but rather to early-normal puberty in the majority of girls.\textsuperscript{2} One wonders why Dr Leiper did not analyse in more detail the growth and development of the well defined group of girls who attained menarche and instead formed a selected group with evidence of early pubertal maturation.

Interestingly enough, the growth pattern of the girls followed to final height who were reported in Dr Leiper's article, agrees very well with the results presented by us.\textsuperscript{2} Dr Leiper and coworkers do not analyse the growth data of the 55 girls who attained menarche. In our study we found that the average height decreased from -0.5 SD before puberty to a mean final height of -1.5 SD. This loss in stature was due to a subnormal growth spurt.

Analysis of spontaneous growth hormone secretion during 24 hours indicates that girls treated for acute lymphoblastic leukaemia, including cerebral irradiation, have severely blunted growth hormone secretion already before puberty (C. Moell et al., unpublished data). Growth at that time, however, is not decreased.

The growth hormone insufficiency of these girls seems to be relative and manifests itself only when the increased demands for growth hormone during puberty cannot be coped with. In patients who lack an adequate growth spurt the onset of early puberty will cause additional impairment of final height. In this respect we agree with Dr Leiper that precocious and premature puberty can be an important factor in contributing to short stature in girls treated for acute lymphoblastic leukaemia.

References


Drs Stanhope, Leiper, and Chessells comment:

Unfortunately, the paper by Dr Moell and coworkers was received after our manuscript had been submitted. We agree with most of their findings but we wish to indicate several important differences.

Four of the 10 patients described in Dr Moell’s paper had received craniospinal irradiation and it is not appropriate to include these children in the analysis of peak height velocity without data on sitting height and subischial leg length growth velocities. Moreover, the fields for spinal irradiation in early childhood may well have included ovarian tissue. No data were given for the onset of puberty in the patients described by Dr Moell’s group. The fact that the patients were all girls is interesting, however, and supports our hypothesis for the difference in timing of the onset of puberty in normal girls and boys.\textsuperscript{2} We excluded patients who had developed early puberty soon after receiving cranial irradiation; as stated in our paper, mean age at treatment was 4.0 years (range, 1.4–7.8) whereas mean age for the onset of puberty was 8.8 years in the girls and 9.3 years in the boys.

Although menarche is an easy event to date precisely, it has little physiological meaning. Menarche does not usually indicate the onset of fertility and the mechanism of a uterine ‘withdrawal bleed’ is simple compared with the intricate endocrine events preceding an ovulatory cycle. We only used menarche retrospectively to correlate the timing of cranial irradiation and sexual maturation.

We agree with many of Dr Moell’s comments about the growth of children with acute lymphoblastic leukaemia treated with prophylactic cranial irradiation; the predominant loss in height of such children is during the pubertal growth spurt. Although sex steroids are synergistic with growth hormone, the onset of the pubertal growth spurt is contemporaneous to a large increase in growth hormone pulsatility and indeed growth during puberty correlates with growth hormone secretion.\textsuperscript{2} It is not surprising that the predominant impairment in the growth of children who have growth hormone insufficiency secondary to cranial irradiation is during the pubertal growth spurt.\textsuperscript{3}

References


Increasing medical burden of child abuse

Sir,

I share Drs Sharma and Sunderland’s concern about the increasing medical burden of child abuse.\textsuperscript{1} In Central Birmingham Health District, with 43,000 children aged up to 15 years, health visitors now attend over 300 child abuse case conferences annually. We too are experiencing an appreciable increase in referrals which subsequently prove to be unfounded. School and nursery staff have heightened awareness, but this has increased the rate of false positive referrals. It is well recognised that identified child abuse is more common in children from socially deprived backgrounds. Such children are also more likely to sustain accidental injuries. My concern is that an unexplained bruise found on the inner city child of inarticulate parents, socially distanced from professional staff, may be much more likely to result in a child abuse investigation than an identical bruise in the advantaged child of middle class parents.

The circumstances which lead families to abuse their children are complex, and not confined to particular classes of society. I cannot agree with Sharma and