LETTERS TO THE EDITOR

Paracetamol overdosage

EDITOR,—Paracetamol is the most common drug used for deliberate self poisoning in adolescents.[1]

Recent legislation in the United Kingdom has limited the number of paracetamol tablets available in a single purchase. We wish to draw attention to another area where we believe legislation might be helpful.

An 11 year old boy was admitted to our care following a deliberate overdose of paracetamol tablets. After a period of observation, and assessment by the child psychiatry team, he was discharged from hospital. He returned a few hours later having taken a further overdose of paracetamol tablets which he had purchased, legally, at a local pharmacy.

English law sets lower age limits for the purchase of alcohol and cigarettes. It seems anomalous that there is no restriction on the age at which a child can purchase a potentially fatal over the counter drug. We recognise that someone with sufficient determination can always circumvent legal obstacles. Nevertheless we believe that reducing the ease of availability of drugs containing paracetamol to children under 16 years might help lessen the numbers of children and young teenagers now using this dangerous route to draw attention to their problems.

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1 Hawton K, Fagg J, Simkin S. Deliberate self-poisoning and self-injury in children and adolescents under 16 years of age in Oxford, 1976–1993. *Br J Psychiatry* 1996;**169**:202–8.

Trying to deliver aerosols to upset children is a thankless task

EDITOR,—The study by Iles *et al*[1] may, once and for all, help dispel the myth that aerosol delivery to the lungs of crying children is enhanced as a result of them taking a deep inspiratory breath.

Compelling evidence that delivery of drug to the lungs of distressed infants is greatly reduced compared with that to the lungs of a relaxed tidal breathing infant has been available for many years. Murakami *et al*[2] illustrated this point extremely effectively in a radiolabelled deposition study using a jet nebuliser, while Tal *et al*[3] also observed a tenfold reduction in dose in crying infants using a pMDI and Aerochamber. More recently we have also reported significantly reduced drug delivery and increased treatment times in the small minority of young children who were upset when using the dosimetric Halolite nebuliser.[4]

There are two principal reasons for poor delivery in distressed children. Failure to achieve a good seal between mask and face when using a face mask results in air entrainment with little or no drug being inhaled.[5] Even if a face mask can be clamped on to the face of a distressed child, the dose inhaled is reduced and the majority of the inhaled dose deposits in the upper airways, to be swallowed as illustrated so elegantly in the paper by Murakami *et al*. This is due to the abnormal breathing patterns adopted by upset toddlers and infants.[6]

As asthma nurses have known for many years, factors relating to the use of devices by patients are of greater importance in determining drug delivery to the lungs than are technical factors such as static in holding chambers which is only one of many sources of variability.[7] To date, pharmaceutical companies and nebuliser manufacturers have conspicuously failed to develop devices that are intrinsically acceptable to young children. It is to be hoped that consideration of factors that would positively encourage young children to cooperate will be incorporated into the next generation of delivery systems.

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School nursing

EDITOR,—May we please correct an unintentional impression of therapeutic nihilism in the article on school nursing.[1] One of us (DH) commented in the article on the work of Bax and Whitmore. These authors described the process by which they assessed school entrants, with the aim of identifying children who might develop learning problems and thereby prevent the adverse outcomes associated with failure in school.

It was implied in the article that there was no treatment for the conditions identified by this process. When Bax and Whitmore carried out their studies this was probably true, but with greater sophistication of diagnosis and management there are many children with neurodevelopmental (and psychological) disorders, such as attention deficit disorder, coordination problems, or reading difficulties who could now benefit from assessment and intervention. The unresolved challenge facing paediatricians, child mental health services, and educationalists is to develop better ways of finding these children and providing effective intervention before it is too late to help them.

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1 Hall DMB. School nursing: past, present, and future. *Arch Dis Child* 1999;**81**:181–4.

GFR estimation in paediatric oncology

EDITOR,—The study by Rees *et al*[1] indicates the low utility of routine glomerular filtration rate (GFR) estimation in paediatric oncology. We have performed a similar study in a regional children's cancer centre, looking at the utility of routine GFR estimation in patients who received high dose methotrexate, alkylating agents, or platinum containing cytotoxics.

The audit involved 110 GFR estimations on 42 patients, aged 5 months to 18 years, 48% of whom had haematological malignancies. The median GFR was 101 ml/kg/1.73 m^2 (interquartile range 81.6 to 111.5 ml/kg/1.73 m^2), with 15 measurements (13%) below 70 ml/kg/1.73 m^2 (see table 1). In only two patients (4%) was treatment changed on the basis of GFR results: one was a child who had suffered mild tumour lysis syndrome, the other a young man who had previously been treated for lymphoma. Both had been exposed to amphotericin; as had eight other patients who did not require a change in management.

We also studied the relation between the ratio of height (cm):serum creatinine (μmol/l) (H:Cr ratio) to abnormal GFR results. This ratio had previously been shown to be a useful screening test for abnormal GFRs in general paediatrics,[2 3] although inaccurate for estimating the absolute value.[4] Setting a lower cut off of normal at 1.5 (on the basis of previously published data[2]) we tested this relation in 22 patients (34 measurements) who had contemporaneous height and creatinine measurements. No patients with abnormal GFR results had a H:Cr ratio greater than 1.5; the patients whose treatment had been changed had H:Cr ratios of 1.1 and 0.8. (The test had a sensitivity of 100% and specificity of 97%.)

We agree that routine estimations of GFR are unhelpful in paediatric oncology patients. The use of the H:Cr ratio is a potentially useful screening test for abnormal glomerular filtration rates.

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Table 1 Test data

	GFR <70 ml/kg/1.73 m^2	*GFR >70 ml/kg/1.73 m^2*
H:Cr <1.5	2	1
H:Cr >1.5	0	31

Meningitis and meningococcal septicaemia

EDITOR,—In May 1999, prior to the meningococcal C campaign, we designed a ques-

Table 1 Results of questionnaires on meningitis and meningococcal septicaemia

	Time questionnaire administered		
	June 1999 (study group before teaching)	*Late October/early November 1999 (study group after teaching)*	*Late October/early November 1999 (control group)*
Number	37	31	32
% who had heard of meningitis and septicaemia	95	100	100
% who knew possible causes of meningitis and septicaemia	22	27	12.5
% who could list important signs and symptoms	59	76	77
% who could state how to do the "glass test"	35	81	64
% who knew what to do if they suspected a friend had meningitis	81	97	87

tionnaire to ascertain knowledge about meningitis and meningococcal septicaemia among 15/16 year olds. Groups of 8–10 volunteers, in each of the local state secondary schools in Bath, independently completed a short questionnaire. The school nurse then led a tutorial session, and provided handouts summarising the teaching.

We planned to return to the participating schools after a 12 month period to re-interview the students, and control groups, in order to determine whether the teaching sessions were effective in raising knowledge and awareness about meningitis. However, in August, the government announced the introduction of the meningitis C campaign. This brought the topic into the public arena. Families of adolescents received leaflets about meningitis and the vaccination programme. We therefore decided to re-administer our questionnaire in the autumn term to the original target groups of adolescents, and to age and ability matched control groups.

Of the seven schools who originally participated, three were unable to schedule sessions for a revisit. The results are tabled for the schools where the full project was completed. The initial level of knowledge shown by the groups in schools where we were unable to revisit was not significantly different from those completing the study.

Knowledge about meningitis and meningococcal septicaemia increased during the study period (see table 1). The glass test was mentioned by only 35% of the study group prior to teaching. By late October/November the level of knowledge in our control group had increased to 64% and in our study group it was 81%.

When asked "what would you do if you suspected a friend has meningitis?", correct responses increased from 81% prior to the interviews to 87% in the control group and 97% in the study group.

The increase in knowledge in the control group has probably been due to the publicity campaign. It would seem likely that the improvement in our study group was due to the campaign plus the added effect of tutorial based teaching.

We are concerned that the positive effect may be short lived. Community paediatricians and school nurses should not feel complacent, but should be aware of the need for ongoing education on this important topic.

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Exploring the scope for advocacy by paediatricians

EDITOR,—Dr Bell shows himself to be as much influenced by prejudice as the paediatricians he criticises, in his comments on "the family" (Commentary, December 1999 issue of *ADC*, p. 517). We agree entirely with him that advocacy should have an evidence base. It also needs to have an achievable focus and be politically attainable: neither are easy to define in relation to family life. Whilst we might agree that a two parent family is desirable, not even the Pope, the Archbishop of Canterbury, nor Mrs Thatcher can influence current trends and the role of politics is minimal.

Two other aspects of his commentary disturb us. First, advocacy as much as counselling requires careful study and applied training; we do not agree that any doctor can do either well, simply "as part of his or her work". Second, Dr Bell disparages speaking out on women's and children's rights in developing countries as it might lead to deportation. However this view ignores the role of the West, through aid policies and transnational corporation activity, in flouting these rights. Our advocacy should be global in concept but local in focus.

Dr Bell goes on to criticise the study for being based on opinion. Certainly it was based on opinion—its very value is that nine paediatricians working in a variety of settings came together in an attempt to analyse the sorts of issues requiring advocacy that arise in the course of a working week, and to set up an agenda that they would like to see addressed. If it only serves as a springboard for discussion and action, something worthwhile will have been achieved.

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Dr Bell comments:

With 38% of our children now born out of wedlock, my comments on the breakdown of family life in the UK are all too evidence based. I note that Drs Rudolf and Waterston, "... might (*might!*) agree that a two parent family is desirable", and whilst I agree that the church leaders named seemed to have been ineffective in stemming the rise, I do not agree that the role of politics is minimal; indeed I fear that it has been legislation (i.e. politics) with respect to support and housing of single parents and changes in marital law and taxation that has fuelled this rise, not to mention the subtle advocacy provided by the constant fare of value-free sex in television programmes.

Some forms of advocacy may need special training, as I stated in my commentary, but many aspects of advocacy are part and parcel of a doctor's job and such skills must be acquired as part of one's general development: surely they are not suggesting that one needs to attend their course before one writes to an MP?

I did not disparage advocating women's and children's rights: what I wrote was, "shouting loudly about women's and children's rights is, in many a third-world country, more likely to result in rapid deportation, than any benefit to the women or children of that country." My point was that to achieve an end one has to soberly consider how best to do it. For example, ideological statements such as, "... the role of the West, through aid policies and transnational corporations ...", seem more likely to frighten off any legislator, not to mention many potential supporters. It is hardly what Berman meant by "identify friends and build coalitions".

If, as Drs Waterston and Rudolf state, their paper was to serve as, " ... a springboard for discussion and action ...", why are they complaining that it achieved its aim?

Perthes' disease and blood manganese levels

EDITOR,—Children with Perthes' disease have aseptic necrosis of the hip and a selective impairment of growth. Trace metal deficiency in chickens leads to a similar condition, and in 1989 Hall *et al* reported low blood manganese levels in 27 children with this disease.[1] With consent from the ethical committee of the same hospital as in their study, we have measured blood Mn of a further group of Perthes' patients, and control patients with minor musculoskeletal trauma matched for age and sex. Blood was drawn through stainless steel needles into plastic syringes and transferred into acid washed tubes containing EDTA. They were frozen upright at −20°C. Whole blood analysis was undertaken as reported previously[1] and statistical analysis was by conditional logistic regression, adjusting for age, sex, and socioeconomic status.

A number of the frozen tubes cracked. Mean Mn levels in the tubes from control children were higher in the cracked tubes (197 (93) nmol/l) than in the uncracked tubes (157 (45) nmol/l; $p = 0.02$). We attribute the raised levels to dehydration or contamination and recommend freezing at an angle to obviate this complication. After exclusion of cracked tubes, results from 21 patients and matched controls remained. Mean blood Mn levels were 179 (77) and 157 (45) nmol/l respectively. Whole blood iron did not differ significantly in the two groups.

These findings do not support the earlier results from this hospital, although the entry criteria to the study and methods of analyses were similar. The difference between the studies may be due to chance, or to an inherent bias in one of them.

We also undertook a concurrent randomised intervention trial in 25 patients with Perthes' disease, all of whom took 3.4 g of flavoured maltodextrin daily, supplemented in 11 patients with 1 mg of manganese chloride. The outcome was determined over a two year period by regular assessment of symp-

toms, by sequential anthropometry, and measurements of femoral head morphology and subluxation. The patients did not like the oral supplement and there was no improvement in the measured parameters in those taking the active supplement. Their mean blood Mn levels at baseline and six months were 177 and 195 nmol/l respectively, whereas for those taking the inert powder the mean levels were 187 and 214 nmol/l. However, the fact that ingestion of 1 mg Mn daily had no effect on blood levels may not be surprising as this is about 25% of the normal daily intake, and manganese has a short biological half life. We found no benefit from supplementary Mn, but assessment was difficult due to the variable presentation and natural history of Perthes' disease.

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Gastrostomy feeding in the disabled child

EDITOR,—The annotation by Dr P Sullivan in the December 1999 issue of *ADC* on gastrostomy feeding in the disabled child and indications for an antireflux procedure, was timely and comprehensive. As nutritional needs of chronically ill children including the neurologically disabled are recognised, there have been ever increasing calls for gastrostomy. There is a trend, perhaps to "overdo" fundoplications in a few centres, particularly, but not exclusively, because of the relative ease of the laparoscopic technique. The facts, as aptly outlined by Dr Sullivan, all say that fundoplication as a treatment for gastro-oesophageal reflux disease is far from ideal. In the short term follow up studies he quoted, the failure and complication rates are high, more so in children with neurological disorders. The only firm indication for fundoplication, with or without gastrostomy, is clearly documented aspiration. This said, feeding via gastrostomy and discontinuing oral intake of fluids might cure recurrent aspiration in some children with palatopharyngeal discoordination. Medical treatment of oesophagitis has made major progress, as we now have effective acid suppression agents and prokinetics.[1] In addition, there are the options of antireflux feeding methods before or after gastrostomy insertion—that is, continuous feeding and transgastricojejunal feeding. Pyloroplasty may be required, either alone or in conjunction with fundoplication, when delayed gastric emptying is documented.[2 3]

The management of feeding disorders and of gastro-oesophageal reflux disease should be approached carefully and requires thorough investigation of the mechanisms of feeding and of reflux. This should be a joint venture between paediatric gastroenterologists and paediatric surgeons. We should not allow the pendulum to swing to the extreme of automatically offering gastrostomy and fundoplication to children with disabilities.

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Liverpool Infirmary for Children

EDITOR,—Dr Black, in his historical paper,[1] mentions that The Hospital For Sick Children, Great Ormond Street, founded in 1852 was the first Children's Hospital in the UK. In fact, The Liverpool Infirmary for Children was founded in 1851. This date has been confirmed by the Liverpool Records Office, although there is a view that it was opened in 1848.[2] In the first ten months, 657 outpatients were treated by the medical staff. At that time the Infirmary was housed in a building in Upper Hill Street.

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1 Black JA. Great Ormond Street in 1941 and 1942. *Arch Dis Child* 2000;**82**:84-7.
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Growth monitoring

EDITOR,—There is a rather subtle statistical error in the Appendix to Professor Hall's paper.[1] It is stated there that "if a child's height is observed to be on the 3rd centile we can be very confident that the true height lies between the 2nd and the 4th centile". This overlooks the fact that the centile charts are derived from observed and not from true heights. If a child's height falls on the 3rd centile we can be confident that just 3% of all healthy children have observed heights which are smaller. The issue of the "true" height does not arise. A similar remark applies to velocity measurements. It is tempting to reduce the substantial measuring errors involved in observed velocities by repeating each of the two measurements and taking the averages, but different centiles would be required to interpret the results, with centile lines closer together than those on the orthodox charts.

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Helicobacter pylori infection with iron deficiency anaemia and subnormal growth at puberty

EDITOR,—The authors of this paper did not indicate the relative proportions of iron deficient patients identified by using hypoferritinaemia as the diagnostic criterion versus those diagnosed by means of transferrin saturation less than 15%.[1] A vast preponderance of the former signifies more robust characterisation of the iron deficient state, given the fact that the area under the curve is significantly greater for the serum ferritin receiver operator characteristic (ROC) than for the transferrin saturation ROC, namely, 0.91 versus 0.79.[2] Inspection of the curves also reveals that the likelihood ratio of true positive versus false positive diagnosis of iron deficiency is likely to be much higher when the serum ferritin is less than 12 ng/ml than when the transferrin saturation is less than 15%.[2] There is no reason to believe that these observations are not generalisable across the entire age range.

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1 Choe YH, Kim SK, Hong YC. Helicobacter pylori infection with iron deficiency anaemia and subnormal growth at puberty. *Arch Dis Child* 2000;**82**:136-40.
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Imaging in scoliosis

EDITOR,—I read with interest the article "Imaging in scoliosis". In addition to the radiological techniques described, I would include surface shape measurement as a simple, safe, repeatable, and clinically relevant assessment.

Associated with the bony deformity there is a cosmetic problem; indeed, idiopathic scoliosis is primarily a cosmetic disorder. The first sign commonly noticed by the clinician or patient is an asymmetry of backshape, resulting from axial rotation of the spine and rib cage. Treatment is aimed at the reversal or arrest of the progression of this visible deformity as well as that of the underlying skeletal abnormality.

Quantitative assessment of scoliosis is necessary both to assess the severity of the condition, and to monitor its progression. The Cobb angle is a reasonably consistent uniplanar measure of spinal deformity, but indicates neither the vertebral rotation/rib prominence which causes the cosmetic deformity, nor any associated changes in kyphosis/lordosis, thus representing only a partial record of the overall disease.

Methods of evaluating back shape or cosmetic deformity in scoliosis include measurement of simple parameters such as the height difference between left and right sides of the back during the forward bend test. However, they too attempt to reduce a complex three dimensional shape to a single number which inadequately represents the full clinical deformity. Furthermore, being posture dependent, the consistency of measurement is poor.

An advancement involves optical measurement techniques, with the advantage of avoiding contact with the patient. The Moire fringe technique[1] produces an instant graphical representation of the whole back, rather

like a contour map, but even a small movement of the subject causes the appearance of the picture to change dramatically. The integrated shape imaging system (ISIS) shape measurement and analysis system[2] uses totally safe visible light to scan the back in one second and produces a printed computer analysis of three dimensional backshape within five minutes. Patient posture is not critical and the system can be operated in a normal office environment. An ISIS scan provides the clinician with a repeatable objective assessment of the surface shape of the back in three planes, and consecutive scans can be examined for any indication of progression. Used as a regular screening aid, it has been estimated to reduce the number of spinal radiographs, and thus radiation dose, by as much as 75%. Chief indications for radiographs become at initial presentation to exclude bony anomalies, preoperatively to assess fusion levels, and postoperatively to check instrumentation and fission mass.

Although the exact relation between back surface shape and skeletal deformity is not well defined, surface shape measurements such as ISIS have been shown to be valuable in assessing the progression of a scoliotic curve, the effect of treatment on backshape, and in giving a reliable indication of the need for spinal surgery.[3]

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Opportunistic immunisation in schools may be more effective than immunisation in hospital

EDITOR,—We support Conway's call for immunisation to be given greater emphasis during medical training, and for opportunistic immunisation in hospital to be more widely practiced.[1] However, even in the context of that study, only 43 of 183 eligible children (23.4%) were offered immunisation before discharge. The reasons for this are not stated, but the pressures on the junior doctors asked to administer the vaccinations must have been a significant factor.

Following a sustained decline in the uptake of MMR vaccine,[2] we explored the feasibility of opportunistic immunisation with MMR vaccine during doctors' routine visits to schools to carry out medical inspections. We selected four primary schools from three counties in North Wales during May 1999. Out of a total of 2145 pupils in the schools, born between 1 September 1987 and 31 August 1994, 492 (22.9%) had not received two doses of MMR vaccine based on data held on the Child Health Computer database. The parents of these pupils were sent a letter describing the complications of measles, strongly recommending immunisation, and inviting them to return written consent to immunisation in school, also offering an opportunity to discuss MMR with the school doctor first if required. Of the 492 pupils given letters to take home, the parents of 242 (49.2%) consented and were vaccinated during a single visit to each the four schools.

A large proportion of children in these primary schools had not received two doses of MMR. If the control of measles is to be maintained and the disease eventually eradicated, 95% of children must receive two doses of measles vaccine.[3] Our findings suggest that opportunistic immunisation during routine visits to schools to carry out school medicals would be an efficient and effective method of targeting children who have not received two doses of MMR.

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Viruses in febrile convulsions

Editor,—A study from Japan has produced evidence of human herpes 6 virus (HHV6) infection in 21 of 105 children with febrile convulsions.[1]

Those with HHV6 infection at the time of a first febrile convulsion were younger and more likely to have had a convulsion with complex features (prolonged, clustering, or partial seizure or post-ictal paralysis) than those in whom there was no evidence of HHV6 infection. The authors have suggested that febrile convulsions associated with this virus might be the result of direct invasion of the central nervous system rather than simple fever.

More than 20 years ago, an investigation in north west London showed that a viral aetiology could be implicated in 86% of 73 children after their first febrile convulsion—by combining the results of techniques available for viral diagnosis (tissue culture, mouse inoculation, electron microscopy, complement fixation tests, and interferon assays).[2]

A disseminated viral illness was shown by isolating a virus from the blood, CSF, or urine in 20 (27%) of the children. The viruses isolated included serotypes of adenovirus, echovirus, Coxsackie virus, parainfluenza, measles, and cytomegalovirus. Children with "complex" convulsions had a similar spectrum of viral and bacterial findings as those with simple convulsions. We concluded that many common viruses can invade the blood or CSF, and that systemic invasion produced both fever and convulsion. The viruses identified were common pathogens in infants in our community. The HHV6 virus had not yet been identified, although it was known that roseola infantum was often associated with febrile convulsions in children less than 1 year old, when roseola had its peak incidence.[3]

Today, the expansion of techniques of rapid viral diagnosis, including the polymerase chain reaction, might lead to recognition of more "neurotropic" viruses and further support the proposition that a febrile convulsion occurs when a child with a genetic predisposition is exposed to a systemic virus or bacterium at a susceptible stage in brain development.

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1 Suga S, Suzuki K, Ihira M, *et al*. Clinical characteristics of febrile convulsions during primary HHV6 infection. *Arch Dis Child* 2000;**82**:62–6.
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Failure to thrive

EDITOR,—On reading Charlotte Wright's excellent review of failure to thrive,[1] I was challenged to consider my own impressions of the long term outlook. In preparing a lecture on the subject recently, I undertook a (non-systematic) review of the literature and concluded that the prospects for growth were generally good, with recent studies suggesting that over 75% and 90% of children respectively achieve weights and heights over the 3rd centile. In contrast the outlook for development and behaviour appeared less good: intellectual problems were reported in 15–67% and behavioural problems in 28–48% of children in the studies I reviewed, with evidence from many studies that these difficulties persisted into school age.

My conclusions were therefore directly opposite those of Wright: "Although the growth consequences of FTT seem to be enduring there is now more reassuring evidence about its impact on cognition". From the references quoted by Wright, we seem to be drawing on a similar body of literature, but have come to very different conclusions. I wonder how much this is down to a difference of perspective and hence interpretation? I do not consider a height differential of three quarters of a centile space (5 cm) particularly worrying, but perhaps I am influenced by being 187 cm tall. In contrast, I do consider a cognitive deficit of 1–1.5 standard deviations below population or control group means to be of far greater concern.[2]

Wright's final conclusion that "slow weight gain does have long term consequences" surely must apply both to growth and to developmental and behavioural outcomes. If the long term consequences only related to growth, there would be less need to intervene. If wider effects are accepted, we should continue to strive to identify growth faltering early and to identify effective interventions for this needy group of children.

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1 Wright CM. Identification and management of failure to thrive: a community perspective. *Arch Dis Child* 2000;**82**:5–9.
2 Wolke D. Sleeping across the lifespan. In: Rutter M, Hay DF, eds. *Development through life: a handbook for clinicians*. London: Blackwell, 1994:517–57.

Nut allergy in children

EDITOR,—True and perceived nut allergy is a problem that paediatricians are seeing ever more frequently and guidelines for adequate diagnosis are needed. We therefore published our experience of children presenting to a general paediatric clinic with this problem.[1] In their letter O'B Hourihane *et al* have some points regarding the methodology we used for the diagnosis of nut allergy[2]; we would like to comment on some of those issues.

The assumption that nut was the "only possible allergen" in a composite food was questioned. Although any allergenic food can cross contaminate other foods, severe or fatal allergic reactions are more likely following exposure to peanuts.[3] Thus any child with a known anaphylactic reaction was excluded from an oral challenge on this basis. However the vast majority of children presented with non-specific symptoms where nut ingestion was implicated as the cause of their symptoms. Further investigation was warranted before a diagnosis of nut allergy could be reached.

Skin testing is known to be unreliable,[4] and given the lifestyle implications some parents and children were unhappy to accept a diagnosis of nut allergy on the basis of serum IgE results alone. Under these circumstances oral challenge was offered as a diagnostic investigation.

Our protocol was also criticised for exposing children to only 2 g of nut when a minimum amount of 8 g should have been administered. The maximum amount administered to each child in our protocol was not 2 g, but rather a portion of the same foodstuff reported to have caused the allergic reaction. In addition, O'B Hourihane *et al*,[5] based on previous publications, have suggested that as little as 50 mg of peanut protein is needed in testing for allergic reactions.[3 6] Children admitted for investigation were required to stay for at least one hour after testing; no child tested in this manner has subsequently returned with allergy to nuts.

True peanut and nut allergy is a devastating illness, but fortunately this is uncommon.[7 8] The incidence of perceived food allergy within the UK population however is as high as 20%,[9] and general paediatricians care for the majority of children with possible nut and other food allergies. Clearly treatment of these children without investigation is inappropriate and direct oral challenge has been advocated in some instances.[4] Our key message remains unchanged—in certain circumstances within a controlled environment it is appropriate to consider oral challenge to obtain a definitive diagnosis of nut allergy.

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1 Armstrong D, Rylance G. Definitive diagnosis of nut allergy. *Arch Dis Child* 1999;**81**:175–7.
2 O'B Hourihane J, Warner JO, Bock SA, Sampson SA, Strobel S. Definitive diagnosis of nut allergy. *Arch Dis Child* 2000;**82**:88.
3 Sampson HA. Managing peanut allergy. *BMJ* 1996;**312**:1050–1 .
4 O'B Hourihane J, Roberts S, Warner JO. Resolution of peanut allergy: case-control study. *BMJ* 1998;**316**:1271–5.
5 O'B Hourihane J, Bedwani SJ, Dean TP, Warner JO. Randomised, double blind, crossover challenge study of allergenicity of peanut oils in subjects allergic to peanuts. *BMJ* 1997; **314**:1084–8.
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7 O'B Hourihane J, Dean TP, Warner JO. Peanut allergy in relation to heredity, maternal diet, and other atopic diseases: results of a questionnaire survey, skin prick testing, and food challenges. *BMJ* 1996;**313**:518–21.
8 Colver A, Cant A, Macdougall C. Fatal/severe allergic reaction to food ingestion. *Royal College of Paediatrics and Child Health British Paediatric Surveillance Unit Annual Report* 1998/99, 19–20.
9 Young E, Stoneham MD, Petrukevitch A, Barton J, Rona R. A population study of food intolerance. *Lancet* 1994;**343**:1127–30.

BOOKS

The surgery of childhood tumors. Edited by Carachi R, Azmy AF, Grosfield JL. (Pp 592, hardback; £175.) London: Arnold, 1999. ISBN 03 40692 693 9

With increasing specialisation, it is getting more and more difficult to remain up to date in every field of paediatrics. This is particularly true in paediatric oncology, with the new understanding of chromosomal abnormalities and their aetiological role in cancer. Added to this, there is the problem of following the various protocols of all the multicentre trials being conducted in paediatric oncology. This requires an IQ of at least 160 and a degree in obsessionalism, both of which are lacking in most paediatric surgeons. The paediatric surgeon's main hope is to regularly attend the SIOP Congress, as it circumnavigates the exotic conference centres of the world. Failing that, this book is a useful and cheaper way of updating one's basic knowledge of surgical oncology. It is a concise book covering most aspects of interest to the paediatric surgeon, a bit brief in places, but generally informative. It starts with a useful précis of the genetic background to tumourigenesis and moves on to individual tumours, both common and rare. What it lacks in great detail it makes up for in readability.

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Childhood Epilepsies and Brain Development. Edited by A Nehlig, J Motte, S L Moshé, P Plouin. (Pp 311; hardback: £59.) John Libbey, 1999. ISBN 0 86196 578 7.

The challenge of the developmental epilepsies is multifaceted. As clinicians, we have all spent time trying to control the anxiety we feel because of our impotence in the face of intractable epilepsy. The mainspring of our concern is usually not the paroxysmal events themselves but the quite unpredictable cognitive impairment which may accompany these events. Despite great strides being made in our understanding of epilepsy in recent years, we are still profoundly ignorant of the crucial relationship between seizure disorder and learning disability. Defining the pathogenetic mechanism of cognitive impairment and developing possible preventions or treatments for these children with epilepsy is of paramount importance to scientists and clinicians alike and is probably *the* challenge for those studying and treating developmental epilepsies.

This volume is compiled from the proceedings of a meeting held in Alsace in 1997 that brought together clinicians and scientists involved in the diagnosis, treatment and research of childhood epilepsies. The theme of the meeting was the inter-relationship between brain development, epilepsy and learning disability, and the conference was an important stepping stone in establishing a dialogue between the different disciplines.

Age related mechanisms involved in the development of seizures and "windows" of vulnerability for developing chronic epilepsy are closely considered. A fascinating multiauthor chapter on neuronal migration disorders, suggesting that neocortical structural abnormalities, due to developmental disturbances, are often the cause of treatment resistant seizure activity is followed by several chapters discussing laboratory details of producing an animal model with neuronal migration disorder and refractory epilepsy. Some of the scientific detail is rather heavy going for the clinician, but the message, of course, is that since clinical studies are technically limited in the analysis of the cellular mechanisms underlying hyperexcitability, rat models of cortical dysplasia are invaluable.

This, in fact, is the pattern of the volume, where we are treated to some excellent clinical chapters (for example, those on the malignant epilepsies, or the effects of asphyxia on the developing brain), which are then followed by scientific chapters attempting to characterise the molecular neurobiology underlying some of our observations.

The clinician will discover some gems in this book, but he will also need to plough through a swathe of scientific information, the relevance and interpretation of which is not always immediately obvious. In some sections the conversation between scientist and clinician is stilted, but there are some passages where the dialogue sparkles (for example, chapters 3, 5, 11, and 21). One is left with a feeling that trying to get on the same wavelength and trying to speak the same language, though challenging, benefits all of us, and of course it is the child with epilepsy and cognitive impairment who will be the ultimate beneficiary of the exercise.

Such multidisciplinary conferences and the volumes they spawn are clearly one of the ways forward in unravelling the complex developmental epilepsies.

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Diseases of the liver and biliary system in children. Edited by Kelly DA. (Pp 375, hardback; £99.50.) Oxford: Blackwell Science Ltd, 1999. ISBN 0 632 04802 6

Complete textbooks about liver disease in children are few compared to the literature available to our colleagues on adult liver disease. Yet because of the broad spectrum of illnesses associated with liver dysfunction in children, and the rapid developments in the speciality over the last 20 years, all paediatri-

cians need a basic knowledge of this area. This book covers the wide spectrum of liver disease in children in a practical, clinical format. It dispenses with indepth descriptions of the pathophysiology of liver disease and concentrates on providing up to date clinical information. There are numerous tables, bullet lists, and flow charts, which give a very didactic but accessible style.

Perhaps the most important chapter in a book about paediatric hepatology is that describing the jaundiced newborn infant, a clinical scenario encountered regularly by neonatologists and general paediatricians as well as those interested in liver disease. Appropriate investigation of jaundice or abnormal liver function tests in neonates may be the only route to early diagnosis and treatment of conditions as diverse as hypopituitarism and biliary atresia. Eva Roberts' chapter "The jaundiced baby" is a thorough exposition of the diagnostic approach to this topic. There is enough detail about each condition to show why it should be considered in the differential diagnosis of prolonged neonatal jaundice, but the clinical message is not swamped by speculations on aetiology. It is complimented by the following chapter about the acutely ill baby presenting as true or apparent liver failure. Here there is an emphasis on metabolic diseases, and a clear alogorithm for initial investigation of these babies.

Unusual chapters include those on "Skin disorders in liver disease" and "Dental care of children with liver disease". They mainly describe problems in the post-transplant, immunosuppressed child, again both reflecting the very practical approach of the book. The atlas section at the back provides 75 plates with excellent clinical photographs, but some of the radiological investigations and histology pictures are poorly demonstrated. Common to all multiauthored books, there is some overlap between the chapters, which is not always clearly cross referenced in the text. This can be frustrating when the reader is trying to quickly check the facts on a specific condition.

In summary, this book provides a good overview of paediatric hepatology for the non-specialist. The style reflects the broad clinical experience of the authors. The information is presented in a clear, uncluttered fashion, but with adequate references at the end of each chapter for those who wish to explore a topic in greater depth.

PATRICIA MCCLEAN
Consultant Paediatrician

CliniCAL paediatrics. By McWilliams A, Wright IC. (Software; £110.) London: Hodder and Stoughton Publishers, 1998. ISBN 0 41282 170 2

Computer aided learning could be everywhere: everyone has access to a computer (and some even carry them around with them) and uses them all the time. It hasn't taken off though, and this package, good as it is, won't be widely used.

CliniCAL paediatrics is a lovely programme. The learner is taken through a series of patients' histories, and must make decisions on diagnostic, treatment, and prognostic aspects of the case. The layout is clear and easy to use. The illustrations in the imaging module are excellent.

The major flaw is in the lack of guts to the package.

Four areas are presented, with a heavy neonatal bias. There are six cases in each of the lung disease and bone disease modules, five chest imaging cases (I'd never heard the diagnosis of four of them before running this programme), and a single ethical case. I'm certainly not suggesting this package is a triumph of style over content. The content is good but skimpy. (Akin perhaps to wearing a fur coat and G-string rather than no knickers.)

The stated aim of the package is "to make the DGH consultant paediatrician better at . . .". It's good for an SHO to learn from, being used to didactic instruction and the partial use of the published literature to support clinical decisions. It may be quite annoying when one is more senior.

A couple of very minor changes to the way the programme runs, and a massive expansion in the areas of medicine covered, would make this excellent. As it is, it's a good tutorial for select areas of neonatal medicine, but nothing more.

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CORRECTION

We are grateful to Dr Theo Fenton from Mayday University Hospital, Surrey, for pointing out the following errors:

Kamat, *et al.* **Raised serum transaminases: not always liver disease**. *Arch Dis Child* 2000;**82**:270. The letter referred to a non-existent enzyme, creatinine kinase. It should, of course, have been creatine kinase.

Thayyil Sudhan S, Gupta. **Dipstick examination for urinary tract infection**. *Arch Dis Child* 2000;**82**:271. More significantly, Dr Fenton points out that this letter confused nitrates with nitrites throughout.