a result of poor documentation of cephalhaematoma in the admission and discharge physical examinations (misclassification bias), but it raises concerns about the use of subjective factors in clinical risk factor scoring systems. Our results suggest that using more objective findings, such as use of vacuum extraction during delivery—a common cause of cephalhaematoma—may provide more accurate information about subsequent risk of hyperbilirubinaemia.

Finally, our finding that breast feeding increases the risk of hyperbilirubinaemia is not new and should not be interpreted as a recommendation against breast feeding. As paediatricians who routinely care for newborn infants, we recognise the benefits of breast feeding and strongly support its use. However, at the same time we are cognisant of the potential risks of dehydration and hyperbilirubinaemia posed by inadequate intake in breast fed infants. The results of our study should be interpreted as another reminder that healthcare systems and providers must work to ensure adequate lactation support for breast feeding mothers and early identification and treatment of breast feeding problems that may result in inadequate intake for infants.

As Drs Kanjilal and Prasad suggest, a prospective validation of alternative risk assessment strategies is needed to confirm the results of our study as well as other studies of alternative screening strategies. Additional studies are also needed to evaluate the incremental benefit of using clinical risk factors in addition to the pre-discharge TSB to predict which infants are at risk of developing severe hyperbilirubinaemia. And finally, more studies are needed to evaluate the cost effectiveness of alternative strategies for screening and tracking infants for their risk of developing severe hyperbilirubinaemia in order to prevent the occurrence of kernicterus, an uncommon but devastating, costly, and entirely preventable condition.

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Biopsychosocial approach to functional abdominal pain

We read with interest the article by Lindley and colleagues[1] outlining their concerns about consumerism in health care focusing on the potential detrimental effects on the child with functional abdominal pain (FAP). All of the children had extensive investigations carried out by the authors according to in house clinical service guidelines for the management of children with abdominal pain.

While this is surprising in itself, it is even of more concern when it is noted that most children already had extensive investigations in other centres. Clinical service guidelines should take into account the fact that children referred with abdominal pain to a tertiary referral practice have a high probability of having a functional disorder. Rather than embark on an extensive, expensive, and traumatic list of procedures, protocols should encompass a biopsychosocial approach to the management of abdominal pain. We are potentially doing a great disservice to children if we first resort to invasive investigations while failing to make a positive diagnosis of FAP.

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Reference

1 **Lindley KJ**, Glaser D, Milla PJ. Consumerism in healthcare can be detrimental to child health: lessons from children with functional abdominal pain. *Arch Dis Child* 2005;**90**:335–7.

Is timing of haemorrhage by spectrophotometry similar for haemorrhages in the subdural and subarachnoid space?

We investigated whether quantifying the spectral peaks for oxyhaemoglobin, methaemoglobin, and bilirubin (and their ratios) and comparing them to established standards for timing subarachnoid haemorrhage, might permit timing of the subdural haemorrhage.

When red cells enter the subarachnoid space, they are visible for a few days to several weeks.[1] Lysis of red cells results in oxyhaemoglobin release predominantly between 2 and 12 hours but continues up to 48 hours. A microsomal enzyme haeme oxygenase, released from macrophages (and the arachnoid membrane) converts oxyhaemoglobin to bilirubin. Bilirubin usually appears after 3–4 days but may exceptionally occur as early as 9–10 hours. The ''bilirubin transforming capacity'' is a rate limiting reaction, and when the concentration of oxyhaemoglobin rises rapidly, additional amounts are oxidised non-enzymatically to methaemoglobin.[2]

Thirty spectrograms performed on centrifuged, undiluted samples of subdural aspirates from 14 infants (mean age 4.6 months) admitted with subdural haematoma/effusion of suspected non-accidental origin, were reviewed retrospectively and peaks of oxyhaemoglobin (absorption peak at 413–415 nm), bilirubin (peak at 450–460 nm), and methaemoglobin (absorption peak at 405 nm) identified.

The haemoglobin (Hb) and bilirubin (Bil) spectral amplitudes were converted to micromoles per litre using a nomogram[3] and the Haemoglobin Index [HBI = Hb (μmol/l)/Bil (μmol/l)] and Haemoglobin Coefficient (HC = arc sine$\sqrt{\{[Hb]/[Hb + Bil + 1]\}}$ + arc sine $\sqrt{\{[Hb + 1]/[Hb + Bil + 1]\}}$) calculated on the 30 spectrograms.[4]

Absorbance indices (HBI and HC) of oxyhaemoglobin and bilirubin in subdural specimens did not correspond with those reported for subarachnoid haemorrhage and were unrelated to the time from admission. Pigment concentrations were higher than those reported from patients with subarachnoid haemorrhage, confirming similar observations of Wahlgren and Lindquist who suggested that this was due to ''packing'' of the erythrocytes and their subsequent lysis.[5] Unlike a subarachnoid haemorrhage which disperses within the cerebrospinal fluid spaces and will dilute and disappear fast, the subdural haemorrhage is in a more encapsulated space without a natural circulation.

We concluded that while spectrophotometry of the subdural fluid can identify fresh blood, oxyhaemoglobin, bilirubin, or methaemoglobin in the aspirate, and the presence of bilirubin indicates that bleeding has occurred between 24 hours and 3 days prior to admission, it is not possible to time the original haemorrhage by using spectral peak data from existing models of subarachnoid haemorrhage.

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doi: 10.1136/adc.2003.036061

Competing interests: none declared

References

1 **Tourtelotte W**, Metz LN, Bryan ER, DeJong RN. Spontaneous subarachnoid haemorrhage. *Neurology* 1964;**14**:302–6.
2 **Wahlgren NG**, Lindquist C. Haem derivatives in the cerebrospinal fluid after intracranial haemorrhage. *Eur Neurol* 1987;**26**:216–21.
3 **Kronholm V**, Lintrup J. Spectrophotomeric investigations of the cerebrospinal fluid in the near-ultraviolet region. A possible aid in diseases of the central nervous system. *Acta Psychiatr Neurol Scand* 1960;**35**:314–29.
4 **Marlet JM**, Barreto Fonseca J de P. Experimental determination of time of intracranial haemorrhage by spectrophotometric analysis of cerebrospinal fluid. *Journal Forensic Sci* 1982;**27**:880–8.
5 **Wahlgren NG**, Lindquist C. Haem derivatives in subdural haematomas. *Acta Neurochir (Wien)* 1988;**95**:109–13.

Melatonin and epilepsy

There have been conflicting reports of the effects on seizure control of prescribing melatonin for people with epilepsy.[1–4] We undertook a retrospective before-and-after observational study of 13 young people prescribed melatonin for sleep disturbances at the David Lewis Centre, a residential school for children and young adults with severe epilepsy and learning difficulties situated in Cheshire, UK, with particular focus on any changes in seizure frequency.

At the David Lewis Centre each patient has a comprehensive record of their daily seizure profiles (seizure numbers and seizure types over 24 hours) carefully documented by care workers. Daily seizure rates were tabulated for each young person at 3 months, 1 month, 1 week, and 24 hours before and after the start of melatonin administration. Data were analysed using the Wilcoxon signed ranks test.

Eleven children (aged 6–18 years, mean age 14.1) and two adults were included. All had severe learning disabilities and behavioural problems, 12 had autistic spectrum disorders, and 11 suffered from severe epilepsy. All of the young people had severe sleep disturbance.

The dose of melatonin ranged from 2–6 mg nocte with a mean dose of 4.8 mg (SD 1.54) (0.1 mg/kg/day, SD 0.05). Eleven of the 13 young people slept better with melatonin. Two discontinued melatonin due to lack of efficacy. For the remainder the mean length of time on melatonin was 2.6 years. One person showed worsening behaviour following melatonin initiation, but no other side effects were observed. Of those with epilepsy three had an increase in seizure rate, seven had a decreased seizure rate, and one patient had no observable difference. The Wilcoxon signed ranks test was applied to the data using a significance level of 0.05. The p value was insignificant (>0.05) for all four time parameters, indicating that in this study melatonin had no effect on seizure frequency.

Our experience has been that melatonin can be helpful for sleep disturbance in young people with significant neurological impairment without a demonstrable influence on seizure control.

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doi: 10.1136/adc.2005.077172

Competing interests: none

References

1 **Sheldon SH**. Pro-convulsant effects of melatonin in neurologically disabled children. *Lancet* 1998;**351**:1254.
2 **Jan JE**, Hamilton D, Seward N, *et al*. Clinical trials of controlled-release melatonin in children with sleep-wake cycle disorders. *J Pineal Res* 2000;**29**:34–9.
3 **Peled N**, Shorer Z, Peled E, *et al*. Melatonin effect on seizures in children with severe neurologic deficit disorders. *Epilepsia* 2001;**42**:1208–12.
4 **Fauteck J**, Schmidt H, Lerchl A, *et al*. Melatonin in epilepsy: first results of replacement therapy and first clinical results. *Biol Signals Recept* 1999;**8**:105–10.

Nutritional rickets is increasingly diagnosed in children of ethnic origin

We noted with interest the article published by Ladhani and colleagues[1] highlighting the problem of vitamin D deficiency. We agree that this remains a problem, especially in ''at risk'' ethnic minority groups.

In Oldham, which has a population of 49 992 children, 20.8% are Asians (Census 2001). Between December 2002 and March 2004, we identified nine cases of hypocalcaemia/rickets secondary to vitamin D deficiency. We excluded those with vitamin D deficiency secondary to other conditions of non-nutritional aetiologies. All of the nine children had biochemical changes of raised alkaline phosphatase and levels of 25-OHD below 10 ng/ml; three had radiological evidence of rickets. Eight were of Asian origin and five were male.

Presentation of these children was divided into those with hypocalcaemic symptoms and those with clinical rickets. Six of them presented with hypocalcaemic symptoms and their ages ranged from 6 days to 13 years of age. These included two neonates who presented with focal seizures; two toddlers under 2 years who presented with generalised seizures; and two 13 year olds who presented with cramps/carpopedal spasms. Three of the nine presented with signs of rickets and were aged 15–19 months.

The two neonates involved were born at term with their birth weights on the 25th centiles. Calcium levels were 1.39 and 1.54 mmol/l respectively. Both were on formula feeds, and tests on maternal blood revealed levels of parathyroid hormone and calcium suggestive of vitamin D deficiency. Four toddlers were still breast fed, all of whom were confirmed from dietary history to have limited solid intake. Of the two teenagers, one had a diet low in calcium and the other had background problems of abdominal pain.

All the children were treated with vitamin D, and three children also received oral calcium supplements. All responded to treatment with normalisation of biochemical bone profiles and vitamin D/parathyroid hormone levels.

There is no information on the prevalence of rickets in the UK; however, there are reports to say that this is growing.[2] Our

experience and reports across the UK show that the ethnic minority population still remains at risk of vitamin D deficiency. Efforts to promote vitamin D supplementation as recommended by the Department of Health[3] need to be implemented and targeted at the risk group.

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References

1 **Ladhani S**, Srinivasan L, Buchanan C, *et al*. Presentation of vitamin D deficiency. *Arch Dis Child* 2004;**89**:781–4.
2 **Mughal MZ**. Resurgence of vitamin D deficiency rickets in the UK. *Osteoporosis Review* 2005;**13**(1):10–13.
3 **Department of Health, Nutrition and Bone Health: with particular reference to calcium and vitamin D**. Report of the Subgroup on Bone Health, Working Group of the nutritional Status of the Population of the Committee on Medical Aspects of the Food Nutrition Policy. *Rep Health Soc Subj (Lond)* 1998;**49**:iii–vii,1–24.

A vaccine scare in 19th century Northampton

The controversy regarding immunisation is longstanding. Records from 1806 concerning a vaccine scare in Northampton give a flavour of events, which strike a contemporary chord.

The revelation of Edward Jenner's 1798 seminal work meant smallpox mortality fell from 31% in unvaccinated children compared to 1.2% in vaccinated.[1 2]

Northampton General Infirmary made cowpox vaccination a high priority and was proactive in its approach, with free cowpox inoculation being undertaken on the hospital premises from 1804 onwards.[3]

On 10 January 1806 the Board of Governors dealt with a growing vaccine scare concerning alleged vaccine failure and one in particular, leading to the death of a child, Peter Bell.

> ''Gentlemen, the public mind having been lately much agitated by reports of the insecurity of the vaccine inoculations, we have endeavoured to investigate those instances of failures we have heard of and have invariably found such reports to be arrived at either by error or misrepresentation.''[3]

However, to defuse the situation an affidavit signed by the parents of Peter Bell denying these rumours was published in the *Northampton Mercury*:[3]

> **Article from the *Northampton Mercury*, 10 January 1806**
> ''Whereas a false and groundless report has been spread about this town and neighbourhood that our son Peter Bell died on the 6th instant of smallpox after having been inoculated for the cowpox by Dr Kerr and the Infirmary now we do hereby declare that neither the above named child nor our child Ann Bell ever had the smallpox or the symptom or appearance of smallpox whatever. Both our said children were inoculated for the cowpox by Mr. Mills and both of them came safely through the disease. The eldest of them has been ever since in perfect health and Peter the youngest having been always a weakly child had better health after the cowpox than ever he had enjoyed before until he was seized with a violent complaint in his bowels of which he died on 20th December last.'' (Signed by William Bell, guard to the Defiance coach; Sarah Bell, his wife[3])

The following week on 17 January the Board of Governors reported.

> ''The Governors…having adopted the resolution of permitting the poor to be inoculated for the cowpox as outpatients…do hereby certify that we know of no incidence of any person having had the smallpox who had been previously inoculated for the cowpox.''[3]

A register was however established with the hope: ''By these means the practice of vaccination and its merits as a complete security against the smallpox will be gradually be brought to the test of unprejudiced experience''.[3]

One could regard this as common sense, which today would be described as ''clinical governance''.

Doctors beleaguered in the present time through similar ''misrepresentations'' regarding immunisations should take heart that this is not a new problem, but perhaps managers could learn from the more robust attitude taken by our medical forebears when dealing with the media in these matters.

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doi: 10.1136/adc.2005.081034

Competing interests: none declared

References

1 **Jenner E**. *An inquiry into the causes and effects of the variolae vaccine*. London: Sampson Low, 1798.
2 **Sheldon W**. *Diseases of infancy and childhood*. London: JA Churchill Ltd, 1948:715.
3 **Waddy FF**. *A history of Northampton General Hospital 1743–1948*. Northampton: The Guildhall Press, 1974:57–8.

More evidence is needed in the antibiotic treatment of *Pseudomonas aeruginosa* colonisation

In presenting various therapeutic approaches for the management of cystic fibrosis (CF), Smyth primarily considers evidence obtained from The Cochrane Library as either systematic reviews of randomised controlled trials (RCTs) or RCTs.[1] The antibiotic treatment of *Pseudomonas aeruginosa* (PA) when first isolated, is still an open question. When discussing this aspect, Smyth considers only the RCT by Valerius and colleagues.[2]

In our critical review of published clinical studies evaluating the early antibiotic treatment in asymptomatic PA colonised CF patients,[3] we identified three relevant RCTs (two versus placebo).[2 4 5] Our study also included eight cohort studies, two of which were with historical controls. Overall, 309 patients (range 7–91) were recruited. There was a high variability between the individual studies for age, outcome measures, duration of follow up, and treatment (three studies: two RCTs; 1 cohort used only aerosol tobramycin, 1 colistin, 4 aerosol colistin plus ciprofloxacin, 1 intravenous treatment, and 2 miscellaneous therapy).

An overall critical evaluation indicated that early antibiotic treatment can reduce the rate of positive cultures and of anti-PA antibody titres. Long term benefit is expected but not yet proven. Moreover, we recently conducted an observational study which found that nearly all CF centres in Italy treat asymptomatic PA colonised patients in order to prevent or postpone chronic pulmonary infection (unpublished data). However, the adopted prescribing practice varies largely even within the same centre, highlighting the existing lack of formal consensus on this subject.

Several therapeutic options (aerosol therapy alone or oral therapy associated with aerosol inhalation) are available for the early treatment of PA colonisation, but no direct comparison has so far been made. Prospective multicentre randomised studies with relevant outcomes measures[6] are needed to investigate which of the different proposed antibiotic schemes has the best benefit/risk ratio and the best patient compliance.

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References

1 **Smyth RL**. Diagnosis and management of cystic fibrosis. *Arch Dis Child Ed Pract* 2005;**90**:ep1–ep6.
2 **Valerius N**, Koch C, Hoiby N. Prevention of chronic Pseudomonas aeruginosa colonisation in cystic fibrosis by early treatment. *Lancet* 1991;**338**:725–6.
3 **Marchetti F**, Giglio L, Candusso M, *et al*. Early antibiotic treatment of pseudomonas aeruginosa colonisation in cystic fibrosis: a critical review of the literature. *Eur J Clin Pharmacol* 2004;**60**:67–74.
4 **Wiesemann HG**, Steinkamp G, Ratjen F, *et al*. Placebo-controlled, double-blind, randomized study of aerosolized tobramycin for early treatment of Pseudomonas aeruginosa colonization in cystic fibrosis. *Pediatr Pulmonol* 1998;**25**:88–92.
5 **Gibson RL**, Emerson J, McNamara S, *et al*. Significant microbiological effect of inhaled tobramycin in young children with cystic fibrosis. *Am J Respir Crit Care Med* 2003;**167**:841–9.
6 **Ramsey BW**, Boat TF. Outcome measures for clinical trials in cystic fibrosis. *J Pediatr* 1994;**124**:177–92.

Community needlestick injuries may still be dangerous

We read with interest the report by Makwana and Riordan on community needlestick injuries in children.[1] We do not believe, however, that the authors have presented sufficient data to support their conclusion that routine follow up after community needlestick injury is unnecessary.

In their study only 25 children had complete serological follow up. Their literature review cites three additional papers in which children were followed up after needlestick injury. Adding all of these children gives a total of 138 children who had

serological testing following needlestick injury. This is an insufficient number to allow one to conclude with confidence that the risk of transmission is low.

If all of these needles contained HIV positive blood, applying the rule of threes[2] to the pooled data, we can say with 95% confidence that the risk of HIV transmission following community needlestick injury in children is less than 2%. The transmission rate in healthcare workers following HIV positive needlestick injury is around 0.35%. Their study, therefore, does not provide sufficient evidence to state that these children are at a lower risk of acquiring HIV following needlestick injury than healthcare workers in similar circumstances. Until such evidence becomes available, there seems to be no good reason to treat these children differently to healthcare workers following needlestick injury.

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References

1 **Makwana N**, Riordan FAI. Prospective study of community needlestick injuries. *Arch Dis Child* 2005;**90**:523–4.
2 **van Belle G**. *Statistical rules of thumb*. New York, NY: John Wiley and Sons, 2002:49.

Authors' reply

We were interested to read this letter. The authors feel that children with community needlestick injuries should be treated the same as healthcare workers. This seems to miss the point of our paper. Hospital needlestick injuries are very different to out-of-hospital needlestick injuries: the blood is generally dry, so therefore less likely to be infectious;[1] the injuries are often superficial—again less likely to be infectious;[1] and, although the HIV status of the needlestick user is often unknown, the incidence outside of London is very low.

The risk of HIV transmission is estimated to be less than 1:100 000.[2] Our study was not designed to show the risk of transmission (which incidentally would need a study of more than 100 000 patients), but showed that only half those offered follow up returned for their appointment. Studies examining needlestick exposure and HIV seroconversion have shown that no children seroconverted despite not receiving HIV post-exposure prophylaxis.[3–5] Within this population of children were included those who sustained injuries from areas with a high prevalence of injecting drug use. Zamora and colleagues[6] evaluated HIV-1 proviral DNA from 28 discarded syringes of intravenous drug users and found no traces of the virus, concluding that the risk of HIV transmission in that setting was zero.

These children are therefore in a low risk group for transmission of infectious viruses, and together with the low rate of attendance for follow up, it is still reasonable, we feel, to offer follow up to those children who have high risk injuries or in whom parents have a high level of anxiety.

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References

1 **Havens PL**. Committee on Pediatric AIDS. Postexposure prophylaxis in children and adolescents for nonoccupational exposure to human immunodeficiency virus. *Pediatrics* 2003;**111**:1475–89.
2 **Anon**. Post exposure prophylaxis (PEP) guidelines for children exposed to blood borne viruses. www.bhiva.org/chiva (accessed 29 June 2004).
3 **Wyatt JP**, Robertson CE, Scobie WG. Out of hospital needle stick injuries. *Arch Dis Child* 1994;**70**:245–6.
4 **Nourse CB**, Charles CA, McKay M, *et al*. Childhood needle stick injuries in the Dublin metropolitan area. *Ir Med J* 1997;**90**:66–9.
5 **Aragon-Pena AJ**, Arrazola-Martinez MP, Garcia de codes A, *et al*. Prevencion de hepatitis B y reisgo de infeccion por VIH en ninos accidentados con agujas y /o jeringuillas abandonadas. *Atencion Primaria* 1996;**17**:18–140.
6 **Zamora AB**, Rivera MO, Garcia-Algar O, *et al*. Detection of infectious human immunodeficiency type 1 virus in discarded syringes of intravenous drug users. *Pediatr Infect Dis J* 1998;**17**:655–7.

Interpreting immunogenicity data in UK studies

It has become increasingly clear that interaction between vaccines is an important consideration for immunogenicity studies. Only full information on all vaccines used in a particular population will allow correct interpretation of immunogenicity data.[1] This is particularly important where comparison is made to historical controls in a rapidly changing schedule such as that used in the UK, or where immunogenicity data obtained using vaccines that differ significantly from those currently in use are subsequently used to guide practice.

It also apparent that the ''best'' combination of specific vaccines, the effects of interactions of conjugate proteins, the optimal timing of the primary course, and the necessity for boosters within the UK schedule are all currently unclear. Certain groups of infants may require separate consideration, for example those born preterm or from specific ethnic backgrounds.

We therefore read with interest the data presented by Booy *et al* of responses to primary series immunisation in Asian infants born in the UK to a population of parents of whom ''most'' (88%) were born abroad.[2]

Based on the achievement of an anti-PRP GMT of 15 μg/ml, Booy *et al* are reassured that vaccination with PRP-T should protect this population from Hib meningitis. We are uncertain as to whether this confidence is justified. There is no clear description of the exact vaccines administered to their population, or of when the study took place. PRP-T and DTP were administered in separate limbs, but the nature of the pertussis component of the DTP (whole cell [DTPw] or acellular [DTPa]) is unspecified. Since DoH advice from 1996 was for combined single limb injection of PRP-T and DTP, we assume that the study predates 1996.[3] Given that DTPa was introduced in 1999,[4] we therefore also assume that the study DTP was DTPw. Separate limb administration of DTP, or using DTPw may result in a higher anti-PRP GMT in comparison to that achieved by infants receiving either combined vaccines[5]) or an acellular DTP[6] (or a combination of this, as with the UK's new vaccine, Pediacel).

While Booy *et al* comment on their study as ''descriptive and uncontrolled'', they do include a historical cohort of controls. Neither the original publication of the control data,[7] nor this present publication clearly describe to the reader the actual (as opposed to planned) timing of important study interventions (vaccine administration, vaccine intervals, blood sampling in relation to vaccines), with the exception of acknowledging that the median time of primary course completion differed between the two groups. Clear descriptions of study timings would allow the reader to consider whether the populations are crudely comparable; alternatively a statistical analysis could have been performed that would take account of these differences. Without this the difference in GMTs is without context. Placing the data in context may help explain the otherwise very surprising finding that Asian infants appear to respond three times as well to PRP-T as Caucasians.

It would also be interesting to know the limits of detection for the anti-PRP assay, and how results above or below these limits were handled—the 28 (or 34) infants having surprisingly tight 95% confidence intervals around their GMT for such small numbers of infants.

Given the recurrence of Hib disease in the UK, the question of how well UK infants respond to PRP-T is clearly very important, as well as whether or not UK infants (like most others) should receive a fourth (booster) dose. Careful studies that help to address these questions are crucial. We would welcome the additional information from Booy and colleagues that would allow this current information to be more readily interpreted.

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References

1 **Berrington JE**, Fenton AC. Interpreting immunisation data requires complete information. *Arch Dis Child Fetal Neonatal Ed* (published online 4 October 2004).
2 **Booy R**, Haworth EA, Ali KA, *et al*. Immunogenicity of routine vaccination against diphtheria, tetanus, and Haemophilus influenzae type b in Asian infants born in the United Kingdom. *Arch Dis Child* 2005;**90**:589–91.
3 **Department of Health**. *Combined Hib/DTP vaccines*, Chief Medical Officers Update 10, 1996.
4 **Department of Health**. *Current vaccine issues: action update*, 1999 PL/CMO/99/9 1–4.
5 **Begg N**, Miller E, Fairley CK, *et al*. Antibody responses and symptoms after DTP and either tetanus or diphtheria Haemophilus influenzae type b conjugate vaccines given for primary immunisation by separate or mixed injection. *Vaccine* 1995;**13**:1547–50.
6 **Bell F**, Heath P, Shackley F, *et al*. Effect of combination with an acellular pertussis, diphtheria, tetanus vaccine on antibody response to Hib vaccine (PRP-T). *Vaccine* 1998;**16**:637–42.
7 **Booy R**, Taylor SA, Dobson SR, *et al*. Immunogenicity and safety of PRP-T conjugate vaccine given according to the British accelerated immunisation schedule. *Arch Dis Child* 1992;**67**:475–8.

Car seat safety for premature and LBW infants

Recent advances in neonatal intensive care have resulted in improved survival rates of premature and low birth weight infants. These infants are frequently transported in the parent's own vehicle when discharged from hospital. Commercially available infant car seats are primarily designed for a typical

infant weight of 3.1 kg and hence may not be suitable for premature and low birth weight infants. We conducted a postal questionnaire survey of 200 neonatal and special care baby units in the UK, to assess current practice of "car seat safety" at hospital discharge for premature and low birth weight infants. They were posted to both the "consultant-in-charge" and "nurse-in-charge" for these units. The response rates for the consultants and nurses were 60.5% and 90.5% respectively. Analysis of the responses suggests that 90% of the neonatal units across the UK do not have a programme for assessing "car seat safety" at discharge for these high risk infants. The typical discharge weight of these infants can range from 1.5 kg to 3.0 kg. A small proportion of these infants are also discharged home on oxygen. If they are not transported in an appropriate car seat with appropriate precautions, these infants may be subject to oxygen desaturation, especially when placed in a semi-upright position.[1–3] They are also at risk of respiratory compromise because of the potential for slumping forward and lateral slouching if they cannot be adequately restrained in the seat.[4] The American Academy of Pediatrics has published recommendations for transport of these infants based on current research and evidence[4] and they recommend that these high risk infants be monitored in their car seats for apnoea, desaturations, and bradycardia for an hour, prior to discharge. This would enable the identification of infants at risk so that parents can be appropriately counselled regarding the suitability of the car seats. Families should be advised to minimise travel for infants at risk of respiratory compromise. Infants failing the test could be retested in a different car seat. There is a paucity of studies in this area and clearly further research is essential to guide us in establishing and implementing an appropriate "car seat safety" programme for these vulnerable infants.

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Copies of the questionnaire used in our survey can be obtained by contacting the corresponding author

doi: 10.1136/adc.2005.080614

Competing interests: none declared

References

1 **Willet LD**, Leuschen MP, Nelson LS, *et al*. Risk of hypoventilation in premature infants in car seats. *J Pediatr* 1986;**109**:245–8.
2 **Willet LD**, Leuschen MP, Nelson LS, *et al*. Ventilatory changes in convalescent infants positioned in car seats. *J Pediatr* 1989;**115**:451–5.
3 **Bass JL**, Mehta KA, Canara J. Monitoring premature infants in car seats: implementing the American Academy of Pediatrics policy in a community hospital. *Pediatrics* 1993;**91**:1137–41.
4 **American Academy of Pediatrics**. Safe transportation of premature and low birth weight infants. *Pediatrics* 1996;**97**:758–60.

Melatonin: prescribing practices and adverse events

Melatonin is currently an unlicensed, "named patient only" medicine in the UK, although it is available as a dietary supplement in the United States and over the internet. It is used for a variety of sleep disorders in children who often have neurodevelopmental impairments.[1 2] There remains a dearth of robust randomised controlled trials to demonstrate its efficacy, while lack of pharmacokinetic, pharmacodynamics, and toxicology data limits knowledge of therapeutic dose ranges, formulations, and adverse effects.

We carried out an anonymous questionnaire survey to examine prescribing practices of members of the British Association for Community Child Health (BACCH) and the British Academy of Childhood Disability (BACD) (see *ADC* website: http://www.archdischild.com/supplemental).

From a newsletter circulation reaching an estimated 926 paediatricians, responses to the questionnaire were received from 148 (about 15%) (table 1).

Of these 98% were currently prescribing, or had prescribed melatonin in the last year; data on a total of 1918 children were obtained.

The dose prescribed (0.5–24 mg) varied widely (table 2).

Autism (68%) and attention deficit hyperactivity disorder (44%) were the most frequent clinical diagnoses in the children prescribed melatonin. On a crude four point scale of perceived effectiveness (never, rarely, usually, always), over 95% of respondents found melatonin "usually" or "always" effective. Adverse events were reported by 18% (n = 27) of respondents including: new onset seizure activity (n = 2), increased seizure frequency (n = 3), hyperactivity (n = 5), agitation/behavioural changes (n = 6), worsening sleep pattern (n = 6), nightmares (n = 2), and constipation (n = 2).

As this survey was opportunistic, and unfunded, we did not have the opportunity

Table 2 Dose of melatonin prescribed

	Median	Range	25–75% interquartile
Starting dose (mg)	2.5	1.0–5.0	2.0–3.0
Lower maintenance dose (mg)	3	0.5–10.0	2.0–3.0
Higher maintenance dose (mg)	6	2.0–20.0	6.0–9.0
Maximum dose used (mg)	8	2.0–24.0	6.0–10.0
	0–2.0 mg	**2.1–3.0 mg**	**>3.0 mg**
Starting dose	63 (44%)	70 (49%)	9 (7%)
Lower maintenance dose	42 (30%)	69 (48%)	31 (22%)
	0–5 mg	**6–9 mg**	**>9 mg**
Higher maintenance dose	34 (24%)	82 (58%)	26 (18%)
	Immediate release	**Slow release**	**Both**
Formulation of melatonin	89 (68.5%)	3 (2.3%)	38 (29.2%)

Table 1 Responses to the questionnaire

	Response					
	Yes	**No**	**Median**	**Mean**	**Range**	**25–75th quartile**
Prescribed melatonin	145 (98%)	3 (2%)	8	14.4	1–150	5.0–20
	Autism	**ADHD**	**Learning difficulties**	**Visual impairment**	**Specific sleep disorders**	
Disorders treated	97 (68%)	63 (44%)	57 (40%)	19 (13%)	7 (5%)	
	Sleep onset difficulties	**Night waking**	**Specific sleep disorder**	**Carer respite**	**EEG**	**Non-specific sleep problems**
Indications for melatonin	53 (39%)	16 (12%)	5 (4%)	4 (3%)	2 (1.5%)	68 (50%)
	Behavioural therapy/sleep hygiene	**Other medication**	**Advice**	**Other**		
Measures prior to melatonin	124 (87%)	32 (22%)	7 (5%)	7 (5%)		

to further interrogate the non-responders and determine to what extent they systematically differed from the responders. Information on frequency of prescribing is also missing on a national level, as exact numbers of melatonin prescriptions are not recorded, but since November 2002, 239 UK hospitals/trust pharmacies have requested melatonin (personal communication, Peter Stephens, IMSHealth, 2004).

Reports of adverse events from our study mirror those in the literature.[2–4] Although 27 respondents in this limited survey reported adverse events, only 13 reports, involving 25 adverse events were notified to the UK Medicines and Healthcare products Regulatory Agency (MHRA) (Committee for Safety of Medicines, Drug Analysis Print: Melatonin; personal communication, 2004) and two notified to the UK Food Standards Agency in the same period (personnel communication, Cath Mulholland, 2004). Whether these ''adverse events'' represent a significant rise above events that would be seen by chance in this population will need much larger studies over a longer time period.

It remains crucial to establish just how effective melatonin is for children with developmental disorders, through large scale, multicentre randomised controlled trials. This survey suggests that problems agreeing appropriate and safe dose ranges, the heterogeneity of underlying developmental problems, and a potentially wide range of underlying sleep disorders are just a few of the hurdles that will need to be overcome.

Acknowledgements

The authors would like to acknowledge the help of Ms Cath Mulholland (Food Standards Agency) for data concerning adverse events reporting in the UK and Mr Peter Stephens (IMSHealth) for NHS prescribing data.

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doi: 10.1136/adc.2005.077289

Competing interests: none declared

The questionnaire can be viewed on the *ADC* website (http://www.archdischild.com/supplemental)

References

1 **Willey C**, Phillips B. Is melatonin likely to help children with neurodevelopmental disability and chronic severe sleep problems? *Arch Dis Child* 2002;**87**:260.
2 **Jan JE**, Freeman RD. Melatonin therapy of circadian rhythm sleep disorders in children with multiple disabilities: what have we learned in the last decade? *Dev Med Child Neurol* 2004;**46**:776–82.
3 **Sheldon SH**. Pro-convulsant effects of oral melatonin in neurologically disabled children. *Lancet* 1998;**351**:1254.
4 **Palmer ME**, Haller C, McKinney PE, *et al*. Adverse events associated with dietary supplements: an observational study. *Lancet* 2003;**361**:101–6.

Hearing impairment: age at diagnosis, severity, and language outcomes

I have read with great interest the original article from Wake and colleagues[1] and I would like to acknowledge and compliment their valuable efforts in such a difficult research area. I felt nevertheless quite concerned with the conclusions of this study and their possible repercussions. Diagnosis and management of childhood deafness is one of my areas of interest and I have also been actively involved in the setting up of NHSP in my local district In the UK, the NHSP is in its final phase of implementation and hopefully there will be no going back. In other areas of the globe, however, where professionals may still be pondering about the importance and need of such a programme, outcomes of research studies like this one may help to tilt the balance in the wrong direction.

Research into deafness and especially childhood deafness is extremely difficult, a real minefield. Severe and profound deafness is relatively rare and the number of variables to take into consideration is huge: age of diagnosis, age of hearing aid fitting, consistent use of hearing aids, cochlear implant, age at start of other forms of intervention such as speech and language therapy, educational input (type of specialist intervention programmes, bilingual versus oral-only programmes), cognitive ability, parents' hearing status, parents acceptance and cooperation with professionals...the list is enormous.

Only a study involving very large populations would allow for improved variable control and still achieve comparison samples large enough to be treated statistically. This would require huge human and financial resources and is usually beyond the possibility of most research centres.

The present study did attempt to control some of these variables, but the inclusion of hearing losses from mild to profound (or even hearing losses above 40 dB HL) may have skewed the results. Severity of hearing impairment is in itself such a stronger predictor of language outcome that it compensates for many other variables including age of diagnosis.

Deaf children with a hearing loss of around 60 or 70 dB HL, may, with consistent use of well fitted hearing aids, achieve enough amplification to be able to hear and discriminate spoken language, essential for spoken language acquisition. A profoundly deaf child with >90 dB HL loss or more will never be able to achieve that much. If comparisons between severe and profound deaf children already cause difficulties, what to say when moderate hearing losses are also included?

I believe this is one of the reasons why, in this study, age of diagnosis did not help to predict language outcome and therefore the conclusion that early diagnosis may not be an important factor in improving outcomes for deaf children may not be correct.

Other factors may also have influenced outcome in this particular study. There is very little information about intervention programmes and since children came from different areas and schools, these may be very different and have significant impact on progress. Also, there is no mention of use of sign language and I wonder if this is not used at all by the children in the sample or just spoken language progress was considered.

I would like to finish with a parent's reply when asked how she felt at the time of her child's late diagnosis (at 9 months of age): ''We were too relieved. We should be upset or shocked but, having battled with someone for five months, it was just a relief that someone believed''. However, later on, she would say: ''I was angry, I was very angry, I don't know I will ever get over the anger''.

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Reference

1 **Wake M**, Poulakis Z, Hughes EK, *et al*. Hearing impairment: a population study of age at diagnosis, severity, and language outcomes at 7–8 years. *Arch Dis Child* 2005;**90**:238–44.

Food challenge tests

Ewan and Clark's helpful commentary provokes further comment on the diagnosis of allergy and the management of the allergic child.[1] The issues raised are controversial because differences in clinical practice exist between countries, between allergy centres in the UK, and between allergists and general paediatricians. Unavailability of skin prick testing outside allergy centres accounts for some of the differences, but neither SPT or RAST distinguish between sensitisation and clinical allergy; scepticism about the meaningfulness of test results will continue until they are validated by oral food challenge (OFC) and correlated with a careful clinical history. Persistence of positive SPT is not always evidence for persistence of allergy[2] and restriction of the OFC to the role of confirming resolution of allergy as suggested by Ewan and Clark will tend to disadvantage patients with indeterminate skin prick results, those with newer food allergies such as kiwi and sesame with uncertain prognosis, and those where the history is open to question. The usefulness of OFC as a tool for exploring allergic thresholds and for defining the characteristics of an individual's allergic reaction has not yet been clearly defined but merits further study. Although OFC should only be recommended and performed by allergists experienced in the selection of appropriate patients, challenge need not be restricted by risks of severe adverse reactions, the incidence of which is reported to be approximately 1% for open challenges in routine clinical practice.[3] Higher rates of severe reactions have been described in studies where larger and cumulative doses of allergen were used in double blind placebo controlled challenges.[4] My own series of patients with higher rates of reactions requiring bronchodilator treatment included a high proportion of asthmatic children and they also received larger doses of allergen.[5]

Establishing the true presence of food allergy is fundamental to clinical management. Allergists are better at making a correct diagnosis than the non-specialist, but the various diagnostic errors and pitfalls suggest that we should be utilising all the available tests more fully in the best interests of the patient. I agree with Ewan and Clark that many more trained paediatric allergists will be required to provide this service.

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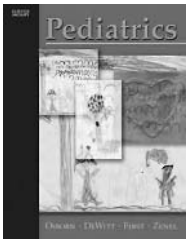
References

1 **Ewan PW**, Clark AT. IgE mediated food allergy: when is food challenge needed? *Arch Dis Child* 2005;**90**:555–6.
2 **Hourihane J**. Roberts SA, Warner JO. Resolution of peanut allergy; case control study. *BMJ* 1998;**316**:1271–5.
3 **Torr T**, Gaughan M, Roberts G, *et al*. Food challenges; a review and audit. *Paediatr Nurs* 2002;**14**:30–4.
4 **Morriset M**, Moneret-Vautrin DA, Kanny G, *et al*. Thresholds for clinical reactivity to milk egg peanut and sesame in IgE dependant allergies. *Clin Exp Allergy* 2003;**33**:1046.
5 **Roberts S**. Challenging times for food allergy tests. *Arch Dis Child* 2005;**90**:564–6.

BOOK REVIEWS

Pediatrics

Edited by Lucy Osborn, Thomas DeWitt, Lewis First, Joseph Zenel. London: Elsevier Mosby, 2005, £76.00 (hardback), pp 2031. ISBN 0 323 01199 3



There are hundreds of textbooks on paediatrics. When I heard of another textbook on paediatrics, the first question that came to my mind was—Do we need another textbook on paediatrics and how does this particular book differ from the rest of the books on the market?

To start with, the authors make it very clear that this book is directed towards the generalists and does not provide in-depth information into rare conditions. This reference was conceived in response to the need for a generalists' text for paediatricians who have not narrowed their focus to a subspecialty of children's care. It does not aim to be an exhaustive review, particularly of unusual or rare conditions, but rather a source of easily accessible information for clinicians who deal most frequently with common complaints and make decisions about when to refer and how to co-manage children with complex chronic diseases.

The most important attribute of the book is its format. The approach is problem based rather than as a narrative of conditions. For example, in the section on the cardiovascular system, the subheadings are: ''Child with a murmur'', ''Child with chest pain'', and so on.

So, unlike the other standard texts that are on the market which give the conditions first and then go onto explain the symptoms and their management, this text by Osborn *et al* starts with symptoms and then gives a structured approach to evaluate the symptoms.

The book is organised in a very friendly manner. It is divided into nine sections. The first two sections deal with fundamentals and health promotion. These sections are unique to this book and provide a good revision of the basics for the generalist.

The next section forms the core of the book. It is divided into organ systems, but the approach is problem based. The book does not provide exhaustive information, but acts as a guide.

For example, in the section 'approach to child with headache' the authors do not provide an exhaustive list of causes of headache and their treatment. They give only pointers. There are boxes highlighted with a red flag, which make sure that a generalist does not miss the salient points in history and examination.

Other than the core medical problems, the book also contains sections on adolescence care, mental health care, and social aspects of childcare. These sections are quite exhaustive. These chapters have been handled with a very practical approach.

Other important features of the book are that it is very colourful. All the sections are colour coded for easy access. The book is well illustrated. In particular, the chapter on skin conditions contain many photographs, which are very informative.

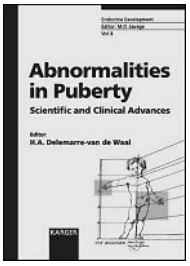
The book also comes with a CD-Rom. The CD is not the textbook in a digital format. The CD contains videos of clinical conditions, medical procedures, colour atlases of dermatological conditions, etc; all the tables and pictures on the CD are provided in a PowerPoint format that can be downloaded for educational use.

All in all, a very useful book to have as a part of the generalist's library.

M S H Madhava

Abnormalities in puberty: scientific and clinical advances

Edited by H A Delemarre-van de Waal. Karger, 2005, €117.00 (hardback), pp 182. ISBN 3 8055 7867 9



This book is described in the foreword as being of interest to paediatric and adult endocrinologists as well as workers involved with puberty. It is one of a series of books on endocrine development and has a strongly European dominated authorship.

The book is set out in 11 chapters which read like scientific papers, have useful explanatory abstracts, and are extensively referenced. A broad range of topics pertaining to puberty are covered, including the potential effects of fetal nutritional status on the timing of puberty, and adolescent topics such as polycystic ovarian syndrome and fertility preservation in cancer sufferers. The chapters are stand-alone articles and the reader is likely to pick and choose specific areas of interest rather than reading from cover to cover.

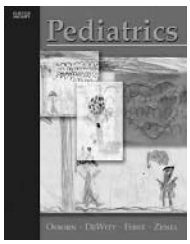
The chapters themselves vary from discussion of theoretical ideas about mechanisms of pubertal abnormalities to evidence based summaries of management of conditions, and presentation of trial data. The subject matter is generally well explained, even for the non-endocrinologist, but is quite scientific and specialised and the main appeal will be for those working within the field. Although background information is given as a reminder of pathophysiology which sets the scene for the new information presented, this is not an easy read and demands full concentration. Perhaps a summary of the points raised would have helped those with shorter attention spans and a desire for easily processed information. However, some relief from the text is provided in the way of data tables and graphs, and there are informative illustrative diagrams of receptors and hormone pathways, as well as clinical radiological images, such as MRI scan pictures of hypothalamic hamartomas.

Overall this book provides new insights into a variety of current topics in pubertal development and will no doubt assist in stimulating further developments in the field. There are interesting nuggets of information such as developments in the understanding of the genetics of hypogonadotrophic hypogonadism and some practical information on the diagnosis and management of precocious puberty; however, those revising for examinations or looking to broaden their knowledge of pubertal problems may wish to consult a more standard textbook first.

A Kelly

Paediatric pulmonary function testing

Edited by J Hammer and E Eber. Basel: Karger, 2005, €120.00 (hardback), pp 278. ISBN 3-8055-7753-2



Paediatricians often encounter patients with respiratory problems so most will have reasonable knowledge about the common and chronic respiratory diseases; virtually every paediatric department will have its collection of peak flow devices, spirometers, and other instruments for measuring and recording pulmonary function. Research departments and tertiary respiratory centres will have specialised lung function laboratories where more sophisticated tests may provide pages of numerical data to help the clinician best treat the child. But what is the place of pulmonary function testing in the broader clinical context and what does it all mean? This book has been written to provide a comprehensive survey of pulmonary function testing and to review the latest developments in the field.

The pleasingly slim tome is one of a series of books entitled ''progress in respiratory research'' and is a multi-author book written by experts in the individual areas that form many of the chapters. Despite this the style is consistent and the content up to date.

As one might expect with a comprehensive review, the book divides logically into lung function testing in infants and toddlers unable to cooperate with most procedures and is followed by analysis of the traditional adult founded techniques as applied to children. Technical and procedural considerations are

discussed; limitations, normative data, and aids to interpretation are proffered. These sections provide much useful theoretical and technical understanding in a reasonably digestible way; diagrams are helpful and equations are kept scarce. The book also covers the newer emerging methodologies such as exhaled breath condensate and exhaled nitric oxide testing, diffusing capacity, work of breathing, and respiratory muscle function. The final section deals with the clinical application of lung function testing to common respiratory disorders such as asthma, cystic fibrosis, neuromuscular disease, and the continuing care of neonatal survivors and lung transplant recipients. Lung function testing in critical care settings is also discussed. I found these sections the most useful part of the book. It was informative, rewarding, and at the same time surprising to read; for example, it finds no evidence to underpin the usefulness of lung function measurements (including peak flow) in the monitoring of asthma. Its suggestions for the care of children with neuromuscular disorders are sensible and appear to be broadly in step with recent American Thoracic Society guidelines.

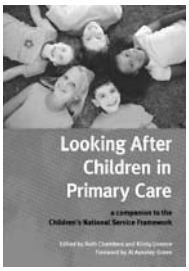
All chapters are kicked off with a pithy abstract that lets the reader know what they are in for, which was useful, although the substance of some of the technical sections proved hard going.

While there is no doubting the quality of the book I do wonder whether it will satisfy the potentially diverse needs of the pulmonologists, technicians, and general paediatricians the book claims it is written for. I suspect general paediatricians may find the last half of the book much more useful than the first, while technicians and researchers may find the converse. I would therefore recommend the book to research departments and specialist units more than to the general readership. For those considering buying the book, the price tag of £80.00 is likely to induce some sharp inspiratory manoeuvres in the purchaser.

A Brooke

Looking after children in primary care: a companion to the Children's National Service Framework

Edited by Ruth Chambers, Kirsty Licence. Oxford: Radcliffe, 2005, £24.95, pp 224. ISBN 1 85775 888 9



The recently published leading article on the National Service Framework (NSF) in *Archives of Disease in Childhood* by our ex-president of this college[1] and the issues such as child poverty, the phenomenal increase in the number of sexually transmitted diseases, teenage pregnancies, and campaigns such as Jamie Oliver's school dinners have highlighted child health issues. The government acknowledged that our youngsters are not just simply ''little adults'' by producing the Children's National Service Framework in 2004. Further government initiatives to improve the lives of the children and their families—for example, Every Child Matters and the Children Act 2004—have been announced. The question arises as to how many professionals understand what the NSF actually entails. The editors have been involved in the evolution of the NSF. They claim this book is a companion and will be beneficial to those working in primary care (health, education, and social services). Is the claim justified?

There are 18 chapters in the book in comparison to 11 standards set in the NSF for young people and maternity services. Of 11 NSF standards, five are meant for primary health care. Each chapter has its own merits: a good introductory overview of the Children's NSF; emphasising the need for involving children and young people in the organisation of health care with good examples and principles; setting out an audit checklist for GPs for creating a child and young person friendly environment; providing a box of core curricula for training people who work with parents; and displaying universally available preliminary support to be available to all parents. But the highlight of the book was chapter 18 (listening to young people's perspectives in relation to adolescent health). It covers smoking, obesity, drinking alcohol, illicit drug use, and sexual health. I liked the poem ''Don't go losing your virginity''. Overall this book is easy to read and doesn't falter in its purpose. It is well written by highly experienced authors. The tables and boxes highlight the key features in each chapter. The references are broad and up to date. The immunisation schedule keeps on changing, therefore the readers should update themselves. It is a pity that there is no chapter on resource implications. This book should be beneficial not only to primary healthcare professionals but also to those in secondary and tertiary care. I would recommend that women's and children's departments, managers involved with care of children, parents, and carers, and each library should have a copy.

G Sinha

Reference

1 **Craft A**. The National Service Framework for Children. *Arch Dis Child* 2005;**90**:665–6.

Book review in Fetal and Neonatal edition

The following book review is published in this month's *Fetal and Neonatal edition*:

- Neonatal formulary, 4th edition. Drug use in pregnancy and the first year of life: a pharmacopoeia

Pre-published book reviews

Book reviews that have been accepted for publication but have not yet been published in the print journal can be viewed online at http://adc.bmjjournals.com/misc/bookreviews.shtml