LETTERS TO THE EDITOR

Emergency management of meningococcal disease

EDITOR,—Pollard *et al* presented a comprehensive personal view on the emergency management of meningococcal disease.[1] I wish however to take issue with one point concerning lumbar puncture. Lumbar puncture should certainly be deferred in certain instances but should not be avoided as could be interpreted from the article. All children with suspected meningitis should, in my opinion, have a lumbar puncture at some stage in their illness. The reasons for lumbar puncture include:

- the presence or absence of meningitis should influence the choice and, perhaps, duration of antibiotic treatment
- the presence or absence of meningitis should influence fluid management once the initial shock is treated
- accurate anatomical diagnosis of meningitis is important for epidemiological purposes
- the presence or absence of meningitis is very relevant to neurodevelopmental prognosis and possible hearing impairment.

I increasingly meet paediatric trainees who seem to accept that a clinical and polymerase chain reaction based diagnosis of meningitis is sufficient. I would prefer if Pollard *et al* replaced (in the figure) the capitalised order DO NOT LUMBAR PUNCTURE (sic) with the instructions DEFER LUMBAR PUNCTURE and discuss its performance later in the illness.

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1 Pollard AJ, Britto J, Nadel S, DeMunter C, Habibi P, Levin M. Emergency management of meningococcal disease. *Arch Dis Child* 1999;**80**:290–6.

Dr Pollard and colleagues comment:

The role of lumbar puncture in the management of children with meningococcal disease deserves scrutiny, and an ongoing study by the Royal College of Paediatrics and Child Health will examine this issue (Ninis N, personal communication, 1999). We are, therefore, pleased that Professor Gill supports our avoidance of lumbar puncture in special circumstances (cardiorespiratory insufficiency or shock, raised intracranial pressure, and coagulopathy).

He mistakenly interprets our article as advocating the complete avoidance of lumbar puncture in all cases of meningitis. We do not consider lumbar puncture necessary in the emergency management of children presenting with the characteristic petechial/purpuric rash of meningococcal disease. Although other pathogens (*Haemophilus influenzae* type b and *Streptococcus pneumoniae*) may also cause a non-blanching rash, because of the potential risks involved in the critically ill child, and the possibility of rapid deterioration in those who appear well on first assessment, we stated that "lumbar puncture should probably be avoided or deferred in the initial assessment of all patients with clinically obvious meningococcal disease".

Early lumbar puncture is not only hazardous but may provide false reassurance as patients with meningococcal septicaemia may have no cerebrospinal fluid (CSF) changes on presentation, even though the organism can be cultured from the CSF sample. CSF changes may develop later and full neurological evaluation at follow up is mandatory in patients with septicaemia or meningitis. Our personal practice is to avoid lumbar puncture in meningococcal disease because we consider that the test adds little useful information to the clinical diagnosis, it could be misleading,[1 2] and does not affect clinical treatment. Alternative microbiological samples (blood cultures, throat swab, skin lesion aspirate) and molecular diagnostic techniques on blood are both essential and helpful in identifying the organism for epidemiological purposes and potentially for identification of antibiotic resistance.

Gill suggests that presence or absence of meningitis in meningococcal disease would influence the choice or duration of antibiotic treatment. We advocate use of a third generation cephalosporin in a child with meningococcal disease for seven days regardless of the predominant clinical syndrome for the reasons described in our article. Central nervous system infection commonly coexists with septicaemia[3] and does not require a unique approach to antibiotic treatment. Furthermore, accurate anatomical diagnosis of meningococcal meningitis does not provide useful epidemiological information, as the collection of separate data for meningococcal meningitis and septicaemia are obscured by the overlap between the two clinical syndromes.

Because of this overlap between meningitis and septicaemia, the emphasis in the acute stage of meningococcal disease presenting with shock, should be on maintaining an adequate mean blood pressure by volume resuscitation and inotropic support, thus ensuring adequate cerebral perfusion pressure. When clinically apparent raised intracranial pressure is present, correction of coexistent shock, followed by cautious fluid management and measures to reduce intracranial pressure are necessary. In children without features of shock or raised intracranial pressure, fluid restriction in the management of meningitis in children has been widely advocated but has been challenged and may even have an adverse effect on outcome.[4 5]

1 Felgenhauer K, Kober D. Apurulent bacterial meningitis (compartmental leucopenia in purulent meningitis). *J Neurol* 1985;**232**:157–61.
2 Onorato IM, Wormser GP, Nicholas P. "Normal" CSF in bacterial meningitis. *JAMA* 1980; **244**:1469–71.
3 Marzouk O, Thomson AP, Sills JA, Hart CA, Harris F. Features and outcome in meningococcal disease presenting with maculopapular rash. *Arch Dis Child* 1991;**66**:485–7.
4 Duke T. Fluid management of bacterial meningitis in developing countries. *Arch Dis Child* 1998;**79**:181–5.
5 Singhi SC, Singhi PD, Srinivas B, *et al*. Fluid restriction does not improve the outcome of acute meningitis. *Pediatr Infect Dis J* 1995;**14**: 495–503.

Recommendations for the management of galactosaemia

EDITOR,—We were pleased to see the publication on behalf of the UK Steering Group "Recommendations for the management of galactosaemia".[1] In particular, we were pleased to see the emphasis on the management of adult women and the prevention of osteoporosis. We have, however, some concerns about the advice on the use of Loestrin 20 (an oral contraceptive preparation containing 20 μg ethinyl oestradiol and a progestogen) for long term oestrogen replacement.

None of the combined oral contraceptive pills, such as Loestrin 20, are licensed for the prevention of osteoporosis, although until the more widespread use of hormone replacement therapy (HRT) preparations, many were widely used for this purpose. In addition to providing oestrogen, they have the advantage of being without prescription charge and are widely accepted, particularly by young adults.

There are disadvantages, however. The main one is the duration of therapy. Women taking combined oral contraceptive preparations receive only three weeks oestrogen out of four, which being extrapolated means that they receive 30 years replacement instead of 40. In women who are producing no oestrogen of their own, this difference may be important. We are also concerned that ethinyl oestradiol, as contained in combined oral contraceptive preparations, is not detected by standard hormone assays. Monitoring of oestrogen replacement is, therefore, dependent on suppression of follicle stimulating hormone and luteinising hormone, which does not allow for appropriate adjustment of oestrogen levels and may result in the woman receiving inadequate oestrogen. Women taking the combined oral contraceptive pill are also exposed to progestogens for longer in the cycle (21 days rather than 12 days) than women on HRT. In some cases, although not with the recommended Loestrin 20, this may also be at higher doses. Progestogens are reported to adversely affect the lipid profile in women receiving oestrogen replacement.

There are particular reasons for advocating the use of HRT rather than combined oral contraceptives in women with galactosaemia. Although it is recognised that the dose of lactose in combined oral contraceptive preparations is very small, it may be unacceptable to some patients. One method of delivery of HRT is via the transdermal patch, which avoids the ingestion of any exogenous lactose.

For these reasons, we believe that oestrogen replacement in the form of HRT preparations are preferable to combined oral contraceptive preparations in the long term management of women with galactosaemia.

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1 Walter JH, Collins JE, Leonard JV, on behalf of the UK Galactosaemia Steering Group. Recommendations for the management of galactosaemia. *Arch Dis Child* 1999;**80**:93–6.

Planning for major incidents involving children by implementing a Delphi study

EDITOR,—The proposed paediatric triage algorithm in Mackway-Jones *et al*'s study[1] has a number of important flaws:

(1) few children younger than 10 months are ambulatory
(2) there is no airway opening manoeuvre
(3) capillary refill time is affected by the ambient temperature; refill time measured at the sternum and forehead

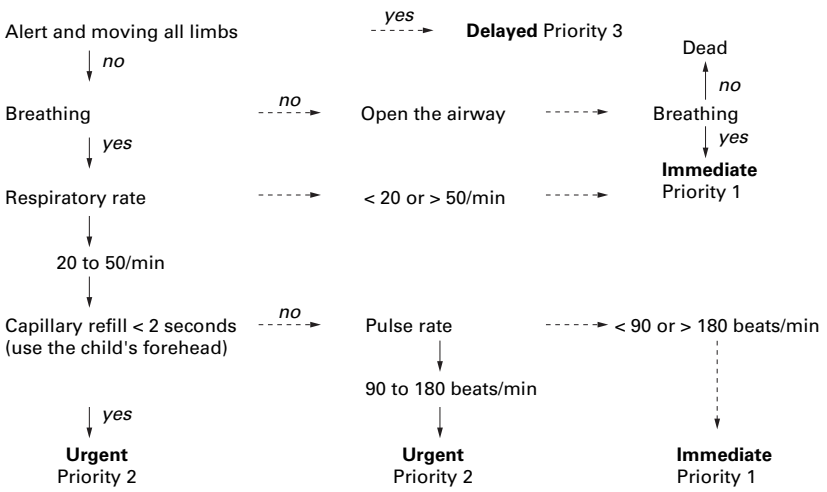

Figure 1 Paediatric triage tape 50–80 cm.

only have a Gaussian distribution.[2] To rely solely on the capillary refill time increases the number of priority 1 (immediate) children especially in cold surroundings

(4) the paediatric trauma score (even with the Eichelberger modification) is inappropriate as the systolic blood pressure is required. The ability to measure it at the incident, the need for different cuff sizes, and the time it takes when faced by a large number of casualties rule this score out

(5) there is no account made for the change in physiological parameters with age.

We have devised a triage system (on a waterproof tape) that overcomes these problems.[3] The changes in normal physiological values with age are shown by dividing the tape into 4 compartments based on body length or weight (50–80 cm or 3–10 kg; 80–100 cm or 11–18 kg; 100–140 cm or 19–32 kg; and > 140 cm or > 32 kg). A child > 140 cm is triaged as an adult.

Each compartment has a triage sieve algorithm corrected for age with the 5th and 95th centiles for respiratory rate and heart rate from all available published literature stated. Figure 1 shows the values for the 50–80 cm compartment.

Triage is a dynamic process and starts in the prehospital setting. Appropriate prioritisation allows limited resources to be diverted to needy children.

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1 Mackway-Jones K, Carley SD, Robson J. Planning for major incidents involving children by implementing a Delphi study *Arch Dis Child* 1999;**80**:410–18.
2 Maconochie I. CRT-it's enough to make you blush! *Pre-hospital Immediate Care* 1998;**2**:95–7.
3 Hodgetts TJ, Hall J, Maconochie I, Smart C. Paediatric triage tape *Pre-hospital Immediate Care* 1998;**2**:155–9.

Pacifier use and SIDS

EDITOR,—Fleming and colleagues state the protective association between pacifiers and cot death is probably real, but feel recommendations on its use cannot be made.[1] Is the association causal? There are several hypotheses that might provide a biological mechanism, and thus strengthen this supposition. Having a pacifier might prevent turning prone face straight down. Or perhaps pacifiers facilitate switching to mouth breathing if nasal occlusion occurs.[2] Both mechanisms would explain why only pacifier use on the last occasion is protective. Usually using a pacifier would then only be significant if highly correlated with use in reference sleep. A dose–response effect of pacifiers could only be expected if the underlying mechanism would need repeated use to be effective—for example, through repetitive sucking which would increase muscle tone and thus oropharyngeal patency. A dichotomy between never or only rarely using a pacifier and using it always or often would then be more logical than the study's dichotomy ever/never.

Studies on the risks and benefits of pacifiers are hampered by the issue of reverse causality. Do pacifiers increase the risk of otitis media? Or do mothers try to soothe their infant with a pacifier when it suffers from (recurrent) otitis? Does pacifier use have an adverse effect on breast feeding? Or is it a marker for breast feeding difficulties or an attempt to wean the baby? A definitive answer can only be given by randomised trials where pacifiers are introduced at a set time, but clearly these are not easy to carry out. It may not be possible to postpone a decision on pacifiers until such trials are conducted, if indeed they ever will be. Surely the major potential disadvantage of pacifier use is its effect on breast feeding. This needs to be explored further. With current knowledge we would think, however, that using a pacifier can be recommended actively for infants that are bottle fed only.

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1 Fleming PJ, Blair PS, Pollard K, *et al*. Pacifier use and the sudden infant death syndrome: results from the CESDI/SUDI case control study. *Arch Dis Child* 1999;**81**:112–16.
2 Swift PGF, Emery JL. Clinical observations on response to nasal occlusion in infancy. *Arch Dis Child* 1973;**48**:947–51.

Use of duvets and SIDS

EDITOR,—In the paper by Mitchell *et al* the use of duvets was associated with sudden infant death syndrome (SIDS) on univariate (odds ratio 1.65 (95% confidence interval 1.31, 2.08)) but not multivariate analysis, and the SIDS risk associated with duvet use was not modified by sleeping position.[1] These findings contrast with our finding of a strong association (adjusted odds ratio 6.16 (2.01, 18.87)) between quilt (duvet) use and SIDS among non-prone infants.[2]

Mitchell *et al* postulated that one reason for the discrepancy may be that our Tasmanian study had not controlled for socioeconomic factors. We did, in fact, adjust for a large number of additional potential confounders that could not be listed in the short report due to space limitations. We found that adjustment for unemployment, maternal education, or maternal parity did not alter the association between duvet use and SIDS among non-prone infants. We agree that the conflicting findings may relate to local differences in duvet characteristics, although alternative explanations may also contribute.

One possibility is non-differential misclassification in that the New Zealand study asked a single question to determine exposure whereas the Tasmanian study employed visual verification of the actual bedding items for cases and controls by interviewer where possible. From 1991 to 1995 this also included a sample of different types of duvets, which the nurse took to the home interview to assist with classification. As non-differential misclassification will bias an association based on dichotomous exposures towards or beyond the null,[3] this may explain the weaker strength of association between duvet use and SIDS in the New Zealand study compared with ours.

It is also critically important not to adjust for any factor that may be on the causal pathway between exposure and disease, as this will lead to an underestimate of the true association.[4] A classic example is adjustment for birth weight when examining any association between maternal smoking and infant mortality. This is clearly inappropriate as the adverse effect of smoking is partially mediated through birth weight.[4]

The causal pathways between duvet use and SIDS are less clear but our data indicated that part of the adverse effect resulted from facial obstruction.[2] Thus, it is not surprising that little adverse effect remained for duvet use on SIDS in the UK study after adjustment for a large number of factors, including head covered during last sleep, and the authors correctly pointed out that duvet use appeared to increased the risk of SIDS partially through a propensity for total covering.[5]

Mitchell *et al* report that duvet use was inversely associated with being tucked in firmly, a protective factor for SIDS. They included "firm tucking in" as a confounder in their analyses, and thus report adjusted odds ratios for duvet use that reflect only the residual effect of duvet use on SIDS, excluding any adverse effect that is actually meditated through looser bedclothes. To rely on these adjusted results may underestimate the true association between duvet use and SIDS.

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1 Mitchell EA, Williams SM, Taylor BJ. Use of duvets and the risk of sudden infant death syndrome. *Arch Dis Child* 1999;**81**:117–19.
2 Ponsonby A-L, Dwyer T, Couper D, Cochrane J. Association between use of a quilt and sudden infant death syndrome: case-control study. *BMJ* 1998;**316**:195–6.
3 Rothman KJ, Greenland S. *Modern epidemiology.* Philadelphia: Lippincott Raven, 1998:128–9.
4 Leon DA. Failed or misleading adjustment for confounding. *Lancet* 1993;**342**:479–83.
5 Fleming PJ, Blair PS, Bacon C, *et al*. Environment of infants during sleep and risk of the sudden infant death syndrome: results of 1993–5 case-control study for confidential inquiry into stillbirths and deaths in infancy. *BMJ* 1996;**313**:91–5.

Dr Mitchell and colleagues comment:

Our results have similarities and differences with those from the Tasmanian study. We have both shown that the risk of SIDS from thermal stress is only among infants sleeping prone.[1 2] Given that there is a positive relation between duvet use and excess thermal insulation, we were surprised that the Tasmanian study subsequently found that duvet use increased the risk of SIDS only among infants sleeping supine.[3]

We suggest several explanations for the difference between the studies:

- the characteristics of the duvets differed between New Zealand and Tasmania
- the Tasmanian study did not adjust for confounders; their letter indicates that they did, but this was not reported in their paper.

Ponsonby and colleagues suggest two additional explanations:

- non-differential misclassification
- adjustment for "firm tucking in" is inappropriate as it may be on the causal pathway.

Misclassification is unlikely; although we used a simple question to determine whether a duvet was used it is unlikely that parents would mistake its use. Furthermore fewer than 2% of cases and controls did not answer this question.

To exclude the possibility that we inappropriately included a factor in the causal pathway we have rerun the multivariate analysis without "firm tucking in". The risk of SIDS with duvet use remains insignificant (adjusted odds ratio 1.18 (95% confidence intervals 0.87, 1.60) compared with "variable included" adjusted odds ratio 1.04 (0.77, 1.58)). Our impression is that duvets in New Zealand are larger than those in Australia and are tucked in at the foot of the cot. This may reduce the possibility of the duvet covering infants' heads. In our study there was no increased risk of being found with head covered when using a duvet compared with those who were not using a duvet.

We conclude that these explanations do not account for our findings and that our study does not support the recommendation to avoid duvets.

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2 Ponsonby A-L, Dwyer T, Gibbons LE, Cochrane JA, Wang YG. Factors potentiating the risk of sudden infant death syndrome associated with the prone position. *N Engl J Med* 1993;**329**:377–82.
3 Ponsonby A-L, Dwyer T, Couper D, Cochrane J. Association between use of a quilt and sudden infant death syndrome: case-control study. *BMJ* 1998;**316**:195–6.

Examination of children who may have been sexually abused

EDITOR,—We were surprised to read of the letter from Hodes *et al* justifying a second examination of three girls suspected of being sexually abused.[1] The triplets were presumably prepubertal? Were the photographs taken by the first paediatrician inadequate, and if so, why? A clinical diagram complemented by good quality photography usually provides adequate documentation, especially for prepubertal girls. Although the girls were compliant and the doctors no doubt sensitive, children do not like being examined and three paediatricians, the girls' mother, and a nurse (recommended by the General Medical Council), and in some areas, a policewoman, suggests an overcrowded examination room.

Colposcopy with integral photography has improved the quality of recording and the photographs are part of the casenotes. Discussion of individual cases by a peer group is well established, and slides or other recorded images are an essential part of this process. They may be used to detect subtle changes when a follow up examination is performed later. They have been shown to assist in differential diagnosis—for example, healing trauma versus evolution of a disease process. The doctor can never guarantee that the photograph will only be used for clinical purposes and teaching. The court has the power to direct that the slides are made available, hopefully to a named paediatrician.

We have used a colposcope mounted video camera with remote television monitor to allow trainees to observe the examination from an adjacent room with consent from parents and children as appropriate. The use of a one-way screen also enables the child's demeanour to be observed during the interview phase of the consultation.

Pretrial meetings clearly have a place in assessing medical evidence, but re-examination, even if the children are well prepared, always needs justification.

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1 Hodes D, Larcher V, Watkeys A. Pretrial liaison between doctors in alleged child abuse [letter]. *Arch Dis Child* 1999;**81**:189.

Dr Hodes and colleagues comment:

We agree that, as a general rule, re-examination should be avoided, but would argue that there are some circumstances in which it may be in a child's best interest. We accept that such circumstances will be controversial and require justification.

The triplets were prepubertal and although the still photographs were adequate there were different opinions at the peer review group as to their interpretation. As we stated, most differences could not be resolved by discussion alone. We did debate the ethical aspects of re-examination in detail because there may be harm to children in this process. However, had there been any evidence of discomfort or distress to the children we would not have proceeded with the examination. We did respect their wishes and a fair decision concerning their placement was possible. On balance, the overall benefits outweighed the harm.

It is well known that examination of the genitalia is a dynamic process and we accept that a videorecording is the gold standard that permits evaluation of changes in hymen configuration. However, not all units have access to video colposcopy facilities so disputes will occur over interpretation of still photography.

We offer review appointments to children and families after medical examinations to clarify understanding of the findings and their significance. In general the response to the experience of examination has been positive, as was indeed the ease with the three children we described.

We are sure that debate in this controversial area will continue.

Prevalence of bruising in babies

EDITOR,—Carpenter has written an important study on prevalence and distribution of bruising in babies.[1] However, there are problems with the terms relating to measures of disease frequency in epidemiology which, arguably, make this descriptive paper misleading.

The terms *point prevalence*, *prevalence proportion*, *prevalence ratio*, and *prevalence rate* are sometimes used to mean the same thing.[2] However, the word *rate* is used more specifically in the context of the number of events per unit time and intuitively suggests a survival rate or an incidence rate. This is strongly purported by the use of the term *rate parameter* to describe the unknown and estimated value associated with a Poisson probability model.[3] The distinction is often not explicit, even in the best textbooks of epidemiology, but I think that it is worth making.

Carpenter's study included infants aged between 6 and 12 months who were opportunistically screened for bruising at the time of routine surveillance checks. It is implied that each child was examined on only one occasion. Bruises were found in 22 infants, and seven had more than one bruise. However, a bruise would be expected to be visible for fewer than 28 days. If we take even this liberal estimate of duration, the infants were observed for approximately 1/6th of the period during which they might be found to have one or more bruises.

Hence, use of the term *point prevalence* in place of *prevalence rate* would have made things clearer, as it emphasises the fact that measurements were made at a single time between the ages of 6 and 12 months—the stated age range of study participants. The appropriate measure of risk depends on the question being asked. If we want to know the probability of a bruise being present at a single, random visit during the second 6 month period after birth, the point prevalence of 12% (0.12) is a useful measure of risk. If, however, we are asking for the probability that a child will develop one or more bruises with regular surveillance during the second 6 month period after birth, the appropriate risk is a *cumulative rate*, which can be derived from an *incidence rate*.

We can use the point prevalence presented in Carpenter's paper and our assumptions about duration to calculate a cumulative incidence from the relation:

$$\text{incidence rate} = \frac{\text{(point prevalence)}}{\text{(duration)}}.$$

The cumulative incidence is obtained from the formula:

$$\text{cumulative incidence} = (1 - \exp(-(\text{incidence rate}) \times (\text{number of periods}))).$$

Assuming that bruising occurs at a constant rate and that the probability of a child having a bruise at one age is independent of the probability of it having a bruise at another age—assumptions that are probably not justified but which excuse a simpler model—the cumulative rate is $(1 - \exp(-0.12 \times 6)) = 0.51$ (that is, approximately 1 in 2). If we assume that a bruise lasts only two weeks and calculate the risk from the 12% point prevalence and 12 time periods, the risk becomes 0.76. It is worth being clear about which risk figure we are going to carry around in our heads and for which purpose: 1 in 8, or 1 in 2, or 3 out of 4.

As bruising is more common in infants who are more mobile, the incidence rate of bruising will increase with age. The age distribution of the sample would thus be important in the calculation of even a summary "average" measure of risk, and mention of risk estimates in appropriate age strata would provide useful information.

Carpenter suggests in the abstract that his study "tested out the methodology which might be used in future research". I hope that these aspects of terminology and study design will be considered in such future studies.

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1 Carpenter RF. The prevalence and distribution of bruising in babies. *Arch Dis Child* 1999; **80**:363–6.
2 Rothman KJ, Greenland S. *Modern epidemiology*. 2nd ed. Philadelphia: Lippincott Raven, 1998.
3 Clayton D, Hills M. *Statistical models in epidemiology*. Oxford: Oxford University Press, 1993.

Dr Carpenter comments:

First, I thank Dr Lux for pointing out that I should have used the term *point prevalence* rather than *prevalence rate*.

Second, my figure (12.4% or 1 in 8) is the one "to carry in our heads". Although it is possible to calculate the cumulate index over a 6 month period, as he suggests, this is not relevant for the clinical situation at presentation when abuse could be considered.

Finally, the study showed that age was barely significant when looking at bruises ($p = 0.05$) whereas mobility was significant ($p < 0.001$, see also table 1). Therefore, age cannot be used as a proxy for mobility and so risk estimates for age would not be helpful.

Adolescent inpatient units

EDITOR,—Although separate dedicated medical inpatient adolescent units have been advocated[1] there is little information on their availability in UK. We report the prevalence of adolescent medical inpatient facilities in England and Wales.

We performed a two stage survey between March and September 1998. In stage I we telephoned all hospitals with paediatric departments in England and Wales to ascertain the provision of inpatient adolescent facilities. In stage II we sent postal questionnaires to hospitals reporting separate inpatient facilities. Factors determined included provision of separate ward or designated bay, number of available beds, groups of patient served, visiting times, and presence of multidisciplinary input for adolescents.

All 225 hospitals surveyed supplied baseline information. Fifty nine (26%) had separate medical inpatient facilities, of which 49 (83%) responded to the written questionnaire. Sixteen hospitals had a separate adolescent ward. Seven of these were in university hospitals; five were specialist oncology units. The other nine units were in district general hospitals and catered for all medical specialities. The remaining 33 units had a designated bay for adolescents. The number of beds in the adolescent wards ranged from 3 to 19 (median 6) while the number of beds in designated adolescent bays ranged from 4 to 12 (median 8). Thirty nine of 49 units had a multidisciplinary policy and 29 had nurses with an interest in adolescent care. The age for admission ranged from 11 to 23, but only seven units took patients over 17.

The justification for adolescent inpatient units is based on catering for the unique developmental and psychosocial needs of adolescents, such as independence, peer contact, privacy, and educational opportunity.[2 3] Teenagers may prefer an adolescent based service.[4] Only a quarter of hospitals in England and Wales have dedicated facilities for adolescents, mostly dedicated bays. There was no geographical pattern and no relation to size of hospital. It is therefore likely that the provision of adolescent inpatient facilities is dependent on other factors such as funding and the presence of interested nurses and clinicians. Neither paediatric nor adult medical specialist training curriculum stipulates adolescent exposure and there may be concern over trainees' exposure to adolescent medicine. We believe that the needs of many adolescent patients are unmet and dedicated adolescent facilities should be increased.

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1 Fisher M. Adolescent inpatient units. *Arch Dis Child* 1994;**70**:461–3.
2 Denholm CJ, Ferguson RV. Strategies to promote developmental needs of hospitalised adolescents. *Child Health Care* 1987;**15**:183–7.
3 Battle CV, Kreisberg RV O'Mahoney K, Chitwood DL. Ethical and developmental considerations in caring for hospitalised adolescents. *J Adolesc Health Care* 1989;**10**:479–89.
4 Jones R, Finley F, Simpson N, Kreitman T. How can adolescents' health needs and concerns be met. *Br J Gen Pract* 1997;**47**:631–4.

Transition from paediatric to adult care. Bridging the gaps or passing the buck?

EDITOR,—We read with interest the article by Viner regarding transition from paediatric to adult care.[1] The need for planned transition is indeed very real[2] and its recognition led us to develop a specific service for young people aged 16 to 25 with physical disability—the Young Adult Team. This multidisciplinary team (doctor, physiotherapist, occupational therapist, speech and language therapist, psychologist, and social worker) was established in 1988 through joint funding from health and social services with the aim of increasing the young person's autonomy while addressing parental concerns.[3] It works in conjunction with the rehabilitation medicine physician and has strong links with paediatric, adult health, education, and social services. Nationally there are similar services run by members of the British Society of Rehabilitation Medicine. The impact and cost effectiveness of this type of intervention is currently the subject of a National Health Service research and development funded controlled study comparing organised transitional services and ad hoc services.

Viner suggests that the most suitable professional for transitional arrangements is a nurse specialist. While this may be appropriate for young people with conditions such as diabetes, the needs of young people with complex physical disability resulting from neurological disease are likely to be best met through a multidisciplinary approach of which the Young Adult Team is an example.

We agree that disease specific combined paediatric–adult clinics can facilitate the transitional period. However disabled young people with various diagnoses often have issues in common such as those relating to life skills, which are often independent of the disease process. These are likely to be best addressed through generic young adult services. Transitional services must acknowledge the need for disabled young people to learn how to monitor their own condition and how to access help. In Leeds we have had jointly run (paediatrician and adult physician) arthritis[4] and cerebral palsy clinics for several years. These clinics have direct links with the Young Adult Team and other adult services. It is important however to recognise that services solely organised around diagnoses may exclude vulnerable young people. Therefore transitional services should be inclusive and developed using both approaches and not one approach solely to the exclusion of the other. By doing so (although we await the evidence!) we believe that the needs of young people and their parents will be best served.

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1 Viner R. Transition from paediatric to adult care. Bridging the gaps or passing the buck? *Arch Dis Child* 1999;**81**:271–5.
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Iron fortified follow on formula from 9 to 18 months improves iron status but not development or growth

EDITOR,—I enjoyed reading the paper by Morley *et al*, which provides evidence for two things that I have long suspected.[1] First, you cannot make children smarter by putting more iron in their milk, and second that I am

the only person who has ever read any of my own publications. The authors say that Stevens and Nelson[2] found that formula milk reduced the incidence of iron deficiency anaemia whereas the study that was designed to look at the effect of iron in formula milk provided no evidence at all to justify this statement. There was no evidence that formula milk was responsible for the low incidence of iron deficiency anaemia in the children who were studied and no evidence that iron in formula milk was an important source of dietary iron for these infants.

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1 Morley R, Abbott R, Fairweather-Tait S, MacFadyen U, Stephenson T, Lucas A. Iron fortified follow on formula from 9 to 18 months improves iron status but not development or growth: a randomised trial. *Arch Dis Child* 1999;**81**:247–52.
2 Stevens D, Nelson A. The effect of iron in formula milk after 6 months of age. *Arch Dis Child* 1995;**73**:216–20.

Dr Morley and colleagues comment:

We apologise for misquoting Stevens' paper; this was an editing error when we amalgamated two papers. The reference for the statement "Iron fortification of milk formula . . . has been shown to reduce the incidence of iron deficiency anaemia" should have been: Moffatt ME, Longstaffe S, Besant J, Dureski C. Prevention of iron deficiency and psychomotor decline in high risk infants through use of iron fortified formula: a randomised trial. *J Pediatr* 1994;**125**:527-34.

The iron content of the three milks was also misquoted and should have been: cows' milk 0.5 mg/litre; iron fortified formula 12 mg/litre; unfortified formula 0.9 mg/litre. This correction strengthens rather than weakens our conclusions.

Estimating the genetic potential in stature

EDITOR,—Midparental height is an important measure in estimating a child's target height—the genetic potential in stature. Height reference values that allow for parental height are more appropriate for growth evaluation in paediatric clinics. We read with interest the recent paper by Wright and Cheetham on the strengths and limitations of parental heights as a predictor of attained height.[1] The authors concluded that midparental height was a useful indicator of the expected height for children when their parents were of average stature but misleading when used to assess short children. We have recently reported the same findings based in 2402 Swedish children.[2] We observed that the regression coefficient between midparental height and a child's final height was approximately 0.6 in standard deviation scores (it was 0.5 for children 8 years of age in the paper by Wright and Cheetham).

We believe that the linear function of midparental height could be used to estimate a child's target height, rather than midparental or corrected midparental height, which Wright and Cheetham implicitly used to represent a child's genetic target height. The meaning of midparental height is different for children with short, average, and tall parents. The parents' heights not only reflect the parents' genotype in stature, but also mirrors the extrinsic influences the parents experienced during their own growth span. This provides a biologically meaningful explanation of the so called "regression to the mean phenomenon". For instance, the intrinsic genetic potential in stature of short parents is usually much greater than their measured heights; consequently, the following generation is usually taller due to a better manifestation of the intrinsic growth potential.[2]

We agree that short children attending paediatric clinics are usually shorter than their target height, whatever method is used for estimation. The height of parents is important for clinical evaluation of short children. A short child with tall parents is certainly more likely to have a pathological cause than a short child of short parents. It is not appropriate to consider midparental height itself as a simple measure of target height. Clearly, midparental height is not misleading for any child if its linear function is used for estimating a child's target height—the genetic potential in stature.

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1 Wright CM, Cheetham TD. The strengths and limitations of parental heights as a predictor of attained height. *Arch Dis Child* 1999;**81**:257–60.
2 Luo ZC, Albertsson-Wikland K, Karlberg J. Target height as predicted by parental heights in a population-based study. *Pediatr Res* 1998; **44**:563–71.

IGFBP-3 as a predictor of growth hormone deficiency

EDITOR,—We read with interest the paper by Mitchell and colleagues[1] and wish to add our own observations on this subject. In 1996 the Regional Endocrine Laboratory started to provide a service for the measurement of insulin-like growth factor binding protein (IGFBP-3) following early reports that this was a good marker of growth hormone secretion. We then undertook a retrospective audit of the measurement of serum insulin-like growth factor (IGF-1) and IGFBP-3 as predictive markers of growth hormone deficiency (GHD) in children undergoing growth hormone stimulation tests (glucagon and insulin tolerance tests). Between October 1996 and January 1998, 93 children had simultaneous measurements of IGF-1 and 78 children had measurements of IGFBP-3. We defined GHD as a peak growth hormone level of < 20 mU/litre and complete GHD as a peak < 10 mU/litre in response to a stimulation test.

The results for IGF-1 and IGFBP-3 were compared to reference ranges for age available in the laboratory and classified as low or normal. The reference range for IGF-1 was constructed by the laboratory using their own assay and that for IGFBP-3 being supplied by the manufacturers of the kit (Nichols Institute, San Juan Capistrano, California, USA). We calculated their sensitivity and specificity as predictors of GHD using the two different cut off levels and the likelihood ratio—that is, the likelihood that the result would be seen in someone with as opposed to someone without GHD (table 1).

Eight children had both a low IGF-1 and IGFBP-3, which produced a sensitivity of 22.2% and specificity of 90.4%, with a likelihood ratio of 2.3 in predicting GHD. Therefore the combination of a low IGF-1 and low IGFBP-3 would be highly suggestive of GHD, but a significant number of children with GHD will have normal values for either of these two markers.

Table 1 Sensitivity and specificty of IGF-1 and IGFBP-3 in predicting growth hormone (GH) deficiency

	Peak GH < 10 mU/l	*Peak GH < 20 mU/l*
IGF-1		
Sensitivity	37.5%	29.5%
Specificity	79.7%	79.6%
Likelihood ratio	1.85	1.5
IGFBP-3		
Sensitivity	31.5%	27.8%
Specificity	76.3%	76.2%
Likelihood ratio	1.33	1.2

Thus it can be seen that a single measurement of IGFBP-3 performed no better than IGF-1 as a marker of growth hormone secretion despite previous claims. Neither marker had a high likelihood ratio and would therefore not be good as a single predictive test. Although we realise that some of the normal IGFBP-3 results could have resulted from the presence of IGFBP-3 protease activity interfering with the assay in children with radiation induced GHD this is not likely to alter our findings significantly.

Thus we agree with Mitchell *et al* and other authors[2] that IGFBP-3 measurements are not good predictive markers of growth hormone secretion and do not replace the need for careful clinical evaluation and growth hormone stimulation tests in short, slowly growing children.

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1 Mitchell H, Dattani MT, Nanduri V, Hindmarsh PC, Preece MA, Brook CGD. Failure of IGF-1 and IGFBP-3 to diagnose growth hormone insufficiency. *Arch Dis Child* 1999;**80**:443–7.
2 Tillmann V, Buckler JM, Kibirige MS, *et al*. Biochemical tests in the diagnosis of childhood growth hormone deficiency. *J Clin Endocrinol Metab* 1997;**82**:531–5.

Raised serum transaminases: not always liver disease

EDITOR,—Too often, the pursuit of detailed investigation supersedes clinical suspicion and decision making. A 3 year old boy was referred to our service for investigation of chronic liver disease. The patient was reported to be a well child, whose development was "within normal limits"; a 2 cm hepatomegaly was found during an admission for a chest infection. Subsequent investigations revealed normal serum bilirubin, γ glutamyl transpeptidase, alkaline phosphatase, and albumin. The only abnormality was a persistently raised alanine aminotransferase (507 IU/litre) and it was this that prompted referral to a liver centre.

Retrospectively it became apparent that the boy had some motor delay, having first walked at the age of 2 years. On clinical examination he was mildly hypotonic and demonstrated a positive Gower's sign. In view of this and the isolated increase in alanine aminotransferase, serum creatinine kinase measurement was requested to determine whether the origin of the transaminase was in fact muscle. The serum creatinine kinase was severely raised at 22 000 μmol/litre and the

boy was diagnosed with muscular dystrophy. His liver, which was not enlarged, was palpable probably because of visceroptosis seen on ultrasound scan.

We see two to three cases a year of muscular dystrophies masquerading as liver disease. This phenomenon has been described in a series of five male patients with raised serum alanine aminotransferase in whom signs and symptoms of hepatic disease were absent but evidence of neuromuscular dysfunction was detectable on clinical examination.[1] A further case of muscular dystrophy has also been diagnosed in a child with coeliac disease and persistently raised alanine aminotransferase.[2] Thinking of alternative sources of alanine and aspartate aminotransferase can help avoid such clinical pitfalls and spare families the anxiety and trauma of unnecessary investigations and delays in diagnosis, which may have prognostic implications.

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1 Zamora S, Adams C, Butzner JD, Machida H, Scott RB. Elevated aminotransferase activity as an indication of muscular dystrophy: case reports and review of the literature. *Can J Gastroenterol* 1996;**10**:389–93.
2 Stenhammer et al. Muscular dystrophy misdiagnosed as hepatic disease in a child with coeliac disease. *Acta Paediatr* 1995;**84**:707–8.

New developments in the treatment of cardiac failure

EDITOR,—The article by Westaby *et al* presented concisely recent developments in the management of infants and children with congestive cardiac failure.[1] We would like to highlight the following additional points.

A better understanding of pathophysiology has shifted medical management from steps that directly improve myocardial function to those that modulate the neuroendocrine profile and peripheral vascular reactivity. Similar advances in therapeutic applications would be assisted by controlled studies and full licensing of drugs for use in children. Medical intervention will remain the cornerstone of management until advances in surgical techniques become more widely available.

Although digoxin does not improve survival it provides symptomatic relief and reduces hospital admissions for exacerbations.[2] Loop diuretics lose efficacy over time; this "breaking phenomenon" can be overcome by combination with metolazone, producing sequential segmental nephron blockade.[3] The recently published results of RALES (randomized aldactone evaluation study)[4] have shown significant survival benefits from the use of spironolactone, an aldosterone receptor antagonist when used with an angiotensin converting enzyme inhibitor and loop diuretic. This combination necessitates careful monitoring for hyperkalaemia, but reduces the need for oral potassium supplements, which have a bitter taste and are poorly accepted by children. Compliance with medication can be enhanced by assisting the family to choose the "best-fit" regimen (concordance).[5]

Attention to psychological problems arising from the restricted lifestyle and frequent diagnostic and therapeutic interventions can improve prognosis and outcome. Additionally, young children may not understand the benefits of treatment, and adolescents may exhibit independence or denial. However, despite many limitations the prognosis for children with severe heart failure has significantly improved over the past decade.[6 7]

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1 Westaby S, Franklin O, Burch M. New developments in the treatment of cardiac failure. *Arch Dis Child* 1999;**81**:276–7.
2 The Digitalis Investigation Group. The effect of digoxin on mortality and morbidity in patients with heart failure. *N Engl J Med* 1997;**336**:525–33.
3 Wells TG. The pharmacology and therapeutics of diuretics in the pediatric patient. *Pediatr Clin North Am* 1990;**37**:463–504.
4 Pitt B, Zannad F, Remme WJ, *et al*, for the Randomized Aldactone Evaluation Study Investigators. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. *N Engl J Med* 1999;**341**:709–17.
5 Bradley C. Compliance with drug therapy. *Prescribers' Journal* 1999;**39**:44–9.
6 Burch M, Siddiqi SA, Celermajer DS, Scott C, Bull C, Deanfield JE. Dilated cardiomyopathy in children: determinants of outcome. *Br Heart J* 1994;**72**:246–50.
7 Venugopalan P, Agarwal AK, Akinbami FO, El Nour IB, Subramanyan R. Improved prognosis of heart failure due to idiopathic dilated cardiomyopathy in children. *Int J Cardiol* 1998;**65**:125–8.

Dipstick examination for urinary tract infections

EDITOR,—Recently dipsticks using nitrates and leucocyte esterase have become available as markers of urinary tract infection (UTI). Leucocyte esterase is an enzyme from neutrophils not normally found in urine and is a marker of pyuria. Nitrates are produced by the bacterial breakdown of dietary nitrates. Most urinary pathogens reduce nitrates to nitrites.[1] Dipsticks have been extensively tested in adults, but there are few reports on their use as a routine screening test for UTIs in children. In children, the method of urine collection is often variable, and UTIs have far reaching implications.[2] This study was conducted to identify which dipstick tests are most accurate for detecting UTIs in routine paediatric practice.

A retrospective study was done of 500 consecutive patients admitted to the children's ward of Hartlepool General Hospital between January and June 1999. All the children admitted to the ward had a dipstick examination using Bayer reagent strips (Bayer, Berkshire, UK) read by an automated colorimeter. Culture of urine was undertaken if the dipstick examination was abnormal. Urine culture was also done routinely before starting antibiotic treatment, if there was any clinical suspicion of UTI, and in children with a history of UTI or renal anomalies, even if the dipstick examination was normal. Dipstick testing was considered abnormal if positive for protein, blood, leucocyte esterase or nitrates. Urine culture was done in 312 (62.4%) children. Of these, the indication for culture was an abnormal urine dipstick testing in 272 (87.2%) cases. In the remaining cases urine was sent for culture even if the dipstick testing was normal, because of the aforementioned criteria.

Urine was collected from pads in infants and young children. Midstream specimens were taken from older children. Urine was sent for culture immediately or stored in a refrigerator if immediate transport was not available. A pure growth of a pathogenic organism with a colony count of $> 1 \times 10^9$/litre was deemed positive. Mixed growth was immediately repeated and subsequent result was taken for analysis.

Nitrates were found to have a very high specificity (92.4%) for detecting UTI, which is similar to previously reported studies (table 1). However, sensitivity of nitrates was very low even when combined with leucocyte esterase (64.5%). If all the indices (blood, protein, leucocyte esterase, and nitrates) were combined, and the urine sent for culture if any of these were positive, the sensitivity increased to 97.7% and the chance of missing a UTI was very small. Specificity, however, then decreased to 15.4%. The sensitivity of urine microscopy in our study was very low (12.5%). This is probably because of the delay in transport of the urine sample to the laboratory.

Unlike the previously reported studies in adults,[3 4] UTIs in children cannot be excluded by a negative dipstick nitrates and leucocyte esterase enzyme reaction. Similar results were observed by Lejeune *et al*.[5] In a study of 243 infants, they reported 97.6% specificity and 16.2% sensitivity for nitrates for detecting UTI. However, when leucocyte esterase, nitrates, and proteins were combined, the sensitivity increased to 89.2% and specificity decreased to 71.8%.

Therefore, urine culture needs to be undertaken if any of the four indices (nitrates, blood, protein or leucocyte esterase) are abnormal, or if there is a clinical suspicion of UTI. If nitrates are positive, starting empirical treatment for UTI seems to be reasonable until cultures are reported. This method helped to reduce the workload of the laboratory on urine cultures by 35.8%. Urinary dipsticks are useful screening tests for detecting UTI, only if their limitations are fully understood.

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1 Fowlis GA, Waters J, Williams G. The cost effectiveness of combined rapid tests (Multistix) in screening for urinary tract infections. *J R Soc Med* 1994;**87**:681–2.

Table 1 Comparison of the results of 312 dipstick examinations with total positive cultures

	Sensitivity (%)	*Specificity (%)*	*Predictive value for a*	
			Positive test (%)	*Negative test (%)*
Leucocyte esterase	46.9	58.6	11.5	90.6
Nitrates	34.4	90.7	29.8	92.4
Leucocyte esterase and nitrates	64.5	52.6	13.2	93
Proteins, blood, leucocyte esterase, and nitrates	96.9	15.4	11.4	97.7
Wet film*	12.5	99.0	66.6	90.9

*Pus cells ⩾ 10 was considered as a positive wet film.

2 Woodward MN, Griffiths DM. Use of dipsticks for routine analysis of urine from children with acute abdominal pain. *BMJ* 1993;**306**:1512.
3 McGlone R, Lambert M, Clancy M, Hawkey PM. Use of Ames SG10 urine dipstick for diagnosis of abdominal pain in the accident and emergency department. *Arch Emerg Med* 1990;7:42–7.
4 Flanagan PG, Davies EA, Rooney PG, Stout RW. Evaluation of four screening tests for bacteruria in elderly people. *Lancet* 1989;i: 1117–19.
5 Lejeune B, Baron R, Guillois B, Mayeux D. Evaluation of a screening test for detecting urinary tract infection in newborns and infants. *J Clin Pathol* 1991;**44**:1029–30.

BOOKS

Homeless Children: Problems and Needs. Edited by Vostanis P, Cumella S. (Pp 202, paperback; £15.95.) London: Jessica Kingsley Publishers, 1999. ISBN 1 85302 595 X

Homeless children have clear needs in their health, education, and welfare that are increasingly being recognised as being of great concern. This book brings together contributions from a number of sources outlining their problems and some possible solutions.

Much of the book is based on a longitudinal study in Birmingham of homeless children and their families. This study, which was funded by the Nuffield Foundation, largely focuses on the mental health and social problems of the children.[1] The book also has a series of vignettes of homeless families and their problems—many are harrowing. Much of the book is compelling reading to anyone who is interested in promoting child health and welfare. The book covers fields of child mental health, domestic violence, the impact on social services, and education. The impact of homelessness on a child's schooling described by Sally Power and colleagues demonstrate the "double disadvantage" of being homeless and the difficulty of maintaining a school place for these children. There are also sections on housing legislation and homeless adolescents.

The main gap in the contributions is one of clinical child health. The contribution from Kath Hutchinson, a health visitor, would be augmented with the collaboration of a community paediatrician. The question that I think paediatricians would like to have answered is whether homeless children are uniquely disadvantaged or whether they form one end of the spectrum of poor children. The data from the Birmingham study suggest the former; however, there were only 29 control families in their study compared to 113 homeless ones. In conclusion, Stuart Cumella and Panos Vostanis produce a series of well thought out recommendations for government, both national and local, to improve the problem. They recommend designated sessions for paediatricians to work with homeless families.

This is a useful inexpensive book that would be helpful to all those working in child health. Nevertheless, it is perhaps an example of how child psychiatry and paediatrics are not collaborating nationally. I find it difficult to envisage producing a multiauthor book like this without the help of a paediatrician.

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1 Vostanis P, Grattan E, Cumella S. Mental health problems of homeless children and their families; a longitudinal study. *BMJ* 1998;**316**:899–902.

Silent Nights: Overcoming Sleep Problems in Babies and Children. By Symon B. (Pp 200, paperback; £8.99.) Oxford: Oxford University Press, 1998. ISBN 0 19550 607 3

Isn't it an often voiced fantasy that young babies should come complete with a "user's manual"? Look no further—one has been produced. It might have to be purchased from the bookshop rather than collected from the delivery room, but that in no-way reduces its appeal.

Silent nights is a highly readable, and often amusing, account of normal sleep (and early feeding) patterns in babies and children, some of the problems that can arise, and how to prevent them from occurring and resolve established problems. The information is clearly based on the author's considerable professional and personal experience.

The content of the advice offered is not greatly dissimilar to other parent manuals that deal with infant sleep problems from an essentially behavioural perspective. There are a handful of such books that cover sleep problems and their treatment, from birth to adolescence. *Silent nights* concentrates primarily on sleeplessness and a limited range of treatment or preventive measures. Its originality is that it focuses on the sleep patterns of babies and young children (although older children are mentioned) and the very specific problems that their parents will have to face. These range from the exhausting and disruptive round of relatives visiting to admire the baby, to sexuality being a "casualty of parenthood". As such, it deals with the wider family context, which might be why parents reading this book, as opposed to other parent manuals, will be less likely to feel they have done something "wrong", and more likely to view any difficulties as understandable and treatable events that they have the power to correct. This instilling of confidence in parents is an important determinant of the success of any intervention or preventive measure. Therefore, although the book is intended for parents, professionals may appreciate and benefit from it.

Minor criticisms relate to the rather cursory treatment of circadian rhythms and no mention of other common paediatric sleep disorders (such as rhythmic movement disorders). Such omissions may be inevitable, given that other aspects of sleep and sleeplessness are given a thorough treatment. Idiosyncratic use of the term "night terrors" and an unhelpfully simplistic diagnostic table in the appendix are potentially misleading.

The author acknowledges that the book offers his opinions, rather than the results of empirical studies. Overall, the information offered is clear and authoritative and the style of the book evokes feelings of receiving advice from a wise friend rather than delivery of a set of prescriptive "do's and don'ts" from a remote expert. Because of this narrative style, it is less easy to find precise pages of particular interest and to quickly identify and extract key action points. However, by assimilating the book in its entirety parents should have a greater understanding of their children's sleep in relation to overall development and family functioning.

How many new parents have concerns about their children's sleeping, eating or crying? The answer to that question should provide some indication of the number of people to whom this book would be of interest.

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Benign Childhood Partial Seizures and Related Epileptic Syndromes. By Panayiotopoulos CP. (Pp 360, hardback; £60.00.) London: John Libbey, 1999. ISBN 0 86196 577 9

The recycled ingredients of texts that disseminate evidence-based medicine, although essential, sometimes acquire the bland heaviness of the worst school dinners. How stimulating, then, to read a book so full of zest that boldly admits to being a highly personalised and opinionated account of a field in which the author is an acknowledged expert. Rather than dwell on the areas of consensus, Panayiotopoulos tells us in the opening pages that he "often has to argue against generally applauded statements" about epilepsy, and contends that "differential diagnosis is seldom undertaken for epilepsies". Throughout the book, the image persists of the author as a lone figure fighting against the philistinism of an establishment that does not perform EEG after a single seizure or always tries sodium valproate first. There are glimpses of personal battles won or lost in this symposium or that classifying committee. He presents a rich and illuminating account of his good fight against "textbook recommendations on drug treatment and management, which perpetuate inappropriate generalisations", supporting his arguments with 44 references to his own work and nearly 800 other references, ranging from antiquity through to many from the 1990s.

The 360 pages are divided into five easily digestible sections: general aspects; Rolandic seizures and centrotemporal spikes; occipital seizures and related epileptic syndromes (including his eponymous syndrome); occipital seizures versus migraine; and other childhood partial seizure syndromes. Different chapters are written to serve different purposes and a couple are an inspection at greater magnification of the material covered in a preceding chapter. Although this makes for some repetition, it helps to make the book a useful source for different types of reader. Less common syndromes such as epilepsy with continuous spikes and waves during slow sleep, acquired epileptic aphasia, benign affective seizures, and others are also well covered. Views other than those of the author are, for the most part, fairly represented, although he never leaves the reader in doubt about what he thinks. Opinions are sometimes presented as facts (for example, "the visual hallucinations of migraine . . .cannot last for [only] seconds").

Panayiotopoulos is both clinician and neurophysiologist and can examine both of the elements that make up the electro–clinical syndromes of epilepsy with astonishing attention to detail. Did you know—for example, that fortification spectra were so named by Herschel in 1866 because they resembled not the castellated appearance of battlements but the star patterned pentagonal shape of earthworks projecting from fortifications and known as bastions? Similar precision in detailed clinical observation is used to underpin distinctions between different epilepsy syndromes (or, in that particular example, a

"purposely overemphasised" argument that the visual phenomena of migraine are quite different from those of occipital seizures). Some genetic and molecular biological information is presented but the prime purpose of the book is to refine the definition of the clinical syndromes (on which genetic study currently depends).

This book is never dull. Nor is it a storm in a teacup. Although he is a well known "splitter" of syndromes, his overall thesis is that these entities are manifestations of a common childhood seizure susceptibility syndrome that affects 2–4% of all children and 25% of children with seizures. Reading this book has already forced me to think in greater depth about several patients I have seen in the last week, and may affect my view of their treatment and prognosis. I recommend it to all clinicians, neurophysiologists, and perhaps to geneticists who see children with the many varieties of epilepsies and migraine. It should provoke its readers into a more precise form of observation-based medicine.

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WESTMINSTER BRIEFING

The following items are from *Children & Parliament*, autumn and winter 1999. *Children & Parliament* is an abstracting service based on *Hansard* and produced by the National Children's Bureau. It covers all parliamentary business affecting children and is available on subscription via the internet (http://candp.ncb.org.uk). The *Children & Parliament* web site provides direct links to full text *Hansard*, government department sites, the sites of the Office for National Statistics, Ofsted, and other relevant organisations. For further details contact Lisa Payne, Editor, *Children & Parliament*, National Children's Bureau, 8 Wakley Street, London EC1V 7QE, UK (tel: +44 (0) 171 843 6000; fax: +44 (0) 278 9512). (The *Hansard* reference is given in parentheses; from 17 November 1999 column numbers for written questions will be followed by W).

- The government is to establish 48 000 new free nursery places for 3 year olds in 1999–2000.
  (27 July 1999, Col 165–167)

- The School Standards and Framework Act 1998 came into force in September 1999 and makes corporal punishment illegal for pupils in maintained and non-maintained schools and for children receiving nursery education.
  (19 Oct 1999, Col 543)

- Recent government action against smoking includes the allocation of up to £60 million over 3 years to help health authorities develop specialist services, £47.5 million over 3 years to prepare a health education programme, NHS smoking cessation services in health action zones, draft regulations to ban tobacco advertising, the Public Places Charter, and consultation on an Approved Code of Practice on smoking in the workplace.
  (19 Oct 1999, Col 468)

- The government is making money available for 1999–2002 to help the recruitment and training of an additional 20 000 (full time equivalent) school assistants, including learning support assistants, for children with special educational needs.
  (19 Oct 1999, Col 540)

- Although £20 million has been allocated to the Schools Access Initiative for 1999–2000, the government is planning large increases for the subsequent two years.
  (19 Oct 1999, Col 534)

- An intended change in the law will mean that local education authorities will have to conduct the "transition review" of a child's statement during academic year 9 instead of after the child's 14th birthday as is the present requirement. This means that statemented 16 year old school leavers will have had at least two and usually three annual reviews at which their transition from school has been planned.
  (19 Oct 1999, Col 535–536)

- The higher rate mobility component of Disability Living Allowance will be extended to 3 and 4 year olds by a clause in the Welfare Reform and Pensions Bill. An additional 8000 children should benefit beginning in April 2001.
  (25 Oct 1999, Col 729)

- The British Dyslexia Association, with the help of a grant from the Department of Education and Employment, has recently produced a schools resource pack called *Achieving dyslexia friendly schools*. Teachers in training will need to demonstrate competence in identifying children with special educational needs including dyslexia.
  (1 Nov 1999; Col 68–69)

- In 1998–99 the Medical Research Council spent some £160 000 on research into juvenile arthritis, and the Department of Health has recently given over £500 000 to projects on the same topic.
  (4 Nov 1999, Col 286)

- The government is to give £22.5 million over the next three years towards education in schools about drugs.
  (10 Nov 1999, Col 578)

- In 1998, 284 sudden infant deaths were recorded in England and Wales.
  (10 Nov 1999, Col 643)

- In 1998–99 Medical Research Council spending on epilepsy research was £3.6 million.
  (11 Nov 1999, Col 816–817)

- The NHS Direct telephone helpline should cover 60% of the population of England by December 1999 and the whole population by the end of 2000.
  (11 Nov 1999, Col 814)

- An Early Day Motion calling for more research into autism and improved services for children and adults with autism was signed by 11 MPs.
  (17 Nov 1999, Early Day Motion no. 24)

- Legislation referred to in the Queen's Speech and likely to affect children includes the following Bills: the Care Standards Bill, the Children (Leaving Care) Bill, the Child Support, Pensions and Social Security Bill, the Crime and Protection of Children Bill, the Freedom of Information Bill, the Learning and Skills Bill, the Race Relations (Amendment) Bill, the Sexual Offences (Amendment) Bill, the Special Educational Needs Bill, and the Local Government Bill.
  (17 Nov 1999, Col 4–7, 1–6)

- In 1996 there were an estimated 4.3 million or more fuel poor households, defined as those who need to spend more than 10% of household income to achieve satisfactory heating.
  (22 Nov 1999, 26 Nov 1999, Col 48 W, 209–210 W)

- The government is to consult on Quality Strategy for Social Services and proposes to establish an institute for excellence in social care early in 2000.
  (23 Nov 1999, Col 92 W)

- Low income single parents may have further education tuition fees reimbursed from the Further Education Access Fund and may be considered for a free or subsidised child care place.
  (26 Nov 1999, Col 251 W)

- Childcare Link, a freephone national child care information line and website was launched by The Under Secretary of State for Education and Employment on 1 December 1999.
  (29 Nov 1999, Col 44–45 W)

Note: from 30 November 1999 adjournment debates will take place on Tuesday and Wednesday mornings in Westminster Hall. They will be reported in Hansard with a separate sequence of columns with the suffix WH.

- In a debate about under age smoking attention was drawn to Gutkha, a sweetened chewing tobacco which, it was claimed, is being cynically marketed at children, especially within the Asian community. A three year £50 million tobacco education programme was to be launched on 13 December 1999. It will be translated into 11 languages and some programmes will be targeted at ethnic minorities.
  (30 Nov 1999, Col 32–39 WH)

- The Children's Fund to be set up in the 2000 spending review will support work with low-income families and their children.
  (6 Dec 1999, Col 450 W)

- People who were sexually or physically abused as children can claim compensation under the Criminal Injuries Compensation Scheme administered by the Criminal Injuries Compensation Authority.
  (7 Dec 1999, Col 499 W)

- Around the world the number of couples having access to modern contraception has risen from 9% to almost 60% in the last 30 years. There is an international commitment to make it 100% by 2015.
  (8 Dec 1999, Col 577 W)

- An investment of $390 million over 3 years (1999–2002) is intended to help achieve the target of free educational places for 66% of 3 year olds by 2002. Priority will be given to areas with the greatest social needs.
  (8 Dec 1999, Col 589 W)

- Key international targets to which the government is strongly committed include sex equality in primary and secondary education by 2005 and universal primary education by 2015. The World Forum on Education is to be held in Dakar, Senegal in April 2000.
  (9 Dec 1999, Col 629 W)