Iron deficiency anaemia, helicobacter pylori infection and delayed pubertal growth

We read with great interest the paper by Choe and colleagues which investigated a possible relationship between Helicobacter pylori infection, iron deficiency anaemia (IDA), and subnormal growth at puberty.1 The authors concluded that H pylori infection and related IDA, rather than bacterial infection alone, might cause delayed pubertal growth. However, we believe that these results need some consideration.

H pylori infection may cause IDA in different ways: (a) the bacterium can cause a decrease in the gastric juice of the concentration of ascorbic acid, which is the best promoter of non-heme iron absorption; (b) H pylori may increase iron demand because iron is an essential bacterial growth factor; (c) H pylori contains a 19.6 kDa protein resembling ferritin with a binding activity for heme iron; (d) acute or chronic infection may increase iron demand because iron in erythrocytes is an essential bacterial growth factor; (e) iron absorption is promoted by ascorbic acid, which is the best promoter of non-heme iron absorption; (f) Helicobacter pylori infection with iron deficiency anaemia and subnormal growth at puberty. Arch Dis Child 2000;82:436–40.

Firstly, when the paper was published, there was prominent media coverage, erroneously concluding that our paper rendered formula unpalatable in the latter 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 suboptimal 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.

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.

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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Iron deficiency anaemia, helicobacter pylori infection and delayed pubertal growth

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 unpalatable in the latter 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 suboptimal 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.

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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” is not made 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 “Targeting such infants for interventions diﬀers who do not read the whole paper. In the abstract, but it is the latter which, reinforced our discussion on this matter was circum-

spect. We point out clearly that poor weight gain itself is not a sensitive marker and that it “should be seen as a thread in a web of factors that render an infant ‘inordinately close’ to SIDS and is both a consequence of adverse health and social conditions”.

We agree that absolute risks must be used for targeted prevention campaigns but do not advocate such a campaign based on our soli-
tary finding. Preliminary analysis of risk scor-
ing on the first two years of our dataset, tested an association between bedding and sleeping position in the third year show that 42% of SIDS differences 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”.

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).4 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 at UTI was diagnosed ranged from 9 days to 15.3 years (mean 3.2 years, SD 2.4 years).

Urine culture results were: UTI (>10^5 cfu/ml) in 82 cases (58%), contaminant (<10^5 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 possible 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 unlikely that these findings are peculiar to our district.

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Dr’s 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-
Iron deficiency anaemia, *Helicobacter pylori* infection and delayed pubertal growth

L CUOCO, G CAMMAROTA, R A JORIZZO, R CIANCI and G GASBARRINI

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