Correspondence

Height and lymphoblastic leukaemia

Sir,

Broomhall et al.1 have shown that in their series of children with untreated lymphoblastic leukaemia (ALL) the mean height was significantly greater than that of the normal population. They suggested that the disease may occur in constitutionally tall children, or that there may be an aberration of growth associated with leukaemia that may in turn be related to abnormal concentrations of growth hormone or somatomedin.

There is perhaps an alternative explanation. It has been shown that ALL of childhood occurs with greater incidence in children from upper social class families.2,3 It is also known that upper social class children are generally taller than those from lower social class backgrounds. For example Goldstein,4 in an analysis of a large cohort of 7 year old British children found that children from social class 1 and 2 were on average 3.3 cm taller than those from social class 5. This difference may well be sufficient to explain the observed increase in height of the leukaemic children in the series of Broomhall et al. Without more information it is impossible to analyse their data further.

We have examined our own data and did not find any significant increase in height in leukaemic children at presentation. We analysed the heights of 117 children with ALL diagnosed between 1975 and 1981 and used the same restrictions as Broomhall et al., namely boys aged 0-12 years and girls aged 0-10 years. We used the growth data for Australian children provided by the National Health and Medical Research Council5 for comparison. The standard deviation score for the whole ALL group was +0.19 (Figure), and was +0.39 for the 59 boys, and -0.02 for the 58 girls. The mean age of the children was 4.62 years. For the 101 cases in whom it was possible to allocate a social class on the basis of the father’s occupation,6 no correlation was found between social class and standard deviation score.

Oakhill and Mann7 noted that Asian children with ALL living in England, who were generally of lower socioeconomic status than native white children, were significantly shorter than the white children with ALL. The findings of Broomhall et al. are certainly of interest but their significance remains unclear, especially as others7 have found that children with ALL are notably shorter than normal at the time of diagnosis.

References


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Dr Lilleyman comments:
Drs McWhirter, McWhirter, and Taylor suggest that our unexplained finding of increased height in children with lymphoblastic leukaemia may be caused by an atypical distribution of the social classes among them. This raises two questions. Was there such a bias in UKALL II, and if so, was it responsible for the children being taller? The first we cannot answer as the relevant information is not available to us. If, however, there were more children...
Earlier recognition of abnormal stature

Sir,

Any attempt to identify and treat short children is to be applauded. I therefore strongly support the Oxford screening chart designed by Drs Aynsley Green and MacFarlane.¹ My concern about this approach is that it identifies a child once he has already become short. In many instances, this is too late because even if the cause is found and treated, the ultimate result depends on how much growth has already been lost in the process of identifying the child.

I think that it is preferable to screen for growth problems on the basis of height velocity measurements because these may identify children who may not yet be absolutely short but who already need attention because they are growing badly. The Figure shows the chart which we have introduced in child health clinics in Brent for this purpose, and I offer it as an alternative to the more absolute screen proposed by Aynsley Green and MacFarlane.

Reference

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Lithium toxicity in the newborn

Sir,

We were interested in the papers by Wilson, Forfar, and Godman¹ and by Morrell et al.² because in 1974 we published a case of transplacental lithium poisoning³ that was diagnosed antenatally by noting an irregular and often slow fetal heart. In that paper we made two points which are worth repeating because of the current interest in this subject.

Firstly, although controlling the mothers’ serum values in pregnancy is desirable, it must be stressed that normal values do not exclude toxicity in the newborn. Normal maternal values were found in the patient we reported and in others in the published reports, although the two in the Archives both had concentrations of lithium in maternal blood above the normal adult therapeutic range.

Secondly, because bradycardia and irregularity of the fetal heart may be caused by lithium toxicity, these signs do not necessarily mean fetal distress if the mother is receiving this drug.

References

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