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

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

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Melatonin: prescribing practices and adverse events

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

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

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

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

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

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

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

Table 2 Dose of melatonin prescribed

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

Table 1 Responses to the questionnaire

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

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

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

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

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

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

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

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

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

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

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

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

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

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Food challenge tests

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

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