LETTERS TO THE EDITOR

Recurrent apparent life threatening events and intentional suffocation

EDITOR,—Intentional suffocation is a difficult diagnosis. Children often appear well in between such episodes and cannot tell their paediatrician that they are being intermittently assaulted. The evidence for intentional suffocation is often circumstantial, which means that doctors may have difficulty substantiating the diagnosis in court without evidence from covert video surveillance. We are surprised, therefore, that Davis *et al* state that a high proportion of suffocation victims were protected effectively without covert video surveillance.¹ It may be that these cases were not contested or had evidence of other harm to the child in addition to the suffocation.

Davis *et al* reinforced the British Paediatric Association working party guidelines that "when there is a very high risk, children should not be exposed to danger simply to achieve a criminal standard of proof."² Covert surveillance has been used in our knowledge primarily to confirm the diagnosis and to ensure effective protection for the child and siblings. Even in civil proceedings, which rely on a balance of probabilities, the more serious the allegations of abuse, the higher the level of evidence the court requires. In a number of cases of suspected suffocation, we have seen children inadequately protected because of a lack of firm evidence from surveillance. Medical experts with little experience of suffocation may fail to convince a judge or confound proceedings with clinical controversies. Inadequate evidence may then result in children suffering long term physical or emotional harm.

We suggest that all infants with recurrent apnoeic-cyanotic episodes are investigated within units that can both perform multi-channel recordings in hospital or at home, preferably during events for diagnostic purposes, and be open to considering the possibility of abuse. In the latter situation, clinicians should follow appropriate child protection procedures, including the use of confidential multiagency strategy meetings and, if required, covert video surveillance.

An approach that aims to record clinical events may help some children with recurrent events avoid unnecessary medical treatment or surgery. These have often been given as a result of abnormal investigations between episodes (for example, fundoplication for reflux). Searching for the pathophysiology during episodes may mean covert surveillance is used earlier in selected cases to ensure that suffocation is confirmed and not dismissed in the court room.

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1 Davis P, McClure RJ, Rolfe K, *et al*. Procedures, placement, and risks of further abuse after Munchausen syndrome by proxy, non-accidental poisoning, and non-accidental suffocation. *Arch Dis Child* 1998;78:217-21.

2 British Paediatric Association. *Report of a working party on evaluation of suspected imposed upper airway obstruction*. London: BPA (now the Royal College of Paediatrics and Child Health), 1994.

Dr Davis and colleagues comment:

We agree with much of what Dr Samuels and Professor Southall say. Deliberate suffocation must be considered in the differential diagnosis of apparent life threatening events (ALTE) in infants. All paediatricians managing cases of this type must be aware of this possibility and be prepared to follow their local child protection procedures.

Clearly in these cases, paediatricians need the best quality evidence, and if definitive evidence exists, then this is most helpful. It is our view, however, that fabricated illness should primarily be a clinical diagnosis. There is often a wide range of medical factors in the child's background as well as ALTE, and there are usually features in the medical histories of siblings. Close examination of this background can usually enable a clear diagnosis on clinical grounds (on the balance of probabilities). Where a clinical diagnosis can be reached it is appropriate to act without subjecting the child to the risk of further harm to obtain definitive evidence.

Covert video surveillance is undoubtedly both ethical and clinically indicated where there is genuine clinical uncertainty about whether the child is suffering "medical" life threatening events, or imposed ones, and where a careful review of the clinical history of the whole family does not support a diagnosis of fabricated illness. However, the fact that covert video surveillance revealed abuse in 33 of 39 suspected cases in Southall's own report suggests that the clinical criteria used to select children for covert video surveillance were in fact good indicators of abuse in themselves.¹ This work has been instrumental in bringing deliberate suffocation of children into the public domain and acknowledging that it is an important variety of child abuse. It has also clarified considerably the clinical elements of diagnosis. In light of these reports it is probable that covert video surveillance has become less necessary and a clinical diagnosis without it should now be more acceptable.

Covert video surveillance was used in only a quarter of the cases of non-accidental suffocation we surveyed as part of the British Paediatric Surveillance Unit (BPSU) study. Of the 26 survivors of suffocation abuse followed up, all of them were initially protected by removal from the home or separation from the perpetrator. At follow up approximately two years later, only three of these 28 children had been allowed home without major conditions being applied (usually involving the exclusion of the perpetrator). Most of these children were subject to Care Orders.

Most of these children were, therefore, protected even though there was no evidence from covert video surveillance. Obviously, the follow up duration and the fact that we obtained our information from paediatricians means we can only draw limited conclusions about the risks of further abuse.

It was not our intention to suggest that clinicians were performing covert video surveillance purely to obtain evidence for a prosecution. However, the BPSU study and anecdotal experience suggest that where covert video surveillance evidence is available prosecution is usual. We agree with Professor

Southall that prosecution has various benefits, but Children Act proceedings should allow the protection of children perfectly adequately in most cases.

It is, of course, entirely appropriate for diagnoses of child abuse, clinical or otherwise, to be challenged in court. The complexity of cases of fabricated illness is such that only paediatricians with a major research background in this area should be undertaking these expert assessments. Courts seem, quite rightly, to be becoming more selective in this respect.

We agree that covert video surveillance requires specialist facilities and clinicians who are able to consider both organic and abuse diagnoses. However, we feel that many of these children can be diagnosed on clinical grounds and that not all infants suffering recurrent ALTE would need this service.

1 Southall DP, Plunkett MCB, Banks MW, Falkov AF, Samuels MP. Covert video recordings of life-threatening child abuse: lessons for child protection. *Pediatrics* 1997;100:735-60.

Rectal biopsy in the investigation of constipation

EDITOR,—Ghosh *et al* concluded that using rectal biopsy to investigate constipation and, thus, rule out Hirschsprung's disease, is unnecessary if symptoms begin after the neonatal period.¹ We agree with the authors that in most cases there is a history of constipation in the first month of life.

However, we saw two children recently whose symptoms began at four and five months old, respectively. They had suction rectal biopsy and were subsequently diagnosed with Hirschsprung's disease, with excellent results after surgery. The parents were unable to provide a neonatal history. A Belgian review supports the view that symptoms can occur first after 1 month of age; they reported that constipation starts, on average, at 38 days.² Thus, we feel that rectal biopsy may still be useful in atypical cases of infantile constipation.

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2 Reding R, de Ville de Goyet J, Gosseye S, *et al*. Hirschsprung's disease: a 20-year experience. *J Pediatr Surg* 1997;32:1221-5.

EDITOR,—Ghosh and Griffiths¹ reviewed 141 diagnostic rectal biopsy samples to develop criteria for performing the procedure in children with intractable constipation. They concluded that a biopsy sample was unnecessary if the child was not in the neonatal period (older than 28 days) at onset of symptoms. This conclusion was based on the study of 17 children with Hirschsprung's disease who had developed symptoms within the neonatal period.

We performed a retrospective study of 122 patients with histologically proven Hirschsprung's disease who were treated in our department between 1988 and 1994 (7 years). We found that 8% of our patients had no symptoms in the neonatal period; age at

onset of symptoms was between 2 and 36 months (mean 7.5 months); this is not a new finding.^{2,3}

Failure to perform biopsy samples in children with late onset of symptoms may delay the eventual diagnosis and could increase the risk of developing enterocolitis. We feel that the study from Southampton¹ is based on a very small cohort of patients with Hirschsprung's disease and that the authors' conclusion regarding the indication for rectal biopsy is incorrect.

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- 1 Ghosh A, Griffiths DM. Rectal biopsy in the investigation of constipation. *Arch Dis Child* 1998;79:266-8.
- 2 Wheatley MJ, Wesley JR, Coran AG, et al. Hirschsprung's disease in adolescents and adults. *Dis Colon Rectum* 1990;33:622-9.
- 3 Swenson O, Sherman JO, Fisher JH, et al. Diagnosis of congenital megacolon: an analysis of 501 patients. *J Pediatr Surg* 1973;8:587-94.

Dr Griffiths comments:

We reviewed three years of rectal biopsies, and Landman¹ reviewed five years of rectal biopsies in children with constipation; 41 children were studied and there were no cases of Hirschsprung's disease that started after the neonatal period.

Babu *et al.*, state that 8% of their patients had no neonatal symptoms and that this is not a new finding. Wheatley and colleagues did not study the onset of symptoms, but found that "each patient had a history of abdominal distension as well as severe, lifelong constipation necessitating regular cathartics or enemas".² Swenson³ conducted a 16 year review of 501 cases of Hirschsprung's disease, in three hospitals, which ended in 1973. He does not comment on when symptoms began, and reports the passing of meconium in only 268 children; this suggests that retrospective neonatal data collection was difficult. However, study of the birth history of the children (which is of unspecified length), revealed that 93% (453 of 495) were constipated and that 87% (316 of 356) had abdominal distension.

Dr Wilschanski and colleagues cite the paper by Reding,⁴ a 20 year review which studied the outcome of surgical management, with the biopsy date for completeness. Unfortunately, Reding *et al.* confused age of presentation with age of onset of symptoms (age at presentation in the legend, and age at onset in the table). Thus, we emphasise that the age of presentation is irrelevant, and in our study, it ranged from 1 day to 3 years. However, we found onset of symptoms in children with Hirschsprung's disease was always within the neonatal period.

We reiterate that if a detailed history focusing on the neonatal period is taken, many children whose constipation began after the neonatal period could avoid unnecessary biopsy, and the most cases of Hirschsprung's disease will be found (in our 41 cases, 100%).

However, if there are other reports of patients with Hirschsprung's disease who were genuinely asymptomatic in the neonatal period, a greater proportion of babies will need a biopsy.

- 1 Landman GB. A 5 year chart review of children biopsied to rule out Hirschsprung's disease. *Clin Paediatr* 1987;26:288-91.
- 2 Wheatley MJ, Wesley JR, Coran AG, et al. Hirschsprung's disease in adolescents and adults. *Dis Colon Rectum* 1990;33:622-9.
- 3 Swenson O, Sherman JO, Fisher JH, et al. Diagnosis of congenital megacolon: an analysis of 501 patients. *J Pediatr Surg* 1973;8:587-94.
- 4 Reding R, de Ville de Goyet J, Gosseye S, et al. Hirschsprung's disease: a 20 year experience. *J Pediatr Surg* 1997;32:1221-5.

Infant feeding and atopic disease

EDITOR,—I was offended by David's observation¹ that many health professionals are "no more equipped to breast feed than they are to give practical advice on the subject" and that the middle class solution would be to "close the door to the health visitor" and call in someone more appropriate.

Health visitors are skilled practitioners and, apart from during the initial 10 day period when midwives are still involved, do more work with new mothers to promote breast feeding than any other health professional, and are aware of the benefits of breast feeding to the infant as well as the mother. However, in the postnatal period it is important that health visitors establish a long lasting professional relationship with the mother. We lose their favour, trust, and respect forever if they think that we are forcing them to make decisions or to continue breast feeding when they are desperate to stop.

Contrary to David's opinion, health visitors are not ill equipped to offer breast feeding advice. However, we work with the mother to help her adopt the type of feeding with which she is most comfortable. As a result, the mother may choose to change from breast to bottle feeding or, indeed, to mix the two types of feeding. Mothers are aware of the benefits of breast feeding to themselves and to the baby, but sometimes they prefer one type of feeding to another because it is more compatible with their lifestyle and commitments. As yet, I have had no success in helping a mother to continue breast feeding when she wants to stop, despite preparations made in antenatal classes.

It is unlikely that doctors will see mothers who feel they should breast feed even though they are frustrated by the baby's demands, and despite the health visitor's reassurance that it is normal for infants to feed almost constantly for long periods in the first weeks after the birth. These women may be exhausted and tearful and may admit to resenting their baby when it cries. All possible help and reassurance cannot persuade them to continue breast feeding, and they feel intense relief when they are given permission to introduce formula milk and, thus, the relationship between mother and baby instantly relaxes and improves.

I feel strongly, as do many of my colleagues, that the most important factor in the first weeks after the birth is that the mother and baby enjoy each other. Therefore, the mother should be able to stop breast feeding if her desire to do so outweighs the pleasure of her baby. Mothers are aware of the long and short term benefits of breast feeding and feel immense guilt when they realise they want to stop. Who are we to make them feel even more guilty?

David should reconsider his comment that health visitors are worse than breast feeding counsellors, as sometimes the worst teacher is a woman who believes that she has had the

best experience of childbirth or breast feeding and that others should adopt the procedures she found most suitable. It is a surprising comment for a doctor to make, as most health care professionals are familiar with the misconception that one should have had the condition oneself to be a good carer.

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- 1 David TJ. Infant feeding causes all cases of asthma, eczema, and hay fever. Or does it? *Arch Dis Child* 1998;79:97-8.

Dr David comments:

In reply to Ms Dion's comments, the following points may be helpful:

- The statement that some families opt for the support of—for example, a National Childbirth Trust trained breast feeding counsellor rather than a health visitor when seeking advice on breast feeding, does not imply criticism of health visitors. It is simply a factual observation.
- It is well known that the quality of practical advice given to breast feeding mothers by all types of health care professionals (including doctors, midwives, and health visitors) is highly variable and often very poor.^{1,2} This may be one reason why some mothers prefer to seek the help of non-health care professionals who fulfil the dual criteria of either having successfully breast fed their own infant or having received training in how to advise breast feeding mothers.
- It is plainly unhelpful to try to force reluctant mothers to continue breast feeding, or to try to impose one's own ideas as to what is correct.
- Having breast fed one's own baby is unlikely to be the best basis for giving advice to other breast feeding mothers who are having problems. Training is essential. Women who have breast fed their own babies, and who have been trained to give practical breast feeding advice, are a valuable resource in the training of health professionals.³
- Ms Dion says that mothers and babies should enjoy one another. I fully agree that this is of fundamental importance. I also fully agree that a mother who wishes to give up breast feeding should be allowed to do so, though I am saddened when I see that this has happened largely as the result of simple problems that have been inadequately or incorrectly addressed by misinformed health professionals.
- It is correct to state that one does not have to have had a particular condition in order to care for someone with that condition. Nevertheless, I cannot comment on whether breast feeding is a condition. However, personal experience should not be dismissed as it can afford special insights—for example, as a paediatrician I have learned an enormous amount from having children of my own.

- 1 Beeken S, Waterston T. Health service support of breast feeding: are we practising what we preach? *BMJ* 1992;305:285-7.
- 2 Malik ANJ, Cutting WAM. Breast feeding: the baby friendly initiative. *BMJ* 1998;316:1548-9.

- 3 David TJ, Taylor R, Buckley A. Teaching medical students the practical aspects of breast feeding. *Maternal and Child Health* 1981;6: 140-4.

Albumin: saint or sinner?

EDITOR,—I disagree with the conclusions of Nadel *et al* that albumin should remain the first choice as a resuscitation fluid in sepsis.¹

The objections have been fairly highlighted by the authors:

- It is a blood product, which may carry an infection risk
- Blood products are in constant short supply
- It costs substantially more than starch without any proven benefit towards the final patient outcome but with added risks
- Although albumin is relatively impermeable to the endothelial barrier under normal conditions, it leaks relatively easily into tissues of critically ill patients.²

Clearly trials are required to address this issue. Meanwhile, there is ample experimental and clinical evidence suggesting that hydroxyethyl starch (HES) of 250 kilodaltons size (pentastarch) is more effective than albumin in reducing capillary permeability oedema in experimental and clinical models of permeability oedema.³ In addition, it has been suggested that the molecular size of the 250 kD HES is optimal for use in capillary leak and sepsis.⁴ It works not only because of its oncotic pressure effects but may retain water in the circulation better by sealing endothelial gaps. The 250 kD HES has also been shown to have no adverse effect on clotting.⁵

Gelatins do not have the same virtues as HES as they have a short half life with rapid leak into interstitial space and have poor colloid osmotic function.

In our paediatric intensive care unit we stopped using albumin more than a year ago. Our standardised mortality (as well as Nadel *et al*'s) is lower than predicted. Does it follow that the choice of colloid has no influence on the survival of a critically ill child? If so, why use a product with so many potential drawbacks?

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- 1 Nadel S, De Munter C, Britto J, Levin M, Habib P. Albumin: saint or sinner? [annotation]. *Arch Dis Child* 1998;79:384-5.
- 2 Wardrop CA, Holland BM, Jacobs S, Jones JG. Optimization of the blood for oxygen transport and tissue perfusion in critical care. *Postgrad Med J* 1992;68(suppl 2):2-6.
- 3 Yeh T Jr, Parmar JM, Rebecka IM, *et al*. Limiting edema in neonatal cardiopulmonary bypass with narrow-range molecular weight hydroxyethyl starch. *J Thorac Cardiovasc Surg* 1992;104: 659-65.
- 4 Waxman K, Holness R, Tominaga G, Chela P, Grimes J. Hemodynamic and oxygen transport effects of pentastarch in burn resuscitation. *Ann Surg* 1989;209:341-5.
- 5 Rackow EC, Mecher C, Astiz ME, Griffel M, Falk JL, Weil MH. Effects of pentastarch and albumin on cardiorespiratory function and coagulation in patients with severe sepsis and systemic hypoperfusion. *Crit Care Med* 1989; 17:394-8.

Transitional care of young disabled people

EDITOR,—Fiorentino *et al* have tried to quantify the widely acknowledged view that the care of young disabled adolescents falters

during their transition to adulthood.¹ They have arrived rightly at conclusions which, when implemented, should improve transitional care of chronically disabled young people; this level of care has been delayed in its development by a lack of enthusiasm and initiative by health professionals and their organisational bodies. Furthermore, a significant number of young people with chronic health problems do not have a "statement of special educational needs".

Last year, I presented findings of a survey of transitional care of chronically ill young people in the South Thames region at a national meeting of community paediatricians.² This survey was questionnaire based, and 161 practising paediatricians and consultant child psychiatrists from the South Thames region took part. The questions dealt with the current level of satisfaction with transitional care and planned changes in this area as perceived by the participants. The data showed that while only 32% were either frequently or always satisfied with the current transitional care for disabled adolescents, only 50% were working towards changing the situation. The lack of equivalent adult services were cited frequently as an unmet need.

Recently, the UK Royal College of Physicians³ and the American Academy of Pediatrics⁴ issued guidance in the organisation of transitional care. We should therefore take this opportunity to persuade the Department of Health and primary care groups to provide organised transitional care for disabled young people. During the past 20 years, the survival of young chronically ill people has improved greatly due to the dedication of, and hard work by, carers as well as health professionals. It would be a disservice to these young people and their carers if we fail to improve service provision during their transition to adulthood.

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- 1 Fiorentino L, Datta D, Gentle F, *et al*. Transition from school to adult for physically disabled young people. *Arch Dis Child* 1998;79:306-11.
- 2 Sarmah A, Fowler A. Transitional care of chronically sick adolescents: how does it happen in the South Thames region? British Association of Community Child Health Annual General Meeting 1998.
- 3 Kurtz Z, Hopkins A. *Services for young people with chronic disorders in their transition from childhood to adult life*. London: Royal College of Physicians, 1996.
- 4 American Academy of Pediatrics. Transition of care provided for adolescents with special health care needs. *Pediatrics* 1996;96:1203-6.

EEG and epilepsy

EDITOR,—We welcome the debate on the timing of EEG examinations.¹ The practice of delaying the EEG until after a second seizure began when epilepsy syndromes were unrecognised. The EEG was then of little help in predicting prognosis or guiding treatment, but has been transformed by the recognition of epilepsy syndromes. Although some of these syndromes may be difficult to diagnose,² others—for example, benign childhood epilepsy with centrotemporal spikes, and juvenile myoclonic epilepsy, have easily recognised clinical and EEG features.³ It may not be possible to predict seizure recurrence in a child who has had a nocturnal tonic

clonic seizure, but if the EEG shows centrotemporal spikes, we can reassure the family that if recurrent seizures occur, they will almost certainly remit and the cognitive outcome will be good. Similarly, a child presenting with a tonic clonic seizure whose EEG shows generalised multiple spike and wave discharges and photosensitivity, can be advised on the likelihood of seizure recurrence, and specific measures to reduce seizure frequency. These are common clinical situations and the advice is specific and practical. Why delay giving it?

A further argument about how to define epilepsy is often used to delay EEG examinations. If epilepsy is a condition where a person experiences recurrent epileptic seizures,² it can be argued that a person who has had a single seizure is not epileptic and, therefore, does not need an EEG. However, the clinical usefulness of this definition should not be overemphasised. Epilepsy is a symptom of a number of different conditions whose only common feature is an increased susceptibility to seizures. This can be shown by the occurrence of recurrent seizures or characteristic EEG abnormalities. In the future, a specific gene or ion channel defect may be the strongest predictor of a decreased threshold. An EEG should not be used to diagnose epilepsy, but its role lies in helping to diagnose which type of epilepsy the patient has.

A third argument for delaying EEG examinations is the quality of EEG services. It is undoubtedly dangerous to rely on a poorly performed and incorrectly interpreted EEG. Is it safer to rely on such an EEG after a second seizure? This is an argument for improving services rather than an argument against adopting best practice.

There are few circumstances in which an EEG would be not appropriate when a child is considered to have had an epileptic seizure.

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- 1 Panayiotopoulos CP. Significance of the EEG after the first afebrile seizure. *Arch Dis Child* 1988;78:575-7.
- 2 Cross JH. Commentary. *Arch Dis Child* 1988;78: 577-8.
- 3 Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for revised classification of epilepsies and epileptic syndromes. *Epilepsia* 1989;30: 389-99.

Attention deficit hyperactivity disorder

EDITOR,—In the management section of his paper on attention deficit hyperactivity disorder (ADHD) which focuses mainly on medication,¹ Hill gives the erroneous impression that managing hyperactivity by diet is difficult and ineffective: those who use it regularly to control ADHD know that this is not the case.

Standard diets are not helpful in the management of this disorder because the foods which provoke hyperactive behaviour are different for each child. Few parents succeed in identifying the foods which affect their child without help, but an elimination diet is effective in most cases. Deficiencies of omega 6 essential fatty acids are common in these children² so, in addition to the calcium supplement given to all children avoiding milk, evening primrose oil, borage oil, and cofactors such as zinc, are also usually

recommended. Under this regimen, the hair-raising first consultation with the child is often followed by a quiet and cooperative second or third visit. If they have had help with finding alternative foods, most parents find it surprisingly easy to keep the child to the diet most of the time after the first few weeks because the child usually prefers to feel well.

There are three good trials³⁻⁵ which report substantial improvement and significant reduction of Connor's scores in hyperactive children on a hypoallergenic diet; over 70% of children responded in each study. The results were confirmed with double blind placebo controlled challenges³⁻⁵; significantly higher scores were recorded during periods on challenge food rather than on placebo. These trials did indeed use a stringent few-food hypoallergenic diet during the investigative phase, with very slow challenges. However, in the clinic, if you start by taking a good history (preferably when the child is not present), it is often possible to see good results in hyperactive children fairly quickly and with relatively few exclusions.

The principles of elimination dieting are set out in a recent text.⁶ Provoking foods or food additives are usually those which are eaten frequently; it is rare for a single item to be responsible for ADHD. Most of the probable provoking substances must be avoided completely and at the same time to get good results. It may be sufficient to avoid additives (especially colours and preservatives in food, drink, medicine, and toothpaste), chocolate, milk, and orange, to which most hyperactive children react.⁴ However, reactions to cheese, wheat, and other fruit are also common and any food may provoke hyperactivity, especially if eaten frequently. An improvement is often seen in children within 3-7 days, and single open oral challenges are usually sufficient if given within three weeks. Foods which cause a distinct deterioration in behaviour should be avoided for several months, by which time they can often be tolerated if not eaten too frequently. The diet may relieve other conditions—for example, glue ear or abdominal pain, which are also present in many of these children^{2,6}; in boys with eczema it is sensible to arrange challenges under supervision as there have been reports of anaphylaxis. Finally, the nutritional quality of longer term diets should be checked by a dietician.

If the diet is effective, behaviour often reverts to normal, to the great relief of all concerned. In view of the potential toxicity of medication in children and its limited effectiveness, all families with hyperactive children should be offered help in detecting offending foods. It is more appropriate to reserve medication for those who fail.

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- 1 Hill P. Attention deficit hyperactivity disorder. *Arch Dis Child* 1998;79:381-5.
- 2 Stevens LJ, Zentall SS, Deck JL, et al. Essential fatty acid metabolism in boys with attention-deficit hyperactivity disorder. *Am J Clin Nutr* 1995;62:761-8.
- 3 Egger J, Graham PJ, Carter CM, et al. Controlled trial of oligoantigenic treatment in the hyperkinetic syndrome. *Lancet* 1985;i:540-5.

- 4 Carter CM, Urbanowicz M, Hemsley R, et al. Effects of few-food diet in attention deficit disorder. *Arch Dis Child* 1993;69:564-8.
- 5 Boris M, Mandel FS. Foods and additives are common causes of the attention deficit hyperactive disorder in children. *Ann Allergy* 1994;72:462-8.
- 6 Anthony H, Birtwistle S, Eaton K, et al. *Environmental medicine in clinical practice*. Southampton: BSAENM Publications, 1997.

Professor Till comments:

Of course an elimination diet works for some children with attention deficit hyperactivity disorder; I use it myself. I agree that there is good evidence for the effectiveness of the few-food diet, but its effect size is small compared with that of medication. Parents committed to dietary treatment have usually tried it. Those who are uncommitted may find it cumbersome. I know of no convincing hard evidence that confirms an allergenic mechanism. The approach recommended by your participants is a mixture of elimination diets, additive restriction, and magic ingredients, which I do not think has been tested scientifically. Nevertheless, I expect that borage oil is very nice.

Doctors as expert witnesses

EDITOR,—Most doctors do not like giving evidence in court, and giving an opinion is becoming more difficult as advocates appear to be increasingly hostile, and judges non-interventional. As care courts have become more adversarial it may be easier to appear as a witness in the criminal court where the rules of evidence are clear. Following the Children Act 1989 the emphasis was to be inquisitorial with "... the child's welfare is paramount" as the fundamental tenet.

Contrary to this concept of the Children Act a group of doctors exists who do not appreciate the need to give evidence in an even handed way and to give all the evidence without omissions (Re AB¹ and Re R²).

The Expert Witness Group³ was established to make clear a doctor's credentials, training, practice, and research interest. A standard form is available to the courts which clarifies the doctor's expertise.

The pretrial meetings in care proceedings aim to clarify and simplify medical opinion to smooth the legal proceedings⁴ and "reduce or obviate the need to attend court". A schedule of agreement and disagreement is drawn up, there may be no significant differences of opinion yet the doctors are still called to court. Why meet, one wonders? There is a debate as to who should chair these meetings (I do not know any practising doctor with the time and secretarial back up to undertake this work).

It is galling that the courts take doctors' time so casually; often spending hours in the witness box—often far longer than with the child and family. What can be done?

- the full implementation of the Pigot report⁵ would allow more children to be heard in criminal proceedings
- universal introduction of the Expert Witness Group's form would clarify the expertise of the witness
- pretrial liaison is useful but only if there is a reduction in doctors' hours in court
- referral of more cases to lower courts to be heard by appropriately trained magistrates
- more joint training between paediatricians and advocates (at all levels).

With more discussion and training paediatricians will become better advocates for children.

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- 1 Re AB. (Child Abuse: Expert Witnesses) [1995] 1 FLR 181.
- 2 Re R. (A Minor) (Experts' Evidence) [1991] 1 FLR 291.
- 3 The Expert Witness Group. *Expert Witness Pack*. Bristol: Jordan Publishing, 1997.
- 4 David TJ, Hershman DA, McFarlane AE. Pretrial liaison between doctors in alleged child abuse. *Arch Dis Child* 1998;79:205-6.
- 5 Advisory Group on Video Evidence. *Pigot Report*. London: Home Office, 1989.

Pretrial liaison between doctors in alleged child abuse

EDITOR,—We were interested in the annotation on pretrial or precourt hearing liaison between doctors acting as expert witnesses in the cases of alleged child abuse.¹ We agree that there is considerable utility in this approach as the following case illustrates.

A set of female triplets were initially referred to one us (DH) for review and examination following a disclosure that they had been sexually abused by their father. The children were examined by colposcopy using a standard technique of gentle labial traction.² Still photographs of the findings were made with the informed consent of the mother who understood that they would be used for teaching and training purposes only. The examination findings were consistent with previous sexual abuse, but there was neither disclosure of, nor findings compatible with, recent trauma.

The photographs were subsequently obtained and discussed at a peer group review meeting held by North East London paediatricians who work in child protection at which we were present. There was a debate as to whether there were changes in the hymens of the triplets consistent with sexual abuse or whether the appearances were due to congenital abnormalities of the hymen. Unknown to the peer group members, one of us (JW) had been asked by the official solicitor to review the medical evidence.

To resolve these important issues it was agreed that there should be a joint medical examination by DH who described the initial findings, JW who had been asked by the official solicitor to comment on the medical report, and VL who gave an independent opinion. The mother and children gave consent. The court granted permission for the medical to be part of a "precourt" review. At the examination we agreed that in two girls the findings were diagnostic of sexual abuse and in the third strongly supportive of it. No doctor had to give evidence in court.

These cases illustrate three points. First, the utility of the peer group review; second, the difficulties that may arise when photographic or even video evidence is used; and third, the positive benefit of joint examination by experienced paediatricians. None of the girls reacted adversely to the process and we were able to discuss the findings with the family and other child protection agencies.

We believe that this case highlights the importance of pretrial liaison in child protection work and illustrates the therapeutic benefit of the joint approach.³

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The way ahead with meningococcal disease

EDITOR,—Hoare has recently highlighted the fragmentation of research into meningococcal disease, and suggested adopting the model of the Medical Research Council trials in acute leukaemia as a potential way forward.¹ Such suggestions are timely as last year meningococcal disease became the most common cause of death in children between the ages of 2 and 5 years.

The Medical Research Council trials in acute leukaemia clearly demonstrated improved survival for children enrolled in collaborative trials and for those cared for in participating centres.² Before these trials the outlook for children with leukaemia was bleak; case fatality was in the order of 70% with children receiving diverse management in a wide range of units. Improving the outcome for meningococcal disease requires a similar approach but is more challenging because of three main differences:

- the sickest children with meningococcaemia are already cared for in a regionalised network of paediatric intensive care units each seeing at least 20 such cases per year
- the case fatality rate of meningococcal disease is already low and declining: from 15% in 1989 to 10% (or less in some centres) in 1997
- in meningococcal disease we are dealing with a rapidly progressive acute illness where early management is crucial to outcome.

A forum of excellence for furthering the management of meningococcal disease could easily be developed as a joint venture between the Paediatric Intensive Care Society, the Royal College of Paediatrics and Child Health, and the Public Health Laboratory Service. The UK Children's Cancer Study Group has already shown us that a simple process of registration can lead to improved patient survival through the introduction of standardised treatment approaches in participating units.

A similar approach could be followed with dissemination of basic protocols through paediatric intensive care units, and the role of novel treatments could be clarified through the application of relevant clinical trials. For this to be successful a group methodology would need to be adopted aimed at achieving consensus. Many fundamental questions require unequivocal answers in meningococcaemia including: the role of human albumin in fluid resuscitation³; the optimal correction of coagulopathy; and the use of haemofiltration.^{4,5}

Currently more than 300 children have been enrolled worldwide in an international multicentre randomised trial of bactericidal permeability increasing protein in severe meningococcaemia. To gain the maximum benefit from the huge amount of data collected, ongoing registration is required between clinical trials. The formation of a central coordinating group is long overdue.

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Is prolonged rotavirus infection a common cause of protracted diarrhoea?

EDITOR,—The possible link between rotavirus and the postenteritis syndrome was highlighted in the recent editorial¹ about Richardson *et al*'s paper concerning the finding of prolonged rotavirus excretion in the stool using reverse transcription-polymerase chain reaction.² We feel it is important to be circumspect about this link as the evidence to date is circumstantial.

The symptom duration in Richardson *et al*'s study ranged up to, but not beyond, 14 days. This is at the minimum limit for diagnosing persistent diarrhoea.³ Following this there were only two intermittent episodes of mild diarrhoea and/or vomiting in three children, one episode in five, and no episode in three of the cases who were shown to have rotavirus RNA in their stool for > 21 days after the onset of severe rotavirus diarrhoea. A comprehensive screen for other pathogens was not carried out, so that infection by another agent cannot be excluded. No growth was obtained in MA104 cells so that the viability of the stool rotavirus could not be established, and the finding may represent the excretion of degenerate particles.

A stronger case would be made if rotavirus had been found in the mucosa of children who had protracted diarrhoea at the time and who had prolonged excretion of rotavirus, as shown in the classic paper of Bishop *et al*.⁴ Extended stool excretion, in the absence of clinical symptoms, has been shown for rotavirus and other gastrointestinal viruses⁵⁻¹⁰ and this may be a common phenomenon in viral gastroenteritis, just as excretion of pathogenic bacteria in asymptomatic individuals (the carrier state) is recognised. This is a potentially important observation in terms of cross infection, but not necessarily in terms of chronic, postenteritis diarrhoea.

Our retrospective review of cases of the postenteritis syndrome in a hospital setting¹¹ has shown intercurrent viral infection and persistent bacterial infection to be important associations with the diagnosis, and our earlier report of degenerate rotavirus particles in the stool of two children with protracted diarrhoea using electron microscopy¹² probably represented nosocomial infection rather

than persistent rotavirus infection. While it may be tempting to conclude that rotavirus can cause the postenteritis syndrome, the evidence presented to date does not make it more than a hypothesis awaiting rigorous examination.

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CMV coinfection and disease progression in vertically acquired HIV infection

EDITOR,—The paper by Boriskin and colleagues¹ provides interesting information regarding the age related contribution of cytomegalovirus (CMV) viral load in HIV infected children. The significantly higher CMV viral load in the youngest age group (0-2 years) could explain the generally higher mortality in this age group due to the accelerated disease progression.² However, the conclusion regarding the progression of HIV infection associated with the CMV viral load in this cross sectional study¹ should be treated with caution.

Between May 1985 and September 1995 we were able to follow from birth 25 vertically HIV infected children staged as A or B. We studied the clinical outcome with respect to CMV infection during 76 patient years.

Patients were divided into CMV negative (CMV IgG antibody negative and negative CMV cultures from urine samples; n = 12; median follow up 42 months (range 9-80)); and CMV infected (CMV IgG antibody positive; n = 13; median follow up 39 months (range 19-68)). In the CMV infected group 10 patients had positive urine samples for CMV on several occasions during observa-

tion. There was no primary CMV infection in the CMV negative group during follow up.

Clinical outcome of CMV negative patients was significantly better than CMV coinfecting patients (4/12 v 11/13 progressed to AIDS; $p < 0.02$). The relative CD4 cell count used to assess disease progression was significantly different between the CMV infected and CMV negative group at the study end point ($p < 0.01$).

Pleskoff *et al* showed that CMV encodes a chemokine receptor, which also serves as a cofactor for the entry of HIV into cells.³ Furthermore, CMV is able to potentiate cellular immunodeficiency in HIV infection directly or due to coinfection with enhancement of HIV replication.⁴ Therefore, a longitudinal study should be performed using CMV viral load to estimate the correlation of HIV and CMV coinfection for disease progression.

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Botryoid neutrophils in unexpected heat stroke

EDITOR.—Heat stroke is a potentially fatal disorder but often difficult to diagnose in children. Peripheral neutrophils with radially hypersegmented nuclei—"botryoid" neutrophils—are known to be characteristic of heat stroke in adults.¹ We describe three children exhibiting botryoid neutrophils who presented with acute encephalopathy.

Patient 1 was a 7 month old girl with severe cerebral palsy who became unresponsive after being kept warm with a hot pack in winter. She was febrile ($\geq 42^{\circ}\text{C}$) and hypotensive, and she had several seizures and bloody stools. After supportive treatment including cooling procedures, she returned completely to her pre-illness state 14 days later.

Patient 2, a previously healthy 9 year old girl with a one day history of cough and fever was found unresponsive and convulsive in winter. Before her collapse, her mother had kept her warm with an electric foot warmer because she had complained of a chill. Her clinical picture was characterised by high fever ($\geq 42^{\circ}\text{C}$), shock, diarrhoea, and bleeding. Despite aggressive treatment she continued to deteriorate and was pronounced brain dead on day 4.

Patient 3 was a previously healthy 14 month old boy with a three day history of diarrhoea who presented with acute encephalopathy in summer. He had been well until three hours earlier. At presentation his temperature was 40.4°C and his blood pressure 36 mm Hg. Bleeding diathesis was outstanding on his skin and oral mucosa.

Table 1 Laboratory findings of patients exhibiting botryoid neutrophils during acute stage

	Patient 1	Patient 2	Patient 3	Reference range
Leucocytes ($\times 10^9/\text{l}$)	7.3	5.9	11.4	8.0 to 15.0
Neutrophils ($\times 10^9/\text{l}$)	3.7	3.0	4.8	1.5 to 6.0
Haemoglobin (g/l)	83*	108*	88*	116 to 140
Platelets ($\times 10^9/\text{l}$)	47	20	124	168 to 400
PT (seconds)	17.0	24.8	> 60	10.7 to 13.3
aPTT (seconds)	51.8	106.2	> 120	25 to 35
Fibrinogen (g/l)	1.8	1.4	< 0.4	2.2 to 4.1
FDP ($\mu\text{g/l}$)	NA	359	722	< 10
AST (IU/l)	508	7260	812	< 40
ALT (IU/l)	301	3780	712	< 30
LDH (IU/l)	2772	13340	4120	250 to 600
CK (IU/l)	4960	15710	3670	< 180
BUN ($\mu\text{mol/l}$)	6.4	14.3	13.2	1.8 to 6.4
Creatinine ($\mu\text{mol/l}$)	62	115	71	27 to 62
CRP (mg/l)	25	3	< 3	< 3
pH	7.30	7.33	6.88	7.35 to 7.45
Pco ₂ (torr)	38.5	15.4	17.5	35 to 45
Base excess (mEq)	-6.7	-14.7	-30.2	-3 to +3

*Fell by more than 30 g/l within 3 days after onset.

PT, prothrombin time; aPTT, activated partial thromboplastin time; FDP, fibrin degenerative products; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; BUN, blood urea nitrogen; CRP, C reactive protein; NA, not assessed.

Figure 1 Peripheral neutrophil with radially hypersegmented nucleus (botryoid neutrophil) observed at admission. (A) patient 1; (B) patient 2; (C) patient 3. (Original magnification $\times 1000$, May-Grünwald-Giemsa staining.)

Despite aggressive resuscitative measures he died 18 hours later.

Table 1 shows the laboratory findings of the three patients. In the presented blood smears neutrophils had botryoid changes of 1% (patient 1), 50% (patient 2), and 2% (patient 3) (fig 1). Patients 1 and 2 did not have botryoid neutrophils on day 2. In patients 1 and 2, all bacterial cultures were negative. In patient 3, blood culture was positive for *Pseudomonas aeruginosa* but no lesions of pseudomonas infection were detectable in any organs at necropsy. In patient 2, a rise in haemagglutination inhibiting antibodies against influenza virus A was detected.

Although the thermal condition was different in each case, these patients shared clinical features typically seen in heat stroke including acute encephalopathy, shock, diarrhoea, raised liver and muscle enzymes, azotemia, coagulopathy, and high fever.² Botryoid neutrophils have been observed in haemorrhagic shock and encephalopathy syndrome (HSES), which is a fulminating encephalopathy of infants and has been suggested to be the same condition as heat stroke.³ Patient 1 had the typical features of HSES. Patient 2 was rather old for HSES and patient 3

conflicted with the diagnosis of HSES because of a positive blood culture.⁴ These latter two patients may represent atypical forms of HSES. Heat stroke may easily be misdiagnosed as infectious encephalopathy because children, as with patients 2 and 3, frequently have clinical evidence of infection. Our observations suggests that detection of botryoid neutrophils may be helpful in the early diagnosis of heat stroke including atypical HSES in children who present with acute encephalopathy.

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BOOK REVIEWS

Living with Haemophilia, 4th ed. By Jones P. (Pp 412; paperback £14.99; hardback £26.95.) Oxford: Oxford University Press, 1995. ISBN 0 192 62961 1 paperback; 0 192 63030 X hardback.

The diagnosis of haemophilia in a newborn son often leaves parents feeling completely overwhelmed. Many mistakenly believe that a simple scratch in a boy with this congenital bleeding disorder can lead to death through exsanguination, or that their son will end up in a wheelchair because of early arthritis. In fact, the prognosis for young people with haemophilia is excellent, and boys in developed countries can look forward to an essentially normal life. The introduction of recombinant coagulation factor concentrates has eliminated the risk of transmission of viral infections such as HIV or hepatitis, and it is now usual practice to give factor VIII on a regular, prophylactic basis to prevent most spontaneous bleeds, and thus long term disability. Haemophilia actually confers protection against coronary artery thrombosis, which is the principal cause of death in middle aged men.

Haemophilia is a relatively rare disorder, affecting approximately 6000 boys and men in the UK. Few doctors and nurses will have experience of dealing with a condition that affects only 1 in 10 000, and parents may be frustrated by difficulty in finding answers to their many questions. This book distils the personal experience of a paediatrician, which extends over 30 years, in the treatment of this rare disorder. The book is primarily aimed at the parents of boys with haemophilia. However, it will undoubtedly also be an invaluable source of reference for all health care professionals or others (such as teachers) who come across children with haemophilia. The value of this book is reflected in the fact that the book is now given as a gift to all parents registering for the first time with the Haemophilia Society, the national patient charity. The basic medical facts about blood clotting and haemophilia are clearly set out, and excellent illustrations help to get the basic messages over. There is comprehensive advice on matters about management of all bleeding problems, with specific advice on exact dosage of coagulation factor concentrate. There is also general advice on issues such as employment and travel. There is also a section aimed at female carriers of haemophilia, with up to date information on the latest methods used for genetic identification of carriers as well as antenatal diagnosis of haemophilia. It may come as a surprise to some to learn that boys with haemophilia are now positively encouraged to participate in sports at school, and this book gives very sensible advice on which sports are to be encouraged (such as swimming and tennis) and those that are best avoided (such as hockey and rugby).

A programme of physical exercise helps to maintain muscle strength and prevent joint bleeds. People with haemophilia should not take aspirin, which exacerbates the bleeding tendency, and I am sure that patients will find the list of almost 600 over the counter products that contain aspirin very useful.

This 5th edition was first published in 1995 but is now available in paperback for the first time. This is a rapidly moving field and some significant recent developments are not covered in the book. Recombinant factor IX is now available for all young patients with haemophilia B, and recombinant factor VIIa is now licensed and has proved to be invaluable in the treatment of the minority of patients with inhibitory antibodies. However, this is an excellent, comprehensive, and well illustrated book that will be invaluable to all who come into contact with boys with this rare condition.

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Immunizing Children: A Practical Guide, 2nd ed. By Mayon-White R, Moreton J. (Pp 104, paperback; £14.95.) Oxford: Radcliffe Medical Press, 1998. ISBN 8 577 5219 8.

Although immunisation is one of the most effective forms of medical intervention, the increasing scepticism with which it is viewed by a significant number of parents means that those involved in the process need to be fully informed.

The authors of *Immunizing children* acknowledge the stimulus provided by parents, their questions, and their reluctance to accept immunisation. This book appears to provide a bridge between the "Green book" *Immunisation against infectious disease* (London: HMSO, 1996) and the consumer.

The early part discusses vaccine development (trial phases), and explains the immunological background, before going on to describe the characteristics of the preventable infections. There is a section on immunisation schedules, considerations and contraindications, immunisation procedures, and care of vaccines. The last part of the book considers reactions and parents' questions.

This book is in many respects less comprehensive than the "Green book". The descriptions of the infectious diseases are irritatingly simplistic and at times show ignorance of current practice: thus epiglottitis resulting from *Haemophilus influenzae* type B infection is described as "... inflammation of the throat which blocks the entrance to the windpipe. Children ... died by suffocation unless they were immediately treated by antibiotics and in some case by a tracheostomy." Tracheostomy has not been used as treatment for many years. Similarly rubella and diphtheria are said to cause heart damage and this rather simple approach has led to important omissions—no mention of the serious consequences of measles in the immunosuppressed child. Egg allergy is a common problem in children yet it is covered in a short, rather uninformative paragraph.

The section on common worries will be useful to health professionals who have to reassure parents about measles-mumps-rubella, Crohn's disease, and autism. Some background references in this section would have been useful.

This book is a useful addition to the literature on immunisation. I suspect its greatest

benefit will be to the professional who needs information for the sceptical parent.

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Differential Diagnosis in Pediatric Radiology. Edited by Ebel K. (Pp 825; hardback.) Stuttgart: Georg Thieme Verlag, 1998. ISBN 3 131 08131 7.

A revised translation of a German textbook first published in 1995 has been transformed into this edition. The book is aimed primarily at radiologists who undertake some imaging of children. It may also be of interest to paediatricians in general as the numerous (over 1500) images cover a wide range of conditions.

The format consists of lists of possible differential diagnoses for a variety of imaging findings—for example, bilateral renal enlargement without pelvicalyceal dilatation. This is a relatively novel approach to a radiological text and by and large it works well. A three column table is employed to discuss the general headings and their differential diagnoses. Some brief additional text and normal anatomical measurements are frequently included at the beginning of each chapter or subchapter. The book also contains numerous anatomical diagrams and schematic drawings, which are superb and greatly facilitate a rapid understanding of the subsequent text. The tables are simply laid out, contain a sensible amount of information, and are easy to sift through. Overall, this is an excellent book. The authors and publishers are to be congratulated for a number of reasons. The English translation is virtually faultless, the images excellent (always hard to achieve with plain radiographs), and the general layout is easy to follow. The aim of the book is as an aid to differential diagnosis when confronted with a radiological finding in a child, although the major strength is really in the large selection of good quality plain films and other images.

A few deficiencies just about merit mentioning. A laudable attempt at brevity has resulted in staccato, one word sentences in the comments section. This approach can occasionally be quite confusing and assumes a good understanding of the disease processes being discussed. Although a relatively large book, the emphasis is on images and so inexperienced trainees will need to look elsewhere for in depth discussion of the pathology illustrated. There is too little nuclear medicine particularly in the genitourinary chapter, which does not reflect modern paediatric radiology or urology practice, which now relies heavily on nuclear medicine techniques. The section on gastrointestinal radiology would benefit from some better editing of the text to match the high standard of the rest of the book. Ultimately these drawbacks do not seriously detract from what is otherwise an excellent and extensive collection of paediatric images allied to relevant sensible tabulated information.

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Eponymists in Medicine: John Langdon Down 1828-1896, a Caring Pioneer. By Ward CO. (Pp 213; hardback; £15.95.) London: Royal Society of Medicine Press Ltd, 1998. ISBN 1 853 15374 5.

The author of this latest addition to the *Eponymists in medicine* series is a distinguished Irish professor of paediatrics, well known for his interest in mental handicap. He has been honorary medical advisor to the Down Syndrome Association of Ireland for 20 years. Conor Ward is to be congratulated on this first full and vivid biography of a remarkable and somewhat neglected Victorian physician.

John Langdon Down, MD, FRCP was born in Cornwall in 1828, the son of an apothecary. Entering medicine in 1853 he was a brilliant student and qualified at the University of London in 1858. At once he was appointed superintendent to the Royal Earlswood Asylum for Idiots. Careful observation and the use of clinical photography enabled him to attempt a classification of children with mental retardation. He was the first to describe both the Prader-Willi syndrome and, in 1866, the characteristics of mongoloid idiocy, which, 95 years later, came to be known eponymously as Down's syndrome.

In 1868 Langdon Down opened his own private institution for mentally retarded children, Normansfield. There he worked till his sudden death at the age of 68 in 1896. His work was then continued by his sons Reginald and Percival, and by a grandson, until the institution was absorbed into the National Health Service in 1952.

Langdon Down was a large handsome man with charming manners and liberal views. A pioneer in the humane caring for and treatment of people with learning disabilities, he had a natural empathy with the handicapped. In this as in other ways he was ahead of his time. He was a strong advocate for the rights of women and for their higher education and entry into the professions. He became a Justice of the Peace and in 1889 Alderman for Middlesex.

This slim volume is well illustrated, referenced, and indexed. It is warmly recommended to all those having an interest in the history of medicine and in the care of those with mental retardation.

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Clinical Management of the Child and Teenager with Diabetes. Edited by Plotnick L. (Pp 268; paperback, £20.50; hardback, £45.50.) Baltimore: John Hopkins Press, 1999. ISBN 0 801 85909 3 paperback; 0 801 85908 5 hardback.

The evidence provided by the diabetes control and complications trial (DCCT) exerts considerable pressure on clinicians to help their patients achieve optimal blood glucose control. This book has been written to provide detailed information for primary care physicians who, due to shifting responsibilities in the USA away from specialist teams, are becoming increasingly involved with the care of children with diabetes.

Initially, the book appears rather wordy, having few diagrams and no pictures. However, it is not always the books with the most

lavish illustrations that prove most use to the novice learning a craft. This book, while not forgetting some scientific information, is about the practical aspects of managing childhood diabetes. It is written in a sensitive manner, is easy to read, and the many brief case studies provide useful and interesting verbal illustrations. I was glad to find that, despite the transatlantic differences, we share a common philosophy of care, and most of the subject matter is as relevant in the UK as in the States. Professor Plotnick's empathy with the children and their families is evident throughout the book, and social and psychological issues feature alongside the day to day diabetes management. There are chapters by a dietitian and nurse educator, and an appendix that contains a variety of sample record forms and letters, a reading list, and web sites.

I get the impression that American families are encouraged to work harder at perfecting their diabetes management than their British counterparts, although it was sobering to note that personal finances and insurance policies may restrict some families' ability to do this. Much is expected of them especially where meal plans and monitoring are concerned. Detailed instructions are given for altering insulin doses, and intensive monitoring and accurate record keeping are emphasised to achieve good control. I was intrigued to learn that when blood glucose concentrations are out of target range it is possible to determine the decrease of blood glucose produced by one unit of insulin by using "the 1500 rule". This entails dividing 1500 by the total daily dose of insulin to calculate how many mg/dl one unit of insulin will decrease blood glucose.

Diabetes nurse educators appear to work in much the same way as specialist nurses in the UK; however, I would have liked to learn more about their teaching methods and aids with perhaps some illustrations. It was interesting to see a whole page devoted to drawings of different sizes of syringes and needles but none of pen devices that are "very popular in Europe" or pumps, which are used more by American children.

This book will be of interest to all practical clinicians caring for children with diabetes, but more will be gained by reading it in its entirety before delving for specific information. Some phrases remain in my mind which could sum up its ethos: "thinking like a pancreas", the "relentlessness of diabetes" and "keeping hope alive".

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Data Interpretation in Paediatrics. F Finlay. (Pp 142, £16.95, paperback.) Blackwell Science, 1998. ISBN 0 632 05044 6.

The written paper of the MRCPC part II examination (member of the Royal College of Paediatrics and Child Health) consists of three sections: case histories or "grey cases", visual material, and data interpretation.

The current market boasts a multitude of excellent reference atlases and examination orientated picture books that between them offer a staggering array of clinical photographs. Conversely, there are only several challenging books of case histories and, as yet, even fewer texts for data interpretation.

Although practice promises to make perfect, this is perhaps a more realistic target to attain in the analysis of data questions than in answering the case history section, which does tend to retain a murky quality peculiar to itself. This book will certainly aid one in developing a critical approach to data problems, and the importance of achieving confidence and maximum scores in this section cannot be overemphasised in an examination which, so often, is lamentably unpredictable.

The questions in this book cover a broad spectrum of topics and manage to include problems referring to recently implemented investigative techniques in medicine as well as to conventional "bedside tests", of which few junior doctors these days have actual clinical experience. In recent years there has been a growing representation of neonatology questions in the examination as well as the increasing employment of figurative data in the form of EEGs and ECGs, and the complex numerical data of cardiac catheter measurements and pulmonary function tests. These trends are adequately reflected by the choice of questions in this book. Subjects such as epidemiology and genetics, which are all too frequent stumbling blocks, are addressed in a comprehensive manner.

The overall standard of questions is comparable to that faced in the actual part II examination and the answers are succinct with appropriate references to the standard paediatric textbooks. Thankfully, it avoids making the common error committed by many textbooks for postgraduate qualifications, which aspire to be so difficult and esoteric that one loses all faith in one's ability ever to pass the examination.

Furthermore, *Data interpretation* fits snugly into a jacket pocket, making it a handy companion on train journeys or for quiet moments on call when revision beckons.

Having recently sat, and fortunately passed, the MRCPC part II examination I would thoroughly recommend this book to potential candidates. It is one of the few question books that I had the time and the heart to reread just before the examination in that critical period when one needs to revise material in the shortest possible time while simultaneously suppressing an ever growing sense of panic.

Finally, I believe that this is a useful text even for those who have left the spectre of postgraduate examinations far behind. I am sure that a surprising number of new facts may be gleaned from perusing these pages and, after all, the process of self education by no means ends with membership.

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