Eur Spine J (1993) 2:29–36

European Spine Journal

Adolescent idiopathic scoliosis: is rising growth rate the triggering factor in progression?

C. J. Goldberg[1], F. E. Dowling[2], and E. E. Fogarty[2]

[1]Children's Research Centre, Our Lady's Hospital for Sick Children, Dublin, Ireland
[2]Department of Orthopaedics, Our Lady's Hospital for Sick Children, Dublin, Ireland

Scoliose idiopathique de l'adolescent: est-ce que la poussée de croissance représente le facteur déterminant de l'évolutivité?

Résumé. Le programme de dépistage de la scoliose à l'âge scolaire pratiqué au "Our Lady's Hospital for Sick Children" de Dublin a fourni le matériel nécessaire à une étude prospective continue de l'histoire naturelle des scolioses idiopathiques de l'adolescent. L'analyse de l'évolution clinique observée chez 339 filles a montré que les progressions d'au moins 10°, survenues chez 46 d'entre elles (13,6%), dépendaient du moment du diagnostic et étaient bien plus liées à la situation de l'enfant sur sa courbe de croissance et à son état pubertaire qu'à la maturité squelettique donnée par le degré d'ossification de la crête iliaque ou l'âge osseux. Cette notion est riche d'implications pour la compréhension de l'histoire naturelle, pour l'établissement des programmes de dépistage et pour l'interprétation des résultats du traitement conservateur.

Mots-clés: Scoliose idiopathique de l'adolescent – Pronostic – Évolutivité – Croissance – Puberté

Summary. The school scoliosis screening programme at Our Lady's Hospital for Sick Children, Dublin, has provided material for an ongoing prospective natural history study of adolescent idiopathic scoliosis. An examination of the clinical course in 339 girls showed that observation of progression of at least 10°, which occurred in 46 girls (13.6%), depended on the timing of diagnosis and related primarily to the child's position on her growth rate curve and her pubertal status, and much less to skeletal maturity as interpreted by iliac crest ossification or bone age. This has implications for the understanding of results in conservative management, screening programmes and natural history.

Correspondence to: Dr. C. J. Goldberg, Children's Research Centre, Our Lady's Hospital for Sick Children, Crumlin, Dublin 12, Ireland

Key words: Adolescent idiopathic scoliosis – Prognosis – Progression – Growth – Puberty

Recent studies at the Children's Hospital, Boston, and Our Lady's Hospital for Sick Children, Dublin [14], have suggested that reports of the outcome of conservative treatment regimes are clouded by the inclusion in the results of large numbers of subjects whose scoliosis may not have been progressive in the first place. While it has been taught that adolescent idiopathic scoliosis may progress until spinal maturity, which can be dated to complete excursion of the iliac crest apophyses, comparison of two matched groups treated and untreated and all with a zero Risser sign, showed no significant difference in outcome [14]. The conclusion was that maturity markers currently in use are too generous and thus the results of orthoses in genuinely progressive scoliosis have been swamped by the large numbers of "good" results in non-progressive scoliosis.

This study was designed as an extension of the previous work, to examine and perhaps refine criteria for progression potential. As a starting point, all previous beliefs were suspended and the null hypothesis, that no form of conservative management has any effect on natural history, was adopted. The subjects were drawn from the school screening data base at Our Lady's Hospital for Sick Children, Dublin, and results are current at December 1992. The aim was to be able to assess treatment more critically and prescribe more appropriately, as well as to contribute to knowledge of natural history and, perhaps, aetiology.

Literature review

Early authors, e.g. Risser and Ferguson [23] in 1938 and Cobb [6] in 1948, have drawn attention to the importance of growth in the natural history of adolescent idiopathic scoliosis. In 1958, Risser [22] described what

has come to be known as the Risser sign as a useful maturity marker, commenting that the risk of progression was considerably reduced at the first appearance of the iliac crest apophysis. James [18] in 1954 and Biondi et al. [2] in 1985 have also drawn attention to be importance of the iliac crest apophyses, considering the risk period to continue until complete excursion, now known as Risser 4.

This is now standard. Thus, criteria for conservative management, e.g. Carr et al. [5] in 1980, Emans et al. [7] in 1986 and Kehl and Morrissey [19] in 1988 on bracing, or Brown, Axelgaard et al. [3] in 1984 and Swank et al. [26] 1989 for spinal stimulation, have dubbed anything less than Risser 3 or 4 "progressive" and so suitable for treatment. Natural history studies, e.g. those by Lonstein and Carlson in 1984 [20] and Bunnell in 1986 [4], have acknowledged the importance of growth potential and menarchal status, but have left pride of place to the Risser sign. Scoles et al. [25] stated in 1988 that the iliac crest appeared at a mean of 8 months after menarche in girls, and writers on endocrinology, e.g. Root [24] in 1973, state that menarche occurs several months after peak growth velocity, when the growth rate is slowing considerably and thus potential for growth is diminishing apace. Thus, "Risser 1" may be almost 12 months after peak growth velocity. The observation that scoliosis does not usually progress after menarche has been made, e.g. by Terver et al. [27] in 1980, suggesting that the limits for anticipated progression should be brought forward. Previously, however, Urbaniak et al. [28] in 1976 found no correlation between progression and menarche, and only some with Risser sign.

Another indication for conservative treatment, as observed by Kehl and Morrissey [19] has been documented progression. A problem with this is that since adolescent idiopathic scoliosis eventually arrests spontaneously, any observed control of curve by treatment may be coincidental, not the result of intervention. As has been stated [4, 11, 28], evidence of past progression is not a guarantee of future progression.

Materials and methods

The subjects for this study are all girls detected on school scoliosis screening between 1979 and 1990 attending Our Lady's Hospital for Sick Children, Dublin. They form part of an on-going prospective study of the natural history of adolescent idiopathic scoliosis. The screening programme began in suburban areas of South Dublin [10], gradually expanding until by 1988 it included all post-primary schools from the River Liffey to the South Wexford coast. The design of this screening programme has been described previously [11, 12]. Computerised records have been maintained on all girls screened and all repeat screening examinations. Investigations and clinic attendance have been on the basis of the perceived clinical need of the individual girl.

For inclusion in this study, girls had to have been between the ages of 8 and 15.5 years at diagnosis, have radiographic evidence of more than 5° scoliosis, although not necessarily at diagnosis, and to have attended the clinic on at least two occasions. All radiographs were taken with the patient standing. A minimum time for follow-up was not specified, since this can exclude those who progress rapidly to surgery as well as those whose follow-up is not yet complete. Girls were excluded if menarchal status was not recorded. The null hypothesis regarding non-surgical intervention was observed and patients treated by conservative means were not excluded. The evidence that any method is beneficial has been found unconvincing at this centre [13, 14], and it was felt that a clear advantage, if it existed, should become obvious as the study proceeded.

The natural history was considered to have been interrupted at operation in those requiring surgery. Progression was considered to be a sustained increase in Cobb angle of at least 10°. Statistical analysis was by standard two-tailed t-test for independent means, Z-test for proportions, χ and λ. The sensitivity, specificity and predictive value of prognostic indicators (iliac crest ossification and menarchal status) were calculated. Values are given to one or two decimal places in this paper, but four decimal places were used in the calculations.

Results

Programme size

Between June 1979 and June 1990 97000 examinations were carried out on 58000 girls. The rate of referral to hospital declined steadily throughout the period in question and by the late 1980s was less than 1%.

There were 599 girls with measurable scoliosis identified on the screening programme. Their mean Cobb

Table 1. Demographic data and Cobb angle of excluded scoliotic subjects

Age < 8 years at diagnosis	$n =$ 7
Age > 15.5 years at diagnosis	$n =$ 13
Lost or discharged on first visit	$n =$ 94
No record of menarchal status	$n =$ *146*
Total	$n =$ 260
Mean Cobb angle: 10.38° (SD 5.2)	
Mean age: 13.28 years (SD 1.44)	

Table 2. Demographic data and Cobb angle in the study group (n = 339)

	Mean	SD
Age at diagnosis (years)	12.8 (range 8.4–15.4)	1.27
Follow-up (years)	2.4 (range 0.2– 7.8)	1.66
Cobb angle (°)	17.37 (range 1 –67)	9.4258
Number progressing >10°:	46 (13.5%)	

Table 3. Cobb angle at diagnosis: progressive vs stable scoliosis groups

	Progressive scoliosis (n = 46)		Stable scoliosis (n = 293)	
	Mean	SD	Mean	SD
Age at diagnosis (years)	12.11	1.1	12.92	1.3
Cobb angle at diagnosis (°)	22.5	13.0	16.5	8.5

Two-tailed t-test to compare Cobb angles: $t = 3.0204$; $P < 0.01$

Table 4. Relationship between Cobb angle at diagnosis and subsequent progression

	Cobb angle						
	<10°	10°–19°	20°–29°	30°–39°	40°–49°	50°–59°	≥60°
Age at diagnosis (years ± SD)	12.4 ± 1.2	12.7 ± 1.3	13.1 ± 1.3	13.2 ± 1.1	13.7 ± 1.1	12.7 ± 1.1	13.3
Progression	6	15	14	6	2	2	1
Stable scoliosis	40	178	49	18	5	3	–
Total	46	193	63	24	7	5	1

$\chi^2 = 22.92847$; $P < 0.01$; $\lambda = 0.00524$ (not significant)

Table 5. Association between outcome and management in patients with an initial Cobb angle of 20° or more ($n = 100$)

	Treatment			Total
	None	Brace	LESS[a]	
Progressive scoliosis	10	5	9	24
Stable scoliosis	54	13	9	76
Total	64	18	18	100

$\chi^2 = 10.78906$; $P < 0.01$; $\lambda = 0$

[a] LESS, Electrospinal stimulation

Table 6. Relationship between progression and presence or absence of ossification of the iliac crest: all subjects[a]

	Progressive scoliosis	Stable scoliosis	Age at diagnosis (years)	
			Mean	SD
Risser sign 0	41	200	12.4	1.2
Risser sign >0	5	88	13.7	0.87
Total	46	289		

$\chi^2 = 7.6509$; $P < 0.01$; $\lambda = 0$

[a] Risser sign was not recorded in 5 girls, 3 of whom had scoliosis 20° or more and none of whom had progression. With iliac crest ossification as the prognostic test: sensitivity: 89%; specificity: 30%; predictive value: 17%

angle at diagnosis was 14.47° (SD 8.92) and mean age was 12.48 years (SD 1.54). Twenty-four or 4% underwent surgery. From this group 260 girls were excluded on the basis of age at diagnosis and inadequate follow-up (Table 1). One of the girls without follow-up went directly to surgery. The mean Cobb angle of the remainder was 10.38° (SD 5.2; range 5°–35°). One who was lost to follow-up had a scoliosis greater than 30° and a total of 5 had a Cobb angle greater than 20°. Their mean age at diagnosis was 13.28 years (SD 1.44).

Study population

Among the 339 girls remaining in the study, the mean age at diagnosis was 12.8 years (SD 1.27) and the mean Cobb angle 17.37° (SD 9.42; range 1°–67°). Follow-up ranged from 3 months to 7.8 years (mean 2.4 years; SD 1.66). This information is given in Table 2. The median Risser sign at diagnosis was zero; 133 were post-menarche at diagnosis and 206 were pre-menarche. Skeletal age was routinely recorded at the first radiological examination, but since it very rarely differed significantly from chronological age, it will not be used here. In 46 girls (13.6%) the scoliosis progressed by 10° or more during the observation period.

Of this group of 339, 23 (6.8%) underwent surgery after a period of observation or conservative treatment (5 with a brace, 9 with electrospinal stimulation and 1 with both). The follow-up time for this operated group was 0.5–4.8 years (mean 2.2; SD 1.2) and not significantly different from that of the main group.

There were 145 thoracic curves with 28 progressing, 77 thoracolumbar with 8 progressing, 70 lumbar curves with 7 progressing and 47 double major with 3 progressing.

Cobb angle and progression

There were 46 girls whose scoliosis progressed by at least 10° during follow-up. The mean Cobb angle at diagnosis was 22.5° (SD 13.02) in progressive and 16.6° (SD 8.44) in stable scoliosis. The results are shown in Table 3. The initial Cobb angle in the progressive scoliosis was significantly higher ($P < 0.01$).

Those girls whose scoliosis progressed under observation had starting curves that were significantly higher than in those with stable scoliosis. The incidence of progression by curve size shows a statistically significant but by no means universal or predictive association between initial Cobb angle and progression (Table 4). Of the 46 girls whose initial scoliosis was more than 10°, 6 had at least a further 10° progression. Five passed 20°, and one stabilised without treatment at 42°.

Of the 13 girls whose scoliosis was 40° or more at diagnosis, 6 were braced and 3 went on to surgery, while 1 received electrospinal stimulation which failed, resulting in surgery. Six had an initial period of observation, by intention in 4 cases (1 of whom opted for surgery after 2 years) and due to problems with scheduling in 2 cases.

Since the question of conservative management cannot be ignored entirely, the results of bracing and electrospinal stimulation, compared to no treatment, are given in Table 5 for girls with scoliosis of greater than 20°. Treated scoliosis had a significantly worse outcome ($P < 0.01$). A total of 36 girls in this category were treated, 18 each with brace and electrospinal stimulation. Half of the girls given electrospinal stimulation and almost one-third in a brace progressed by more than 10°, compared

Table 7. Relationship between progression and presence or absence of ossification of the iliac crest: subjects with Cobb angle >19°

	Progressive scoliosis	Stable scoliosis	Age at diagnosis (years)	
			Mean	SD
Risser signs 0	22	36	12.6	1.3
Risser signs >0	3	36	13.8	0.7
Total	25	72		

$\chi^2 = 11.1460$; $P < 0.01$; $\lambda = 0$
With iliac crest ossification as the prognostic test: sensitivity: 88%; specificity: 50%; predictive value: 38%

Table 8. Relationship between progression Risser sign

	Risser sign						Total
	0	1	2	3	4	5	
Progression	41	4	1	–	–	–	45
Stable scoliosis	200	29	28	13	17	1	290
Total	241	31	29	13	17	1	334

$\chi^2 = 9.76036$; $P = 0.0823$ (not significant); $\lambda = 0$

to only 16% of those untreated. This gives a statistically significant association between management and outcome.

Risser sign and progression

Of the 334 girls whose iliac crests were visible on the diagnostic radiograph, 241 or 72% showed no evidence of iliac crest ossification (Table 6). Of the girls with scoliosis greater than 20° at diagnosis, 60% had a zero Risser sign (Table 7). There is a statistically significant association between progression and the absence of any ossification of the iliac crest, but this is not predictive in any way ($\lambda = 0$) since the majority of scolioses did not progress under observation. This association (Table 8) did not hold when the population was broken into smaller groups by the conventional [18, 28, 29] staging for iliac crest excursion. There was no association between risk factors and conventional stages of iliac crest ossification (Table 8). The increased risk applied only to those who had not begun the ossification process, and of those only 16% showed progression.

Menarche and progression

The relationship between menarchal status at diagnosis and curve progression is shown in Tables 9 and 10. In the whole group of 339 girls, 206 (61%) had not passed menarche at the time of diagnosis. Of the 46 girls with progressive scoliosis, 5 were post-menarche at diagnosis and 3 of these had starting curves of 20° or greater. Fifty-eight percent of the girls with stable scoliosis were pre-menarche at diagnosis, but this percentage drops to 31% when only those with scoliosis 20° or more are included.

Table 9. Relationship between progression and menarchal status: all subjects

Status	Progressive scoliosis	Stable scoliosis	Age at diagnosis (years)	
			Mean	SD
Pre-menarche	41	165	12.3	1.2
Post-menarche	5	128	13.6	0.9
Total	46	293		

$\chi^2 = 17.9593$; $P < 0.01$; $\lambda = 0.0056$
With menarchal status as the prognostic criterion: sensitivity: 89%; specificity: 44%; predictive value: 20%

Table 10. Relationship between progression and menarchal status: subjects with a Cobb angle >19°

Status	Progressive scoliosis	Stable scoliosis	Age at diagnosis (years)	
			Mean	SD
Pre-menarche	22	23	12.4	1.0
Post-menarche	3	52	13.4	1.2
Total	25	75		

$\chi^2 = 24.9024$; $P < 0.01$; $\lambda = 0.26$
With menarchal status as the prognostic criterion: sensitivity: 88%; specificity: 69%; predictive value: 49%

Table 11. Comparison of progressive and stable scoliosis: patient age at diagnosis

	Progressive scoliosis	Stable scoliosis
n	46	293
Age at diagnosis (years; mean ± SD)	12.1 ± 1.1	12.9 ± 1.3

$t = 4.3645$; $P < 0.01$

Table 12. Comparison of progressive and stable scoliosis: patient age at menarche

	Progressive scoliosis	Stable scoliosis
n	44	283
Age at menarche (years; mean ± SD)	13.5 ± 1.2	13.1 ± 1.0

$t = 2.3207$; $P < 0.01$

For these higher curves, λ for predictability is 0.26 – still low for prognostication and the initiation of radical treatment, but enough to indicate a trend. The χ^2 test shows that for all degrees of curvature, there is a positive association between pre-menarchal status and scoliosis progression.

Relationship between pubertal staging and diagnosis

Girls with progressive scoliosis were diagnosed at a mean age of 12.1 years, 0.8 years or almost 10 months

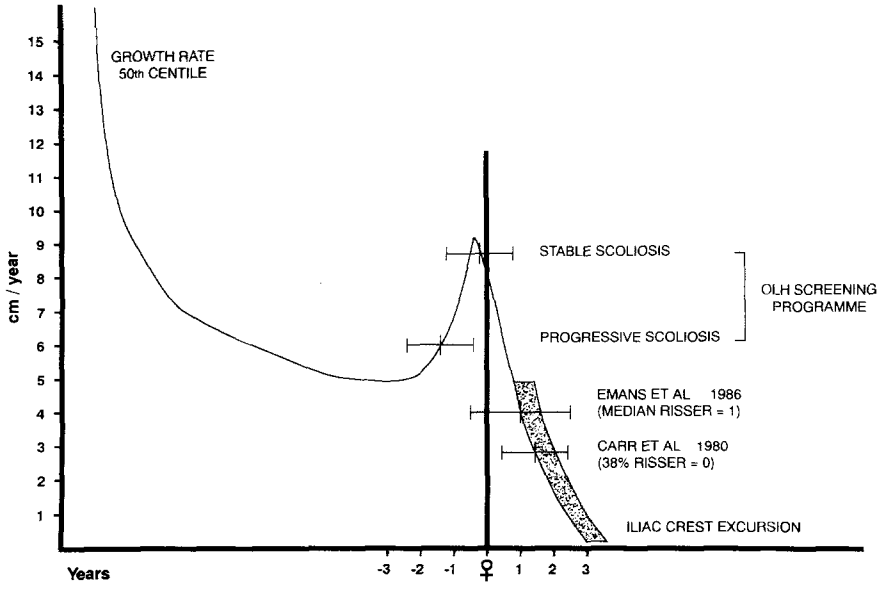

Fig. 1. Relationship between diagnosis and growth rate for progressive and stable scoliosis in the present study, compared with reported statistics of conservative treatment [5, 7]

Table 13. Height and growth rates related to menarchal status: progressive scoliosis ($n = 43$)

	Height at diagnosis (cm)		Height at last visit (cm)		Growth rate (cm/year)	
	Mean	SD	Mean	SD	Mean	SD
Years pre-menarche						
>4.49 ($n = 1$)	131	–	160	–	6	–
4 –4.49 ($n = 0$)	–	–	–	–	–	–
3.5–3.9 ($n = 0$)	–	–	–	–	–	–
3 –3.49 ($n = 4$)	141.3	5.4	161.5	2.7	5.6	1.7
2.5–2.9 ($n = 0$)	–	–	–	–	–	–
2 –2.49 ($n = 4$)	146.4	3.7	164.3	3.0	5.1	1.2
1.5–1.9 ($n = 10$)	147.4	5.4	163.4	4.3	4.4	1.1
1 –1.49 ($n = 8$)	152.5	5.7	163.2	5.6	5.0	2.9
0.5–0.9 ($n = 8$)	153.7	5.8	164.9	5.6	4.8	2.5
0 –0.49 ($n = 4$)	149.3	5.6	157.7	7.8	7.5	5.7
Years post-menarche						
0 –0.49 ($n = 2$)	143.5	1.5	145.5	0.5	0.65	0.05
0.5–0.9 ($n = 1$)	170	–	174.5	–	1.9	–
1 –1.49 ($n = 1$)	158	–	163.5	–	1.8	–

Mean increase in height: 12.2 cm (SD 7.5)
Mean final height: 162.2 (SD 6.8)
Mean increase as percentage of final height: 7.5% (SD 4.4)
Mean follow-up time: 3.11 years (SD 1.7)
Mean growth rate: 4.8 cm/year (SD 3.0)
Mean age when last seen: 15.9 years (SD 1.6)

before those with stable scoliosis (Table 11). This difference is statistically significant ($P < 0.01$). The median Risser sign for both groups was zero. Two girls from the progressive group were still pre-menarche at last review, but for the remainder, mean age at menarche was 13.55 years, half a year later than in girls with non-progressive scoliosis. This is also statistically significant (Table 12).

The mean age at menarche for Irish girls, calculated on a status quo method, is 13.52 years [15]. Of the girls with progressive scoliosis ($n = 24$), 53% had passed menarche at this age; of the girls with stable scoliosis ($n = 187$) 67% had passed menarche by the age of 13.52 years, a statistically greater proportion than the national norm of 50% ($P < 0.01$). Girls whose scoliosis did not progress under observation passed menarche significantly earlier than the national mean, at 13.09 years.

When the amount of time elapsing between diagnosis and menarche is compared for the two groups, a statistically significant discrepancy is seen ($P < 0.01$). Girls

Table 14. Height and growth rates related to menarchal status: stable scoliosis ($n = 244$)

	Height at diagnosis (cm)		Height at last visit (cm)		Growth rate (cm/year)	
	Mean	SD	Mean	SD	Mean	SD
Years pre-menarche						
>4.49 ($n = 3$)	139.4	13.49	161.8	3.9	3.8	1.8
4 –4.49 ($n = 0$)	–	–	–	–	–	–
3.5–3.9 ($n = 4$)	141	5.2	160.9	6.7	3.8	1.7
3 –3.49 ($n = 6$)	139.7	1.6	158	7.3	3.7	1.6
2.5–2.9 ($n = 3$)	143.8	5.3	162.5	1.9	3.6	0.3
2 –2.49 ($n = 13$)	144.1	6.4	159.3	5.0	3.2	1.3
1.5–1.9 ($n = 25$)	145.7	7.5	159.4	6.2	3.1	1.3
1 –1.49 ($n = 23$)	150.8	1.5	159.8	6.6	3.0	1.5
0.5–0.9 ($n = 27$)	153.1	4.4	159.9	5.1	3.3	1.5
0 –0.49 ($n = 45$)	155.9	6.7	160.0	6.9	2.9	1.6
Years post-menarche						
0 –0.49 ($n = 32$)	158.2	7.0	160.7	7.1	1.7	1.3
0.5–0.9 ($n = 23$)	157.2	6.1	159.7	6.1	1.0	1.1
1 –1.49 ($n = 19$)	158.6	3.4	160.1	3.9	1.1	1.4
1.5–1.9 ($n = 4$)	165.8	3.6	166.7	3.2	0.9	0.8
2 –2.49 ($n = 7$)	159.9	7.1	161.1	7.2	1.1	1.5
2.5–2.9 ($n = 7$)	163.6	9.1	164.5	8.4	0.5	0.5
3 –3.49 ($n = 3$)	157.6	4.2	158.7	4.6	0.8	0.5

Mean increase in height: 5.9 cm (SD 6.3)
Mean final height: 160.1 (SD 6.5)
Mean increase as percentage of final height: 3.7% (SD 3.9)
Mean follow-up time: 2.5 years (SD 1.8)
Mean growth rate: 2.5 cm/year (SD 1.8)
Mean age when last seen: 15.4 years (SD 1.5)

with stable scoliosis were diagnosed quite close to the onset of menses, at a mean time of 64 (SD = 1.5) days before. The combination of earlier diagnosis and later menarche results in the progressive group being diagnosed at a mean of 534 (SD = 1.3) days before menarche ($t = 6.0514$; $P < 0.01$).

Extrapolating back from menarchal status at diagnosis to the growth rate curve (from [16]) puts the mean diagnosis in the progressive group at the beginning of the acceleration phase of the growth spurt, while in the non-progressive group mean diagnosis was after peak growth velocity (Fig. 1). Tables 15 and 16 give the growth data on 287 girls for whom starting and finishing heights were available, relating diagnosis and menarche. They also compare increase in height between the groups, as an absolute figure, a percentage of final height and as rate of increase. The progressive group have longer follow-up, which is predictable since they were younger at diagnosis and, being progressive would warrant closer observation. The children whose scoliosis progressed under observation grew more ($P < 0.01$) to reach the same final height at the same final age. The overall growth rate was higher ($P < 0.01$) and the percentage of final height achieved under observation was greater ($P < 0.01$). The numbers if divided into age groups are too small to permit valid graphical representation.

Discussion

Study population

The patient group in this study has been described before [10–12]. It consists of school girls, predominantly in the 12- to 14-year age group (average 12.5 years). The referral rate varied significantly during the time in question, falling in proportion to the rise in knowledge of the natural history. In particular, it became practice as the programme proceeded to identify girls with minor asymmetry and return them to the screening pool for follow-up without radiographic investigation at any stage. These, of course, do not appear in this report. The subjects excluded for reason of inadequate data or follow-up had a significantly lower Cobb angle than those included ($P < 0.01$).

The decision to use chronological rather than skeletal age was prompted by the observation that only a very few girls showed a discrepancy between the two, and that those who did were as likely to have advanced as retarded skeletal age. It was observed by Marshall in 1974 [21] that while skeletal age predicts the approximate percentage of total growth remaining, "the role of bone age in height prediction apparently is not to indicate whether the peak of adolescent growth spurt will be reached early or late" (page 38). The present study

found the critical landmark to be the growth spurt and this was more usefully identified by consulting the child's height record. In the final analysis, while estimation of skeletal age is subjective and has an error of plus or minus half a year, chronological age is potentially accurate to within an hour and is backed by the objectivity of ineluctable law.

Cobb angle at diagnosis

Table 3 shows a statistically significant difference in initial Cobb angle between stable and progressive scoliosis. However, it is not so great as to make an instant diagnosis possible on the basis of curve size alone. Similarly, progression was not inevitable, although it was increasingly probable, in the girls with the more pronounced curves. The results for the 100 girls whose starting Cobb angle was 20° or more show no significant difference that can be related either to management or to curve size alone.

A Cobb angle of less than 10° is usually disregarded. Six of the 46 study subjects in this category had significantly progression, by at least 10°. Four of these stabilised between 20° and 25° and one reached 42° before stabilising. None were treated. It was decided to include this low group in the final picture, as to exclude those who proved non-progressive would give an unjustifiable bias.

Examination of results in those treated conservatively shows that there was a statistically significant association between progression and treatment. This is not a controlled trial and these figures cannot be interpreted as evidence for or against any form of management. Selection for treatment on the basis of progression risk showed an increased risk of progression in those selected. However, since this is not in favour of the treated group, it is presumed that it reflects the fact that the prognostic indicators were more reliable than the treatment, not that the treatment itself was actually harmful.

Progression and the staging of the iliac crest apophysis

The findings on this point are in agreement with those of Risser in 1958 [22], when he stated that progression risk was significantly reduced once the first appearance of the iliac crest apophysis was noted. What is noteworthy is that the majority of girls were at Risser stage zero when scoliosis was detected and yet showed no serious progression, even those with curves above 20°. This "all or nothing" approach to the significance of iliac crest ossification is supported by Table 8, which breaks down the results by Risser staging, and all association is lost.

Progression and menarche

In contrast to Urbaniak et al. [28], this study demonstrated a very clear association between progression and menarchal status. It was non-predictive, thus not alone useful in prognosis, although for the greater curves it was becoming more so ($\lambda = 0.26$). The majority of cases of scoliosis remain non-progressive from the time of diagnosis. Since menarche usually occurs 6–8 months before Risser 1 [25], this finding makes it possible for a tentative prognosis to be made considerably earlier than by reliance on iliac crest ossification.

Time between diagnosis and menarche

Tables 15 and 16 show a clear difference between growth rates in the group. This is not surprising, since the group with progressive scoliosis reaches farther back in time to encompass more of the pubertal growth spurt. Unfortunately, the numbers in the individual subgroups are too small to permit statistical analysis. It is noteworthy that the growth rate falls off dramatically after menarche in both groups. It is not clear from this data whether the higher growth rate observed in the progressive group is a genuine (perhaps aetiological?) difference or an artefact caused by the disproportion of early diagnoses in the progressive group.

The relationship between pubertal status at diagnosis and prognosis in this series was highly significant. Clearly, there is a wide variation, and equally clearly the association between the different maturity markers is more statistical than causal. What is also certain is that the precise time of diagnosis of adolescent idiopathic scoliosis is determined less by the natural history and more by extraneous factors in the child's life; in the cases reported here, entirely by the date for screening arranged between the school and the Orthopaedic Department of Our Lady's Hospital for Sick Children. Thus, diagnosis could be said to occur at a random point along the normal maturation curve determined by events that are immaterial to the individual scoliosis.

Prognosis then depends not only on such obvious characteristics as radiological findings and pubertal status, but also on dynamic concepts of growth rate and acceleration which can only be determined over time. A child diagnosed with 16° of scoliosis at the beginning of her growth spurt might be observed to progress to perhaps 35° or more after 6 months, to about the time of her menarche, by which time she has stabilised. If she were not diagnosed until this stage, she would not progress further and would be deemed "stable", yet it would be the same child, the same scoliosis.

This observation raises some serious points. Firstly, in the two major brace reviews [5, 7], while menarchal status was not considered specifically, the median Risser sign was 1. Reference to Fig. 1 shows that growth potential by this time is considerably reduced. Since at least 50% of the subjects in these reviews had a Risser sign of 1 or more, the validity of the conclusions in these papers must be questioned. Orthotic treatment may be effective, but this has not been demonstrated.

Another problem raised by these findings is of the effectiveness of a scoliosis screening programme. The programme discussed here is organised as an integral part of the activities of the Orthopaedic Department at Our Lady's Hospital for Sick Children. Consequently, the delay between screening and clinic review can be less than a week if necessary, and was less than 1 month for most of the cases considered here. Nevertheless, for

86.4% of subjects, diagnosis occurred too late for progression to be observed. For the remaining 13.6%, it seems unlikely that early diagnosis brought any tangible benefit to the patients. The gain has been an increased knowledge of the natural history, which in turn undermines the validity of its own source.

Finally, it is acknowledged that this study concentrates largely on the acute progression seen in the growth spurt. Slow but equally disfiguring progression may occur later, and it is well known that severe curves continue to progress throughout life. The precise incidence of such later progression is difficult to pin down as it is slower and may not be significant from the management point of view. Very long-term follow-up (see, e.g., [1]) has shown that scoliosis may improve, deteriorate or remain unchanged over protracted periods of adult life. Howell et al. [17] have shown that growth, far from halting completely in adolescence, has a long slow decrescendo for many years. Even then, when growth in the sense of "increase in size" has finished, structural metabolic activity in bone does not cease. Remodelling turnover continues throughout life [8]. The underlying cause of idiopathic scoliosis remains unknown: if the forces that resulted in primary bone formation produced the deformity of scoliosis at a rate proportional to this formation, these same forces will presumably continue into adult life and affect the shape of secondary bone remodelling.

Conclusion

This report describes observations suggesting that the acute progression of adolescent idiopathic scoliosis occurs in the early, acceleration phase of the adolescent growth spurt. This period in a child's maturation is unique in that it is the only instance of a consistent and sustained increase of growth rate. This poses the question: is progression a function of the acceleration itself or is increasing deformity more likely above a certain growth rate, whether accelerating or slowing down? Alternatively, is the growth spurt only relevant in that its very speed forces the progressing deformity to our attention? If, in some alternative universe, children grew at a slow steady pace to the same final height by age 20, would spinal deformity progress equally irrevocably to the same final outcome?

References

1. Ascani E, Bartolozzi P, Logroscino CA, Marchetti PG, et al (1986) Natural history of untreated idiopathic scoliosis after skeletal maturity. Spine 11:784–789
2. Biondi J, Weiner DS, Bethem D, Reed JF (1985) Correlation of Risser sign and bone age determination in adolescent idiopathic scoliosis. J Paediatr Orthop 5:698–701
3. Brown JC, Axelgaard J, Howson DC (1984) Multicenter trial of a non-invasive stimulation method for idiopathic scoliosis: a summary of early treatment results. Spine 9:382–387
4. Bunnell WP (1986) The natural history of idiopathic scoliosis before skeletal maturity. Spine 11:773–776
5. Carr AW, Moe JW, Winter RB, Lonstein JE (1980) Treatment of idiopathic scoliosis in the Milwaukee brace. J Bone Joint Surg 62-A:599–612
6. Cobb JR (1948) Outline for the study of scoliosis. Am Acad Orth Surg (Instructional course) 7: 261–275
7. Emans JB, Kaelin A, Bancel P, Hall JE, Miller ME (1986) The Boston bracing system for idiopathic scoliosis. Follow-up results in 295 patients. Spine 11:792–801
8. Frost HM (1979) A chondral modelling theory. Calcif Tissue Int 28:181–200
9. Goldberg C, Dowling FE (1986) Natural history of idiopathic scoliosis. Proceedings of the Scoliosis Research Society, Bermuda, Sept, pp 22–26
10. Goldberg C, Thompson F, Dowling F, Regan BF, Blake NS (1980) Pilot study for a screening programme in South Dublin. J Irish Med Assoc 73:265–268
11. Goldberg C, Fogarty EE, Blake NS, Dowling F, Regan BF (1983) School scoliosis screening: a review of 21,000 children. Irish Med J 76:247–249
12. Goldberg C, Blake NS, Fogarty EE, Dowling FE, Regan BF (1987) School scoliosis screening: a report. Irish Med J 80: 325–326
13. Goldberg C, Dowling FE, Fogarty EE, Regan BF, Blake NS (1988) Electro-spinal stimulation in children with adolescent and juvenile scoliosis. Spine 13:482–484
14. Goldberg C, Emans JB, Hall JE, Dolan M, Dowling FE (1992) Scoliosis, bracing and the Risser sign. A statistical comparison of bracing and natural history. J Bone Joint Surg [Br] 74 [Suppl]:85
15. Hoey HMCV, Cox LA, Tanner JM (1986) The age of menarche in Irish girls. Irish Med J 79:283–285
16. Hoey HMCV, Tanner JM, Cox IA (1987) Clinical growth standards for Irish children. Acta Paediatr Scand Suppl 338
17. Howell FR, Mahood JK, Dickson RA (1992) Growth beyond skeletal maturity. Spine 17:437–440
18. James JIP (1954) Idiopathic scoliosis: the prognosis, diagnosis and operative indications related to curve patterns and the age of onset. J Bone Joint Surg 36-B:36–49
19. Kehl DK, Morrisey RT (1988) Brace treatment in adolescent idiopathic scoliosis. An update on concepts and technique. Clin Orthop 229:34–43
20. Lonstein JE, Carlson JM (1984) The prediction of curve progression in untreated idiopathic scoliosis during growth. J Bone Joint Surg 66-A:1061–1971
21. Marshall WA (1974) Interrelationships of skeletal maturation, sexual development and somatic growth in man. Ann Hum Biol 1:29–40
22. Risser JC (1958) The iliac apophysis: an invaluable sign in the management of scoliosis. Clin Orthop 11:111–118
23. Risser JC, Ferguson AB (1936) Scoliosis: its prognosis. J Bone Joint Surg 18:557–670
24. Root AW (1973) Endocrinology of puberty. I. Normal sexual maturation. J Paediatr 83:1–19
25. Scoles PV, Salvagno R, Villalba K, Riew D (1988) Relationship of iliac crest maturation to skeletal and chronological age. J Paediatr Orthop 8:639–644
26. Swank SM, Brown JC, Jennings MV, Conradi C (1989) Lateral electrical surface stimulation in idiopathic scoliosis. Experience to two private practices. Spine 14:1293–1295
27. Terver S, Kleinman R, Bleck EE (1980) Growth landmarks and the evolution of scoliosis: a review of pertinent studies and their usefulness. Dev Med Child Neurol 22:675–684
28. Urbaniak JR, Schaffer WW, Stelling FH (1986) Iliac apophyses. Prognostic value in idiopathic scoliosis. Clin Orthop 116:80–85
29. Zaoussis AL, James JIP (1958) The iliac apophysis and the evolution of curves in scoliosis. J Bone Joint Surg [Br] 40:442–453