LETTERS TO THE EDITOR

"Cot death" rates on different days of the week

EDITOR,—The series of reprints that have arisen from the New Zealand cot death study, which took place around 1990, such as the article by Williams and colleagues[1] are replicating work that we carried out in Sheffield in the 1950s.[2–4] In the Sheffield studies, as well as noting the significance of prone sleeping, we also saw an increase in children presenting as unexpected deaths at the weekend. In addition, we found a relative increase on the night after the day when family doctors took their traditional half day off and did not hold afternoon or evening surgeries. This feature became most apparent when the cot death rates were seen as part of the total pattern of deaths—that is, a diminution in acute deaths following admission to hospital was replaced by greater numbers of home cot deaths.

This pattern of deaths in Sheffield changed after we introduced the prevention programme identifying children at increased risk of unexpected death.[5] We found that we had largely eliminated the partially explained group of cot deaths, and the total infant and cot death rates in the city fell considerably.

The point that needs to be made is that such sociopathological studies on child deaths should always be carried out in relation to the pattern and site of deaths, and to the total infant death rate in the local community. Much of the confusion related to risk discriminants results from the false assumption that with cot deaths one is dealing with a single cause. Particular causes can be increased, reduced, or eliminated. This has been particularly striking during the past 50 years relating to what are almost certainly accidental suffocation deaths. An increase in the unexpected death rate occurred following propaganda arising from neonatologists recommending prone sleeping in children. The recent complete reversal of that policy seems to have resulted in the elimination of that group and hence the fall in "cot death" rates to their original level.

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1 Williams SM, Mitchell EA, Scragg R, and the New Zealand National Cot Death Study Group. Why is sudden infant death syndrome more common at weekends? *Arch Dis Child* 1997;**77**:415–19.
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Nitrate and nitrite content of meat products

EDITOR,—Having read the case report by Kennedy and colleagues,[1] we would like to point out some aspects of the production of dry fermented sausage, salami, and sausage. We agree with Kennedy *et al* that food manufacturers should order ingredients specifically, in writing, and preferably by their approved chemical name.

Nitrate and nitrite are widely used as additives in meat products for effects such as reddening, as preservatives, and as antioxidants. Prolonged ingestion of nitrates and nitrites may cause methaemoglobinaemia and favour the formation of carcinogenic nitrosamines.[2 3] The use of nitrates and nitrites as meat curing agents is restricted in Turkey by the *Regulations of food additives*,[4] but it does not prevent the use of overdose by food processors as the residual quantities in the end products are not limited.

To investigate nitrate and nitrite contents in meat products for human consumption we collected 65 dry fermented sausages, 83 salamis, and 60 sausage samples from markets in Istanbul and analysed them with spectrophotometric methods.[5] The average nitrate concentrations were 87.0 mg/kg (range 0–362.9) in dry fermented sausage, 102.4 mg/kg (0–390) in salami, and 147.4 mg/kg (0–370.9) in sausage. The average nitrite concentrations were 42.8 mg/kg (0376.9) in dry fermented sausage, 87.6 mg/kg (0–375) in salami, and 102.8 mg/kg (0–420) in sausage. The nitrate contents in 3.6% of salamis and 11.7% of sausages were above 300 mg/kg. The nitrite contents in 3.0% of dry fermented sausages, 15.6% of salamis, and 20% of sausages were above 150 mg/kg. Therefore, nitrates and nitrites used during the production of meat products were higher than the concentrations indicated by the *Regulations of food additives* and this might be detrimental to human health. Therefore, the concentrations of nitrate and nitrite in the end product should be limited and controlled.

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1 Kennedy N, Smith CP, McWhinney P. Faulty sausage production causing methaemoglobinaemia. *Arch Dis Child* 1997;**76**:367–8.
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Incidence of coeliac disease

EDITOR,—We were interested to read the article by Challacombe *et al* reported a declining incidence of coeliac disease in West Somerset.[1] Our observations on the incidence of coeliac disease in South Glamorgan over a 15 year period have revealed no such decline. We determined the frequency of new cases of coeliac disease from 1981 to 1995 in patients resident in South Glamorgan (1995 total population 415 900; population 14 years or younger 83 500; total live births 5700 per year). Cases of coeliac disease were ascertained from hospital activity data, pathology, dermatology, and dietetic records, general practitioner lists, and the local coeliac society.

All cases satisfied the revised ESPGAN diagnostic criteria.[2] Over the three five-year periods (1981–85, 1986–90, 1991–95) the number of new cases in children younger than 14 were 8, 10, and 9, respectively—annual incidences of 2.08, 2.53, and 2.15 per 100 000. The incidence of childhood coeliac disease has therefore remained constant over the 15 year period at approximately 1 in 2500 to 1 in 3000 live births. In contrast, the incidence of adult coeliac disease has increased over the three time periods from 1.3 to 2.15 and 3.08 per 100 000. The incidence of adult dermatitis herpetiformis has remained between 0.3 and 0.43 per 100 000.

The age at diagnosis of children with coeliac disease has risen from a median of 4 years (1 to 10) between the period 1981 to 1990, to 7.6 years (1.7 to 14.9) between 1991 and 1995, whereas the age at presentation of adult patients has remained constant with a median of 49.5 years (19 to 88). From 1981–90 the predominant presenting symptoms were gastrointestinal, with 70% of the children having diarrhoea, and only three of the 18 children being anaemic. Between 1991–95 anaemia associated with vague abdominal symptoms (such as discomfort or bloating) became a more common presentation (44%) and diarrhoea was noted in only 11%.

Anaemia as the sole presenting feature remained rare at diagnosis (one of 27) compared with a figure of 25% of the adult coeliac population over the 15 year period. Two asymptomatic children were diagnosed following screening for IgA antigliadin antibodies in siblings of affected probands.

We may be missing asymptomatic cases or those that present later with symptoms such as anaemia, so the true incidence is likely to be much higher. Many adult cases are now identified from duodenal biopsies taken during upper gastrointestinal endoscopy for investigation of iron deficiency anaemia and non-specific gastrointestinal symptoms. The incidence of adult dermatitis herpetiformis, which shares the same genetic basis as coeliac disease,[3] has remained stable, suggesting the increased diagnosis of adult coeliac disease primarily because of increased clinical awareness.

We consider that although the classic gastrointestinal presentation of coeliac disease may be decreasing in children, the overall incidence may not have altered, and is likely to be much higher than previously recognised once screening tests become more widely employed. It is thus vital that we remain aware of the diagnosis and how subtle its presentation may be, and screen actively for cases using IgA antigliadin antibody and antiendomysial antibody, particularly in populations at higher risk (for example, family history, Down's syndrome, insulin dependent diabetes mellitus).

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1 Challacombe DN, Mecrow IK, Elliott K, Clarke FJ, Wheeler EE. Changing infant feeding practices and declining incidence of coeliac disease in West Somerset. *Arch Dis Child* 1997;77:206–9.
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Dr Challacombe comments:

The letter by Jenkins *et al* on the incidence of coeliac disease in children younger than 14 years in South Glamorgan raises some interesting questions. They have reported a constant incidence over three five-year periods (1981–85, 1986–90, 1991–95) of approximately 1:2500 to 1:3000 live births. An earlier study in West Somerset reported a declining incidence of coeliac disease between 1971–80 and 1981–92, in which the annual incidence peaked in 1974 and then decreased, and further patients were not diagnosed annually for six years between 1980 and 1992. The cumulative incidence of coeliac disease in West Somerset was 0.68 per 1000 live births during 1971–80 and 0.09 during 1981–92. The different findings could have been caused by different sampling times. A higher incidence of coeliac disease in the late 1960s and early 1970s, possibly caused by the early introduction of dietary gluten, could have been followed by a relative decline in incidence during the late 1970s and 1980s with changing infant feeding practices. These were characterised by the later introduction of dietary gluten, an increased use of baby rice and gluten free foods for weaning, and a higher incidence of initial breast feeding. The age at diagnosis of children with coeliac disease also increased in South Glamorgan and West Somerset, which could have been yet another result of delaying the introduction of dietary gluten in infancy.

Although some children still present with classic symptoms and signs of coeliac disease, others present at school age or adolescence with mild or atypical symptoms and signs. As a result, the diagnosis of coeliac disease has become more covert and difficult to recognise. The development of methods using serum IgA antibodies to gliadin and to endomysium to diagnose and follow up patients with coeliac disease has been opportune. In association with small bowel intestinal biopsy these methods will enable the true incidence of coeliac disease to be determined more precisely and will shed further light on the natural history of this disease in children and adults.

Oestrogen treatment of tall stature

EDITOR,—We deplore the publication of a paper that lends credibility to a therapeutic regimen that is not only obsolete but also dangerous.[1] High dose oestrogen treatment has an unacceptable incidence of side effects, which the authors record, and an unknown risk of thromboembolic problems[2] and carcinoma of the breast, ovary, and uterus.

The prevention of excessive adult stature is attained much more benignly by the induction of puberty using low doses of sex steroid at an age and height judged to achieve a satisfactory end point.

Final height is determined by the height attained at onset of puberty,[3] a constant amount of height (30 cm) being added to that height. This is why children with precocious puberty end up short; they have an insufficient amount of time to grow along the childhood curve of growth. For example, if a girl is not to exceed a final height of 180 cm, she needs to have started breast development (spontaneously or induced with low dose oestrogen) when she has attained a height of 150 cm. The only problem is if she is too young to induce puberty when she has attained this stature. This is a very rare situation, which is probably pathological. Exactly the same arithmetic applies to boys whose puberty can be induced with low dose testosterone.

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1 Weimann E, Bergmann S, Böhles HJ. Oestrogen treatment of constitutional short stature: a risk-benefit ratio. *Arch Dis Child* 1998;**78**:148-51.
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Dr Weimann and Professor Böhles comment:

We were particularly concerned about this harsh criticism because not all necessary points of view have been taken into consideration. Our paper is the result of a retrospective analysis of our data from 1985 to 1994. It describes our experience with high dose oestrogen treatment for the prevention of excessive adult stature, which has been used worldwide over the past 42 years. We were well aware of the risk of hypercoagulability. There was increased platelet aggregation in 60% of the 50 girls we examined. All other coagulation parameters such as activated prothrombin time, partial thromboplastin time, fibrinogen, and antithrombin III were normal. Platelet aggregation can easily be avoided with low dose aspirin supplementation. The risk of carcinoma of the breast, ovary, and uterus is a hypothetical speculation when natural oestrogens are used, as in our patients.

The interesting approach of using low dose oestrogen in girls at risk of attaining excessive adult stature when they reach 150 cm may be theoretically better with respect to possible side effects; however, practically it may be applicable only in some patients because they usually present at a later age when they are deeply concerned about their possible final height. In our study the mean age and mean height at first presentation was 12.8 years and 175.5 cm. In addition, in most cases a height of 150 cm in girls above the 97th percentile is accompanied by a chronological age of less than 10 years, when an accurate adult height prediction is still difficult.

We know of no sufficiently evaluated study on the efficacy of low dose oestrogen treatment for the prevention of excessive adult stature in girls. We were therefore very surprised that such critical emphasis has been placed by our colleagues on an unevaluated opinion.

When patients present with a height above the 97th percentile and ongoing puberty, a decision has to be made, and reports about the experience with treatment regimens other than low dose oestrogens will be welcome. As we are counselling healthy young girls, the indication for treatment is always the result of a thorough appraisal of all known treatment risks, and the consequence of the psychological impact of an excessive final height.

Down's syndrome in infants of diabetic mothers

EDITOR,—In a recent paper Narchi and Kulaylat[1] analysed the prevalence (erroneously termed incidence[2]) of trisomy 21 in children of mothers with diabetes (7/1870) and in non-diabetic mothers (28/20 430) and found it to be significantly higher in the former. All seven cases in the diabetes group occurred in mothers with gestational diabetes.

The authors concluded, that: (1) Maternal gestational diabetes is an independent risk factor for Down's syndrome, irrespective of maternal age, as in an analysis of their data stratified by age, all five age groups showed a higher relative risk for Down's syndrome in diabetic mothers; (2) Down's syndrome should be added to the list of congenital anomalies known to occur more frequently in infants of diabetic mothers.

Table 2 in their paper does not fully support their hypothesis. As can be seen from their data, age is a confounding factor for gestational diabetes as fewer than 5% of pregnant women develop this condition in the age group below 30 years but more than 23% in those over 44 (assuming that females with pre-gestational diabetes are distributed randomly over the age groups—the authors combine both types of diabetes in this table). An analysis of their age stratified groups using the Mantel-Haenszel method reveals an odds ratio of 2.33 with a 95% confidence interval (CI) of 0.99 to 5.48 for the whole study population, as opposed to the authors' unstratified analysis of the whole group (relative risk 2.75; 95% CI, 1.2 to 6.29).

Much more important for the discussion of the results is a violation of the rules of causal inference. The authors laudably made a sharp distinction between mothers with gestational diabetes (n = 1748) and pre-gestational diabetes (n = 122) and found cases (seven in all) with trisomy 21 only in the former group. Thus their analysis of diabetes as a risk factor for Down's syndrome is only valid for gestational diabetes. Although the use of the term risk factor in the literature is rather loosely defined, in aetiological research for an exposure in the broadest sense (gestational diabetes in this study) to become a genuine risk factor it must precede the occurrence of the outcome (here, trisomy 21). As non-dysjunction leading to trisomy 21 occurs before or shortly after fertilisation, gestational diabetes with onset during pregnancy can hardly be a risk factor for trisomy 21. The study at hand does not justify adding trisomy 21 to the known congenital anomalies associated with pre-gestational diabetes in the mother.

What Narchi and Kulaylat may have shown is that a woman with a trisomy 21 conceptus is more likely to develop gestational diabetes, although the attributable risk may be small, and age, as can be seen from their data, seems to be much more important.

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1 Narchi H, Kulaylat N. High incidence of Down's syndrome in infants of diabetic mothers. *Arch Dis Child* 1997;**77**:242–4.
2 Hook EB. Incidence and prevalence as measures of the frequency of birth defects. *Am J Epidemiol* 1982;**116**:743–7.

Drs Narchi and Kulaylat comment:

We read with interest Pelz and Kunze's comments on our study and thank them for the opportunity to clarify our findings.

We agree that the term prevalence is more appropriate than incidence, but our data do not support maternal age as a confounding factor. Although maternal diabetes (mainly gestational) was more common with advancing age, when the prevalence of Down's syndrome was broken down by maternal age, it remained 1.6 to 3.01 times more common in infants born to mothers with gestational diabetes within each age group. If maternal age was a confounding factor, the prevalence of Down's syndrome, although increasing with advancing maternal age, would not be expected to be different within the same age group regardless of the presence of gestational diabetes. Even using the Mantel-Haenszel method for age stratified groups as suggested by Pelz and Kunze, the relative risk for a diabetic mother to have a baby with Down's syndrome was 2.34 (95% CI, 1.02 to 5.33), not very different from the crude data relative risk of 2.35 (95% CI, 1.2 to 6.2) in our initial analysis.

We also disagree that the rules of causal inference were violated: we implied an association rather than a causal relation, as we could prove none. We made it very clear that as mothers with gestational diabetes were euglycaemic at conception, hyperglycaemia was ruled out as a potential mechanism for non-dysjunction; and we suggested the need for further studies of advanced biological aging, autoimmunity, and the role of biochemical factors such as apolipoprotein E. The lack of a direct causal relation should not lead to complacency in ignoring the increased prevalence of Down's syndrome in infants of mothers with gestational diabetes.

Neurological complications of Kawasaki disease

EDITOR,—A 14 week old boy was referred for neurological review with a 24 hour history of an isolated, left sided, lower motor neuron facial nerve palsy. Twelve days earlier, he had presented with fever, irritability, and a macular rash. A septic screen was negative, including normal cerebrospinal fluid indices. The fever settled on day 5, the rash improved, and he was discharged. He remained irritable, re-presenting when facial asymmetry developed.

Computed tomography and magnetic resonance imaging of his brain were normal. Initial haematology was unremarkable. Over the next five days he remained afebrile. His facial palsy resolved, but there was an increase in acute phase reactants and an evolving thrombocytosis. On day 17 of the illness, desquamation of the toes was noted. Echocardiography showing aneurysms of the left anterior descending and circumflex coronary arteries confirmed the diagnosis of Kawasaki disease.

Neurological complications of Kawasaki disease are well recognised. Hemiplegia, epilepsy, and myositis have been reported.[1] In one large series, neurological complications arose in 1.1% of cases.[2] There have been 18 previously reported cases of facial nerve palsy in Kawasaki disease.[3] A review of these cases[3] noted that six of the 10 children in whom cerebrospinal fluid was sampled had a pleocytosis. At diagnosis, coronary artery involvement was found in 55%, compared to 20–40% of all cases of Kawasaki disease.[4]

This is the first case reported in the UK presenting with this complication of Kawasaki disease. He presented at an early age; the peak incidence of Kawasaki disease is 9–11 months[4] and only one of the reported cases of facial palsy arose in a child younger than 6 months.[3] Kawasaki disease should be considered when an acquired facial palsy occurs as an isolated neurological finding in an infant, particularly where fever has occurred in the previous month.

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1 Laxer RM, Dunn HG, Flodmark O. Acute hemiplegia in Kawasaki disease and infantile polyarteritis nodosa. *Dev Med Child Neurol* 1984;**26**:814–21.
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Factors involved in the rate of fall of thyroid stimulating hormone in treated hypothyroidism

EDITOR,—We would like to comment on the recommendations on the management of congenital hypothyroidism by Raza *et al.*[1] Although thyroxine dosage recommendations have been available for the treatment of congenital hypothyroidism for many years[2] it has not been possible to answer the crucial question as to what concentrations of circulating thyroid hormones and degree of thyroid stimulating hormone (TSH) suppression are required to provide the optimal environment for maximising neurobehavioural and intellectual development. The working group on congenital hypothyroidism of the European Society for Paediatric Endocrinology has emphasised that it has not been possible to measure a dose–response relation, and it is coordinating a prospective study to assess the adequacy of treatment and outcome using two different dosage schedules for the first 24 months after birth.

In the absence of randomised, prospective outcome studies, previous authors have recommended that any treatment strategy should aim to "achieve euthyroidism as soon as possible"[3 4] and there is a persuasive argument that TSH suppression is the only and most relevant neurobiological marker of effective or optimal thyroid hormone concentrations and hypothalamic feedback.[5 6]

Surprisingly, Raza *et al* chose an unusual neonatal dosage regimen based on body surface area (100 μg/m^2/day) with all its inaccuracies, and sought to evaluate whether the lack of TSH suppression was influenced by the underlying thyroid disease or basal TSH concentrations, studies of which have been reported previously.[3] It would have been more helpful to have chosen a dosage schedule similar to those recommended previously (8–15 μg/kg/day) and to have accepted TSH suppression to < 10 mU/l to compare with other studies.

When screening for congenital hypothyroidism started in the Trent Region in 1980 several consultant paediatricians discussed a "best guess" dose of thyroxine; in Leicester we decided that as suspensions of thyroxine have questionable stability[7] and the smallest tablet available is 25 μg we would initially use 25 or 50 μg on alternate days (that is 37.5 μg/day), which would be near to 10 μg/kg/day for most infants.

We have looked at the results of our last 29 cases of congenital hypothyroidism to compare TSH suppression with Raza *et al*'s and other reports (table 1).

The starting dose in our infants weighing 2.5–4.7 kg ranged from 8–15 μg/kg/day (mean 10.5 μg/kg/day) compared with a calculated dose of 5–8 μg/kg/day based on Raza *et al*'s recommendation using body surface area. Using our regimen the concentrations of circulating total thyroxine at the time of TSH suppression ranged from 103 to 279 nmol/l (mean 174 nmol/l) and we saw no clinical evidence of hyperthyroidism. These data and a recent French study,[6] where frequent dose titration was used, demonstrate that TSH suppression is related to thyroxine dosage and that there is considerable variation in thyroxine concentrations, presumably owing to variability in thyroxine absorption and metabolism. We therefore agree with Touati *et al* that early and regular individualisation of dosage is required to achieve TSH suppression.[6] Thyroid hormone concentrations often seem to be high but fall within reported normal ranges for infants.[9]

We are concerned that following Raza *et al*'s recommendations will lead to an acceptance that significant non-suppression of TSH is unimportant, whereas it almost certainly

Table 1 Comparison of TSH suppression

Study	n	Thyroxine dose/day	Cases of TSH suppression (%)			Definition of TSH suppression
			3 months	6 months	12 months	
London[1]	32	100 μg/m^2	19	37	72	< 6 mU/l
SW England[8]	42	Variable	48	62	67	< 10 mU/l
Leicester	25*	37.5 μg	52	80	88	< 10 mU/l
Norway[5]	42	50 μg	86	–	–	< 10 mU/l

*Four cases excluded: one because of definite non-compliance and three because of late treatment in mildly affected cases.

represents undertreatment and so may not encourage the best achievable long term intellectual development.

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1 Raza J, Hindmarsh PC, Brook CGD. Factors involved in the rate of fall of thyroid stimulating hormone in treated hypothyroidism. *Arch Dis Child* 1997;**77**:526–7.
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Drs Brook and Hindmarsh comment:

We agree with Dr Swift and colleagues that a prospective randomised trial of different regimens of thyroxine in congenital hypothyroidism is needed. As the European Society for Paediatric Endocrinology trial he mentions will not look at long term sequelae it will not answer the question; we are trying to set up a UK multicentre trial that will address the problem.

One factor that Swift *et al* has not considered is whether feedback mechanisms of thyroxine on TSH are the same in infancy as in older patients. We never use suspensions of any hormone replacement and it seems intrinsically inappropriate to overtreat one day and undertreat the next.

We remain as concerned as ever about the effects of pursuing TSH suppression by increasing free thyroxine concentrations: we reiterate that the long term effects of clinically undetectable hyperthyroidism are much more damaging than the same degree of hypothyroidism. There are simply no data to support the contention of Swift *et al*'s last paragraph.

Post-pyloromyotomy emesis caused by concomitant urinary tract infection in pyloric stenosis patients

An association between infantile hypertrophic pyloric stenosis (IHPS) and concomitant urinary tract infection (UTI) has been reported previously.[1] Two consecutive infants with IHPS who continued to vomit after successful surgical and medical treatment in our institution were found to have concomitant UTI.

This prompted us to examine all cases of radiologically proven IHPS diagnosed in our hospital in whom vomiting, which may be a manifestation of UTI in infants, did not resolve after surgical treatment, to assess the possibility that concomitant UTI is the cause for post-pyloromyotomy emesis in IHPS infants. We examined all records of IHPS patients aged 2 to 7 weeks admitted to our hospital during a 10 year period between 1985 and 1995.

In all, 170 infants (138 male, 32 female) who presented within the first seven weeks of life (mean 4.4 (2.6) weeks) with progressively worsening emesis and clinical signs compatible with IMPS, had radiological confirmation of the diagnosis and underwent Ramstedt pyloromyotomy.

Of them, 24 (14.1%) patients had post-pyloromyotomy emesis and were evaluated for the possibility of UTI; urine analysis and cultures were obtained by either suprapubic aspiration or bladder catheterisation. If urine analysis suggested the presence of UTI, empiric antibiotic therapy was initiated with urine culture until urine culture results were obtained.

Four patients (three male, one female) out of 24 post-pyloromyotomy emesis patients (16.6%) were found to have concomitant clinically manifested UTI and IMPS. Symptomatic UTI occurs in 0.14% of live newborns.[2]

In a previous report of 276 infants with IMPS, two of them (0.72%) had confirmed UTI.[1] In our series of 170 IHPS patients, four of them (2.35%) were found to have concomitant UTI, clinically manifested by continuity of vomiting after surgical repair of the IHPS.

This figure is 17-fold higher than the expected incidence of UTI in young infants and it makes one wonder about the true aetiology of vomiting at the presentation of symptoms. Thus, as post-pyloromyotomy emesis occurs in 5–15% of surgically treated infants,[3] we recommend that any child who continues to vomit after adequate surgical treatment of IHPS be evaluated for the possibility of concomitant UTI.

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1 Atwell JD, Levick P. Congenital hypertrophic pyloric stenosis and associated anomalies of the genitourinary tract. *J Pediatr Surg* 1981; **16**:1029–35.
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Child Health: A Manual for Medical and Health Workers in Health Centres and Rural Hospitals. 2nd ed. Edited by P Stanfield, B Balldion, Z Versluys. (Pp 525; KSh360.) African Medical and Research Foundation, 1997. ISBN 9-966-87407-0.

Rural hospitals in Africa sometimes have a small library, often in the senior doctor's office, where the books are not accessible to the staff who would benefit from reading them. They are usually old editions of standard British or American textbooks, long winded, with drug doses in minims and guidance for making tincture of belladonna. Occasionally, there would be a paperback edition of Clayton's *Ten Teachers*, Bailey and Love's *Textbook of Emergency Surgery*, or one of the other subsidised books of the admirable English Language Book Society, now sadly defunct. Many of the hospitals have no money for books or journals and rely on infrequent donations.

If one asked rural health workers what educational material they had, they might produce a well thumbed thin pamphlet produced by AMREF (the African Medical and Research Foundation). Those who were very fortunate will have a copy of *Child Health*. A new edition is now available, 20 years after the first. The contents move easily between primary health care, curative medicine, and social medicine. It is an intensely practical book, although not as rich in diagrams as *Where There is No Doctor* (David Werner) or *Primary Health Care* (WHO). However, it is aimed at practitioners who know how to do a lumbar puncture but need advice on when, why, and what to do with the result. The clarity of the English makes the text accessible to health workers whose first language is not English or who have not attended secondary education.

If your rural medical aides, clinical officers, and nurses know this book inside out, then they will have the knowledge to manage what comes in front of them. It does not tackle the lack of resources, but having this knowledge will help them to prioritise situations.

Sections of the book acknowledge the great gaps in levels of health care in Africa by accepting that neonatal special care is now practised in larger centres. This still sits well beside advice on how to control the flow of patients through a clinic and safeguard the vaccine cold chain.

The final chapter is on child abuse and neglect. When I worked in rural Tanzania 15 years ago people told me that child abuse did not exist as "there is no word for it in our language". It does exist, particularly in the deprived urban sprawls, and in the refugee communities. A short section on the particular problems of refugee children—physical and emotional—would be a welcome addition next time round.

Sunlight, rain, termites, and frequent use will take their toll on this book. The price is 360 Kenyan Shillings (£3.60). The next time you want to assist colleagues in a developing country, do not send them your former professor's weighty tome. Send them 10 copies of *Child Health*. It will make a difference.

PAUL EUNSON
Consultant paediatrician

Paediatric Images. Case Book of Differential Diagnosis. By E Blank. (Pp 1260; £142.) Lippincott-Raven Publishers, 1997. ISBN 0-316-09991-0.

There are over 1000 images in this large book covering the whole spectrum of childhood illnesses. Some of the radiological examinations are no longer performed, such as pneumoencephalography, but it seems likely that such

studies are included for historical interest. Each case contains a detailed history, biochemical and other laboratory data, radiological images, and a final diagnosis or diagnoses.

There are too many deficiencies to recommend this book. To begin with, the title is misleading as no differential diagnostic possibilities are ever considered in the text. Little or no justification is made for any diagnosis, and yet in some cases diagnostic accuracy is debatable. A true differential diagnostic approach would have strengthened the text. For example, a number of neuroradiological cases could have been caused by non-accidental injury—an important diagnosis to consider and exclude for obvious reasons. The chapter grouping and organisation are somewhat unusual—for example, the larynx, pharynx, and oropharynx sections contain only one case each and the abdominal wall section (chapter) has just two cases. The first chapter is entitled "Skull and brain", the second "Brain" yet both include similar cases with computed tomography or magnetic resonance brain images. More significantly, the chapter on the urinary tract places too much emphasis on intravenous urography with no nuclear medicine studies whatsoever—serious omission considering the particular importance of isotope studies in modern paediatric urology. The chapter on the "Normal skeleton" includes disorders such as Perthé's disease and osteochondritis dissecans. The single greatest weakness, however, is the excess of unnecessary and frequently irrelevant information in the form of historical, biochemical, and laboratory data. Better editing could have helped in this regard. In addition, according to the author, some laboratory errors are deliberately included in the text but it is not clear when they appear or what relevance they have.

The radiology images overall are of good quality. Arrows on selected radiographs would have helped identify some of the more subtle abnormalities. Myelography is no longer contemplated in the diagnosis of discitis nor angiography to diagnose a hepatoblastoma. Many ultrasound images are reversed with a white background, which is no longer the convention.

The author has achieved what he set out to do, to present many fascinating paediatric stories and images "free of opinion and pronouncement". There is a commendable meticulousness throughout the text. Many of the cases are interesting and worth browsing through; however, the book is not topical nor is the intended target audience clearly defined.

KIERAN MCHUGH
Consultant radiologist

Paediatrics: An Illustrated Colour Text. Edited by D J Fields, J Stroobant. (Pp 120; £17.50) Churchill Livingstone, 1997. ISBN 0-443-05254-9.

When I teach medical undergraduates I pitch at three levels: things that if you do not know, you will fail (for example, what is Kernig's sign); things to know that will secure you a safe pass (for example, knowing the ABC of basic resuscitation); and things that will get you into the honours class (for example, use of DNAses in cystic fibrosis). I have yet to see a core textbook based on these key desiderata of the student mind set.

Paediatrics: An Illustrated Colour Text is an enjoyable multiauthor book that offers help to students in a symptom based approach. It wants its photographs and (excellent) illustrations to do the teaching. These and a three column format of text enable it to boast comprehensive cover of paediatrics in a slim 120 pages.

To the student familiar with browsing the internet this book is a boon for a paediatric attachment—much of the layout has a *Windows* feel to it. But one student's path of least resistance is another's dumbed down soft option and its A4 size and lack of margin space make carrying and annotating "on the hoof" difficult.

What of the symptom based approach? It is true that much of paediatric diagnosis rests on the history. Plus, history taking is what a student does most of (and is most comfortable with). Unit headings, such as "noisy breathing" and "spots and rashes" deal with common problems and reflect the language of concerned parents. These criteria break down somewhat with "oliguria" and "abdominal lumps" (not classic symptoms) where it seems disease entities have been shoe horned in for completeness.

What students find difficult is presenting cases, either on consultant ward rounds or eventually at finals' long cases. Terminology, bandied effortlessly by senior house officers and registrars, can be daunting. The authors are to be applauded therefore for taking the trouble to define and distinguish basic terms such as respiratory noises (snuffles, stridor, wheeze, grunting, etc) and the terminology of rashes.

The diagrams are well designed to stay in the memory, although the usefulness of this book as a revision aid is thwarted by a lack of depth in all areas. The problem arose when—for example, I wanted to read up, as students are often asked to, on meningitis. There are five references in the index, each to rather meagre entries in the text, while dehydration is not listed at all in the index. The air of superficiality is compounded by the absence of "further reading" sections.

Overall, *Paediatrics: An Illustrated Colour Text* would be a useful companion for a first year clinical student. Its format is undoubtedly alluring, even addictive, but may frustrate students with designs on a career in paediatrics. If book budgets are tight, it cannot truly be recommended as a sound investment.

NANU GREWAL
Senior house officer, paediatrics

Organelle Diseases. Clinical Features, Diagnosis and Management. Edited by D A Applegarth, J E Dimmick, J Hall. (Pp 454; £150 hardback). Chapman and Hall Medical, 1997. ISBN 0-412-54910-7.

Hands up all of you who know what an organelle is. I asked a colleague, a general practitioner, what he thought one was. "A small thing with long arms", he replied. That is not exactly right but patients rarely present stating "It's my organelles doctor". Knowledge of organelle disease is not really required in general practice. However, at least a rudimentary knowledge of organelle function and the recognition of organelle disorders are becoming increasingly important for paediatricians.

This book deals primarily with three organelles: lysosomes, peroxisomes, and mitochondria. Some lysosomal diseases, such as the mucopolysaccharidoses, have been well known to paediatricians and pathologists for many years. In contrast to disorders affecting intermediary metabolism, the slow accumulation of substrate may lead to progressive neurological disease often with associated dysmorphic features.

The peroxisome was first identified in the 1950s by a Swedish PhD student but its significance in human disease was only identified in 1973 when peroxisomes were found to be absent in cerebrohepatorenal (Zellweger) syndrome. We now know that disorders of peroxisomal function are responsible for at least 15 different disorders including X linked ALD, rhizomelic chondrodysplasia punctata, Refsum disease, and hyperoxaluria type 1. Investigations of these and other peroxisomal disorders have led to identification of new biochemical pathways and an understanding of their importance in human metabolism. Mitochondria are primarily responsible for cellular energy production. There has been an enormous increase in our understanding of mitochondrial disease and the rate of acquisition of knowledge is likely to increase. New diseases with strange acronyms, such as NARP, MELAS, MERFF, and MNGIE, have appeared over recent years. Additionally there are now new genetic concepts to understand—for example, maternal inheritance and heteroplasmy.

Organelle Diseases provides an in depth review of our present knowledge of these three organelles. As stated in the subtitle it deals with clinical features, biochemical and molecular diagnosis, pathogenesis, and management. Professor Charles Scriver, in his foreword, describes this book as linking science with medical practice. Science he describes as an attack on ignorance, and medical practice as a private relationship between practitioner and patient. Certainly there is a great deal of detailed science, and for those with a particular interest in this field it is a delight to see it brought together so well. Medical practice is also covered in some depth. Unfortunately our new knowledge has not yet translated into effective treatment for most organelle disorders. There are important exceptions, such as enzyme replacement treatment in Gaucher disease, but sections on treatment are, as a consequence, somewhat limited.

This book is a rather curious mixture of detailed science and more basic clinical practice. For example, there is a section on the stoichiometry of ATP synthesis and, in contrast, a chapter on how to take a family history. However, the format works well and I would certainly strongly recommend it to clinicians, biochemists, and anyone with an interest in biochemical genetics. Any book that has its text positioned between a foreword by Professor Charles Scriver and a postscript by Professor Victor McKusick is likely to have a lot going for it!

What is an organelle? Fortunately *Organelle Diseases* contains an excellent glossary and provides the following definition "A membrane bound intracellular cytoplasmic structure having specialised functions". Now you know.

JOHN H WALTER
Consultant paediatrician

MEETINGS

1998

XXII International congress of pediatrics
9-14 August, Amsterdam
Further details: XXII International Congress of Pediatrics, Eurocongres Conference Management, Jan van Goyenkade 11, 1075 HP Amsterdam, Netherlands

British Association of Perinatal Medicine and Neonatal Nurses Association: perinatal care towards the millennium
3-5 September, Cambridge
Further details: Conference Secretariat, Bell Howe Conferences (BAPM/NNA), 1 Willoughby Street, Beeston, Nottingham NG9 2LT, UK

Training in child public health, social, and community paediatrics in Europe
10-12 September, Bordeaux, France
Further details: Congress Rive Droite, 28, rue Baudrimont, 33100 Bordeaux, France

Paediatric Research Society
11-13 September, Elgin
Further details: Dr A Attenburrow, Consultant Paediatrician, Dr Gray's Hospital, Elgin, Morayshire IV30 1SN, UK

11th Congress of the International Pediatric Nephrology Association
12-16 September, London
Further details: IPNA 98, Concorde Services Limited, 10 Wendell Road, London W12 9RT, UK

8th International child neurology congress
13-18 September, Slovenia
Further details: President of the Organising Committee, Department of Developmental Neurology, University Paediatric Hospital, Vrazov trg 1, 61104 Ljubljana, Slovenia

European Society for Pediatric Research conference
13-17 September, Belfast
Further details: Project Planning International, Montalto Estate, Spa Road, Ballynahinch, Northern Ireland BT24 8PT, UK

RCPCH Accident and Emergency Group: international aspects of paediatric accident and emergency medicine
22 September, Liverpool
Further details: Dr W J Robson, Consultant in Paediatric A&E Medicine, Royal Liverpool Children's Hospital, Alder Hey, Liverpool, Merseyside L12 2AP, UK

Diabetes mellitus
30 September, London
Further details: Scientific Meetings Officer, Royal College of Pathologists, 2 Carlton House Terrace, London SW1Y 5AF, UK

Theoretical and practical approaches to the management of eating and drinking difficulties in people with learning disabilities from infancy to adult life
30 September, London
Further details: Lisa Spicer, Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE, UK

Drugs in school
2 October, London
Further details: Symposium Office, Institute of Obstetrics & Gynaecology, Queen Charlotte's & Chelsea Hospital, Goldhawk Road, London W6 0XG, UK

Women and children with HIV and AIDS
12 October, London
Further details: Symposium Office, Institute of Obstetrics & Gynaecology, Queen Charlotte's & Chelsea Hospital, Goldhawk Road, London W6 0XG, UK

Childhood onset diabetes and disordered metabolism
15 October, Bristol
Further details: Dr Ruth Williams, Institute of Child Health, Royal Hospital for Sick Children, St Michael's Hill, Bristol BS2 8BJ, UK

British Paediatric Rheumatology Group: autumn meeting
15-16 October, Canterbury
Further details: Dr Alison Leak, Consultant Rheumatologist, Queen Elizabeth The Queen Mother Hospital, St Peters Road, Margate, Kent CT9 4AN, UK

International conference on adolescent health
22-23 October, London
Further details: Youth Support Conference Administration, Youth Support House, 13 Crescent Road, London BR3 2NF, UK

Ninth annual course in paediatric gastroenterolgy
26-28 October, London
Further details: Professor J A Walker-Smith, University Department of Paediatric Gastroenterology, The Royal Free Hospital, Pond Street, London NW3 2QG, UK

Joint RCP/RCPCH conference: alcohol and the young
27 October, London
Further details: Miss Amanda Ambalu, RCPCH, 50 Hallam Street, London W1N 6DE, UK

Fetal, neonatal, and childhood haematology: paradoxes, problems, and progress
28 October, London
Further details: Scientific Meetings Officer, Royal College of Pathologists, 2 Carlton House Terrace, London SW1Y 5AF, UK

Bone marrow transplantation in childhood
28-30 October, Manchester
Further details: Index Communications Meeting Services, Crown House, 28 Winchester Road, Romsey, Hampshire SO51 8AA, UK

Neonatal study day
6 November, London
Further details: Christine Massey, Postgraduate Centre Manager, Hillingdon Hospital, Uxbridge UB8 3NN, UK

The child's perspective: a collaborative approach
8-10 November, London
Further details: The Training Department, The Institute of Family Therapy, 24-32 Stephenson Way, London NW1 2HX, UK

Molecular biology for paediatricians
9 November, London
Further details: Symposium Office, Institute of Obstetrics & Gynaecology, Queen Charlotte's & Chelsea Hospital, Goldhawk Road, London W6 0XG, UK

British Society for Paediatric Endocrinology and Diabetes autumn meeting
12-13 November, Cardiff
Further details: Dr J W Gregory, University Department of Child Health, Heath Park, Cardiff CF4 4XN, UK

Community child health
13 November, London
Further details: Symposium Office, Institute of Obstetrics & Gynaecology, Queen Charlotte's & Chelsea Hospital, Goldhawk Road, London W6 0XG, UK

British Society for Paediatric Dermatology annual meeting
13-14 November, London
Further details: Rosemary Cope, Academic Secretary to Dr D Atherton, Great Ormond Street Hospital for Children NHS Trust, Great Ormond Street, London WC1N 3JH, UK

Controversies in paediatrics
17 November, London
Further details: Symposium Office, Institute of Obstetrics & Gynaecology, Queen Charlotte's & Chelsea Hospital, Goldhawk Road, London W6 0XG, UK

Eating disorders: mysteries, paradoxes, and challenges
20 November, London
Further details: The Training Department, The Institute of Family Therapy, 24-32 Stephenson Way, London NW1 2HX, UK

Infection and immunological disorders in childhood
20 November, London
Further details: Dr M Abinun/Dr S Hoare, Consultant Paediatricians, Newcastle General Hospital, Westgate Road, Newcastle-upon-Tyne NE4 6BE, UK

Neonatal course for senior paediatricians
23-27 November, London
Further details: Symposium Office, Institute of Obstetrics & Gynaecology, Queen Charlotte's & Chelsea Hospital, Goldhawk Road, London W6 0XG, UK

International symposium: periventricular leucomalacia—a research priority for neonatology and public health
30 November to 1 December, Paris, France
Further details: Professor J C Gabilan, Département of Neonatology, Hôpital A Béclère, 157 rue de la Porte de Trivaux, 92141 Clamart, France

Autistic disorders in people with learning disability: diagnosis and management
3 December, London
Further details: Lisa Spicer, Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE, UK

1999

British Paediatric Neurology Association Annual Scientific Meeting
8-10 January, Belfast
Further details: Dr David Webb, Consultant Paediatric Neurologist, Royal Belfast Children's Hospital, Grosvenor Road, Belfast BT12 6BE

5th International congress of tropical paediatrics
10-15 February, Jaipur, India
Further details: Dr Ashok Gupta, Secretary General, 25, Chetak Marg, M. D. Road, India Jaipur-302 004 (India)

Royal College of Paediatrics and Child Health 3rd annual scientific meeting
13-16 April, York
Further details: Miss Amanda Ambalu, RCPCH, 50 Hallam Street, London W1N 6DE, UK

Joint Meeting of British and Italian Societies of Paediatric Gastroenterology and Nutrition
22-24 September, Oxford
Further details: Dr Peter Sullivan, Department of Paediatrics, John Radcliffe Hospital, Oxford OX3 9DU, UK