Archives of Disease in Childhood, 1986, **61**, 464–471

Growth in thyrotoxicosis

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SUMMARY To study the effect of thyrotoxicosis on growth, 46 children and adolescents presenting with thyrotoxicosis have been reviewed and followed up for several years, in most cases until adult height was achieved. At presentation the children were underweight for height but were above expected heights even if already well advanced in puberty. Despite bone ages often being very advanced at diagnosis, the condition had no adverse effect on ultimate stature, which was commonly greater than that expected on the basis of parental height. Weight was rapidly regained on treatment, but nevertheless the younger subjects continued to be underweight despite adequate prolonged control of thyrotoxicosis.

For many years it has been well recognised that thyroid hormones have a great influence on growth and bone maturation. Most of the evidence about this has been accrued, however, from observations on hypothyroidism.[1] Overtreatment with thyroid hormones in hypothyroidism results in an excessive acceleration of bone maturation, and thyrotoxicosis in infants has been reported to cause premature closure of skull sutures.[2 3] There are, however, few reports of the effect of hyperthyroidism on final height.[4–6] The reason for this lack of information is that thyrotoxicosis is a rare condition in childhood and most cases develop shortly before or during puberty, when the normal acceleration of growth will be superimposed on any effects of the disease and will make interpretation difficult. A recent monograph on hyperthyroidism gives no special consideration to the condition in childhood or adolescence.[7]

The present study has been undertaken to provide a large series of cases of thyrotoxicosis in children and adolescents and, by following them up over a prolonged period, to document the long term effect of this condition on growth. This collaborative study has been undertaken in two widely separated centres, Leeds and Leipzig, reviewing cases presenting since 1968.

Patients and presentation

Forty six children and adolescents from Leeds and Leipzig who developed thyrotoxicosis after infancy have been studied, and details of their ages and clinical presentation are shown in Tables 1–3. The condition is much commoner in girls than in boys, and only four of these children were boys. The mean age of presentation was about 11 years but with a wide range of 2·6–14·1 years. Twenty three of the cases already showed some signs of puberty, and the stages reached according to the standard description of Tanner[8] are indicated in Table 1.

In children the condition is most common around the age of puberty, and though 20 children were prepubertal at the time of presentation, of whom 16 were aged less than 10 years, in 10 of the girls and one boy the onset of puberty was in the year after the development of the first signs of thyrotoxicosis. In case 3, who had longstanding thyrotoxicosis, there was severe deterioration in the control with medical treatment at the time that puberty occurred. Eleven of the girls had already reached menarche at the time of starting treatment. Five of these developed amenorrhoea at the onset of thyrotoxicosis, but this subsequently responded to antithyroid treatment. Two others became amenorrhoeic during the course of their illness at a time of relapse associated with attempted withdrawal of treatment.

The age at which puberty occurred did not seem to be influenced by either thyrotoxicosis or its treatment, in that the age distribution was similar to normal standards. In those 22 girls in whom the age of occurrence of first signs of puberty was known this averaged 11·3 (SD 0·9) years, and in the 30 whose age of menarche was known, occurring before or while under observation, this averaged 12·9 (SD 1·01) years.

The duration of symptoms before treatment averaged seven months, with a range of 0·1–2·3

years, but the history is often subjective and unreliable as a true indication of the duration of disease. Diagnosis was based on symptoms, clinical signs (Table 3), and biochemical values of thyroid hormones. In the early stages the biochemical estimates were of protein bound iodine and butanol extractable iodine but latterly by radioimmunoassay estimates of free and total thyroxine, triiodothyronine, and thyroid stimulating hormone. Height, weight, and skeletal age were measured just before starting treatment and subsequently at regular, usually three monthly, intervals.

Measurements and methods

Growth standards used in this study were normal British values.[8–10]

Heights. These were measured accurately by Tanner's method.[11] The data are presented in terms of standard deviation score* for height, derived from the standards of Tanner. This gives an indication of the stature of the subject relative to the average population of that age; a value of 0 being average, and positive and negative values representing, respectively, the degrees of tallness or shortness.

Weights. These are also represented as standard deviation scores, derived similarly.

Skeletal age. This has been estimated by the TW2 (20 bones) method of Tanner and Whitehouse[12] by the same two observers, with comparable results. The values have been expressed as the bone development quotient (skeletal age divided by chronological age), giving an indication of the degree of advancement or retardation of skeletal development.

Treatment

All but two subjects were adequately controlled by medical treatment as judged by biochemical and clinical criteria. Two girls (cases 3 and 37) ultimately required thyroidectomy as, after several years of control with antithyroid drugs, florid thyrotoxic symptoms developed at puberty despite increased dosage. There has been considerable variation in the duration of treatment and follow up (Table 1).

It has been customary to continue treatment initially for at least 18 months before attempting withdrawal or progressive reduction of antithyroid drugs. Table 1 shows the outcome of these attempts undertaken in 37 of the 46 subjects. Remission occurred on complete stoppage of treatment in 15 and was maintained during the periods of subsequent observation, which varied from six months to two years or more. In some of these, however, the ultimate outcome is not known, for others of our subjects have shown that relapse can occur after months or even years of apparent remission. In five additional subjects it has also been possible to stop treatment, but so far for periods of less than six months.

*Standard deviation score$=\frac{\bar{X}-X}{Sx}$ where $\bar{X}$=child's height; X=mean height at that age; Sx=standard deviation at that age.[10]

Results

At time of presentation.

Heights

Anthropometric and skeletal age data for all 46 patients at the time of starting treatment are included in Table 1. With the exception of three children with congenital conditions involving short stature (which are excluded from the overall analysis), most of the children had heights above average, and some were very tall. Pooling the combined values for all 40 normal girls showed a small positive standard deviation score for height—that is, +0·75 (SD 1·19). This total, however, included several girls who were already almost fully grown, yet separate analysis of those girls alone whose ages were less than 12·5 years (cases 1–24) at time of starting treatment showed a similar positive standard deviation score of +0·84 (SD 1·31).

Bone development

Skeletal age is usually advanced more than height age, but to a variable degree. The youngest child at presentation (case 1) was aged 2·6 years with a bone age greatly advanced at 7·8 years. This exceptional degree of advancement had a pronounced effect on the overall composite values.

Weights

These, also expressed as standard deviation scores at presentation, are included in Figure 1. The Figure indicates the relation of height to weight standard deviation scores at the time of diagnosis and subsequently. The children have been grouped according to their pubertal state. These children were markedly underweight at the time of diagnosis and start of treatment (mean standard deviation score for weight −0·32 (SD 1·05)), and only three had a higher standard deviation score for weight than height.

During treatment. The changes in standard devia-

Table 1 *Data on 46 thyrotoxic patients at start of treatment and subsequently*

At time of starting treatment							*Follow up*						
Case No	*Age (yrs)*	*Pubertal rating**	*Standard deviation score height*	*Bone age (yrs)*	*Duration of symptoms (yrs)*	*Age of first sign of puberty (yrs)*	*Age of menarche (yrs)*	*Age when last seen (yrs)*	*Adult height (cm)*	*Standard deviation score height when last seen*	*Target height† (cm)*	*Remission*	*Comments*
									Girls				
1‡	2·6	I	+2·81	7·8	0·7	9·0	10·4	13·4	160·2	+0·27	160·9	No relapse after 2 years	Developed rickets at 3·4 years of age
2	3·4	I	−0·18	4·1	0·4	11·7	not yet	11·8	—	+0·27	162·0	No relapse after 3·5 years	
3‡	4·3	I	+1·27	7·4	0·3	11·0	13·9	14·2	165·8	+0·85	167·8	Uncontrolled, thyroidectomy at 12·9 years	
4‡	4·6	I	+1·07	8·6	0·5	not yet	not yet	6·3	—	+1·06	165·0	Not attempted	
5‡	5·9	I	+1·27	8·4	0·7	12·3	14·1	17·2	168·6	+1·07	163·7	No relapse after 1 year at 3rd attempt	
6	7·3	I	+1·94	—	0·7	not yet	not yet	11·7	—	+1·75	164·3	Relapsed after 6 months remission	
7	7·6	I	+0·04	8·9	0·3	9·7	11·4	12·9	>156	0·0	155·0	No relapse after 1 year, 2nd attempt	
8‡	8·2	I	−0·12	7·2	0·5	10·4	12·5	13·2	>156	−0·24	159·7	Not attempted	
9	8·4	I	+1·30	8·1	0·3	10·6	13·0	17·1	169·0	+1·13	159·3	Relapsed after 5 years remission	Thyrotoxic crisis at presentation
10	9·0	I	+0·91	10·3	0·9	11·5	12·6	13·9	>160·1	0·0	160·8	No relapse after 1·5 years	
11‡	9·2	I	+0·28	10·7	0·5	11·7	not yet	12·2	>155	+0·67	147·0	Relapsed after 4 months remission	
12	9·3	I	+2·23	10·3	0·9	11·6	13·0	15·7	175·6	+2·26	167·8	[No relapse after 3 months]	
13	10·6	I	+0·43	12·4	1·0	10·8	not yet	12·5	>154	0·0	158·8	On reducing dose at present	
14	10·7	II	+2·25	11·2	0·8	10·6	13·7	13·9	172	+1·97	157·8	Not attempted	
15	10·7	I	−0·83	11·4	1·0	12·2	not yet	13·1	—	−1·25	156·8	On reducing dose at present	
16	11·0	II	−1·46	10·1	0·5	<10·9	13·2	17·7	156·2	−1·00	149·8	No relapse after 1 year, 3rd attempt	
17‡	11·1§	IV	+1·03	12·7	0·2	—	11·4	16·3	160·9	0·0	160·1	No relapse after 6 months, 2nd attempt, 1·5 years on previous attempt	
18	11·2	I	+1·97	12·3	0·5	11·8	13·2	15·4	171·1	+1·49	164·8	No relapse after 2 years	
19	11·3	II	+2·43	12·4	0·3	<11·3	13·0	15·7	178·0	+2·64	170·8	No relapse after 1 year	
20	11·7	III	−0·04	12·4	0·4	<11·3	13·3	15·1	>161·5	−0·07	156·3	No relapse after 1 year but nodule excised	
21	11·8	I	+2·67	—	0·6	12·3	13·2	14·4	172·8	+2·07	adopted	Small dose still required	
22	12·0	I	−1·2	12·2	0·7	12·5	13·9	14·6	154·2	−1·19	153·8	[No relapse after 3 months]	
23	12·1	I	+1·09	13·2	0·5	12·3	13·8	16·3	172·9	+1·78	171·0	Small dose still required	
24	12·3	III	−0·94	11·7	2·3	<12·0	14·0	15·1	163	+0·18	162·8	Not attempted	
25	12·8	V§	+2·0	13·8	0·5	—	12·2	16·4	174·1	+1·98	176·8	No relapse after 1 year	
26	12·8	III	+0·13	13·4	0·2	—	14·0	16·2	159	−0·53	161·3	Relapsed on reducing dose	
27‡	13·2	V§	+0·57	adult	1·1	—	12·0	15·5	162·3	0·0	165·2	[No relapse after 2 weeks only]	
28	13·2	IV§	−0·80	—	0·7	—	12·3	13·9	153·9	−1·02	—	Not attempted	
29	13·3	V§	+2·14	adult	0·6	—	11·5	15·2	171·7	+1·62	167·8	No relapse after 2 years	
30	13·3	III	+0·81	—	0·1	—	14·4	15·2	>173·0	+1·84	163·8	Not attempted	Thyrotoxic crisis at presentation
31	13·3	III	+1·29	13·6	0·3	—	15·0	15·3	170·0	+1·33	161·8	Small dose still required	
32‡	13·4	V§	+1·93	14·3	0·4	—	12·5	15·7	173·2	+1·78	174·0	Relapsed after 0·7 years remission	
33	13·6	V§	+0·96	adult	0·2	—	11·5	16·6	166·8	+0·77	166·8	Small dose still required	
34	13·7	III	+1·10	—	0·3	—	13·9	16·6	170·6	+1·4	166·8	Not attempted	
35	13·8	IV§	−1·29	14·3	0·7	—	<13·0	16·7	153·5	−1·45	—	Small dose still required	
36	13·8	V§	+0·11	14·7	0·8	—	<13·0	16·3	161·5	−0·12	157·9	No relapse after 8 months	
37‡	13·8	IV	+0·42	14·0	0·3	—	14·1	21·0	165·4	+0·54	166·0	Uncontrolled. Thyroidectomy at 18·3 years. Medical treatment still required until 20·5 years [No relapse over next 0·5 years]	

38	13·9	V§	0·0	adult	0·2	—	12·5	14·8	>161·2	0·0	159·8	Small dose still required	
39‡	14·1	V§	−0·30	14·6	0·3	—	13·5	17·6	164·0	+0·30	162·8	[No relapse after 4 months]	
40	14·1	V§	+0·75	15·4	0·4	—	<13·7	18·4	166·2	+0·67	161·8	[No relapse after 3 months] Previous unsuccessful attempts	
										Boys			
41	12·0	I	+0·38	13·1	0·7	13·0		14·7	—	0·0	174·3	[No relapse after 3 months]	
42	13·8	II	−0·42	14·7	0·4	13·5		16·1	>172·6	0·0	180·0	Not attempted	
Patients with thyrotoxicosis and additional conditions													
										Girls			
43‡	9·0	I	−2·07	9·6	1·2	not yet	not yet	13·4	—	−2·92	155·0	Unsuccessful	Turner's syndrome. Cardiac failure at presentation
44	13·2	I	−3·49	12·8	0·1	13·9	15·2	16·6	145·1	−2·85	164·8	No relapse after 2 years	Intrauterine growth retardation. Thyrotoxic crisis at presentation
										Boys			
45‡	10·5	I	−1·30	10·1	0·6	not yet		12·6	—	−1·38	176·0	Not attempted	Diabetes for 6 months, poor diabetic control at presentation
46‡	12·2	I	−2·14	13·7	0·5	13·4		17·3	155	−2·64	174·0	No relapse after 1 year, 2nd attempt	Down's syndrome

*According to description of Tanner.[8]
†Based on mid-parental centile.
‡Leeds patients.
§Already post-menarchal.
[ ] Indicates too short a duration to be confident of maintained remission.

Table 2 *Age of the 46 thyrotoxic patients at start of treatment*

		Years
		Mean (SD)
Total	46	10·94 (3·05)
Total Leipzig	32	11·59 (2·40)
Total Leeds	14	9·44 (3·87)
Girls:		
Total	42	10·82 (3·15)
Prepubertal	20	8·52 (3·07)
Pubertal	11	12·28 (1·17)
Post-menarchal	11	13·55 (0·41)
Boys	4	12·15 (1·34)

Table 3 *Prevalence of presenting clinical features*

	No (%) of patients
Goitre	42 (91)
Eye signs, lid retraction, or exophthalmos	35 (76)
Palpitations and disproportionate tachycardia	23 (50)
Nervousness	22 (48)
Weight loss	22 (48)
Amenorrhoea	5/11* (45)
Heat intolerance and sweating	17 (37)
Irritability and hyperkinesis	17 (37)
Tremor	9 (20)
Diarrhoea	8 (17)
Increased appetite	6 (13)
Weakness and tiredness	5 (11)
Insomnia	4 (9)
Thyrotoxic crisis	3 (7)
Loss of hair	3 (7)
Total patients	46 (100)

*Prevalence in 11 patients who reached the menarche before presenting with thyrotoxicosis.

tion score and bone development quotient through the first three years after diagnosis are shown in Figures 1 and 2 for the 40 girls who, apart from thyrotoxicosis, would be considered normal.

The normal process of puberty greatly influences the pattern of growth in all children, with a pronounced spurt preceding the slowing down and stopping of growth. These changes confuse any interpretation of the effects of thyrotoxicosis and its treatment in these children at that stage of development. Even the use of standard deviation scores is not reliable, being compared with children in whom pubertal changes are happening at the average ages. As pubertal changes in a high proportion of healthy normal children occur earlier or later than average their standard deviation scores would be expected to deviate correspondingly from the average. In an attempt to clarify this complicated situation, the girls have been divided in analysis into three groups as follows:

(1) those who were completely prepubertal at the time of starting treatment (and in whom the pubertal process would not, at least initially, be influencing growth);

(2) those already in early puberty (in whom this growth aspect would be considerable);
(3) those already sufficiently well advanced in puberty to be past the menarche (at which stage growth in stature would be complete or nearly so).

Insufficient numbers from groups 2 and 3 were followed up long enough to provide three year data for these groups individually.

Heights

The positive standard deviation score for height at

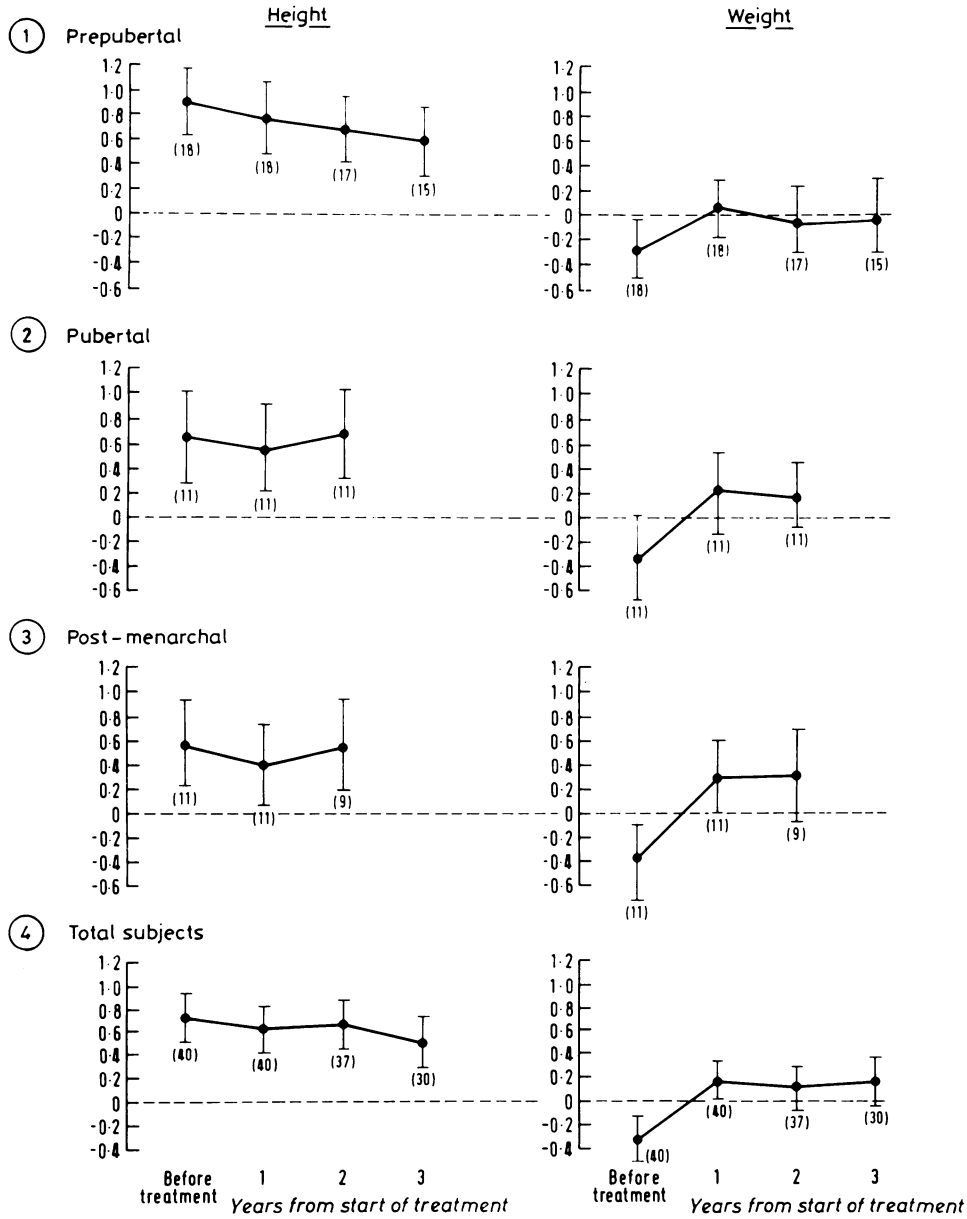

Fig. 1 *Growth measurements (standard deviation scores) of height and weight of 40 otherwise normal thyrotoxic girls before treatment and throughout ensuing years. Development state at presentation: (1) Prepubertal; (2) In puberty; (3) Adolescents past the age of menarche; (4) Total group. (Number of subjects in each group in brackets.)*

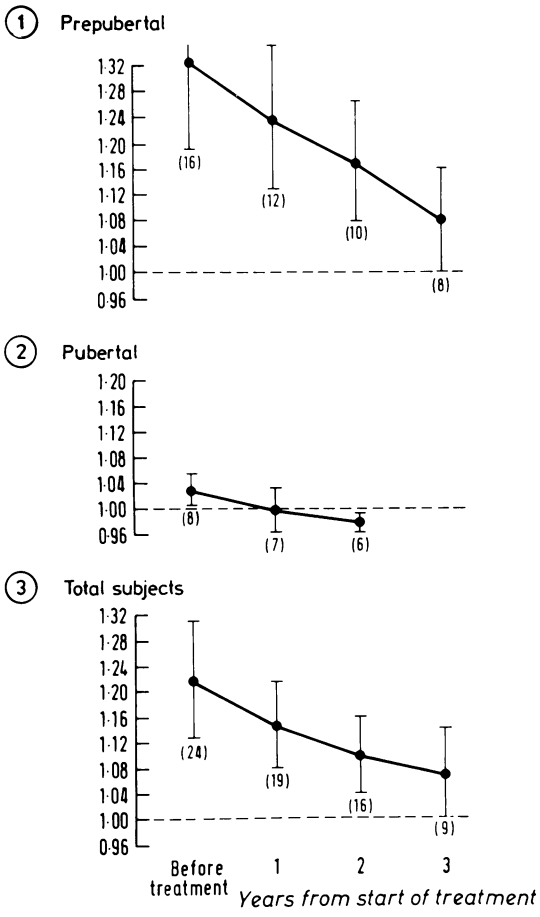

Fig. 2 *Bone development quotient (see text) for 24 otherwise normal thyrotoxic girls before treatment and throughout ensuing years. Development state at presentation: (1) Prepubertal; (2) In puberty; (3) Total group. (Number of subjects in each group in brackets.)*

presentation is seen in all groups but most markedly in the younger children. Over the course of the ensuing years in which the children were maintained clinically and biochemically euthyroid for the vast majority of time, the standard deviation score for height remained positive, showing a slight reduction with time, which was most evident in the younger children.

Bone development

It is not easy to compare precisely skeletal age with height quantitatively, but in terms of height age this was less advanced than skeletal age in most children at presentation. In the adolescents of group 3, who were well advanced physically at the onset of thyrotoxicosis, skeletal age was already adult or nearly so. Because (as with height) appreciable changes will not be possible over the ensuing years, skeletal age data are not included for this group. The very pronounced skeletal age advancement in the younger children (group 1), as indicated by bone development quotient, does not persist over ensuing years and there is a progressive decline towards a value of 1—that is, that state when skeletal age and chronological age are the same. This occurs without a comparable fall off in the height standard deviation score. (The bone development quotient picture is exaggerated because of case 1, but even without this girl the overall trend is unchanged.) The advancement in skeletal age in relation to chronological age as indicated by bone development quotient was proportionately much less in those older children, already in puberty, whose bone development was that much more advanced anyway.

Weight

In most patients on treatment weight increased markedly, and after one year of treatment all but seven had an increased standard deviation score for weight (Fig. 1). In most children this improvement in weight state contrasted with a fall in the standard deviation score for height, and in all but four children there was an overall weight gain compared with height in terms of the standard deviation score. It would be expected that weight, which changes so much more acutely than height, should be regained rapidly. In ensuing years after the first there was no overall further gain in standard deviation score weight, and perhaps surprisingly in two thirds of the children this never fully caught up standard deviation score height, so that these subjects overall remained tall and thin.

Ultimate stature

It has been possible to follow most of these children to their full adult stature. Of the 40 girls who were normal, apart from having thyrotoxicosis, 34 who were ultimately post-menarchal were seen until growth was complete or almost so. Their mean (SD) height then was 165·3 (6·9) cm, which corresponds to a standard deviation score of +0·54 for adult women. Parental heights were known in most subjects and when available were used as a basis for a 'target height' (based on the mid-parental centile values). The mean target height was 162·4 cm, which is very close to the overall 50th centile (standard deviation score=0) height for (British) women (162·0 cm). In 31 of the girls who could be effectively considered fully grown, measurements of parents' heights were available to give a target.

Twenty two out of 31 (71%) achieved heights above target, and three of the remaining nine were within 1 cm of it.

Subdividing the groups according to pubertal state at presentation identifies those in whom there was time for growth to be appreciably affected by the thyrotoxicosis, but such subdivision results in too small numbers in the subgroups for dogmatic conclusions to be drawn. Nevertheless, in all subgroups ultimate stature is above average:

Group 1 (those prepubertal at presentation). Of the 12 who were followed up until completion or near completion of growth, ultimate stature averaged 165·2 (SD 7·5) cm, which is equivalent to a standard deviation score of +0·5.

Group 2 (those in early puberty initially). Of the 11 in this group ultimate stature was 166·3 (SD 6·8) cm, equivalent to a standard deviation score of +0·67.

Group 3 (those already post-menarchal). Curiously, the 11 in this group also ended up above average in height, though to a slightly lesser degree, their stature being 164·4 (SD 6·9) cm, equivalent to a standard deviation score of +0·35.

Discussion

Our observations agree with most reports of childhood thyrotoxicosis in showing increased height velocity as a result of the condition.[4 13–15] Body measurements and bone ages at the time of starting treatment, however, reflect the duration of the disease, which cannot be reliably evaluated on the basis of history. The effects of thyrotoxicosis on height as indicated by standard deviation score and skeletal age as indicated by its relation to chronological age (bone development quotient) are likely to be much greater the younger the patient, and these values for younger children may contribute excessively to the overall means. Skeletal ages in those studies in which they have been reported were nearly always advanced, whatever the age of presentation, and often to a greater degree than the height ages.[3 4 6 16]

Most children with thyrotoxicosis present in or shortly before puberty, although the age of puberty itself does not seem to be affected by the disorder. In subsequent years growth rate is much influenced by the pubertal process with acceleration of height in the earlier stages followed by a slowing down and ultimate stopping of growth. Interpretation of changes in growth velocity in relation to a disease process in these ages is therefore difficult. Like height, bone maturation shows a dramatic acceleration at the appropriate stage of puberty,[17] and the magnitude and chronological timing of this will vary greatly within the normal population. Despite these difficulties certain inferences may reasonably be drawn from this study.

At the time of presentation most thyrotoxic children are relatively tall, though skeletal age is often advanced to an even greater extent than height. Unlike other conditions, however, in which there is premature acceleration of skeletal age with growth—for example, exposure to sex hormones—this study shows that thyrotoxicosis does not have an adverse effect on ultimate stature; the converse is in fact the case as about 70% ended up with heights above that anticipated on the basis of parental stature. This observation agrees with most of the published reports in showing that there is no detrimental effect on subsequent growth and ultimate stature in treated thyrotoxicosis.[4–6]

The tallness of thyrotoxic children, both at diagnosis and ultimately, is not restricted to those presenting at a young age who have many years of growth potential remaining. It is also apparent in those who present already in puberty and even at an age when growth is almost complete. This suggests either that there is a predisposition to thyrotoxicosis in tall individuals, or that a subclinical process has been affecting growth for some time before it is clinically obvious. The implication also is that the effects of exposure to excessive thyroxine have some permanent effect in producing increased stature.

Weight is markedly reduced at diagnosis in contrast to the increased height. Not unexpectedly this weight loss is rapidly improved within the first year of treatment. Yet overall, even when subjects are euthyroid over many years of follow up, the weight standard deviation score continues at a lower level than height, and subjects mostly remain thin (though to lesser degree in those diagnosed at a younger age). This again implies some permanent effect of past thyrotoxicosis on body composition. Those subjects who are tall and thin at the initial presentation of hyperthyroidism mostly remain tall and thin subsequently.

It is not clear to what extent the growth patterns described are dependent on good treatment of thyrotoxicosis or the duration of the disease before treatment is begun. It is of interest that von Harnack *et al*[1] showed that the final adult height of children treated long term for hypothyroidism averaged the 70th centile. This occurred even despite an early advancement in bone age in many early treated cases and presents a similar picture to that of thyrotoxicosis in childhood. All but one of our subjects were maintained strictly euthyroid throughout except for trial periods to assess the need for

continuing treatment, but even in our one subject (case 3) whose control was very inadequate in the later years there was little adverse effect on ultimate stature. Whether maintenance of these children in a euthyroid state by medical treatment (on clinical and biochemical grounds) renders them truly normal is open to question. Many centres in other parts of the world advocate early surgical treatment, and it would be of interest to compare the influence on growth of this other form of treatment.

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Received 5 February 1986