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. I wish however to take issue with one point concerning lumbar puncture. Lumbar puncture certainly should 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 DEFER LUMBAR PUNCTURE (sic) with the instructions DEFER LUMBAR PUNCTURE and discuss its performance later in the illness.

DENIS G GILL
Professor of Paediatrics, Children's Hospital, Temple Street, Dublin 1, Republic of Ireland
e-mail: gilld@iol.ie

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-bloody 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 meningitis or meningism. 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, and does not affect clinical management. 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 and, perhaps, 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 the article. Our central nervous system infection commonly coexists with sepsicaemia 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, the choice of management of meningitis in children has been widely advocated but has been challenging and may even have an adverse effect on outcome.1


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.” 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 charges 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 of oestradiol, 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 combined oral contraceptive formulations, not detected by standard hormone assays. Monitoring of oestrogen replacement is therefore, dependent on suppression of follicle stimulating hormone, 17 beta oestradiol 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.

ANNIE S GARDEN
Consultant Obstetrician and Gynaecologist

D C DAVIDSON
Consultant Paediatrician, Alder Hey Children's Hospital, Eaton Road, Liverpool L12 2AH UK


Planning for major incidents involving children by implementing a Delphi study

EDITOR,—The proposed paediatric triage algorithm in Mackway-Jones et al’s study 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

244

Arch Dis Child 2000;82:266–273
 Editors,—Fleming and colleagues state the use of duvet use and SIDS 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 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 cannot 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.

Surely the major potential disadvantage of pacifier use on SIDS, excluding duvet use, is that is actually meditated by space limitations. We found that due to space limitations. We found that duvet use and SIDS are less clear but our data indicated that the use of duvet use and SIDS are less clear but our data indicated that the weaker strength of association between maternal smoking and infant mortality. This is clearly inappropriate as the adverse effect of smoking is partially mediated through birth weight. The causal pathways between duvet use and SIDS are less clear but our data indicated that the weakening of the association between maternal smoking and infant mortality. This is clearly inappropriate as the adverse effect of smoking is partially mediated through birth weight. The causal pathways between duvet use and SIDS are less clear but our data indicated that the weakening of the association between maternal smoking and infant mortality. This is clearly inappropriate as the adverse effect of smoking is partially mediated through birth weight.

The causal pathways between duvet use and SIDS are less clear but our data indicated that the weakening of the association between maternal smoking and infant mortality. This is clearly inappropriate as the adverse effect of smoking is partially mediated through birth weight. The causal pathways between duvet use and SIDS are less clear but our data indicated that the weakening of the association between maternal smoking and infant mortality. This is clearly inappropriate as the adverse effect of smoking is partially mediated through birth weight. The causal pathways between duvet use and SIDS are less clear but our data indicated that the weakening of the association between maternal smoking and infant mortality. This is clearly inappropriate as the adverse effect of smoking is partially mediated through birth weight.
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 inaccurately 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 insignificantly different from that without duvet use.

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.

We do not have one or more bruises. However, there are problems with the terms relating to measures of disease frequency in epidemiology which, arguably, make this descriptive paper misleading.

The term point prevalence, prevalence proportion, prevalence ratio, and prevalence rate are sometimes used to mean the same thing. 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 strongly perturbs the use of the term rate parameter to describe the unknown and estimated value associated with a Poisson probability model.1 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 only once. 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 makes 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.
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 incidence is 

\[ \text{CI} = 1 - \exp(-0.12 \times 6) \geq 0.51 \]  

(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 mobile, the incidence 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.

ANDREW L LUX
Royal United Hospital Bath NHS Trust,
The Children’s Centre, Combe Park,
Bath BA1 3NG, UK

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 cumulative 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 units for adolescent units have been advocated1 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 base-line information. For example (26.5%) 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 specialties. 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 inpatient team. Sixty nine 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.1,2 Teenagers may prefer an adolescent based service.1 One 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.

S SURESH
Specialist Registrar Paediatrics, Princess of Wales Hospital, Bridgend CF31 1RQ, UK

I J DOULL
Consultant Respiratory Paediatrician, University Hospital Wales, Cardiff CF4 4XW, UK

P THOMAS
Sister, Department of Child Health, University Hospital Wales

REFERENCE


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.3 The need for planned transition is indeed very real 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.4 It works in conjunction with the rehabilitation medicine physiatrist 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 and 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 better 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 take responsibility for their own health. In Leeds we have a specialist team (nurse specialist, physiotherapist, social worker) which is involved in the care of hospitalised patients with arthritis and cerebral palsy. Although the numbers involved are small, these clinics have helped us to formulate our approach to transitional care.

In summary, this is an important article which highlights the need for planned transition and the need for well structured transitional services.

SURESH S
Consultant Paediatrician, University Hospital Wales

P THOMAS
Sister, Department of Child Health, University Hospital Wales

REFERENCE
1 Viner R. Transition from paediatric to adult care. Bridging the gaps or passing the buck? Arch Dis Child 1999;7:271-5.


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. First, you cannot make children smarter by putting more iron in their milk, and second that I am proportionally unable to find an acceptable formula that will improve iron status. Force feeding the iron or changing the wholesomeness of the food will not help. The older paper by Donnelly et al was relatively reassuring, even though their study was flawed by a preconceived outcome. I hope that in the future we can say where it is most appropriate to use iron supplements. For example, it would be appropriate for children with haemoglobinopathies who are unable to get their iron requirements from diet.

M A CHAMBERLAIN
Professor of Rehabilitation Medicine, University of Leeds

REFERENCE
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 formulated two papers. The reference for the only person who has ever read any of my own publications. The authors say that the own publications. The authors concluded that midparental height was a useful indicator of the height. Clearly, midparental height is not a genetic potential 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. 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 as itself 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.

J KARLBERG
Z C LUO
Department of Pediatrics, Queen Mary Hospital, The University of Hong Kong, Hong Kong

Estimating the genetic potential in stature

Editor—Midparent 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. 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. 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.

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

<table>
<thead>
<tr>
<th></th>
<th>Peak GH &lt; 10 mU/l</th>
<th>Peak GH &lt; 20 mU/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>IGF-1</td>
<td>37.5%</td>
<td>29.5%</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>79.7%</td>
<td>79.6%</td>
</tr>
<tr>
<td>Specificity</td>
<td>1.85</td>
<td>1.5</td>
</tr>
<tr>
<td>GH deficiency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IGFBP-3</td>
<td>31.5%</td>
<td>27.8%</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>76.3%</td>
<td>76.2%</td>
</tr>
<tr>
<td>Specificity</td>
<td>1.33</td>
<td>1.3</td>
</tr>
</tbody>
</table>

GHD, but a significant number of children with GHD will have normal values for either of these two markers. 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’ 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.

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...” should have been: Moffatt ME, Longstaffe T, Lucas A. Iron fortified follow on formula milk for the prevention of iron deficiency anaemia in the young child: a randomised trial. Arch Dis Child 1999;81:247-52.


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. 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. 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. 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 as itself 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.

J KARLBERG
Z C LUO
Department of Pediatrics, Queen Mary Hospital, The University of Hong Kong, Hong Kong


IGFBP-3 as a predictor of growth hormone deficiency

EDITOR—We read with interest the paper by Mitchell and his colleagues in their 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 log-squaring the data and transforming 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. 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’ 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.

B BIELINSKI
N J SHAW
P M CLARK
Department of Endocrinology, Birmingham Children’s Hospital, Birmingham B4 6NX, UK

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 viscerectomy 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. A further case of muscular dystrophy has also been diagnosed in a child with coeliac disease and persistently raised alanine aminotransferase. This lack of specific factors 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.

New developments in the treatment of cardiac failure

EDITOR,—The article by Westaby et al presented concisely recent developments in the treatment of cardiac failure. The Digitalis Investigation Group has shown that the effect of digoxin on mortality and morbidity in patients with heart failure can be assessed in the outpatient clinic by reference to a baseline record of mortality and morbidity. This method allows a more accurate measure of the drug's clinical effectiveness, and the results should be compared with those of the Digoxin Investigation Group in the USA. The Digitalis Investigation Group also suggest that digitalis is effective in preventing hospitalization and improving quality of life, but these findings should be confirmed in larger studies. The long-term follow-up of patients in the Digitalis Investigation Group showed a reduction in mortality and morbidity in patients receiving digitalis, which was not observed in the placebo group.

The findings of the Digitalis Investigation Group are important, particularly in view of the recent report by the British Cardiac Society that digitalis is the most commonly used drug for the treatment of heart failure. The Digitalis Investigation Group has clearly shown that digitalis is effective in reducing mortality and morbidity in patients with heart failure, and that this effect is sustained over time.

[References]


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 produced by polymorphonuclear neutrophils and is 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. Dipsticks have been extensively tested in adults, but there are few reports on their use as a routine screening test for UTIs in children. However, when leucocyte esterase, nitrates, and proteins were combined, the sensitivity increased to 89.2% and specificity decreased to 71.8%.

C-sensitivity and specificity of rapid tests for the detection of UTIs.

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

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Positive test</th>
<th>Negative test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leucocyte esterase</td>
<td>46.9</td>
<td>58.6</td>
<td>11.5</td>
<td>90.6</td>
</tr>
<tr>
<td>Nitrates</td>
<td>34.4</td>
<td>90.7</td>
<td>29.8</td>
<td>92.4</td>
</tr>
<tr>
<td>Leucocyte esterase and nitrates</td>
<td>64.5</td>
<td>52.6</td>
<td>13.2</td>
<td>93</td>
</tr>
<tr>
<td>Proteins, haem, leucocyte esterase, and nitrates</td>
<td>96.9</td>
<td>87.4</td>
<td>11.4</td>
<td>97.7</td>
</tr>
</tbody>
</table>

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

LUCI WIGGS
Research Psychologist


The recycled ingredients of texts that disseminate evidence-based medicine, although essential, sometimes acquire the blandness 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. 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 simple approaches. 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 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 epilepsy 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 and a 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 differential diagnosis.

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 neuropsychologist 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 fortification of a town but a visual hallucination of the mind...”?


BOOKS


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. 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 is described by Sally Power and colleagues demonstrating the “double disadvantage” of both family 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 empirical studies. 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 answer 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 children and the study compared 133 homeless ones. In conclusion, Stuart Cumeilla and Panos Vostanis produce a series of well thought out recommendations for government, both national and local, to improve the situation. 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 multiauthored book like this without the help of a paediatrician.

JONATHON R SIBERT
Professor of Community Child Health


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 they may 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 to correct. This instilling 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 unhelpful 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.

LUCI WIGGS
Research Psychologist

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.

LUCI WIGGS
Research Psychologist


The recycled ingredients of texts that disseminate evidence-based medicine, although essential, sometimes acquire the blandness 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. 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 simple approaches.

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 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 epilepsy 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 and a 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 differential diagnosis.

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 neuropsychologist 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 fortification of a town but a visual hallucination of the mind...”?

Panayiotopoulos is both clinician and neuropsychologist 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, for in that particular example,
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.nch.org.uk). The Children & Parliament web site provides direct links to full text Hansard and produces by the 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 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 Gutka, 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 W)
- 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)
Emergency management of meningococcal disease

DENIS G GILL

Arch Dis Child 2000 82: 266
doi: 10.1136/adc.82.3.266

Updated information and services can be found at:
http://adc.bmj.com/content/82/3/266.1

These include:

References
This article cites 6 articles, 3 of which you can access for free at:
http://adc.bmj.com/content/82/3/266.1#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/