LETTERS TO THE EDITOR

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Iron deficiency anaemia, *Helicobacter pylori* infection and delayed pubertal growth

We read with great interest the paper by Choe et al described by the authors.

Iron status and development

**Editor,—** You have recently published a response by Dr Morley to Dr Stevens concerning our paper on iron status and development.1 We wish to raise two matters not discussed in Dr Morley’s letter.

Firstly, when the paper was published, there was prominent media coverage, erroneously concluding that our paper rendered formula unnecessary in the later part of the first year, and that cow’s milk was just as satisfactory. We emphasise that the reason for avoiding cow’s milk in the first year (in babies not breast fed) is based on several considerations, including subclinical nutrient content or bioavailability (for example, for vitamins D and C, iron, and copper), unnecessarily high protein content, and, possibly, increased risk of subclinical gastrointestinal bleeding. Our paper does not provide evidence that should change present policy on cow’s milk feeding.2 Secondly, the haemoglobin data we included, which were not central to the focus of the paper, were from one centre only and should not be taken as typical of those for the UK population. A more complete presentation of iron status in children in the trial is now published.3

The current evidence on the importance of iron in infancy is much debated and frequently cited in policy documents. We wish the interpretation of our study to be as clear as possible.

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**Coeliac screening just as important!**

**Editor,—** The paper by Noble et al on capillary dried spot testing for thyroid stimulating hormone measurement is a welcome advance for children with Down’s syndrome (DS).1 In reducing the number of venepunc
tures this patient group needs, we hope that those professionals caring for children with DS do not omit screening for coeliac disease (CD). This condition is equally prevalent and can be as difficult to diagnose as hypothyroidism in DS, as it too is often asymptomatic. Screening for CD should be done by the measurement of antigliadin (AGA) and antiendomysial (EmA) antibodies. Their use as a screening tool is well described in DS with reported prevalence between 3.9% and 16.9%.2 Diagnosing CD has important consequences with regard to preventing long term complications and maximising growth potential.3 We would like to highlight that community based testing is also feasible for CD.

A study at our centre investigating the prevalence of coeliac disease in type I diabetic utilised patient self sampling for screening blood samples.4 Blood was drawn into a lithium heparin capillary tube (Monovette, Sarseldt Ltd, Germany) or onto filter paper. The in house assays used for AGA and EmA were performed on 10-20 µL of serum or plasma; thus capillary samples were more than adequate. This method could easily be incorporated into the “at school” testing described by the authors.

Annual screening for hypothyroidism is recommended.1 How often screening should be performed for CD is still a matter of debate. With their proposal to establish a Scottish register of school aged children with Down’s syndrome, Noble et al provide an opportunity to perform a Scottish-wide population study for the prevalence of coeliac disease in Down’s syndrome and, more importantly, to identify those children who may benefit from early detection. Community based screening with capillary samples would make that a very realistic prospect.

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Consider absolute risks in SIDS prevention

EDITOR,—The demonstration by Blair et al of an association between poor postnatal growth and an increased risk of sudden infant death syndrome (SIDS),1 is a useful addition to our understanding of the aetiology of this condition. It is unfortunate that the conclusion in the abstract that “Poor postnatal weight gain was independently associated with an increased risk of SIDS and could be identified at the routine six week assessment” goes beyond the data presented.

It can be estimated from the data in this study that the overall risk of SIDS was 0.77/1000 live births. The risk in babies with birth weights greater than the 15th centile, the group in whom the relation with postnatal growth was detected, was 0.68/1000. Given the reported odds ratio of 1.75 associated with being in the lowest growing 16% who might be identified if six weeks, the data suggest that the absolute risk of SIDS among this group would be about 1.1/1000. Even a programme targeted at infants below the 2nd centile for growth at six weeks, would identify a group whose absolute risk of SIDS was about 4.2/1000—that is, for every infant who might benefit from the intervention, there would be 20 who would not.

Yet the authors suggest that this level of risk was sufficient to trigger an intervention, the nature of the intervention remains unclear. To the best of our knowledge, with the exception of the “Back to Sleep” campaign, there is no convincing evidence of the effectiveness of any intervention aimed at preventing SIDS. None of the intervention programmes described in the accompanying commentary1 have been evaluated in appropriately controlled studies.

The discussion in the paper is rather more abstract, but it is the latter which, reinforced by the accompanying commentary,1 has been translated into appropriately controlled studies. The effect of these studies is that conjuring up of an intervention has become the conclusion of the study. We agree that absolute risks must be used for targeted prevention campaigns but do not advocate such a campaign based on our solitary finding. Preliminary analysis of risk scoring for the first two years of our dataset, tested on the third year show that 42% of SIDS families can be identified from 8% of the population using univariate factors. Incorporating postnatal factors such as weight gain may improve the specificity and sensitivity of such a scoring system.

To our knowledge the “Back to Sleep” campaign initiated in October 1991 has not been evaluated in an appropriately controlled study yet the SIDS rate in England and Wales more than halved from 1.7 deaths per 1000 live births in 1990 to 0.77 deaths in 1992.” Findings from our study have helped build on the advice regarding sleeping practices; parents are now advised not only to avoid placing infants in the prone position but also the side position, bed sharing under certain circumstances, sharing a sofa to sleep, and to avoid head covering. In the study, none of the infants at the foot of the cot. In 1998 the SIDS rate fell a further 25% in England and Wales to 0.45 deaths per 1000 live births. There is no direct evidence that these recommendations and fall in SIDS rates are linked although the dramatic fall in the prevalence of parents placing their infant in the prone position but also the side position, bed sharing under certain circumstances, sharing a sofa to sleep, and to avoid head covering. In the study, none of the infants at the foot of the cot. In 1998 the SIDS rate fell a further 25% in England and Wales to 0.45 deaths per 1000 live births. There is no direct evidence that these recommendations and fall in SIDS rates are linked although the dramatic fall in the prevalence of parents placing their infant in the prone position but also the side position, bed sharing under certain circumstances, sharing a sofa to sleep, and to avoid head covering. In the study, none of the infants at the foot of the cot. In 1998 the SIDS rate fell a further 25% in England and Wales to 0.45 deaths per 1000 live births.

Are we requesting too many DMSA scans?

EDITOR,—The recent article by Christian et al highlights the value of clinical features in assessing the risk of renal scarring and therefore the need for dimericcepsuccinic acid (DMSA) scan after urinary tract infection (UTI). We recently performed a pilot study to assess the recording of fever, malaise, recurrent UTI, and urine culture results in children investigated with DMSA scan after UTI. Between April 1996 and October 1997 there were 171 DMSA scans in our hospital that fitted these criteria; 30 case notes could not be traced. There were 105 girls (74%) and 36 boys. Age when UTI was diagnosed ranged from 9 days to 15.3 years (mean 4.2 years, SD 3.2). Urine culture results were: UTI (>10⁵ cfu/ml) in 82 cases (58%), contaminant (<10⁵ cfu/ml) in 27 cases (19%), no growth in 21 cases (15%), and no urine culture in 11 cases (8%). There were 17 (12%) cases of definite or probable renal scar, none of which followed a sterile or contaminated urine culture. Of the 141 case notes, there was no mention of fever in 48 (34%), and no mention of malaise in 76 (54%). In 69 case notes reviewed there was no previous history of UTI in 14 (20%) cases. Of those with a history of fever, 19% (10/53) had an abnormal DMSA scan compared to 10% (4/40) in those without fever. Eighteen per cent (9/50) of those unwell at the time of UTI had an abnormal scan compared to 13% (2/15) of those not ill.

These data suggest that in a substantial proportion of cases, the decision to request a DMSA scan is apparently not influenced by salient clinical features and urine culture results. In this series, it is far more likely that those children with sterile or contaminated urine cultures should not have had a DMSA scan. This would have saved the cost and burden of 48 scans, 34% of this series, over an 18 month period. It is likely that these findings are peculiar to our district.

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Dr Blair, Fleming, and Platt comment:

Poor postnatal weight gain was a significant factor in the multivariate analysis despite controlling for low birth weight, prematurity, neonatal problems, poor socioeconomic sta-

dus, and many other potential confounding factors, and remained significant when further highly predictive covariates of SIDS such as infants put down prone, infants found with head covered, and tobacco exposure were added to the model. In this sense postnatal weight gain was independently associated with an increased risk of SIDS. We also found the difference in weight gain between the SIDS and control infants measured from birthweight to the last known weight was equally apparent if measured from birth to the six week assessment. We therefore stand by our conclusion in the abstract that poor postnatal weight gain “was independently associated with an increased risk of SIDS” and also that poor postnatal weight gain “could be identified at the routine six week assessment.”

We find it difficult to understand how this conclusion advocates an intervention campaign on the basis of these two statements and disagree with Logan and colleagues that our discussion on this matter was circumspect. We point out clearly that poor weight gain itself is not a sensitive marker and that “should be seen as a thread in a web of factors that render an infant vulnerable to SIDS and is both a consequence of adverse health and social conditions”.

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