Clinical assessment of neonatal hyperbilirubinaemia

The study by Keren and colleagues1 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 cephalhaematoma. In cases where the cephalhaematoma 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 emphasis on “effective” breast feeds. The AAP guidelines also focus on the rarity of these events, such as vacuum extraction and cephalhaematoma, which 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 emphasis on “effective” breast feeds. The primary outcome measure was the post-discharge TSB.

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, $5 743 909 for routine pre-discharge 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 benefit.

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.5

They recommend a systematic clinical assessment before discharge and an early and focused follow up based on the risk assessment.6 Finally we must remember that we are all clinicians and we should use the lab report as an adjunct to our clinical knowledge.

Kanjilal and Prasad suggest that effective breast feeding can reduce significantly increased healthcare costs with uncertain benefit.

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References

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 mothers 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 “inter-related” 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 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 colleagues2 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
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. Lysis of red cells results in oxygenated blood, bilirubin and haemoglobin, 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 transformating capacity” is a rate limiting reaction, and when the concentration of oxyhaemoglobin rises rapidly, additional amounts are oxidised non-enzymatically to methaemoglobin.

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 431–415 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 microbels per litre using a nomogram and the Haemoglobin Index (HBI = Hb (µmol/l)/Bil (µmol/l)) and Haemoglobin Coefficient (HC = arc sine, (HBI/[Hb + Bil + 1]) + arc sine, (Hb/MM/[Hb + Bil + 1]) calculated on the 30 spectrograms.

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. Nonsignificant differences in the mean age, and in the number of infants with clinical and biochemical signs of rickets were noted. This suggests that the difference in pigment concentrations is due to “packing” of the erythrocytes and their subsequent lysis.

Unlike a subarachnoid haemorrhage which disperses within the cerebrospinal fluid space 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 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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References

Melatonin and epilepsy

There have been conflicting reports of the effects on seizure control of prescribing melatonin for people with epilepsy. 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 1 day 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 disturbances.

The dose of melatonin ranged from 2–6 mg nocte with a mean dose of 4.8 mg (SD 1.54 mg/kg/day, SD 0.05) for all four time points. Eleven of the 13 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 centile. 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 responding 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. 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 need to be implemented and targeted at the risk group.

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References

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.


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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 noted that consistent evidence obtained from The Cochrane Library as either systematic reviews of randomised controlled trials (RCTs) or RCTs. The antibiotic treatment of Pseudomonas aeruginosa (PA) when first isolated, is still an open question. When discussing this point she considers only the RCT by Valerius and colleagues.

In our critical review of published clinical studies evaluating the early antibiotic treatment in asymptomatic PA colonised CF patients, we identified three relevant RCTs (two versus placebo). 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 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

Community needlestick injuries may still be dangerous

We read with interest the report by Makvana and Riordan on community needlestick injuries in children. 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. Indeed, all these needles contained HIV positive blood, applying the rule of threes’ to the pooled data, we can say with 95% confidence that the risk of HIV transmission following 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

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; the injuries are often superficial—again less likely to be infectious; 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 in 100 000.1 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.2 Within this population of children were included those who sought care from areas with high prevalence of injecting drug use. Zamora and colleagues’ 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 with high risk injuries or in whom parents have a high level of anxiety.

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Interpreting immunogenicity data in UK studies
It has become increasingly clear that interaction between ethnic groups 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. 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 used 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 whom “most” were born abroad.

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 1996.4 Given that DTPa was introduced in 1999,5 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’ or an acellular DTPa (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 not include a historical cohort of controls. Neither the original publication of the control data, 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 of importance 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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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 mother’s own vehicle or discharged from hospital. Commercially available infant car seats are primarily designed for a typical

References
3 Department of Health. Combined Hib/DTP vaccines, Chief Medical Officers Update 10, 1996.
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 ²

They are also at risk of respiratory compromise due to the potential for slumping forward and lateral slouching if they cannot be adequately restrained in the seat.³ The American Academy of Pediatrics has published recommendations for transport of these infants based on current research and evidence⁴ 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 seat. 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
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Competing interests: none declared

References

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.² ³ 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 to conduct a full efficacy trial. We report our findings with the caveat that rates may be higher than those seen in our population; however, the prevalence of melatonin use in these children, and the frequency of adverse events is comparable to that documented in other studies.

Table 1 Responses to the questionnaire

<table>
<thead>
<tr>
<th>Response</th>
<th>Prescribed melatonin</th>
<th>No</th>
<th>Median</th>
<th>Mean</th>
<th>Range</th>
<th>25–75% quartile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>145 (98%)</td>
<td>3 (2%)</td>
<td>8</td>
<td>14.4</td>
<td>1–150</td>
<td>2.0–20</td>
</tr>
<tr>
<td>Autism</td>
<td>97 (68%)</td>
<td>63 (44%)</td>
<td>57 (40%)</td>
<td>19 (13%)</td>
<td>34 (24%)</td>
<td>30 (20%)</td>
</tr>
<tr>
<td>ADHD</td>
<td>3 (2%)</td>
<td>57 (40%)</td>
<td>19 (13%)</td>
<td>7 (5%)</td>
<td>5 (4%)</td>
<td>4 (3%)</td>
</tr>
<tr>
<td>Learning difficulties</td>
<td>16 (12%)</td>
<td>5 (4%)</td>
<td>4 (3%)</td>
<td>2 (1.5%)</td>
<td>68 (50%)</td>
<td></td>
</tr>
<tr>
<td>Visual impairment</td>
<td>32 (22%)</td>
<td>7 (5%)</td>
<td>7 (5%)</td>
<td>2 (1.5%)</td>
<td>68 (50%)</td>
<td></td>
</tr>
<tr>
<td>Specific sleep disorders</td>
<td>30 (21%)</td>
<td>5 (4%)</td>
<td>4 (3%)</td>
<td>2 (1.5%)</td>
<td>68 (50%)</td>
<td></td>
</tr>
<tr>
<td>Specific sleep disorder</td>
<td>16 (12%)</td>
<td>5 (4%)</td>
<td>4 (3%)</td>
<td>2 (1.5%)</td>
<td>68 (50%)</td>
<td></td>
</tr>
<tr>
<td>Carer respite</td>
<td>3 (2%)</td>
<td>5 (4%)</td>
<td>4 (3%)</td>
<td>2 (1.5%)</td>
<td>68 (50%)</td>
<td></td>
</tr>
<tr>
<td>EEG</td>
<td>2 (1.5%)</td>
<td>68 (50%)</td>
<td>124 (87%)</td>
<td>32 (22%)</td>
<td>7 (5%)</td>
<td>7 (5%)</td>
</tr>
</tbody>
</table>

Table 2 Dose of melatonin prescribed

<table>
<thead>
<tr>
<th>Dose prescribed</th>
<th>Median</th>
<th>Range</th>
<th>25–75% quartile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Starting dose (mg)</td>
<td>2.5</td>
<td>1.0–5.0</td>
<td>2.0–3.0</td>
</tr>
<tr>
<td>Lower maintenance dose (mg)</td>
<td>3</td>
<td>0.5–10.0</td>
<td>2.0–3.0</td>
</tr>
<tr>
<td>Higher maintenance dose (mg)</td>
<td>6</td>
<td>2.0–20.0</td>
<td>6.0–9.0</td>
</tr>
<tr>
<td>Maximum dose used (mg)</td>
<td>8</td>
<td>2.0–24.0</td>
<td>6.0–10.0</td>
</tr>
<tr>
<td>Immediate release</td>
<td>0.2–2 mg</td>
<td>2.1–3.0 mg</td>
<td>3.0 mg</td>
</tr>
<tr>
<td>Slow release</td>
<td>0.5 mg</td>
<td>6–9 mg</td>
<td>9 mg</td>
</tr>
<tr>
<td>Both</td>
<td>0–5 mg</td>
<td>34 (24%)</td>
<td>82 (58%)</td>
</tr>
<tr>
<td>Formulation of melatonin</td>
<td>89 (68.5%)</td>
<td>3 (2.3%)</td>
<td>38 (29.2%)</td>
</tr>
</tbody>
</table>
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. 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 (personal communication, Cath Mulholland, 2004).

Whether these “adverse events” represent a significant rise above events that would be expected in the population 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 and type of 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 progress. Rates of severe reactions have been described for open face (OFC) and correlated with a careful clinical history. Persistence of positive SPT is not a good predictor of language outcome that it compensates for many other variables including age of diagnosis.

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 determining 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 centres these may be very different and 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 went on: “I was angry, I was very angry, I don’t know I will ever get over the anger”.

Reference

Food challenge tests
Ewan and Clark’s helpful commentary provokes further comment on the diagnosis of allergy and the management of the allergic child. The issues raised are complex 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 and the lack of a gold standard 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 outcome. Science provides a real minefield. Severe and profound 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 and type of 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.

The issues raised are controversial and the balance in the wrong direction. Research studies like this one may help to tilt the balance in the wrong direction.
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 generalist’s 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 diseases.

The most important attribute of the book is its format. 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 contains 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, i.e., 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 of puberty it is not central to 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 anatomical and 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 summarising how 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


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

The recently published leading article on the National Service Framework (NSF) in Archives of Disease in Childhood by our ex-president of this college 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).

There are 18 chapters in the book in comparison to 11 standards set in the NSF for young people and maternity services. Of the 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 curriculum 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