LETTERS TO THE EDITOR

The vitamin K debacle: cut the Gordian knot

EDITOR,—In his letter[1] about our annotation on vitamin K,[2] Dr Williams questions the incidence quoted for vitamin K deficiency bleeding among babies given no prophylaxis. We thank him for drawing our attention to what was a typographical error [on our part]. The incidence of vitamin K deficiency bleeding should have read 10/100 000 live births. There was an additional error: the manufacturer of Orakay is BMS Laboratories, Beverley, Yorkshire, UK. We apologise for relying on an older Department of Health recommendation for vitamin supplementation.[3]

We remain of the opinion that recommending breast feeding mothers to provide vitamin supplements to their infants is unlikely to reduce breast feeding rates; it has not in the Netherlands (Visser H, personal communication). Most mothers are aware that formula feeds contain supplements and accept vitamin K prophylaxis. We do not state that 25 µg vitamin K is "safe", meaning that it is proved beyond reasonable doubt to be fully effective with no adverse effects—that cannot be claimed for any regimen. We argue that this regimen is logical and practical. There are theoretical reasons to believe (and some reports confirm) that it is more effective than current UK regimens and, in relation to vitamin K, it should be no less safe than formula feeding. We are faced with a dilemma—no proposed solution is perfect, but we need a better policy than we have at present.

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1 Williams AF. The vitamin K debacle [letter]. *Arch Dis Child* 1999;**80**:579.
2 Tripp JH , McNinch AW. The vitamin K debacle: cut the Gordian knot but first do no harm [editorial]. *Arch Dis Child* 1998;**79**:295–7.
3 Department of Health. *Present day practice of infant feeding*. London: HMSO, 1980.

Reduced bone mineral density at completion of chemotherapy for a malignany

EDITOR,—The paper by Arikoski and colleagues[1] provides useful information on bone mineral density (BMD) in survivors of childhood acute lymphoblastic leukaemia (ALL), and the results are consistent with other reports.[2 3] However, we are disappointed in the heterogeneity of the cohort of other malignancies and the major differences in each patient's treatment, as potentially the disease type and the nature of the cancer therapy may have an effect on BMD.

A significant proportion of the other malignancy cohort had radiation directly to or near the spine; the latter may have affected spinal BMD directly, giving a false impression of reduced BMD for the whole cohort. If these patients are removed from the total cohort studied, what impact does this have on the mean BMD?

In the five patients who had received cranial/auricular radiotherapy, what was their growth hormone status? It is not clear from the methods if this was formally assessed.

It is likely that the reduction in BMD in survivors of ALL and even other malignancies is multifactorial, and hence it is only by studying groups of patients treated in a homogenous fashion that it will be possible to tease out the causative factors and hence try to prevent this happening in the future.

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1 Arikoski P, Komulainen J, Riikonen P, Jurvelin JS, Voutilainen R, Kröger H. Reduced bone density at completion of chemotherapy for malignancy. *Arch Dis Child* 1999;**80**:143–8.
2 Nysom K, Holm K, Michaelsen KF, *et al*. Bone mass after treatment for acute lymphoblastic leukaemia in childhood. *J Clin Oncol* 1998;**16**: 3752–60.
3 Brennan BMD, Rahim A, Adams JA, *et al*. Reduced bone mineral density in young adults following cure of acute lymphoblastic leukaemia in childhood. *Br J Cancer* 1999;**79**: 1859–63.

Dr Arikoski and colleagues comment:

Long term studies have shown reduced bone mineral density (BMD) and bone mass in survivors of childhood acute lymphoblastic leukaemia (ALL),[1 2] which might predispose these patients to osteoporosis and diminished resistance to fractures later in life. In our study, we measured BMD at completion of chemotherapy for a childhood malignancy. Because of the heterogeneity of the diagnoses, we analysed the patients in two groups, those with ALL and those with other malignancies. Our finding was that lumbar BMD was reduced only in patients with ALL whereas femoral BMD was reduced in both groups (fig 3).

We understand the concern of Dr Brennan and Professor Shalet that the effect of spinal radiation might give a false impression of reduced spinal BMD for the whole cohort of other malignancies. However, our study showed that lumbar BMD was not reduced in the group of other malignancies, part of whom had received spinal radiation (fig 3). Lumbar BMD was reduced in only those with ALL, and none of the patients with ALL had received radiation in or close to the spinal region (table 1).

The growth hormone status was not analysed formally in our study. We agree entirely with the opinion that the causes of reduced BMD in children with malignancies are multifactorial including treatments with glucocorticosteroids and methotrexate, decreased physical activity, malnutrition, radiotherapy, and growth hormone deficiency.

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2 Arikoski P, Komulainen J, Voutilainen R, *et al*. Reduced bone mineral density in long-term survivors of childhood acute lymphoblastic leukemia. *J Pediatr Hematol Oncol* 1998;**20**: 234–40.

Effect of environmental tobacco smoke on peak flow variability

EDITOR,—In their study on the effect of various indoor pollutants on peak flow variability, Fielder and colleagues[1] should have considered several methodological issues before concluding that environmental tobacco smoke increases airway variability.

Low parental social class is known to be associated with a high prevalence of both parental smoking and respiratory diseases including asthma. Hence, social class is likely to be an important confounding variable, and could easily have accounted for the 5.5% variability in peak flow detected. If parents in the city centre were generally of lower social class than those in the village, it would also have explained higher peak flow variability in children attending school at the city centre. The authors should have included parental social class as a predictor variable in their multiple logistic regression model.

As children spend a considerable proportion of their time at school, and the peak flow measurements were carried out at school, peak flow variability could have been significantly affected by the school air quality. Hence, the air quality should have been measured at the four schools and taken into account in the multiple logistic regression model.

True peak flow variations due to airways variability and apparent variations due to imperfect techniques in the use of peak flow meters both contribute to variations in peak flow measurements. As the winter measurements took place before the summer measurements, the higher peak flow variations in winter could also be explained by increased experience in the use of peak flow meters. Similarly, the lower coefficients of variation in older children may be explained by their relatively better techniques in the use of peak flow meters. Hence, the expected associations between peak flow variations with age and season should not necessarily give confidence in the results showing that poorer lung function was associated with passive smoking.

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1 Fielder HMP, Lyons RA, Heaven M, Morgan H, Govier P, Hopper M. Effect of environmental tobacco smoke on peak flow variability. *Arch Dis Child* 1999;**80**:253–6.

Dr Fielder comments:

Wai-Ching Leung raises some interesting points. For social class to confound the relation between the prevalence of asthma and parental smoking it must be related to both. The literature suggests that the prevalence of asthma may not follow a social gradient,[1] although the severity of symptoms and admissions to hospital are often associated with deprivation.[2 3] Our findings are in agreement with other studies showing that passive smoking has an adverse effect on respiratory function, and therefore it seems reasonable to conclude that some of the variability in peak flow measurement could be attributable to passive smoking. Inclusion of social class may have improved the amount of variability predicted by the model.

The sources of indoor air pollutants were recorded by questionnaire and assumed to remain constant for the study period. Outdoor air quality was measured during the

study using monitors placed at each school. The association of peak flow measurements with fluctuations in atmospheric particulates and gases were studied in a time series analysis, to be reported in a separate paper.

The children and staff were trained in the technique of peak flow measurement by a respiratory nurse in the autumn term before the study started. During the study, the teachers supervised each recording session in the morning to observe the correct technique. Members of the study team visited each school at least once a week to check that the protocol was followed. Measurement of peak flow is not difficult and we did not observe any differences in technique with age or experience.

1 Power C, Hertzman C, Matthews S, Manor O. Social differences in health: life-cycle effects between ages 23 and 33 in the 1958 British cohort. *Am J Publ Health* 1997;**87**:1499–503.
2 Burr ML, Verrall C, Kaur B. Social deprivation and asthma. *Respir Med* 1997;**91**:603–8.
3 Watson JP, Cowen P, Lewis RA. The relationship between asthma admission rates, routes of admission and socioeconomic deprivation. *Eur Resp J* 1996;**9**:2087–93.

Difficult asthma: beyond the guidelines

EDITOR,—I write to draw attention to the fact that in his review "Difficult asthma: beyond the guidelines" Balfour-Lynn[1] has omitted any reference to one of the most important components. Although he refers to problems of adherence to treatment, this is by no means the only important psychosocial component of difficult asthma. Psychophysiological responsivity to emotional challenges in children with asthma and the role of emotional factors in childhood asthma have been well documented over very many years.[2 3] The role of psychological factors in fatal childhood asthma have been clearly established.[4]

Numerous forms of psychological interventions have been shown to have clinically significant effectiveness in randomised controlled trials.[5–8] There can no longer be any dispute about the relevance of psychosocial factors in the triggering and maintenance of episodes of asthma.[9] What is now required is for paediatricians to pay more attention in practice to current knowledge, for so long as they do not, there will continue to be avoidable instances of "difficult asthma."

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1 Balfour-Lynn I. Difficult asthma: beyond the guidelines. *Arch Dis Child* 1999;**80**:201–6.
2 Pinkerton P. Childhood asthma. *Br J Hosp Med* 1971:331–8.
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Dr Balfour-Lynn comments:

I agree entirely with Dr Lask that there are important psychosocial components to difficult asthma. For this reason I stated in the article that it was not uncommon for psychosocial aspects to play a large role in the wellbeing of a child, and particularly an adolescent with asthma. I also emphasised that asthma and psychological disturbance must both be treated on their own merits without necessarily deciding which is primary and which secondary. In the paediatric respiratory unit at the Royal Brompton Hospital in London, UK, our clinical psychologists are a valued part of our multidisciplinary approach to these difficult patients.

Rheumatic heart disease in school children in Samoa

EDITOR,—Rheumatic heart disease (RHD) is a major cause of morbidity and mortality in developing countries,[1] and its true importance may be much greater than is generally recognised. We conducted a prevalence survey of RHD in 8767 school children aged between 5 and 17 years in Samoa; the analysis was weighted to adjust for stratification by urban and rural populations and school level clustering. The prevalence of RHD in our survey was 77.8 per 1000 (95% confidence intervals (CI) 64.0 to 91.6).

The diagnosis of RHD was made on clinical rather than echocardiographic findings by a paediatrician with extensive experience in RHD, and a general physician with cardiology training. Echocardiography increases the specificity of diagnoses of RHD; previous studies suggest that we would have found a prevalence of RHD of about 30 per 1000 if we had used echocardiography. The prevalence of 77.8 cases of RHD per 1000 found in Samoa is the highest reported prevalence in the world to date. A World Health Organisation study estimated that the mean prevalence of RHD among children in developing countries is 2.2 per 1000.[1]

Three factors may explain the very high prevalence of RHD in Samoa. First, we found that the risk of RHD was significantly higher in rural areas in Samoa (odds ratio 1.86; 95% CI 1.37 to 2.53; $p < 0.001$) where access to medical services is poor. This is at odds with findings in other studies conducted in developing countries in Africa,[2] where urban residence has been identified as an important risk factor in RHD.

Second, RHD and acute rheumatic fever appear to be particularly common in the Pacific region. The highest annual incidence of acute rheumatic fever in the world was found in Australian Aborigines (254 per 100 000)[3] followed by Samoans in Hawaii (206 per 100 000).[4] Whether this predominance in the Pacific region is due to environmental or genetic factors is not known.

Third, the Samoan children in our study had a high prevalence of pyoderma: the overall prevalence was 43.6% (95% CI 36.7 to 50.2%) and it was highest in the rural regions (57.2%). Unlike the situation in Australian Aboriginal populations,[5] the high prevalence of pyoderma was not caused by secondary infection of scabies; the prevalence of scabies was only 4.9% (95% CI 3.0 to 7.0%) in our survey, perhaps because ivermectin has been widely used to eradicate filiariasis in Samoa.

In a subgroup of children, the carriage rate of group A streptococci was 2.4% in the pharynx and 51.3% in pyoderma lesions. This indicates that group A streptococci are more often found in the skin than in the pharynx of Samoan children, as observed in Australian Aborigines.[5] The role of group A streptococci from pyoderma lesions in the pathogenesis of acute rheumatic fever and RHD is not clearly understood, although a possible mechanism has been suggested.[5]

RHD is a major cause of morbidity and mortality in Samoa. Further studies of the epidemiology of RHD are needed in the Pacific region, preferably with echocardiographic confirmation of the diagnosis.

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1 Nordet P. WHO programme for the prevention of rheumatic fever/rheumatic heart disease in 16 developing countries: report from phase 1 (1986–90). *Bull WHO* 1992;**70**:213–18.
2 Maharaj B, Dyer RB, Leary WP, Arbuckle DD, Armstrong TG, Pudifin DJ. Screening for rheumatic heart disease amongst black schoolchildren in Indana, South Africa. *J Trop Pediatr* 1987;**33**:60–1.
3 Carapetis JR, Wolff DR, Currie BJ. Acute rheumatic fever and rheumatic heart disease in the top end of Australia's Northern Territory. *Med J Aust* 1996;**164**:146–9.
4 Chun LT, Reddy DV, Yamamoto LG. Rheumatic fever in children and adolescents in Hawaii. *Pediatrics* 1987;**79**:549–52.
5 Carapetis JR, Currie BJ. Group A streptococcus, pyoderma and rheumatic fever. *Lancet* 1996;**347**:1271–2.

Acute appendicitis in children after renal transplantation

EDITOR,—Acute appendicitis is the most common surgical emergency in children, estimated to occur at a rate of 4 per 100 per annum. Its prompt diagnosis is important because the risk of perforation increases with time, occurring in 10% of patients by 24 hours and up to 50% by 48 hours.[1] The technique for renal transplantation in children is similar to that in adults, the extraperitoneal placement of the kidney being sited in the right (or more rarely left) iliac fossa (RIF). The renal artery is anastomosed to the common iliac artery or aorta and the renal vein to the external iliac vein or inferior vena cava. The differential diagnosis of RIF pain in this group of children will therefore include conditions frequently affecting the graft: acute obstruction, urinary tract infection, and acute allograft rejection. This may result in a delay in diagnosis of acute RIF pain secondary to other causes.

A 10 year old boy was transferred from his local hospital where he presented with a history of suspected gastroenteritis and severe hypo-osmolar dehydration. He had received a cadaveric renal transplant eight months previously. He was in renal failure from pseudo-prune-belly syndrome (grossly dilated urinary tract but good rectus muscles),[2] and extensive urological surgery had been undertaken in the past. Four days before presentation, he developed a pyrexial illness associated with headache, diarrhoea, and vomiting. There was notable right sided abdominal and suprapubic pain. After 12 hours at the local hospital he was transferred with a presumptive diagnosis of acute allograft rejection.

On examination he was febrile (38.5°C) and his blood pressure was 110 mm Hg systolic. He had a tachycardia and a capillary refill time of two seconds. His abdomen was diffusely tender with localisation over the graft in the RIF with guarding. A rectal examination was not done. Haemoglobin was 102 g/l; white cell count 22 000 (90% neutrophils); plasma sodium 113 mmol/l, plasma creatinine concentration 78 µmol/l compared to a baseline of 60 µmol/l. His urine microscopy was negative for leucocytes and bacteria.

Following resuscitation with intravenous fluids he underwent urgent laparotomy; a perforated appendix was resected. There was subhepatic and subphrenic collections of free pus. He was given 50 ml/kg body weight colloid and blood during surgery, and postoperatively he required ventilation for 12 hours. He was kept nil by mouth for six days and received immunosuppression and nutrition intravenously. Intravenous antibiotics were continued for one week. He was allowed home on the ninth postoperative day with normal renal function.

There are few reports of appendicitis in kidney transplant recipients. Eight patients, all of them adults, have been reported in the literature to date.[3–6] Appendicitis in kidney transplant recipients is an important diagnosis to make promptly because of the risk of perforation. A delay in the diagnosis of acute appendicitis in preschool children has been highlighted in a recent review.[7] Of 132 children treated for acute appendicitis, 36 (27%) had an uncomplicated illness, 63 (48%) had perforation, and 29 (22%) had an appendix mass. The mean duration of symptoms before admission was 39 hours in the uncomplicated group, 53 hours for perforation, and 82 hours for appendix mass. A misdiagnosis was made in 53 cases (40%) leading to a delay in management.

Renal transplantation is a relatively uncommon clinical subject for the general paediatrician, who will be unfamiliar with many of the acute problems presenting in a transplant patient. It is uncommon for acute allograft rejection, obstruction, or urinary tract infection to cause peritonism. However, acute appendicitis can occur in the transplant recipient just as in the general population; any delay in diagnosis may complicate the illness.

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1 Oski FA, ed. *Principles and practice of pediatrics.* 1st ed. Philadelphia: JB Lippincott, 1990.
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4 Lao A. Colonic complications in renal transplant recipients. *Dis Colon Rectum* 1988;**31**: 130–3.
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Mothering to death

EDITOR,—Professor Sir Roy Meadow paints a spine chilling tableau of the lethal effects of abnormal mothering in three fascinating case reports,[1] conspicuous by the lack of involvement of paediatric, education, and social services. He goes on to ask "whether there are similar children today being kept in bed, or being kept at home, and being prevented from participating in normal school and social life, and from growing up as healthy independent adults?" The simple answer is yes—there are children today who grow up believing that they are disabled or unable to attend school because of their parents' disordered perception. It has been our experience that Social Services have great difficulty in proving "significant harm", even when alerted by health professionals, and they feel powerless to act in such "borderline cases". The problem is compounded further by at least two unrelated pieces of legislation, respectively the Disability Living Allowance (DLA)[2] and the Education Otherwise[3] regulations; the finer details of which do not usually form part of child protection training.

Currently, it is perfectly legally possible for a parent to label their child as disabled and to receive Disability Living Allowance for the child's "severe disability", without the child having ever been assessed by a paediatric medical/surgical/psychiatric specialist. The (unsuspecting) general practitioner's report of "mild asthma, eczema, behaviour problems and scholastic difficulties" is interpreted as "severe disability" by the Benefits Agency (who has surprisingly very little leeway in the matter), and the child acquires a sometimes lifelong, entirely inappropriate and potentially extremely damaging lucrative tag. We propose the term "DLA abuse" for those cases where the child is thus inappropriately labelled, as in the cases described by Professor Meadow (which one could call disability abuse), with the additional complication of a financial incentive for the disability status. It should also be noted that, at the other end of the spectrum, the current disability regulations do not serve the needs of some seriously learning disabled but physically normal children, whose parents' application for an allowance is often turned down, even when a paediatric specialist has recommended they apply for it.

It is not generally known to paediatricians or social workers that it is also perfectly legal for a parent to either never send a child to school, or to remove their child from school at any stage, provided they undertake to make alternative provisions. There is no requirement on the education authority or anyone else to assess the alternative provision (apart from an initial visit, about to be abolished), or its outcomes. There is no current mechanism for an older child's choice in such matters to be established. It is our experience that some parents, when pressed by a network of heath, education, and social services for poor school attendance, suddenly discover the existence of the "Education Otherwise" provisions and use them to avoid prosecution (even when the child has a statement of special educational needs). These children then become "lost" and are invisible to the usual agencies; they are only ever seen by their general practitioner, or accident and emergency services. Some of them resurface a few years later in adult psychiatry services, by which time their emotional health is beyond repair.

This is not meant to ignore the fact that many children are appropriately in receipt of Disability Living Allowance and some children receive excellent education outside a normal school environment; but it is equally undeniable that there are a number of cases in which, in our view, the current legislation is misused and the letter of the law is adhered to while its spirit is ignored, with the result that children are suffering avoidable significant harm.

All professionals concerned about the welfare of children need to ask for a rationalisation of the regulations that lead a child to be labelled as severely disabled and a monitoring system for children Educated Otherwise. Unless this happens, the lost children of today will, 10 years after the Children's Act, continue to lead their hidden and unhappy lives, regardless of "the welfare of the child is paramount" principle.

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1 Meadow R. Mothering to death. *Arch Dis Child* 1999;**80**:359–62.
2 Steadman P. The new Disability Living Allowance. *Arch Dis Child* 1993;**68**:73–4.
3 *The Education Act, 1996.* Part 1, chapter 1, section 7 and 9. London: HMSO.