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

We read with great interest the paper by Choe et al. (1997) that concluded that *H. pylori* infection may cause IDA in diabetics utilised patient self-sampling for screening with blood samples. Blood was drawn into a lithium heparin capillary tube (Monovette, Sarsedd Ltd, Germany) or onto filter paper. The in house assays used for AGA and EmA were performed on 10–20 ml 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. 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 syndrome, Noble and colleagues 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.
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 at 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. Even if it were accepted 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 commentary2 have been evaluated in appropriately controlled studies.

The discussion in the paper is rather more circumspect than the conclusion in the abstract, but it is the latter which, reinforced by the accompanying commentary, is likely to have disproportionate impact on those readers who do not read the whole paper. In the commentary Dr Carpenter acknowledges that the low rates of SIDS make such interventions difficult to justify but then suggests that “Targeting such infants for intervention di

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 dimercaptosuccinic acid (DMSA) scan after urinary tract infection (UTI). We recently performed a 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).

Urinary 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 mention of 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% (2/13) 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 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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References