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 the need for 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 DO NOT LUMBAR PUNCTURE (sic) with the instructions DEFER LUMBAR PUNCTURE and discuss its performance later in the illness.

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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 meningitis 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 misleading2 and can 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 of antibiotic treatment. We advocate use of a third generation cefalosporin in a child with meningococcal disease for seven days regardless of the predominant clinical syndrome, for the reasons described in our article and because central nervous system infection commonly coexists with septicaemia3 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 these syndromes.

Because of this overlap between meningitis and sepsis, 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 the 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, nil by mouth. If the management of meningitis in children has been widely advocated and has been challenged 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 they have been promoted as such. 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 prejudice to fertility, 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 the benefit 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 all aware of the incompatibility of 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 progesterones 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. Progesterones 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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Planning for major incidents involving children by implementing a Delphi study

EDITOR,—The proposed paediatric triage algorithm in Mackway-Jones et al’s study1 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
Alert and moving all limbs ↓ no → yes → Delayed Priority 3
Breathing ↓ yes → no → Open the airway → Immediate Priority 1
Respiratory rate ↓ 20 to 50/min → < 20 or > 50/min → Immediate Priority 1
Capillary refill < 2 seconds (use the child’s forehead) ↓ no → yes → Pulse rate ↓ < 90 or > 180 beats/min → 90 to 180 beats/min → Immediate Priority 1

Figure 1 Paediatric triage tape 50–80 cm.

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Breathing ↓ yes → no → Open the airway → Immediate Priority 1
Respiratory rate ↓ 20 to 50/min → < 20 or > 50/min → Immediate Priority 1
Capillary refill < 2 seconds (use the child’s forehead) ↓ no → yes → Pulse rate ↓ < 90 or > 180 beats/min → 90 to 180 beats/min → Immediate Priority 1

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 on multivariate analysis, and the SIDS risk associated with duvet use was not modified by sleeping position.2 These findings contrast with our finding of a strong association (adjusted odds ratio 6.16 (2.0, 18.87)) between quilt (duvet) use and SIDS among non-prone infants.3

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 marital status strongly associated with 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 used 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.5

The causal pathways between duvet use and SIDS are less clear but our data indicated that part of the adverse effect results from facial obstruction.6 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.7

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

2 Macnonachie I, GRT’s enough to make you blush! Pre-hospital Immediate Care 1998;2:95–7.

References

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. Were the photographs taken by the first paediatrician inadequate, and if so, why? A clinical diagram complemented by a quality photograph 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 court would need a 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 the role of the examiner, even if the children are well prepared, always needs justification.

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Dr Hodes and colleagues comment:

We agree that, as a general rule, re-examination should be avoided, but we 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, there had 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 26 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 make 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:

\[
\text{cumulative incidence} = \frac{(1 - \exp(-\text{incidence rate} \times \text{number of periods})))}{(point \ prevalence)/(duration)}.
\]

The cumulative incidence is obtained from the formula:
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 (1 – exp(–0.12 × 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 in 1988 through joint funding from health and social services the with the young person’s autonomy while addressing their developmental and psychosocial needs of hospitalised 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. 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 best be 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 cope with illness. In Leeds we have had jointly run (paediatrician and adult physician) arthritis 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.

Adolescent inpatient units

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

In a representative 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. Furthermore (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 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 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. Teenagers may prefer an adolescent based service. 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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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 < 0.05) whereas mobility was significant (p < 0.05) whereas mobility was significant (p < 0.05).

The probability of it having a bruise at another time point, as he suggests, this is not relevant for the clinical situation at presentation when abuse could be considered.

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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. ‘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 nurse) 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. It works in conjunction with the rehabilitation medicine physi-

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
the only person who has ever read any of my own publications. The authors say that Stevens and Nelson 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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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, Durecki 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 than 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 a short parent 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 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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IGFBP-3 as a predictor of growth hormone deficiency

EDITOR,—We read with interest the paper by Mitchell and colleagues 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 IGFBP-3. We defined GHD as a peak growth hormone level of < 10 mU/litre in response to a stimulation test. We 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 at 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.

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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 developmental history 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 nmol/litre and the

<table>
<thead>
<tr>
<th>Peak GH</th>
<th>Peak &lt; 10 mU/litre</th>
<th>Peak &lt; 20 mU/litre</th>
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</thead>
<tbody>
<tr>
<td>Sensitivity</td>
<td>37.5%</td>
<td>29.5%</td>
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<tr>
<td>Specificity</td>
<td>79.7%</td>
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<td>Likelihood ratio</td>
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<tr>
<td>Sensitivity</td>
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<td>Specificity</td>
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<tr>
<td>Likelihood ratio</td>
<td>1.33</td>
<td>1.3</td>
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</table>

Table 1 Sensitivity and specificity of IGF-1 and IGFBP-3 in predicting growth hormone (GH) deficiency
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 decreases 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 which is 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 dependence or denial. However, despite many limitations the prognosis for children with severe heart failure has significantly improved over the past decade.6

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**E A WORTHING**
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**Table 1** Comparison of the results of 312 dipstick examinations with total positive cultures

<table>
<thead>
<tr>
<th>Test</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>
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<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>91.4</td>
<td>11.4</td>
<td>97.7</td>
</tr>
<tr>
<td>Wet film</td>
<td>12.5</td>
<td>99.0</td>
<td>66.6</td>
<td>90.9</td>
</tr>
</tbody>
</table>

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

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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. Rather than an unhelpfully simplistic diagnostic table or 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 differentiating the 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 petit mal . . . 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 fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resembled not fortification spectra were so named by Herschel in 1866 because they resemble...
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 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).

- 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 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)