Sir,

I would like to present our experience with copper deficiency in neonates of very low birthweight and to comment on the paper by Sutton et al.1 Over the previous two years we diagnosed five cases of neonatal copper deficiency (birthweight: 740–1200 g, gestational age 26–34 weeks) at the postnatal age of 8–20 weeks. Three neonates had bone changes, but all had anaemia and severe neutropenia in the absence of infection. Only two neonates had required prolonged periods of ventilation and parenteral nutrition. All had received a milk formula with a relatively high copper content. At the time of diagnosis their serum copper concentration was <0.4 μmol/l (2.5 μg/100 ml) and their caeruloplasmin concentration was <1.5 g/l (1.4 mg/100 ml). They were treated with 5 mg/day of copper sulphate solution (1% solution) for six months with excellent results. The earliest and most sensitive indicator to treatment was the response of the neutrophil count (increase in 48 hours after treatment and return to normal in two weeks). With this experience and reviewing previous reports I should like to make the following comments.

(1) Although serum copper and caeruloplasmin concentrations provide some information, they do not adequately reflect the copper stores of the body. Therefore treating very low birthweight infants with copper sulphate because of low serum copper and caeruloplasmin concentration in the absence of any other finding is questionable.

(2) The most sensitive and early index of copper deficiency is in our experience haematological. Bone changes will eventually occur in all patients, but they are a relatively late finding. In preterm neonates anaemia is a common finding of varied aetiology and can be masked by transfusion. Severe neutropenia in the absence of infection, on the other hand, is an unusual and impressive finding. I would agree with Sutton et al that facilities for copper and caeruloplasmin estimation should be available to neonatal units. I think, however, that these variables should be measured in all very low birthweight neonates over the age of 4–6 weeks. If selective measurements are preferred, I would favour neutropenia to be the indicator rather than the findings suggested by Sutton et al, which are either relatively late (osteoporosis) or unusual (oedema).

(3) With respect to treatment I think that neither the dose of copper sulphate nor the length of treatment is presently known. In the absence of balance studies involving at least copper and zinc I would not favour the administration of excessively high doses of copper over a very short period as described by Sutton et al. It is probable that in order to replenish copper stores a longer period of copper administration is necessary.

Reference


H D DELLAGRAMMATICA

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Dr Sutton and co-workers comment:

We were interested to hear of this experience of copper deficiency in Greece and assume that the caeruloplasmin concentrations were less than 0.15 g/l (0.14 mg/100 ml) rather than as stated in the letter. We agree that neutropenia is a useful finding in copper deficiency, but feel that we should try to make a diagnosis before this stage is reached. Growth failure and oedema were present up to four weeks before significant neutropenia or anaemia was seen, and osteoporosis was reported at an earlier stage also. We would dispute the statement that bone changes are a relatively late finding and would suggest that this may be a consequence of the rather insensitive methods of detection.

With regard to treatment, there is an unfortunate error in the dose of copper that we used which should read 4 μmol (254 μg) copper/kg/day. This was based on the experience of Yuen et al and has proved to be satisfactory when given for 1–2 weeks in the six cases we have treated. This dose is approximately four times the minimum daily requirement for the preterm infant, and we would not consider it excessive. The dose of 5 mg per day of copper sulphate (presumably pentahydrate solution, equivalent to 1.25 mg of elemental copper) given by Dr Dellagrammaticas is approximately five times the amount we used. There is no evidence that this amount of copper needs to be given for such a prolonged period, and indeed, as there is competition for absorption with other essential elements it might be detrimental.

Our intention was to increase the general awareness of copper deficiency so that ideally it can be prevented by adequate supplementation, or at least diagnosed at an early stage to reduce the risks from the metabolic effects of a deficiency state. We would therefore strongly disagree with the suggestion by the author of the letter that neutropenia should be the indicator for diagnosis.

Posthaemorrhagic hydrocephalus in newborn term infants

Sir,

Dr Hill and Morgan1 draw attention to the occurrence of intraventricular haemorrhage in previously well, full term infants but in their discussion of aetiology fail to mention the possibility of α1 antitrypsin deficiency. Readers of this journal will know of the association between α1 antitrypsin deficiency and a bleeding diathesis in the newborn2 and of the occurrence of intracranial haemorrhage in such infants.3 4 We have also seen α1 antitrypsin deficiency presenting as intracranial haemorrhage in a previously well, full term infant who had received vitamin K at birth. We would suggest that the diagnosis of α1 antitrypsin deficiency is considered in full term infants presenting with intracranial haemorrhage.

References

...And children first?

Sir,

I can assure Professor Smithells¹ that most women in Liverpool (and presumably in Leeds also) go to work not because of what they have read from female journalists but to keep themselves or their family out of poverty. It is not uncommon for the woman to be the only breadwinner in a family.

It is disappointing that while Professor Smithells is happy to criticise teachers and journalists his criticism of paediatricians is less forthright. The truth is that the BPA and its journal Archives of Disease in Childhood have remained silent on the major social issues that affect the health of children for too long. To give but a few examples:

(1) Smoking. Why is it only the BMA that is issuing press statements on tobacco advertising when it is in childhood (primary and secondary school) that smoking is started. Paediatricians should be at the forefront of the medical profession's campaign against smoking.

(2) Car safety. The excellent article by Sprigg² would have benefited from a short editorial endorsing the suggested recommendations and an accompanying press release. If it is not BPA policy to try and save children's lives by legislation then it should be.

(3) Breast feeding. This is emphasised by David Morley as a priority in the Third World;³ is it not also a priority in the United Kingdom? The silence of British paediatricians with regard to the WHO code of marketing of breast milk substitutes is scandalous.

(4) Poverty. With nearly one in three children living in poverty⁴ this is a major issue that directly affects child health—both physically and mentally. Should we not put pressure on the government to increase child and supplementary benefits?

(5) Unemployment. It is now accepted by a majority of schoolchildren in inner city areas that they will not get a job when they leave school. What is the psychological effect of the threat of unemployment on these children? Again the discussion on unemployment and health within the medical profession has been ignored by paediatricians.

The medical profession is one of the most powerful pressure groups within the country. There is no doubt in my mind that paediatricians if seen to be campaigning for the rights of children could be very effective—both in terms of changes in legislation and also by utilising the mass media to get our message across to children. The question that remains is what will it take to get paediatricians into action?

Professor Smithells comments:

Sir,

I would not take issue with anything Dr Choonaara writes. I appreciate that mothers may have to go out to work for economic reasons, although my comment specifically excluded single parent families. The breast feeding/milk advertising issue is of principal importance in the Third World, whereas I was writing about English children. The other four specific issues mentioned by Dr Choonaara are not primarily medical. Nevertheless, I asked 'Are paediatricians doing all they can...?' the implication being that I think not; and publication in Archives makes clear to whom my comments were primarily directed.

Sir,

Dick Smithells' provocative opening remark in his 'Point of View' paper¹ evokes a response in one who some years ago gave up the somewhat unequal struggle of supporting children against some aspects of British society today. He says 'Why do the English dislike children?' Why stop at the English: the inclusive term British might have been more appropriately used.

Professor Smithells only partially answers his own question, which reminded me of a comment made by a contemporary of mine many years ago in reply to a similar question. My colleague said 'The British don't actually dislike children, they just like dogs more.' Near as we are to the 21st century many British parents (some of whom are politicians and leaders of one sort or another) still cling to the tenets of the 19th century in rearing their children: 'don't spoil the brats, bring them up tough, build their character'. In short the 'stiff upper lip' syndrome is alive and well.

It has always seemed to me not without significance that our country has the Royal Society for the Prevention of Cruelty to Animals, the Royal Society for the Protection of Birds, and the National Society for the Prevention of Cruelty to Children.

Children have no votes and some politicians are content to leave them in a subsidiary, if not second class, position: