Clinical assessment of neonatal hyperbilirubinaemia

The study by Keren and colleagues 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. 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 hyperbilirubinaemia.

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.

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. 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.

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

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 “interpersonal factors” such as 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 examination (miscategorisation 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 breastfeeding 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 et al 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 oxyhaemoglobin release predominantly between 2 and 12 hours but continues up to 48 hours. A, enzymatical 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 transformating capacity” is a rate limiting reaction, and when the concentration of oxyhaemoglobin rises rapidly, additional amounts are oxidised non-enzymatically to methaemoglobin.

Spectrophotometry similar for subarachnoid haemorrhage, confirming similar observations reported for subarachnoid haemorrhage and subdural haematomas. We concluded that while spectrophotometry of subdural fluids 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 from 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 disturbances. The dose of melatonin ranged from 2–6 mg nocte with a mean dose of 4.8 mg (SD 1.54). 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 declared

References

3. Peled N, Sharer Z, Peled E, et al. Melatonin effect on seizures in children with severe neurologic non-convulsive deficit disorders. 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 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 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.

Nutritional rickets is increasingly diagnosed in children of ethnic origin

We noted with interest the article published by Ladhani and colleagues highlighting the rise in the incidence of vitamin D deficiency. We agreed 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 Asian (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 aetiology. 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 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 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.

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 the problems involved in a contemporary child.

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

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.”1

However, to defuse the situation an affidavit signed by the parents of Peter Bell denying these rumours was published in the Northampton Mercury.4

Article from the Northampton Mercury, 10 January 1806

“Whereas a false and groundless report has been spread abroad 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 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)

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.”5

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 brought to the test of unprejudiced experience.”1

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

Doctors bequeathed 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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Competing interests: none declared

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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 projects a consistent 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 multicentre randomised studies with relevant outcomes measures6 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 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. Needlesticks containing 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 0.3%. 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 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. 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. With this population of children were included those who suffered injuries 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 who have 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 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 groups.

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” 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 (wholecell [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. Given that DTPa was introduced in 1999, 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 DTPw (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 also include a historical count 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 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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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-incharge” 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 because of 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 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.

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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.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 = 5), 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>
<thead>
<tr>
<th>Table 1 Responses to the questionnaire</th>
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<tbody>
<tr>
<td><strong>Response</strong></td>
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<tr>
<td>Prescribed melatonin</td>
</tr>
<tr>
<td>Yes (98%)</td>
</tr>
<tr>
<td>No (2%)</td>
</tr>
<tr>
<td>Median 14.4</td>
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<tr>
<td>Mean 3.2</td>
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<tr>
<td>Range 1-150</td>
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<tr>
<td>25-75th quartile 5.0-20</td>
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<tr>
<td>Disorders treated</td>
</tr>
<tr>
<td>Autism (68%)</td>
</tr>
<tr>
<td>ADHD (44%)</td>
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<tr>
<td>Learning difficulties 57 (40%)</td>
</tr>
<tr>
<td>Visual impairment 19 (13%)</td>
</tr>
<tr>
<td>Specific sleep disorders 7 (5%)</td>
</tr>
<tr>
<td>Indications for melatonin</td>
</tr>
<tr>
<td>Sleep onset difficulties 53 (39%)</td>
</tr>
<tr>
<td>Night waking 16 (12%)</td>
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<tr>
<td>Specific sleep disorder 5 (4%)</td>
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<tr>
<td>Carer respite 4 (3%)</td>
</tr>
<tr>
<td>EEG 2 (1.5%)</td>
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<tr>
<td>Non-specific sleep problems 68 (50%)</td>
</tr>
<tr>
<td>Measures prior to melatonin</td>
</tr>
<tr>
<td>Behavioural therapy/sleep hygiene 124 (87%)</td>
</tr>
<tr>
<td>Other medication 32 (22%)</td>
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<tr>
<td>Advice 7 (5%)</td>
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<tr>
<td>Other 7 (5%)</td>
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<table>
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<tr>
<th>Table 2 Dose of melatonin prescribed</th>
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<tbody>
<tr>
<td><strong>Immediate release</strong></td>
</tr>
<tr>
<td>Starting dose (mg) 2-5</td>
</tr>
<tr>
<td>Lower maintenance dose (mg) 3</td>
</tr>
<tr>
<td>Higher maintenance dose (mg) 6</td>
</tr>
<tr>
<td>Maximum dose used (mg) 8</td>
</tr>
<tr>
<td>0-2.0 mg 2.1-3.0 mg 3&gt; mg</td>
</tr>
<tr>
<td>Starting dose 63 (44%) 70 (49%)</td>
</tr>
<tr>
<td>Lower maintenance dose 42 (30%) 69 (48%) 31 (22%)</td>
</tr>
<tr>
<td>Higher maintenance dose 0-5 mg 6-9 mg 9 mg</td>
</tr>
<tr>
<td>Immediate release 89 (68.5%) 3 (2.3%) 38 (29.2%)</td>
</tr>
<tr>
<td>Slow release 89 (68.5%) 3 (2.3%) 38 (29.2%)</td>
</tr>
<tr>
<td>Both 89 (68.5%) 3 (2.3%) 38 (29.2%)</td>
</tr>
</tbody>
</table>

www.archdischild.com
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 (per-
sonal communication, Peter Stephens,
IMSHealth, 2004).

Reports of adverse events from our study
mirror those in the literature.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 (personal com-
mutation, Cath Mulholland, 2004). Whether these “adverse
events” represent a significant rise above
events that would be seen by chance in this population will need
whether these “adverse events” represent a
mortality 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.

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

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events associated with dietary supplements: an

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

I have read with great interest the original
article from Wake and colleagues1 and
would like to acknowledge and compliment
their valuable efforts in such a difficult
research area. I felt nevertheless quite con-
cerned with the conclusions of this study and
their possible repercussions. Diagnosis and
management of hearing impairment 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 NHS 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 their 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, consis-
tent use of hearing aids, cochlear implant,
age at start of other forms of intervention
such as speech and language therapy, educa-
tional input (type of specialist intervention
programmes, bilingual versus oral-only pro-
grammes), cognitive ability, parents’ hearing
status, parents acceptance and cooperation
with professionals...the list is enormous.

Only a study involving very large popula-
tions 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 possibil-
ity of most research centres.

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 pro-
grammes and since children came from
different areas and even vecors, 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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Food challenge tests

Ewan and Clark’s helpful commentary pro-
vokes further comment on the diagnosis of
allergy and the management of the allergic
child.1 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 across the
country, for some of the differences, but neither SPT or
RAST distinguish between sensitisation and
clinical allergy; scepticism about the mean-
ingfulness of test results will continue until
they are validated by objective methodology
(OFC) and correlated with a clinical
history. Persistence of positive SPT is not
always evidence for persistence of allergy2
and restriction of the OFC to the role of
confirming resolution of allergy as suggested
by Ewan and Clark will tend to decrease
suitable 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
controlled challenges.6 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.7 My own series of
patients with higher rates of reactions requir-
ing bronchodilator treatment included a high
proportion of asthmatic children and they
also received larger doses of allergen.3

Establishing the true presence of food
allergy is fundamental to clinical manage-
ment. 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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BOOK REVIEWS

Pediatrics

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 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 on to explain the symptoms and their management, this book 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 head ache 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 adolescent 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 condition, medical procedures, colour atases 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. We 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 and history, the approach 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 hypopituitarism 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

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, spiroimeters, 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). 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


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...