difference may be explained by variable gene expression or by the mother's taking of an oestrogen-containing contraceptive pill until two to three months before her serum thyroxine binding globulin was estimated. This may have served to raise her intermediate thyroxine binding globulin values. Congenital deficiency of thyroxine binding globulin in association with hypothyroidism has been described but we cannot find any previous reports of hereditary excess associated with congenital hypothyroidism, a combination which, unfortunately on this occasion, has been associated with serious long term disability.

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Pubertal growth in diabetics

S L STEWART-BROWN, T J LEE, AND D C L SAVAGE

Royal Hospital for Sick Children, Bristol

SUMMARY Mean values for two variables of the pubertal growth spurt, peak height velocity and age at peak velocity, of children attending the diabetic clinic in Bristol are reported. The growth spurt was normal both in timing and intensity in boys, but the peak velocity was reduced and age at peak velocity more variable among girls.

There have been few studies of pubertal growth in diabetic children, and those that have been published show conflicting results. Despite this reports that pubertal growth is abnormal are still found. Stimulated by this observation we carried out a study of growth during puberty of children attending the diabetic clinic at our hospital.

Methods

At the end of 1974 a children's diabetic clinic was established in Bristol under the care of a paediatric endocrinologist. Although two of the general paediatricians working in Bristol continue to care for diabetics who are specifically referred to them or who present to them on the emergency rota, most children with diabetes in Bristol are seen at the diabetic clinic.

All children attending the clinic had their height measured by trained nursing staff using a Harpenden Stadiometer. Measurements were taken at each visit; rate of attendancy varied from one to six months according to clinical requirements. At the beginning of 1981 the records of all children attending the clinic who could have experienced a pubertal growth spurt within the previous six years were examined. Girls born between the beginning of 1960 and the end of 1971 and boys born between the beginning of 1958 and the end of 1969 were selected for study. The wide limits were used to prevent the omission of children with unusually early or late growth spurts.

A total of 168 records were examined. Yearly growth velocities were calculated using all available measurements of height that had a corresponding measurement within 0-8 and 1-2 years. Velocities were plotted on Tanner and Whitehouse charts against the midpoint of the year in which the velocity
had been recorded. Pubertal growth spurts were identified in 49 children (25 girls and 24 boys). The remaining children were judged not to have reached their pubertal growth spurt by 1981 or to have completed it before 1975 or at the time of diagnosis of the disease. For most children the indentification of a pubertal growth spurt was straightforward, but some children appeared to have experienced two growth spurts, and in others a peak was not clearly defined and could only be identified from a series of measurements. Children were therefore excluded from the study if there was only one measurement before or after an apparent peak as it was impossible to be certain whether the observed peak velocity was the true one.

Each child’s growth spurt was characterised by two variables: the peak height velocity and the age at which the child reached peak velocity. The peak height velocity was taken as the maximum observed annual velocity and the age at the midpoint of the year that it occurred. When two peaks were observed the peak height velocity was taken as the maximum velocity in the higher of the two peaks.

Mean values for peak height velocity and age at this peak in these diabetic children were compared with those of the normal children from the Harpenden growth study by Student’s t test. The F test was used to test the significance of differences between variances.

Results

The Table summarises the results of this study. The peak height velocity and the age at peak velocity among diabetic boys was the same as that among control children. In girls the average age at peak velocity did not differ between the two groups, though the variance was significantly higher among diabetics. The peak velocity among diabetic girls was 0.5 cm/year lower than that in controls.

<table>
<thead>
<tr>
<th></th>
<th>Boys</th>
<th>Girls</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Diabetics</td>
<td>Controls</td>
</tr>
<tr>
<td>No of children</td>
<td>24</td>
<td>49</td>
</tr>
<tr>
<td>Mean (SD) peak velocity</td>
<td>6.8 (1.5)</td>
<td>9.0 (1.1)</td>
</tr>
<tr>
<td>Mean (SD) age at peak</td>
<td>14.1 (0.9)</td>
<td>14.1 (1.1)</td>
</tr>
</tbody>
</table>

* P<0.05 between diabetics and controls. ** P<0.01 between diabetics and controls.

Discussion

An earlier study of growth among children attending our clinic showed that diabetic boys were tall relative to their peers at the time of diagnosis of their disease and that prepubertal growth was poor in both sexes. This suggests that diabetes can interfere with growth even if the disease is adequately controlled.

This study provides evidence that diabetes does not interfere with pubertal growth in boys as the growth spur of the study group was normal both in timing and intensity. In contrast, the growth spurt among this sample of diabetic girls was not normal; the peak velocity was lower and the age at which it occurred more variable.

Several reasons may explain why results from this study differ from those of earlier reports. Earlier studies were based on smaller samples of children, and the methods of analyses varied, therefore, the differences could be due to methodology.

Alternatively, the effects of diabetes on pubertal growth may have changed over time with improved treatment. Growth in diabetes is widely thought to be related to control of disease; control, though difficult to measure, has probably improved over the last two decades. If we postulate that poor control is the cause of our findings we would have to suggest that during puberty disease control was worse among girls than boys. This does not accord with clinical impression, but such impressions could be misleading.

A third possible explanation for our findings is that some aspect of diabetes that is not necessarily related to blood sugar control interferes with pubertal growth among girls but not among boys.

References

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Correspondence to Dr S L Stewart-Brown, Royal Hospital for Sick Children, Bristol BS2 8BJ.

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