Adolescent idiopathic scoliosis (AIS) affects 1–3% of children in the at-risk population of those aged 10–16 years. The aetiopathogenesis of this disorder remains unknown, with misinformation about its natural history. Non-surgical treatments are aimed to reduce the number of operations by preventing curve progression. Although bracing and physiotherapy are common treatments in much of the world, their effectiveness has never been rigorously assessed. Technological advances have much improved the ability of surgeons to safely correct the deformity while maintaining sagittal and coronal balance. However, we do not have long-term results of these changing surgical treatments. Much has yet to be learned about the general health, quality of life, and self-image of both treated and untreated patients with AIS.

Introduction
Adolescent idiopathic scoliosis (AIS) is a structural, lateral, rotated curvature of the spine that arises in otherwise healthy children at or around puberty. The diagnosis is one of exclusion, and is made only when other causes of scoliosis, such as vertebral malformation, neuromuscular disorder, and syndromic disorders, have been ruled out. Patients are generally screened with Adams’ forward bend test and a scoliometer, although a definitive diagnosis cannot be made without measuring the Cobb angle on a standing coronal radiograph (figure 1). When defined as a Cobb angle of at least 10°, epidemiological studies estimate that 1–3% of the at-risk population (children aged 10–16 years) will have some degree of spinal curvature, although most curves will need no intervention.1,2 In this Seminar we discuss present notions about aetiopathogenesis, natural history, non-operative treatment, and surgery.

Aetiopathogenesis
Despite much clinical, epidemiological, and basic science research, the aetiopathogenesis of AIS remains unknown.3–5 AIS is often seen in multiple members of one family, which strongly suggests that it has a genetic component. A meta-analysis of studies of twins showed concordance for AIS in 73% of monozygotic twins and 36% of dizygotic twins.6 Several studies that assessed large pedigrees showed different methods of inheritance, such as autosomal dominance, maternal factors, multiple gene inheritance, multifactorial inheritance, and X-linked dominance.7–9 However, Axenovich and colleagues8 analysed pedigrees of 101 families (778 individuals) and undertook a complex segregation analysis. They noted that genetic control of severe forms of AIS could be attributed to an autosomal dominant, major gene diallelic model with incomplete sex-dependent penetrance of the genotypes. Moreover, four genome-wide screens of AIS have been reported.

Overall, results from these studies are disappointing since no one locus has been identified.2,10,11 This absence can be explained by phenotypic or genotypic heterogeneity, incomplete penetrance, and variable expressivity. Multiple genes could be implicated in the disorder and a phenocopy could also arise in some families. Precise measurement of the degree of curvature in each affected individual and identification of any scoliosis in the family are of paramount importance for future genetic studies. Several investigators have reported candidate-gene analyses of connective tissue genes. Results of these studies have excluded genes for fibrillin 1 (FBN1) and 2 (FBN2); collagen type I (COL1A1 and 2 (COL1A2); elastin (ELN); aggregan (ACAN); and heparan sulfotransferases (HS3ST3A1 and HS3ST3B1) as causes of AIS.12–14 Inoue and colleagues15 studied polymorphisms in the genes for vitamin D receptor (MED4), oestrogen receptor (ESR1), and CYP17A1 in relation to curve progression. The results suggested that XbaI site polymorphism in ESR1 was associated with curve progression.15,16 However, Tang and colleagues17 did not show an association between ESR1 and AIS.

On the basis of the hypothesis that AIS is related to an abnormality in melatonin metabolism, Morcuende and colleagues18 investigated several known receptors, such as hMel1A, hMel1B, and ROR-α, but reported no evidence of mutations in the coding regions of these genes. Gao and colleagues19 discovered a potentially functional polymorphism in CHD7 that is over-transmitted to affected offspring and predicts disruption of a caudal-type (Cdx) transcription-factor binding site. They suggest that this gene is associated with the susceptibility of AIS and propose an overlap of causes between the rare, early onset CHARGE syndrome and common AIS.20 Another potentially involved gene is CHD8, which is linked to CHARGE syndrome.21

Aetiology
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syndrome (coloboma of the eye; heart defects; atresia of the choanae; retardation of growth and development, or both; genital and urinary abnormalities, or both; and ear abnormalities and deafness) and AIS.19

Szappanos and colleagues20 reported a family with multiple familial occurrences of AIS coupled with the same anomaly of the karyotype [inv(10)(p11q26)]. However, analysis of restriction fragment length polymorphism showed no abnormality. Bashiardes and colleagues21 investigated another family in whom a pericentric inversion of chromosome 8 seemed to cosegregate with AIS in three generations. They used fluorescent in-situ hybridisation and established that the p-arm break did not interrupt any known gene, but the q-arm break took place between exons 10 and 11 of the syntrophin, gamma-1 (SNTG1) gene. Mutational analysis of SNTG1 exons revealed a 6-bp deletion in exon 10, but this deletion did not consistently cosegregate with the disease in this family.21

What is the role of melatonin in the development of AIS? Several investigators have shown that pinealectomy in chickens, rats, and hamsters leads to scoliosis, and have attributed this effect to decreased melatonin production. However, other reports have failed to show this development of scoliosis.22–26 Additionally, patients with AIS do not have an inability to form melatonin, or impaired sleep or immune function. Moreover, several diseases associated with an abnormal melatonin rhythm do not have an obvious effect on the development of scoliosis. Thus scoliosis is unlikely to result from a simple absence of melatonin; rather, it might be the result of melatonin’s effect on other unknown growth mechanisms.5

Abnormalities in the structure and function of platelets have been noted in patients with AIS. Evidence suggests that raised concentrations of calmodulin result in altered skeletal muscle activity and subsequent progressive curvatures.27 Burwell28 formulated an integrative platelet-skeletal hypothesis whereby a small curve loads intervertebral discs and vertebral body growth plates creating mechanical microinsults, vascular damage, and platelet activation with changes in calmodulin, leading to the release of growth factors that affect the compromised vertebral endplate. The end result of this postulated chain of events is a relative anterior spinal overgrowth and curve progression. However, the key questions of how and why the initial small curve develops have not been answered.

Several MRI studies have renewed interest in abnormal neuroanatomy associated with AIS. Chiari type-I malformations and spinal cord syrinx have been noted in cases of AIS; and in younger patients, drainage of the syrinx leads to resolution of the deformity.29–31 Additionally, the relation between impaired autonomous nervous system function and aetiopathogenesis of AIS has received some attention. Sympathectomy of intercostal nerves in growing rabbits results in hypervascularity of the soft tissues, increased rib growth on the side of the operation, and thoracic scoliosis convex to the opposite side.32,33

The hypothesis that an abnormality of the paravertebral muscles contributes to the development of AIS has been discussed for many years. Several have been recorded, including decreased number of type-II fibres (fast-twitched), fibre splitting, tubular bodies, myofilament disarray consistent with myopathy, and generalised membrane defect (namely, impaired calcium pump). However, no definitive conclusion can be reached about these abnormalities, though they are likely to be secondary to the deformity itself.

Progressive AIS is attributed to relative anterior spinal overgrowth during the adolescent growth spurt. MRI studies of vertebral thoracic morphometry show that longitudinal growth of the vertebral bodies in patients with AIS is disproportionate and faster than age-matched and sex-matched controls, and takes place mainly by endochondral ossification. By contrast, the circumferential growth by membranous ossification is slower in both the vertebral bodies and pedicles.34–36 However, the mechanisms of this growth asymmetry are not well understood.

Natural history

The natural history of scoliosis varies with the aetiopathogenesis and curve pattern. Early long-term studies of so-called idiopathic scoliosis presented a grim prognosis, perpetuating the common misperception that all types of idiopathic scoliosis inevitably lead to disability from back pain and cardiopulmonary compromise.37–39 Shortcomings of these earlier studies were (1) the inclusion of patients with other causes of scoliosis or of early onset idiopathic scoliosis; and (2) failure to assess the outcomes related to the location of the curvature. Patients with AIS and their families are often unnecessarily upset by the long-term picture painted by these early studies. Although the natural history of scoliosis...
clearly varies according to the aetiopathogenesis and curve pattern, screening and treatment policy for AIS has been formed on the basis of this misinformation.

Treatment of any disorder is an attempt to alter its natural history; therefore, long-term studies of both the natural history and treatment outcomes are necessary. Treatment should be proven to prevent the negative outcomes of natural history without introducing iatrogenic complications. The outcomes of untreated AIS through adulthood have been studied in Britain, Italy, Sweden, and the USA. The most frequently noted long-term sequelae of untreated AIS are curve progression, back pain, cardiopulmonary problems, and psychosocial concerns. Although present in most untreated patients, the severity of these sequelae and their effect on overall health and function is very variable.

The size of the curve tends to increase over the entire lifetime, but the degree of progression over a lifetime and the time-at-risk varies with many factors. Clinicians and patients need to be aware of the risk of curve progression as they make treatment decisions. Factors predicting curve progression include maturity (age at diagnosis, menarchal status, and the amount of skeletal growth remaining), curve size, and position of the curve apex. Many investigators agree that curves with a thoracic apex have the highest prevalence of progression, ranging from 58% to 100%. The more skeletally and sexually immature the patient is, the greater the probability of curve progression.

Table 1 summarises the risk of progression that results from the combined effects of maturity and curve size. Progression equations have also been developed to quantify the risk of progression. Peterson and Nachemson include Risser sign, level of apex, presence of trunk imbalance, and chronological age; the equation developed by Lonstein and Carlson includes the Cobb angle, Risser sign, and chronological age. These methods could be helpful to clinicians and patients who want to base their treatment decisions on the risk of progression.

The goal of non-operative treatment during adolescence is to prevent curve progression; the goal of surgical treatment is curve correction and maintenance. However, the only negative outcome of AIS strongly and consistently associated with curve size is pulmonary function. Besides the degree of lateral curvature, other factors such as high degrees of thoracic lordosis and vertebral rotation and decreased respiratory muscle strength affect pulmonary function. Unlike early onset (age 0–8 years) idiopathic scoliosis in which substantial loss of vital capacity and forced expiratory volume in 1 s could cause pulmonary hypertension, right heart failure, and death, these problems rarely arise in AIS. Large curves (greater than 50°) with a thoracic apex have been associated with reduced vital capacity and more frequent shortness of breath, but rarely, severe cardiopulmonary compromise.

Most adults have back pain in some form during their lifetime. About 50% of adults have an episode of low back pain in any particular year and 15% report frequent back pain or pain lasting for more than 2 weeks in a year. Ascani and others reported the frequency of pain in adults with AIS (61%) to be similar to that of the general population, whereas another report showed that chronic pain was more frequent and of greater intensity and duration in scoliotics than in the general population. However, both groups of investigators agreed that back pain does not seem to cause excessive disability and, overall, patients work and undertake everyday activities similarly to their peers. Over a lifetime, most patients with AIS, as well as the general population, will develop clinically important radiographic osteoarthritic changes. However, history of backache seems to be unrelated to the presence or absence of osteoarthritis or curve severity. Back tenderness on palpation has not been related to the curve type or severity of osteoarthritis on radiography except for areas of lateral liss thesis in lumbar and thoracolumbar curves. These curves, especially those with lateral liss thesis at the caudal end, tend to cause a higher frequency of back pain than do other curve patterns.

Untreated AIS, and its possible sequelae such as back pain and pulmonary limitations, affects overall function and self-esteem. In this respect, publications are scarce and research is conflicting. Some studies show that patients perceive themselves to be less healthy and restricted in physical and social activities. Ascani and colleagues reported “real psychological disturbances” in 19% of their sample, 94% of whom had curves greater than 40°. Other studies have noted no significant difference between people with AIS and controls in their ability to undertake activities or in quality of life. With respect to psychosocial aspects of AIS and presence of clinical depression, scoliosis patients fare as well as age-matched and sex-matched controls. However older, untreated AIS patients are much less satisfied than are controls with their body image, and appearance in clothes and swimsuits. About a third of patients believe that their curvature has restricted their life in some way, such as difficulty in purchasing clothes, reduced physical ability, and self-consciousness.

Table 1: Percentage of curve progression: magnitude of curve at initial detection vs age

<table>
<thead>
<tr>
<th></th>
<th>10–12 years</th>
<th>13–15 years</th>
<th>16 years</th>
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<tbody>
<tr>
<td>&lt;19°</td>
<td>25%</td>
<td>10%</td>
<td>0%</td>
</tr>
<tr>
<td>20–29°</td>
<td>60%</td>
<td>40%</td>
<td>10%</td>
</tr>
<tr>
<td>30–39°</td>
<td>90%</td>
<td>70%</td>
<td>30%</td>
</tr>
<tr>
<td>&gt;40°</td>
<td>100%</td>
<td>90%</td>
<td>70%</td>
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*Data reproduced from reference 56 with permission of authors.
As they age, most patients with untreated AIS can have back pain and cosmetic concerns. Patients with untreated AIS can function well as young adults, become employed, get married, have children, and become active older adults. Unfortunately, patients can develop substantial deformity, and the cosmetic aspect of this disorder cannot be disregarded. Treatment recommendations—watchful waiting, physiotherapy, bracing, and surgery—should be made on an individual basis, and the patient and family should be well informed about the natural history of the disease.

Non-operative treatment

Although AIS is diagnosed and treated worldwide, treatment approaches vary internationally. In patients who still have growth remaining, watchful waiting (observation), followed by bracing if the curve progresses to greater than 25°, is the general course of care in North America.1 Physical therapy (outpatient and inpatient rehabilitation) has been recommended as the first line of treatment for small curves and those with a low risk of progression by various, mostly European, clinicians.66,67 No definite evidence has shown that physical therapy or bracing reduces the risk of curve progression, corrects the existing deformity, or decreases the need for surgery. Patients should be aware of the absence of evidence for these treatments, and be provided with the opportunity to decide for themselves which treatment, if any, they believe will best fit with their own personal assessment of risk, side-effects, and benefits.

The popularity of physical therapy for AIS has waned and waned over time, but it continues to be favoured in France, Germany, and Spain. The aim of physiotherapy is to prevent aggravation of the deformity in mild scoliosis (ie, curves less than 25°) and to enhance the effect of a brace and counteract its side-effects in moderate scoliosis (ie, curves between 25° and 45°). These aims are met, theoretically, by a combination of prescribed and monitored sports activities and kinesitherapy to increase coordination, spinal proprioception, and movement control.80,81 Neither of these braces have been widely tested, but the inclusion of dynamic forces in addition to the original braces. Novel to this brace is the addition of expansion room to allow for active correction by respiratory movements.79 The SpineCor and TrIAC (Boston Brace International, Avon, MA, USA) deliver a higher degree of initial correction through the use of a hypercorrected mould and pads to provide derotational forces throughout the trunk than did the original braces. Neither of these braces have been widely tested, but the inclusion of dynamic forces in addition to the traditional three-point control shows a more sophisticated understanding of the growing spine.

The expectation of treatment with bracing is to prevent progression of the curve until the patient reaches skeletal maturity, at which time the risk of curve progression (and hence the risk of surgery) greatly diminishes. Over the years, the medical community has arrived at two different conclusions. On the one hand, some believe that the weight of evidence favours a positive effect of bracing on the natural history of AIS. On the other hand, the US Preventive Services Task Force,79 its Canadian counterpart,78 and many individual practitioners worldwide believe that evidence to support bracing is poor or inconclusive. In fact, the effectiveness of bracing is being tested in two randomised trials in North America and the Netherlands.79,72

### Table 2: Surgery rates after TLSO treatment

<table>
<thead>
<tr>
<th>Brace Type</th>
<th>Total sample size</th>
<th>Surgical rate (%)</th>
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<tbody>
<tr>
<td>Wilmington</td>
<td>147</td>
<td>31%</td>
</tr>
<tr>
<td>Wilmington</td>
<td>79</td>
<td>11%</td>
</tr>
<tr>
<td>Providence</td>
<td>102</td>
<td>18%</td>
</tr>
<tr>
<td>Boston</td>
<td>212</td>
<td>13%</td>
</tr>
<tr>
<td>TLSO15</td>
<td>54</td>
<td>26%</td>
</tr>
<tr>
<td>Boston or Charleston17</td>
<td>319</td>
<td>22%</td>
</tr>
<tr>
<td>TLSO19</td>
<td>24</td>
<td>13%</td>
</tr>
<tr>
<td>Boston or Charleston17</td>
<td>120</td>
<td>43%</td>
</tr>
<tr>
<td>Boston19</td>
<td>276</td>
<td>30%</td>
</tr>
<tr>
<td>Charleston19</td>
<td>95</td>
<td>18%</td>
</tr>
<tr>
<td>Rosenberger19</td>
<td>71</td>
<td>31%</td>
</tr>
<tr>
<td>Charleston19</td>
<td>42</td>
<td>7%</td>
</tr>
<tr>
<td>Boston19</td>
<td>151</td>
<td>13%</td>
</tr>
<tr>
<td>Boston21</td>
<td>50</td>
<td>30%</td>
</tr>
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Blount first used the Milwaukee brace (originally a postoperative orthotic) in 1958.78 Thoracolumbosacral orthoses (TLSO) were then developed, which improved on the Milwaukee brace by removal of the cervical component; use of lighter materials; and customisation to improve comfort, cosmesis, and compliance.84 Some braces have been developed for use only part-time or at night-time,75,76 whereas most others are worn for 18 or more hours per day.

Modifications to the typical rigid TLSO include the Cheneau bracing programme and the SpineCor dynamic brace (SpineCorporation, Chesterfield, UK). The Cheneau brace27,78 delivers a higher degree of initial correction through the use of a hypercorrected mould and pads to provide derotational forces throughout the trunk than did the original braces. Novel to this brace is the addition of expansion room to allow for active correction by respiratory movements.79 The SpineCor and TrIAC (Boston Brace International, Avon, MA, USA) are non-rigid braces for AIS treatment. Straps are placed to coincide with a specific correcting movement for each kind of curve, producing a progressive positional change, dynamic curve correction, and more appropriate muscle balance.83,84 Neither of these braces have been widely tested, but the inclusion of dynamic forces in addition to the traditional three-point control shows a more sophisticated understanding of the growing spine.

The nature of the dose–response relation between brace wear and outcome is not well established, most probably because of previous inability to accurately and reliably measure time that a brace is worn. On the basis of review of records and other subjective indicators of compliance, in a meta-analysis Rowe and colleagues85 reported that a 23-h per day protocol was more successful than 16-h or night-time protocols. Mechanical devices—mainly measuring temperature, pressure, and proximity sensors—to measure brace...
wear are now available. These devices create the opportunity to critically assess the relation between brace dose (ie, wear time) and curve response. Investigators used objective measures to show compliance to be much lower than that reported by the patient. However, a positive correlation between the time that a brace is worn and outcome is partly evident. In Rahman and colleagues’ study, 34 patients had temperature monitors mounted in their braces for the duration of treatment. Of those patients who were highly compliant, 11% had progressive curves compared with 56% of those patients who were less compliant.

Many investigators have examined radiographic outcomes of TLSOs for AIS over the past half-century. Many regard a progression of greater than 5° to suggest a true change in curvature status, and often use this radiographic benchmark as their definition of treatment failure. This outcome has been used in many case series reports, although a few studies used improved designs such as case–control comparison and one prospective cohort study has been reported. The only prospective trial showed a lower rate of curve progression in the group with braces as compared with the non-treated group. Although the most rigorous so far, this study was non-randomised, non-blinded, with baseline differences between the groups not statistically adjusted for, and the results have not been replicated.

Another definition of brace treatment failure is the number of surgical procedures required despite treatment. The results of studies tracking surgical rates vary widely, most probably caused by different sample characteristics. Table 2 gives an overview of the studies of patients who meet present indications for bracing treatment with the rate of surgery despite bracing. The surgical rate ranges from 7% to 43%.

**Surgery**

The primary objectives of surgical treatment with instrumentation are to (1) arrest progression, (2) achieve maximum permanent correction of the deformity in three dimensions, (3) improve appearance by balancing the trunk, and (4) keep short-term and long-term complications to a minimum. The generally agreed indication for surgery in adolescents is a primary curve greater than a Cobb angle of 45°. In adults, the surgical indications are pain related to the curvature that is unresponsive to non-operative management, curve progression that has exacerbated symptoms, and functional capabilities. Although adolescent patients can choose to delay surgery until they are adults, adults often have less flexible curves than seen in children, which could need anterior and posterior (staged or sequential) procedures. Additionally, the rate of complications in adults is much higher for the same procedures than it is in the adolescent population. Complications in adults are pseudarthrosis, proximal and distal junctional kyphosis, and extended recovery.

Improved preoperative assessments supplemented by intraoperative neurophysiological monitoring and blood salvage techniques have made modern scoliosis surgery safer than previously. Enhancement of solid bony fusion over the surgical instrumentation can now be achieved through autogenous bone graft, allograft, demineralised bone matrix, or the latest biological bone substitutes.

Posterior instrumentation remains the mainstay of treatment for most idiopathic curves. From the first generation of Harrington instrumentation introduced in the 1960s to modern third-generation instrumentation evolved from the Cotrel-Dubousset system in the 1980s, much progress has been achieved by improved multi-

![Figure 2: Radiographs of a teenager with progressive AIS treated by posterior instrumentation by hybrid (rods, hooks, and screws)](A) Preoperative standing posterior-anterior (PA); (B) preoperative standing lateral; (C) postoperative standing PA; and (D) postoperative standing lateral.
planar (coronal, sagittal, and transverse plane) correction, stable fixation, reduced levels of fusion, and avoidance of postoperative immobilisation with a cast or brace. Such progress has been possible through combinations of wires, hooks, and lumbar pedicle screw constructs over contoured rods, and interconnecting systems applied over several purchase sites posteriorly (the lamina, pedicle, transverse processes, and spinous processes)\(^{107,108}\) (figure 2).

The extended use of many segmental pedicle screws in the thoracic spine, originally pioneered by Suk\(^ {109}\) allows even better three-column mechanical fixation, multiplanar corrections, such as the rib prominence reduction, and saving of fusion levels compared with standard hook-wire constructs\(^ {110–112}\) (figure 3). The disadvantages of thoracic pedicle screws are a steep learning curve, increased cost, safety concerns, and difficulties associated with accurately placing pedicle screws within dysplastic pedicles\(^ {113–115}\). At present time, no conclusive evidence exists about improved radiographic outcomes in patients with AIS correlate with enhanced function, self-image, or health\(^ {104}\).

Anterior instrumentation has been used mainly for isolated thoracolumbar and lumbar curves. Evolving from the early Dwyer cable to the vertebral screw system\(^ {116}\), Zeilke ventral derotation spondylodesis, screw-single rod system\(^ {117}\) and other similar systems have been used satisfactorily with good correction of the frontal and transverse plane, restoration of truncal balance\(^ {118,119}\) and reduction of fusion levels. The main disadvantages are the kyphosing tendency of these systems, higher rates of implant breakage, pseudarthrosis, and the necessity of postoperative cast or brace protection\(^ {120,121}\). The latest dual-rod, multiple vertebral screw system provides good rigid fixation, improved correction in the sagittal plane, and minimum need for postoperative protection\(^ {122,123}\).

Anterior instrumentation has been extended to thoracic scoliosis to achieve improved sagittal plane correction and reduced number of fused levels, and to prevent crankshafing (continued anterior spinal growth in the face of a posterior fusion) in the immature patient (figure 4)\(^ {124–126}\). The disadvantages of anterior open thoracotomy approach are the rate of implant breakage, pseudarthrosis, the surgical scar, and the unfavourable effect on lung function\(^ {124,127}\). Video-assisted thoracoscopic anterior instrumentation has been used to reduce the surgical scar. Such surgery, however, entails a very steep learning curve, risk of encroaching on adjacent vital structures, and problems related to anaesthesia in one lung\(^ {128–127}\). Hence, whether this method is useful cannot be addressed without clearly documented advantages over the latest posterior instrumentation with thoracic pedicle screws\(^ {133}\).

A novel way to modulate the growth of the anterior vertebral epiphyses through thoracoscopic stapling of the convex apical vertebrae for scoliosis in skeletally immature patients is under investigation after promising preliminary work in animals (figure 5)\(^ {134,135}\). The theoretical advantages are preservation of growth, and motion and function of the spine caused by non-fusion. However, apart from very limited experience, the need to refine the staple design, and other essential improvements, the most important questions relate to indications. So, who is the ideal candidate? In the absence of objective measures to reliably predict curve progression, one might be unnecessarily treating patients with curves that will never progress.

**Figure 3:** Radiographs of a teenager with progressive right thoracic adolescent idiopathic scoliosis treated by posterior segmental pedicle screws

(A) Preoperative standing lateral; (B) preoperative standing posterior-anterior (PA); (C) postoperative standing lateral; and (D) postoperative standing PA. Reproduced with permission of Dr Peter Newton, San Diego, CA, USA.
Treatment outcome versus natural history

Typically, three types of treatment outcomes are reported: (1) radiographical (coronal and sagittal radiographic measurements); (2) clinical (complications, pulmonary function and symptoms, mobility, muscle strength, back function); and (3) self-reported outcomes (general health and health concerns specific to scoliosis and its treatment).

Several workers have reported that curve size or curve correction do not correlate with quality of life. Moreover, several retrospective long-term studies have established that patients treated surgically or with a brace have nearly the same quality of life, both mentally and physically, after treatment as do healthy controls or national norms. However, this evidence is not conclusive.

Some retrospective investigations have shown that AIS causes disturbances in body image and in other indicators of mental health and adjustment. Each investigation used different instruments to measure psychological indices and adherence. Despite these shortcomings, bracing clearly causes some psychological stress to the patient, at least at the initiation of treatment and possibly in the long term. Tones and colleagues reported a systematic review of publications about health-related quality of life and psychosocial issues in AIS. They concluded that adolescents can have poorer psychosocial functioning, body image, and health-related quality of life than have their peers, but that adults generally do not have psychological distress. However, adults are at risk of disability and concerns about body image. Stress as a result of treatment needs to be considered in the decision-to-treat equation, and the benefits of watchful waiting, bracing, or surgery should outweigh the risk of adverse psychological sequelae.

A long-term study from Sweden corroborates studies of the natural history of back pain and function. compared patients who previously wore a brace and those who had undergone surgery each with a set of age-matched controls. They reported little evidence that either patient group was greatly impaired relative to their peers when the 36-item short form health survey and Oswestry low back pain disability questionnaire was used. The mean curve size in both groups was greater than 30°. Another follow-up report of more than 20 years showed no difference in quality of life, including back pain and function, between AIS patients who had undergone surgery and those who remained untreated, as inferred from several instruments, such as the Oswestry, Roland-Morris, and the EuroQol-5D. This finding is in contrast with that of Mayo and colleagues’ survey. Their sample consisted of patients who were untreated, had a brace, or had undergone surgery. The frequency of back pain did not differ by Cobb angle, and these workers concluded that...
the deformity could induce back pain and that surgery did not induce additional back pain beyond that caused by scoliosis itself. However, on the basis of their answers in the Oswestry questionnaire, people with severe curves or those who had been surgically treated felt restricted in their ability to manage pain, lift, and socialise. Dickson and colleagues compared outcomes of symptomatic, untreated patients who were offered arthrodesis and fusion as adults with those who had arthrodesis and fusion. At an average of 5 years after surgery, 35% had substantial or severe pain compared with 70% of the patients who declined surgery and 7% of a non-scoliotic control group. They concluded that operative intervention, although associated with improvements in quality of life, self-image, pain, and disability, did not alleviate all symptoms or restore function to that of non-scoliotic individuals.

Conclusion

Although many controversies and unanswered questions surround AIS, the most difficult one is the absence of information about its aetiopathogenesis. We do not know if AIS is one entity or a manifestation of several causes. We are uncertain of all the factors that lead to progression and secondary manifestations of spinal deformity. The identification of aetiopathogenetic factors will enable improved prediction of progression and could aid in the development of more specific treatments.

Because of this absence of fundamental information, all treatment efforts are aimed at prevention or correction of the primary manifestation of the disorder—ie, spinal deformity. Although bracing has been regarded as the standard of care for patients at a high risk of progression (curves between 25° and 40° in skeletally immature patients), it has never been subjected to rigorous assessment of either its efficacy or effectiveness. Both the North American and Netherlands’ clinical trials are standard of care for patients at a high risk of progression—ie, spinal deformity. Although bracing has been regarded as the standard of care for patients at a high risk of progression (curves between 25° and 40° in skeletally immature patients)—it has never been subjected to rigorous assessment of either its efficacy or effectiveness. Both the North American and Netherlands’ clinical trials are standard of care for patients at a high risk of progression—ie, spinal deformity. Although bracing has been regarded as the standard of care for patients at a high risk of progression (curves between 25° and 40° in skeletally immature patients). We declare that we have no conflict of interest.

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