Review Article

Current concepts and controversies on adolescent idiopathic scoliosis: Part I

Alok Sud, Athanasios I Tsirikos[1]

Abstract

Adolescent idiopathic scoliosis is the most common spinal deformity encountered by General Orthopaedic Surgeons. Etiology remains unclear and current research focuses on genetic factors that may influence scoliosis development and risk of progression. Delayed diagnosis can result in severe deformities which affect the coronal and sagittal planes, as well as the rib cage, waistline symmetry, and shoulder balance. Patient's dissatisfaction in terms of physical appearance and mechanical back pain, as well as the risk for curve deterioration are usually the reasons for treatment. Conservative management involves mainly bracing with the aim to stop or slow down scoliosis progression during growth and if possible prevent the need for surgical treatment. This is mainly indicated in young compliant patients with a large amount of remaining growth and progressive curvatures. Scoliosis correction is indicated for severe or progressive curves which produce significant cosmetic deformity, muscular pain, and patient discontent. Posterior spinal arthrodesis with Harrington instrumentation and bone grafting was the first attempt to correct the coronal deformity and replace *in situ* fusion. This was associated with high pseudarthrosis rates, need for postoperative immobilization, and flattening of sagittal spinal contour. Segmental correction techniques were introduced along with the Luque rods, Harri-Luque, and Wisconsin systems. Correction in both coronal and sagittal planes was not satisfactory and high rates of nonunion persisted until Cotrel and Dubousset introduced the concept of global spinal derotation. Development of pedicle screws provided a powerful tool to correct three-dimensional vertebral deformity and opened a new era in the treatment of scoliosis.

Key words: Adolescent idiopathic scoliosis, natural history, clinical examination, radiological assessment, treatment

Introduction

Scoliosis is defined as a lateral curvature of the spine on the frontal plane which is greater than 10° when measured on a standing radiograph by the Cobb method.[1] The deformity also includes lateral inter-vertebral tilting and rotation of the vertebral bodies across the apex toward the convexity of the curve in the axial plane. Idiopathic scoliosis is the most common type accounting for up to 80% of structural coronal deformities. This can be classified into three types according to the age of onset: infantile (0 to 3 years), juvenile (4 to 9 years), and adolescent (10 years to skeletal maturity).[2] Adolescent is the most frequent type of idiopathic scoliosis and is the spinal deformity which any orthopedic surgeon is likely to encounter in his practice. Knowledge of the principles of presentation, evolution, and treatment is essential in order to provide an effective patient consultation and select candidates who can benefit from existing and developing treatment strategies.

The purpose of our review is to present the current knowledge and debates on the etiology, natural history, and treatment of adolescent idiopathic scoliosis (AIS) with emphasis on the evolution of surgical management and its controversies.

Etiology and pathogenesis

A better understanding of etiology is required to determine accurate prognosis and develop effective treatment protocols. Despite considerable research advances in the recent past, there is no agreement in regard to the proposed theories for the etio-pathogenesis of AIS. Etiological factors include genetics, relative anterior spinal over-growth (RASO), biomechanical growth modulation, dorsal shear forces and axial rotation instability, uncoupled spinal neuro-osseous growth, postural abnormalities and hind brain dysfunction, motor control problem, neuro-developmental concept,

Commonwealth Travelling Spinal Fellow, [1]Consultant Orthopaedic and Spine Surgeon, Honorary Clinical Senior Lecturer, Scottish National Spine Deformity Center, Royal Hospital for Sick Children, Sciennes Road, Edinburgh, EH9 1LF, UK

Address for correspondence: Mr. Athanasios I. Tsirikos,
Honorary Clinical Senior Lecturer-University of Edinburgh, Scottish National Spine Deformity Center, Sciennes Road, Edinburgh, EH9 1LF, UK.
E-mail: atsirikos@hotmail.com

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systemic melatonin- signaling pathway deficiency, and systemic platelet calmodulin dysfunction.[3-14] Established biologic risk factors include growth velocity and remaining spinal growth. It appears that AIS has a multifactorial etiology with skeletal growth contributing to its presentation and curve progression.

The role of genetic factors is well documented; however, the mode of inheritance remains unclear.[15] Several studies indicate AIS to be a single gene disorder.[6] The genetics of idiopathic scoliosis seem to be similar in all age groups. The incidence rate is 11% among first-, 2.4% among second-, and 1.4% among third-degree relatives.[16] Concordance rates among monozygotic and bi-zygotic twins have been reported to range from 73% to 92% and 36% to 63%, respectively.[17,18] Gao *et al*.[19] suggest evidence of linkage and association with the 8q12 loci. Through further investigations they discovered the first gene (CHD7) associated with susceptibility to AIS.[19] Gurnett *et al*.[20] published a report of a single multi-generational family in which AIS and pectus excavatum segregated as an autosomal dominant condition. A clear mode of inheritance has not been confirmed; however, autosomal dominant,[3] dominant major gene bi-allelle model,[4] X-linked[5] or multifactorial[6] inheritance patterns have been reported. Recent investigations have disclosed a 30-marker genetic panel which can predict AIS curve progression early in the course of the disease.[21]

Relative anterior spinal overgrowth (RASO) resulting in lordosis due to relative growth failure of posterior elements has been recognized as one of the prime initiating factors in the development of AIS by several authors.[7,8] Fixed lordotic area and concave peri-apical rib over-growth pose a biomechanical risk for curve progression.[9] This may occur as part of general skeletal over-growth or due to uncoupled endochondral-membranous bone formation. The differential growth thus created results in lateral shear forces driving the apical vertebrae out of the midline resulting in the typical vertebral and rib deformities.

Deacon and Dickson[10] give primacy to the lordotic segment which results in vertebral rotation and the development of a scoliosis. Castelein *et al*.[11] hypothesize upright posture resulting in backward inclination of the vertebrae in the sagittal plane and an increase in dorsal shear forces. The axial plane instability thus introduced enhances asymmetric loading of the posterior part of the vertebrae leading to asymmetric growth in the pedicles, vertebral bodies, and arches involving the neuro-central synchondrosis in accordance with the Hueter-Volkmann effect.

Abnormalities have been detected in the skeletal, nerve, and endocrine systems and in the connective tissue. It is not clear whether these are primary or secondary. MRI studies have revealed neuro-anatomical abnormalities in about 20% of the younger children with putative idiopathic scoliosis and in patients with curves > 20°.[12] AIS has been speculated to occur as a result of dis-proportionate vertebral-neural growth (asynchronous neuro-osseous growth concept).[13] The initiation and progression of the deformity could result from the vertebral column overgrowth through a lordo-scoliotic adaptation of the spine to the sub-clinical tether of a relatively shorter spinal cord.[22] Neuromuscular conditions producing an asymmetry of the transverse-spinalis muscle, abnormality in visual, vestibular, proprioceptive and postural control have also been implicated in AIS.[9] In addition, tonsillar ectopia with abnormal somato-sensory evoked potentials, larger foramen magnum, and left-right brain asymmetries point to neural origin of AIS.[14] These findings explain a poor performance in combined visual and proprioception, as well as spatial orientation tests and impaired postural balance in AIS patients.[14]

The higher prevalence of AIS observed in females may be due to the fact that girls attain adolescent skeletal growth spurt in relative postural immaturity, compared to boys who go through their pubertal rapid growth at later age when their posture is more mature.[9] Spine slenderness and ectomorphy are other risk factors in girls.[9] In addition, XbaI site polymorphism of the estrogen receptor gene makes girls more susceptible to AIS.[23]

Development of scoliosis has been linked to bipedal gait and the deficiency of melatonin after removal of the pineal gland in chicken.[24] Moreau *et al*.[25] reported melatonin-signaling transduction to be impaired in osteoblast, myoblasts, and lymphocytes caused by inactivation of Gi proteins. Promotor polymorphisms of the gene for melatonin receptor 1B are associated with the occurrence of AIS but not directly with curve severity.[26] None of these factors has been specifically proven in humans as the levels of melatonin are normal in most patients with AIS.[27] Calmodulin, a calcium-binding receptor protein regulates the contractile properties of the muscle and platelet function. Lowe *et al*.[28] suggested that calmodulin levels are higher in patients with curves >30°. A small scoliotic curve transmits axial loads directly to the vertebral body growth plates in which micro-insults are created. This causes dilatation of the juxta-physeal vessels which in turn activate platelet-calmodulin and subsequent growth factor release. The growth factors then enhance the effects of RASO in already compromised vertebral end plates.

Prevalence and natural history

The prevalence of AIS has been reported to range between 0.9% and 12% in the general population with minimum

10% of patients requiring treatment.[29] Severe curves needing active intervention are 7.2 times more common in girls than boys.[29] For curves > 30° the female to male ratio approaches 10:1.[30] Males generally present at a later age (12-15 years) than females (11-14 years) and tend to have more rigid curves.[30] The risk of curve progression is greater in boys than girls.[31] Scoliosis deterioration also depends on curve size and pattern, as well as the amount of remaining spinal growth. Skeletal maturity is usually assessed by the Risser sign (radiographic measurement based on the growth plate along the iliac apophysis), closure of triradiate cartilage, Tanner's index, and menarchal status.[32-34] Patients with complete capping of the iliac apophysis, menses for at least 1 year, and no increase in body height over 6 months are considered skeletally mature.[35] Scoliosis on a skeletally immature patient is more likely to deteriorate than on a patient who has already been through adolescent growth spurt.

Curves of up to 30° are likely to stabilize in skeletally mature patients in contrast to younger patients in whom remaining growth increases the risk of progression.[36] In addition, scoliosis greater than 50° (either thoracic or lumbar) at completion of growth may progress into adult life.[37] Of all curve patterns, double thoracic and lumbar and single thoracic curves are more likely to deteriorate.[36] Thoracic curves may progress by 1°/year after spinal growth; however, an untreated lumbar curve tends to produce more symptoms into adulthood.[37-39] Edgar and Mehta[39] reported that in most untreated double scoliotic deformities, the thoracic curve increased less than the lumbar after skeletal maturity; the thoracic component of a double curvature also progressed less than single thoracic curves.

Curve deterioration can cause pain, reduced pulmonary function, increased mortality, and a negative impact on quality of life. The incidence of chronic back pain is higher in untreated scoliotic patients; however, the ability to perform activities of daily living and work are similar to that of the normal population.[38] Dickson *et al*.[40] compared a group of adult patients with idiopathic scoliosis who were surgically treated with another who declined treatment. At latest followup, the treated patients had significantly reduced pain and fatigue and an increased ability to perform physical, functional, and positional tasks.[40] Significant backache in untreated scoliosis may occur due to progressive curve deterioration and disc degeneration producing translatory intervertebral shift and retrolisthesis.[41] Lower pulmonary function in the form of restrictive lung disease may be present in patients with severe thoracic curves due to significant hypokypkosis.[42,43] Thoracic hypokyphosis may also predispose to cervical kyphosis as an attempt to achieve global sagittal balance and this can accelerate the development of cervical spondylosis.[44] The mortality rates for patients with untreated AIS are comparable to the normal population except in severe thoracic curves (>100°) which increase the risk for cor pulmonale or right ventricular failure.[41] Reproduction has not been affected by untreated scoliosis.[45]

Clinical and radiological assessment

The diagnosis of AIS remains that of exclusion and other likely causes of scoliosis including spinal infections and neoplasms, neuromuscular, and syndromic conditions, as well as congenital anomalies of the vertebral column or the neural axis, should be excluded. The scoliosis can be assessed with the patient in the erect position or with the Adam's test which shows the sharp lateral curve and associated convex rib/chest wall prominence when the patient bends forwards to 90° till the spine becomes horizontal. The Adam's forward test can differentiate a structural deformity from postural or compensatory curves which tend to spontaneously correct. In the presence of neurological signs and symptoms or skin stigmata, an MRI of the spine must be obtained to rule out intraspinal abnormalities. In severe thoracic curves, pulmonary function must be assessed with spirometry and sleep studies preoperatively, especially when anterior release or thoracoplasty is anticipated.

Routine radiological analysis includes standing long cassette (36 inches) postero-anterior and lateral radiographs of the spine including the pelvis to assess the *Cobb angle* (measured between the superior surface of the proximal and inferior surface of the distal end vertebra maximally tilted into the curve),[1] *skeletal maturity* (Risser Grade and triradiate cartilage), and *curve pattern* (based on the region of the spine where the curve apex is located). A postero-anterior radiograph significantly reduces radiation exposure to the breasts and thyroid tissue. In addition, the stable and neutral vertebrae are identified which assists in selection of fusion levels if surgery is planned. *Stable vertebra* refers to the inferior vertebra which is bisected (or most closely bisected) by the central sacral vertical line (CSVL) on standing radiograph.[46] CSVL is a line drawn upward from the center of the sacrum perpendicular to the line joining the iliac crests. *Neutral vertebra* is one which is minimally rotated according to the Nash-Moe criteria in the coronal plane.[47] Potter *et al*.[48] found good to excellent intraobserver but poor interobserver reliability on radiographic determination of end, neutral, and stable vertebra. Difficulties in identifying these vertebral levels represent a potential obstacle to reproducible patient-specific fusion level determination and consequently to the optimization and uniformity of patient care. Relative ratios between the thoracic and lumbar curves with regard to Cobb angle, apical vertebral rotation (AVR), apical vertebral translation (AVT), and trunk shift (TS) are also determined.[49]

AVT designates the distance between the midline and the apex of the curve, AVR refers to maximum rotation across the apical vertebra, and TS defines the distance between the midline and the C7/T1 level.

Flexibility radiographs are obtained to determine the structural nature of the curve. The methods used include supine maximum side-bending, fulcrum bending, push-prone or traction radiographs taken with or without general anesthesia. Disc space neutralization defined as opening of the disc space across both sides on bending radiographs helps decide the distal extent of the fusion.[46] These techniques are useful during surgical planning. Hamzaoglu *et al*.[50] carried out a prospective comparative evaluation of the commonly accepted radiological techniques to assess curve flexibility (supine lateral bending and traction, fulcrum bending radiographs) and compared the results to those obtained by supine traction radiographs under general anesthesia. Flexibility obtained by traction radiographs under general anesthesia was found closer to the amount of surgical correction for curves >65°.[50] Corrective predictability of traction radiographs was also found to be superior to side bending radiographs for main thoracic and proximal thoracic curves.[51]

Classification systems

Classification systems for scoliosis should be comprehensive and reproducible including both the coronal and sagittal alignment with good inter/intraobserver reproducibility. King *et al*.[46] described a classification for AIS based on the coronal characteristics of the deformity [Table 1]. This classification was developed for primary thoracic curves, did not incorporate sagittal spinal balance, and was found to have a low inter/intraobserver reliability.[52,53] The most common curve pattern in AIS includes a thoracic curve (usually right) with or without a compensatory thoracolumbar or lumbar curve (usually left). A thoracic curve has the apex at T10 or above and a thoracolumbar/lumbar has the apex at T11 or below. The patient with a thoracic scoliosis develops chest wall asymmetry with rib prominence adjacent to the convexity of the curve, as well as elevation of the ipsilateral shoulder. Severe thoracic curves produce thoracic translocation and listing of the trunk toward the convexity of the curve and a subsequent waistline asymmetry with prominence of the contralateral iliac crest. King type III and IV describe single thoracic curves. King type II includes a primary thoracic and secondary lumbar scoliosis, while King type I defines a greater thoracolumbar/lumbar deformity with a secondary thoracic curve. King type V describes double thoracic curves (usually left upper-right lower); the structural nature of the proximal curve is clinically indicated by shoulder asymmetry (left shoulder usually higher than the right). Double major curves are not included within this classification and have an equal thoracic and lumbar deformity component. Double thoracic and thoracolumbar/lumbar curves produce often a lesser cosmetic deformity as one curvature tends to balance the other and are frequently diagnosed late.

Table 1: King classification for adolescent idiopathic scoliosis

Type I	S-shaped curves in which both thoracic and lumbar curves cross mid-line. Lumbar curve is larger. The thoracic curve is more flexible than the lumbar on supine-bending films
Type II	S-shaped curves in which both thoracic and lumbar curves cross mid-line. Thoracic curve is equal to or larger than the lumbar curve. The thoracic curve is more flexible than lumbar curve on supine-bending films
Type III	Single Thoracic curve in which the lower level does not cross the mid line
Type IV	Single long thoracic curve in which L5 is centered over the sacrum but L4 tilts into the long thoracic curve
Type V	A double thoracic curve in which T1 is tilted into the convexity of the upper curve (the upper curve is structural on supine-bending film)

AIS = Adolescent idiopathic scoliosis

The global coronal and sagittal balance, as well as the concept of trunk decompensation was not included in King's classification. Lenke *et al*.[49] developed a more detailed system to incorporate not only coronal curves but also sagittal balance across the thoracic and lumbar segments with the aim to predict fusion levels more accurately, thereby reducing the risk of postoperative decompensation [Table 2]. Curves are classified according to location (proximal thoracic, main thoracic, and thoracolumbar/lumbar), curve size and flexibility, as well as coronal lumbar and sagittal thoracic modifier. Types 1 and 2 refer to structural thoracic scoliosis; type 5 refers to structural thoracolumbar/lumbar scoliosis; types 3, 4, 6 include structural thoracic and thoracolumbar/lumbar curves. The lumbar spine modifier based on the relationship of the CSVL to the apex of the lumbar curve and the sagittal thoracic spine modifier based on kyphosis measurement between T5 and T12 on a lateral standing radiograph have been added to better predict global coronal and sagittal alignment. A curve is designated by a triad created on the basis of curve type (1 to 6), lumbar spine modifier (A, B, C), and thoracic sagittal modifier (-, N, +). For example, "1A-" describes a single thoracic structural scoliosis without or with minimal lumbar coronal deformity and hypokyphosis. This classification has been reported to have a good inter/intraobserver reliability.[49]

Conservative treatment

Participation in sports and trunk exercises is beneficial for postural balance, as well as the overall well being of the patient; however, these measures do not change the natural history of scoliosis.[54] There is also no sufficient data to support that chiropractic or osteopathic treatments, acupuncture, or electrical stimulation have any therapeutic effect on scoliosis. The most widely used method of

nonsurgical treatment with documented results in AIS remains bracing.

Brace types

Brace treatment is based on the principle of applying external corrective forces across the curve in order to preserve and modulate growth of the spine with the aim to arrest deformity progression, produce an acceptable sagittal and coronal contour, and delay or avoid surgical treatment.[55,56] There are two basic types of braces used in AIS: a) cervico-thoraco-lumbo-sacral orthoses (CTLSO), and b) thoraco-lumbo-sacral orthoses (TLSO). A CTLSO (such as Milwaukee brace) is used for thoracic scoliosis with the apex above T8. A TLSO (such as Boston, Wilmington, Lyon, Cheneau, Rigo-Cheneau, Malaga, SPoRT) is used for thoracic scoliosis with the apex at or below T8, for thoracolumbar/lumbar scoliosis and for double thoracic and lumbar curves.[56] A third type of over-corrective brace was developed for night wear (Charleston brace) in order to enhance patient compliance. As compliance with CTLSO is generally very poor, we use mainly Boston braces which are custom-molded using the Risser frame to allow reduction of lumbar lordosis and a tight fit [Figure 1]. The brace is then fabricated using the plaster mold and standing radiograph as template and pads are added against the apex of the scoliosis to provide correction.

Brace biomechanics

All spinal braces have a basic design comprising of a pelvic mold over the iliac crests extending anteriorly to the pubis symphysis, which is the lowest part of the 3-point fixation applying bending moments across the curve. A gluteal extension to reduce lumbar lordosis and a trochanteric extension to correct trunk imbalance may be added. Uprights and lumbar/thoracic pads exerting transverse corrective forces across the scoliosis apex are attached to the basic mold. The thoracic pad is placed over the postero-lateral region of the rib cage. If placed posteriorly, it encourages hypokyphosis and when placed direct laterally it

Table 2: Lenke classification for adolescent idiopathic scoliosis

Proximal thoracic apex at T3, T4 or T5	Main thoracic apex between T6 and the disc between T11/T12	Thoracolumbar/lumbar-apex between T12 and L1 and apex between L1-L2 disc and L4, respectively	Curve type	Lumbar spine modifier	Central sacral vertical line to lumbar apex
Nonstructural	Structural (major)	Nonstructural	Main thoracic (MT)	A	Central sacral vertical line between pedicles
Structural	Structural (major)	Nonstructural	Double thoracic (DT)	B	Central sacral vertical line touches apical body/bodies
Nonstructural	Structural (major)	Structural	Double major (DM)	C	Central sacral vertical line completely medial
Structural	Structural (major)	Structural	Triple major (TM)	Thoracic sagittal modifier	Measurement (T5-T12)
Nonstructural	Nonstructural	Structural (major)	Thoracolumbar/lumbar (TL/L)	--(Hypo)	<10°
Nonstructural	Structural	Structural (major)	Thoracolumbar/lumbar-main thoracic (TL/L-T)	N (Normal)	10°–20°
				+(Hyper)	>40°

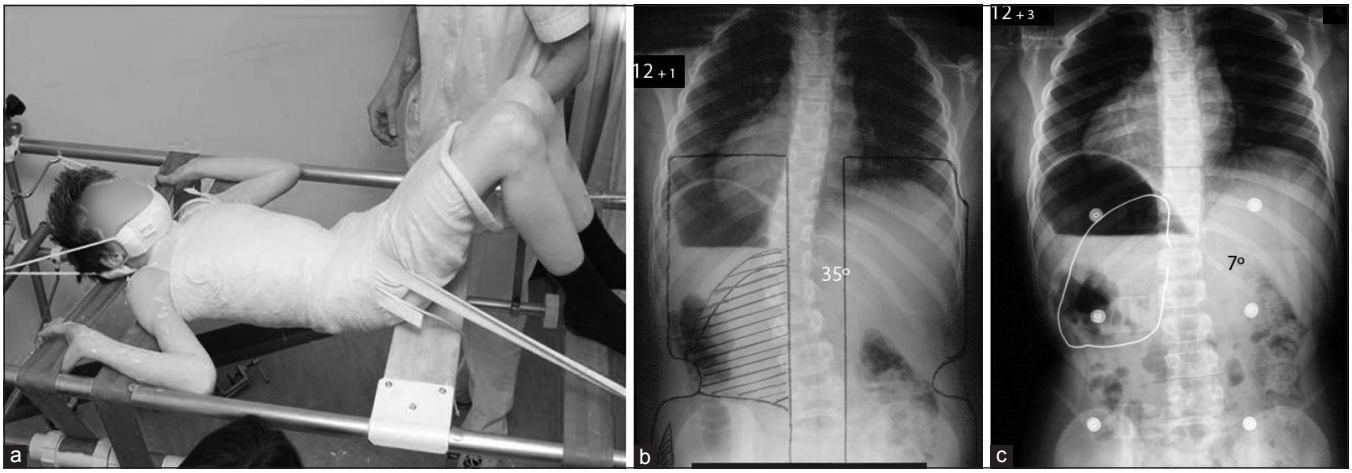

Figure 1: (a) Clinical photograph of a male patient showing a thoracolumbar scoliosis undergoing plaster molding for a Boston brace using the Risser frame and longitudinal traction (b) initial posteroanterior radiograph of the spine showing the template for positioning of the corrective pad. (c) repeat radiograph of the spine (at 6 weeks) which shows adequate location of the apical pad (wire marker) and good support of the spine with the scoliosis corrected from 35 to 7°

fails to produce derotation. For thoracic curves with a higher apex producing shoulder asymmetry a trapezius strap may be added to depress the elevated shoulder.

Brace indications

Brace treatment is designed to prevent curve progression during the growing years and is not indicated in patients who are already skeletally mature. It is generally recommended for curves 25-40° in skeletally immature patients with Risser sign 0-1. Growing children with curves less than 25° and documented progression of at least 5° between two consecutive assessments 6 months apart are also candidates for bracing. Curves above 40° will usually not benefit from bracing.[57] Bracing could be attempted in younger patients with significant remaining growth and curves above 40°as a temporary measure to slow down curve deterioration and preserve growth while delaying surgical treatment for an older age. On the whole, bracing is able to alter curve progression in smaller curves of 20-35°.[58]

Bracing protocol

Braces are required to be worn full time, to be effective. Wiley *et al*.[59] reported significant curve improvement in patients with full time (18-23 hours/day) when compared to part-time brace wear (12-18 hours/day). The brace may be taken off for 2-4 hours/day to allow for personal hygiene, as well as participation in recreational and sports activities. After first application, the brace is progressively adjusted to a tight fit over a period of a few weeks. A standing X-ray is taken to assess positioning of the corrective pads 4-6 weeks after continuous brace wear. Thereafter, the patient attends the clinic every 6 months for brace check and a repeat radiograph. Complications related to brace treatment include pain, skin irritation and pressure sore, renal and pulmonary dysfunction, nerve irritation in the axilla, meralgia paraesthetica, and psychosocial effects.[60,61] The brace is continued until skeletal maturity when it is gradually weaned off over a period of a few weeks.[57]

Outcome of bracing

Previous studies report the effectiveness of Milwaukee and Boston braces in patients with AIS but this is generally limited to smaller curves.[57,62] Rowe *et al*.[63] showed that bracing altered the natural history of scoliosis and that full time was significantly more effective than part-time bracing. Nachemson and Peterson[58] reported that Boston bracing was effective in girls with 25-35° curves and survivorship analysis demonstrated that the success rates of bracing and observation alone were 74% and 34%, respectively. More recently, Danielson *et al*.[64] completed a 16-year followup of Swedish patients who were included in the study by Nachemson and Peterson.[58] The authors concluded that the group of unbraced patients had a mean scoliosis progression of 6°; the group of braced patients demonstrated no curve progression. An earlier study confirmed three-fold decline in requirement for surgical treatment for AIS following early bracing after school screening.[65]

In contrast, a meta-analysis by Dolan and Weinstein[66] showed no difference in surgical rates between braced (23%) and unbraced patients (22%). Janicki *et al*.[67] compared the effectiveness of thoracolumbosacral and providence orthosis in AIS; 79% of patients in the group and 60% in the latter group required scoliosis surgery. A Cochrane systematic review reported very low quality evidence in favor of brace treatment.[55] Preoperative bracing had a negative effect on the outcome of spinal fusion possibly due to the fact that braced patients had more pain and lower activity levels before surgery. Lower satisfaction rates and lower SRS-30 outcome scores were recorded at 2 years after scoliosis surgery in braced patients.[68] Bracing may limit scoliosis progression but at the same time can produce a stiffer spine which in turn reduces the ability for deformity correction by surgical treatment. Possible negative effects of bracing include a temporary decrease in urinary sodium excretion and the obvious psychological effects of wearing a brace on teenagers.[69,70] In addition, Boston brace has been shown to reduce lung volume and pulmonary compliance compared to pre-brace levels.[71] Brace wear has been found to have no effect on bone mineral densitometry.[72]

Highest risk of progression during bracing has been observed in single thoracic curves followed by double thoracic/lumbar and thoracolumbar curves.[66] Brace treatment is less useful in overweight patients, patients with high thoracic curves (apex above T8) or a lordotic thoracic spine, and those who have major medical co-morbidities. Male patients are often less compliant with bracing. Patients who have passed their peak height velocity, those who are within 1 year of skeletal maturity or 1 year postmenarche are unlikely to have an alteration in the natural history of scoliosis by bracing.[73]

The best indication for brace treatment in AIS is probably young patients with progressive curves in whom delaying surgical treatment will preserve spinal and chest growth. A more successful brace outcome can be expected in rapidly growing children with curves between 25 and 45° and Risser grade 0-1. Patients with Risser grade 2-3 and curves of 30-45° may have a lesser effect from brace treatment.[73] Ogilvie *et al*.[74] reported a 30 marker predictive genetic panel which may allow evidence based decisions on the effectiveness of braces and appropriate patient selection.

To allow comparability between studies, the Scoliosis Research Society Committee on Bracing and Nonoperative Management has suggested that all research on bracing

for AIS should meet the standard criteria for inclusion and outcome reporting. Inclusion criteria include age > 10 years, Risser sign 0-2, female patients who are pre-menarchal or < 1 year postmenarche and curve size 20-39°.[75] The committee recommends followup for at least 2 years after skeletal maturity.[75] Accordingly, assessment of brace effectiveness may be reported as: 1) percentage of patients who progressed < or equal to 5°; 2) percentage of patients who progressed > or equal to 6° at maturity; 3) percentage of patients who progressed beyond 45° at maturity; 4) percentage of patients for whom surgery was recommended and undertaken. These criteria set the benchmark for present and future studies on brace effectiveness in patients with AIS.

Surgical treatment

Indications

Previous natural history studies have demonstrated scoliosis progression into adult life for curves of 50° and above at completion of growth;[37-39] therefore, surgical treatment is generally indicated if the curve reaches 50° and it produces a significant deformity with or without associated pain. Curves less than 50° in skeletally immature patients, progressive double curves, thoracolumbar/lumbar curves producing significant trunk imbalance, as well as curves not controlled by bracing are also indications for surgery.

The aim of surgical treatment is to correct the coronal, rotational and chest wall deformity, restore global sagittal balance, and achieve a solid fusion across the instrumented levels. This should be associated with minimal morbidity and limited complications in order to allow early patient mobilization and return to normal function. Preservation of motion segments, especially across the lumbar spine, and improvement in pulmonary function are additional goals of surgery.

Posterior spinal fusion

A solid spinal fusion is required to prevent loss of correction and recurrence of deformity. A good fusion mass cannot be achieved without an adequate surgical technique regardless of type of instrumentation used. Many modifications have been developed since the initial technique of posterior spinal arthrodesis was introduced by Hibbs[76] and Albee.[77] We perform a meticulous sub-periosteal exposure of the spine out to the tips of the transverse processes followed by extensive decortication of the posterior elements in order to create a bleeding cancellous bone bed which will allow consolidation of the bone grafts. The spinous processes are divided into half and then removed at their base to be used for bone graft. In the thoracic region, each inferior facet is excised bilaterally, the cartilage and sub-chondral bone of the exposed superior facet is removed, and a piece of cancellous bone graft is placed to achieve an interfacetal fusion.[78] In the lumbar region, the capsule and opposing cartilage is removed from the facets to mobilize the spine and create space for bone grafting.

Bone grafting

Three types of bone graft have been used in scoliosis surgery: a) autologous bone, b) allograft bone, and c) bone substitutes. Autologous bone remains the "gold standard" as it has similar biological properties to the spine and is osteoinductive/osteogenic and osteoconductive.[79] The source of autograft depending upon the approach includes posterior iliac crest, ribs, and spinous processes. Potential disadvantages and complications include greater blood loss, increased localized postoperative pain which can persist for a few months, risk of nerve and vascular injury (superior gluteal bundle and cluneal nerves), fracture of the pelvis, and the complications associated with thoracotomy or thoracoplasty (pleural tears causing pneumo-thorax, hemothorax, and respiratory compromise).[79] In our experience, iliac crest bone grafting is a safe technique which provides sufficient bone to achieve fusion across the instrumented scoliosis levels.

The use of allograft bone has reduced donor site morbidity; however, this is only osteoconductive acting as a scaffold for the spine to form new bone.[80] Therefore, spinal fusion may take longer compared to the use of autograft. Previous studies suggest that both autograft and allograft are equally effective in achieving spinal fusion in pediatric patients.[79] A recent report by Betz *et al*.[81] has proposed that spinal fusion in scoliosis surgery can be achieved following facetectomies and multisegmental instrumentation fixation even without bone grafting. These results need to be confirmed on long term followup to exclude late presentation pseudoarthrosis.

Synthetic bone graft including ceramics (hydroxyapatite, beta-tricalcium phosphate, mono-calcium hydroxylate) have been used as an osteoconductive scaffold to reduce the need for harvesting autologous bone graft. These increase significantly the cost of surgery, are nonbiological, have slow resorption and doubtful consolidation to the host bone. Human-recombinant bone morphogenic proteins have also been used in human clinical trials mainly in adult patients successfully but long term results are still pending.[82]

Posterior spinal instrumentation systems

The role of instrumentation is to be used as a scaffold to maintain the spine in the corrected position while the fusion occurs. It consists of vertebral anchors (hooks, wires, and screws), longitudinal (rods-smooth, threaded and knurled) and transverse members (cross connectors), as well as the attachments that connect the anchors to the rods. Correction techniques have changed over the years from the initial concave distraction Harrington rods to

multi-fixation systems which apply a variety of techniques including rod rotation, rod translation, cantilever correction, and segmental or en bloc vertebral derotation through segmental anchor points in order to realign the spine.

Harrington instrumentation

Paul Harrington[83] developed a system which was based on a single concave rod which applied distraction forces against a proximal and distal point of hook fixation; this offered limited ability to correct the coronal plane but with no effect on the axial spinal and rib deformity. The system was later coupled with a compressive rod on the convex side of the deformity. Laminar and pedicle hooks with stainless steel distraction and compression rods were used. As the rods were secured to the spine only at two levels (caudal and cephalad vertebrae of the fusion segment), postoperative immobilization was necessary. The system was extensively used because it was inexpensive, easy to apply due to well-defined principles, and offered an easy identification of fusion levels. The surgical procedure was relatively short and included limited blood loss. An extensive surface area was available for bone grafting bilaterally due to very low implant density compared to modern segmental instrumentation.[84] However, the rates of implant failure and pseudarthrosis were high because the system provided a nonsegmental fixation.[85] In addition, pure distraction and compression forces between the points of fixation produced flattening of the spine across the curve and a flat back deformity leading to sagittal decompensation and postoperative pain.[86,87] This often required revision surgery through a pedicle subtraction osteotomy with the aim to recreate lumbar lordosis and correct global sagittal spinal balance. Harrington rods also had high failure rates when extended to the sacrum.

L-rod instrumentation

L-rod instrumentation was developed by Luque[88] who used sublaminar wires for segmental spinal fixation and L-shaped smooth rods to prevent migration at proximal and distal ends of the construct. The primary indication of this system was neuromuscular spinal deformity where the fusion can be extended to the pelvis using the Galveston technique;[89] it has also been widely used in AIS [Figure 2]. There was no need for postoperative immobilization and the system was effective in patients with osteopenia co-existing with renal osteodystrophy, osteogenesis imperfecta, severe osteoporosis or osteomalacia, and neuromuscular conditions where the lamina provided a stronger fixation point.[90] The Luque instrumentation could be used in children as young as 6 years of age when the spinal canal achieves its adult diameter.[91] The most obvious disadvantage is the risk of neurological injury (reported up to 17%) and increased intraoperative blood loss due to invasion of the epidural space.[92] The Luque system did not provide axial stability and was difficult to remove in the case of infection or nonunion increasing significantly the neurological risk.

Harri-Luque system

In order to reduce the complications of the Harrington system, segmental sublaminar wires were used along with the distraction rods to provide multilevel stabilization. Addition of transverse fixation to the distractive forces on the concave side reduced the risk of producing flat back syndrome and thoracic hypokyphosis allowing for a more physiological correction in the sagittal plane.[93] Segmental fixation reduced the incidence of pseudarthrosis and the need for postoperative immobilization. There were several

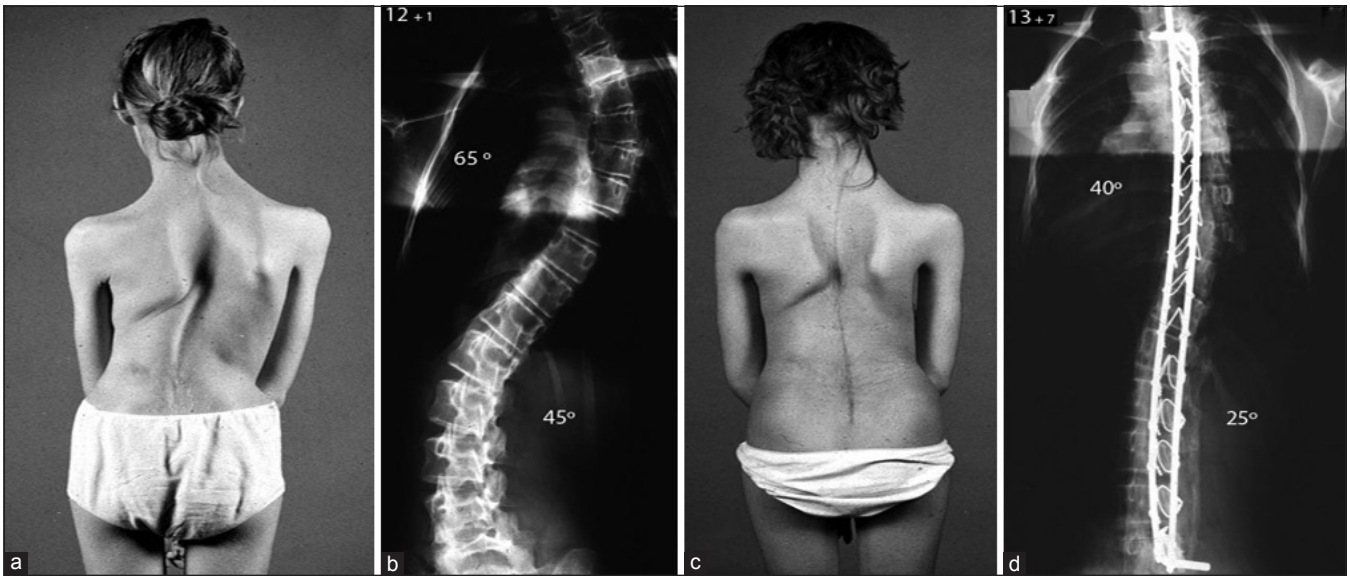

Figure 2: Clinical photograph (a) and spinal radiograph (b) on a female adolescent patient shows a severe right thoracic and left lumbar scoliosis. (c-d) a posterior spinal fusion with the use of Luque segmental wire/rod instrumentation and autologous iliac crest bone graft produced a balanced spine in the coronal plane with level shoulders and symmetrical waist line

theoretical advantages over the Luque system which used only sublaminar wires. While the Luque system relied exclusively on transverse loading, the Harri-Luque instrumentation could apply both axial distraction and transverse correction. The wires were used only on the concave side thereby reducing the risk of neurological injury allowing a large area for bone grafting on the convexity in order to achieve fusion.[94] When compared to the original Harrington rods, the Harri-Luque system increased considerably surgical time and did not eliminate the risk of flat back deformity.

Wisconsin system

The interspinous instrumentation using buttoned wires at the base of the spinous processes was developed to avoid the risk of neurological injury associated with sublaminar wires, while retaining segmental fixation.[95] Flat back syndrome, high rates of pseudarthrosis, need for postoperative immobilization, long levels of fusion to achieve balancing of the spine and limited ability of deformity correction were the main disadvantages of these first generation systems. They had no effect on rib hump reduction and could not address the rotational component of vertebral deformity.

Derotation (hybrid) systems

Instrumentation such as Cotrel-Dubousset (CD) attempted three-dimensional correction of the spine to produce more physiological contours in the coronal and sagittal planes.[96] These systems involved segmental fixation techniques with an array of bony anchors including hooks, wires, and screws and allowed segmental re-alignment of the spine incorporating rod derotation, apical translation, and distraction/compression maneuvers. In a thoracic scoliosis, which is generally producing a hypokyphotic spine, the concave rod is bent into kyphosis, fixed to the spine through the bony anchors, and then rotated clockwise by 90°. This maneuver can convert the scoliosis into kyphosis correcting simultaneously the coronal and sagittal plane deformities (CD maneuver). Similarly, in a thoracolumbar curve, the bent rod is applied on the convex side and then rotated by 90° to produce physiologic lumbar lordosis. Three-dimensional corrections can be achieved through en bloc rotation of the spinal segment held between the anchors, but individual derotation of the vertebra is minimal.[97] Compression and distraction can still be performed across the proximal and distal ends of the construct.

Unlike Harrington rods, hybrid instrumentation could restore sagittal balance but occasionally produce spinal decompensation in the coronal plane. This was due to overcorrection of the major curve (usually thoracic) for which the minor curve (usually lumbar) was unable to balance selection of too short fusion segments and inability to derotate individual vertebrae.[98] Rib deformity correction was limited because the technique could perform global derotation of a segment of the deformed spine but not of the individual vertebrae.[97] Simple rod derotation had a strong postero-medialization effect on the spine that restored coronal and sagittal contours but was unable to correct the rotational deformity necessary to reduce rib prominence.[97] In addition, medial migration of the hooks due to the strong forces applied during rod rotation maneuvers was a potential cause for neurological complications.[99]

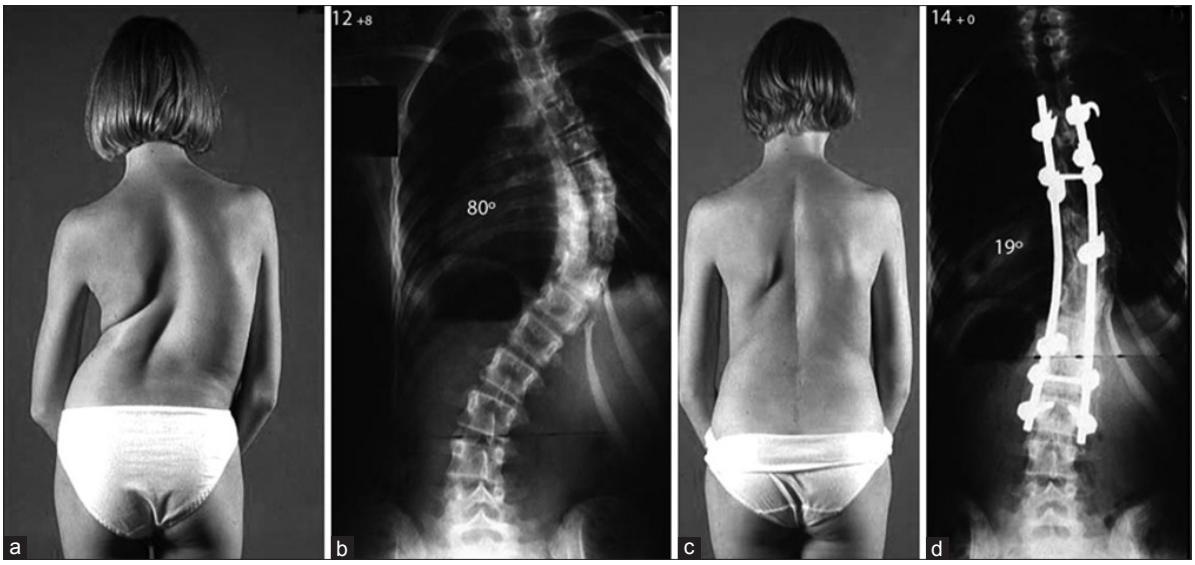

Figure 3: (a,b) Clinical photograph and spinal radiograph of a female adolescent patient show a severe right thoracic scoliosis producing elevation of the right shoulder, prominence of the scapula and ribs adjacent to the convexity of the curve, as well as thoracic translocation to the right and waistline asymmetry with prominence of the left side of the pelvis. (c, d) a posterior spinal fusion with the use of hybrid hook/screw/rod instrumentation and autologous iliac crest bone graft achieved a balanced spine in the coronal plane with level shoulders and symmetrical waist line

Instrumentation like Texas Scottish Rite Hospital, Isola, Colorado, and Universal Spine System were subsequently developed following the same principles with increasing use of more secure bony anchors including distal pedicle screws and these produced improved anatomical and functional results [Figure 3].[100-103] Following a long and steep learning curve pedicle screw fixation could be performed safely in the thoracic spine and the development of pedicle screw-based techniques revolutionalized the surgical treatment of AIS. The clinical safety of these techniques, their advantages, complications, and methods of deformity correction are described in the second part of this paper.

References

1. Cobb JR. Outline for the study of scoliosis. In: American Academy of Orthopaedic Surgeons, ed Instructional Course Lectures. Ann Arbor, MI: JW Edwards, 1948;5:261-75.
2. James JI. Idiopathic Scoliosis: The prognosis, diagnosis and operative indications related to curve patterns and age of onset. J Bone Joint Surg Br1954;36:36-49.
3. Wynne-Davies R. Familial (idiopathic) scoliosis: A family survey. J Bone Joint Surg Br 1968;50:24-30.
4. Axenovich TI, Zaidman AM, Zorkoltseva IV, Tregubova IL, Borodin PM. Segregation analysis of idiopathic scoliosis: Demonstration of a major gene effect. Am J Med Genet 1999;86:389-94.
5. Justice CM, Miller NH, Marosy B, Zhang J, Wilson AF. Familial idiopathic scoliosis: Evidence of an X-linked susceptibility locus. Spine (Phila Pa 1976) 2003;28:589-94.
6. Riseborough EJ, Wynne-Davies R. A genetic survey of idiopathic scoliosis in Boston, Massachusetts. J Bone Joint Surg Am 1973;55:974-82.
7. Guo X, Chau WW, Chan YL, Cheng JC. Relative anterior spinal overgrowth in adolescent idiopathic scoliosis. Results of disproportionate endochondral-membranous bone growth. J Bone Joint Surg Br 2003;85:1026-31.
8. Guo X, Chau WW, Chan YL, Cheng JC, Burwell RG, Dangerfield PH. Relative anterior spinal overgrowth in adolescent idiopathic scoliosis-result of disproportionate endochondral-membranous bone growth? Summary of an electronic focus group debate of the IBSE. Eur Spine J 2005;14:862-73.
9. Burwell RG. Aetilogy of idiopathic scoliosis: Current concepts. Pediatr Rehabil 2003;6:137-70.
10. Deacon P, Dickson RA. Vertebral shape in the median saggital plane in idiopathic thoracic scoliosis. Astudy of true lateral radiographs in 150 patients. Orthopedics 1987;10:893-5.
11. Castelein RM, van Dieen JH, Smit TH. The role of dorsal shear forces in the pathogenesis of adolescent idiopathic scoliosis-a hypothesis. Med Hypotheses 2005;65:501-8.
12. Dobbs MB, Lenke LG, Szymanski DA, Morcuende JA, Weinstein SL, Bridwell KH, *et al*. Prevalence of neural axis abnormalities in patients with infantile idiopathic soliosis. J Bone Joint Surg Am 2002;84:2230-4.
13. Chu WC, Lam WW, Chan YL, Ng BK, Lam TP, Lee KM, *et al*. Relative shortening and functional tethering of spinal cord in adolescent idiopathic scoliosis?: Study with multiplanar reformat magnetic resonance imaging and somatosensory evoked potential. Spine (Phila Pa 1976)2006;31:E19-25.
14. Veldhuizen AG, Wever DJ, Webb PJ. The aetilogy of idiopathic scoliosis: Biomechanical and neuromuscular factors. Eur Spine J 2000;9:178-84.
15. Dickson RA. The etiology and pathogenesis of idiopathic scoliosis. Acta Orthop Belg 1992;58:21-5.
16. Risser JC, Norquist DM, Cockrell BR Jr, Tateiwa M, Hoppenfeld S. The effect of posterior spine fusion on the growing spine. Clin Orthop Relat Res 1966;46:127-39.
17. Carr AJ. Adolescent idiopathic scoliosis in identical twins. J Bone Joint Surg Br 1990;72:1077-83.
18. Kesling LK, Reinker KA. Scoliosis in twins: A meta analysis of literature and report of six cases. Spine (Phila Pa 1976) 1997;22:2009-14.
19. Gao X, Gordon D, Zhang D, Browne R, Helms C, Gillum J, *et al*. CHD7 gene polymorphisms are associated with susceptibility to idiopathic scoliosis. Am J Hum Genet 2007;80:957-65.
20. Gurnett CA, Alaee F, Bowcock A, Kruse L, Lenke LG, Bridwell KH, *et al*. Genetic linkage localizes on adolescent idiopathic scoliosis and pectus excavatum gene to 18q. Spine (Phila Pa 1976) 2009;34:E94-100.
21. Ward K, Nelson LM, Chettiar R, Braun JT, Ogilvie JW. Genetic profile predicts curve progression in adolescent idiopathic scoliosis. 4th Paper. Abstracts of the 43rd Scoliosis Research Society (SRS) Annual meeting and conference; 2008. Available from: http://www.srs.org/professionals/meetings/am08/doc/oral-abstracts.pdf.
22. Porter RW. Can a short spinal cord produce scoliosis? Eur Spine J 2001;10:2-9.
23. Wu J, Qui Y, Zhang L, Sun Q, Qui X, He Y. Association of estrogen receptor gene polymorphisms with susceptibility to adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2006;31:1131-6.
24. Machida M, Dubousset J, Imamura Y, Iwaya T, Yamada T, Kimura J. Role of melatonin deficiency in development of scoliosis in pinealectomised chickens. J Bone Joint Surg Br 1995;77:134-8.
25. Moreau A, Wang DS, Forget S, Azeddine B, Angeloni D, Fraschini F, *et al*. Melatonin signaling dysfunction in adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2004;29:1772-81.
26. Qui XS, Tang NL, Yeung HY, Lee KM, Hung VW, Ng BK, *et al*. Melatonin receptor 1B (MTNR1B) gene polymorphism is associated with the occurrence of adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2007;32:1748-53.
27. Machida M, Dubousset J, Imamura Y, Miyashika Y, Yamada T, Kimura J. Melatonin: A possible role in development of Adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 1996;21:1147-52.
28. Lowe TG, Lawellin D, Smith D, Price C, Haher T, Merola A, *et al*. Platelet calmodulin levels in adolescent idiopathic scoliosis: Do the levels correlate with curve progression and severity. Spine (Phila Pa 1976) 2002;27:768-75.
29. Rogala EJ, Drummond DS, Gurr J. Scoliosis: Incidence and natural history. A prospective epidemiological study. J Bone Joint Surg Am 1978;60:173-6.
30. Suh PB, MacEwan GD. Idiopathic scoliosis in males. A natural history study. Spine (Phila Pa 1976) 1988;13:1091-5.
31. Karol LA. Effectiveness of bracing in male patients with idiopathic scoliosis. Spine (Phila Pa 1976) 2001;26:2001-5.
32. Risser JC. The iliac apophysis: An invaluable sign in the management of scoliosis. Clin Orthop 1958;11:111-9.
33. Sanders JO, Little DG, Richards BS. Prediction of the crankshaft phenomena by peak height velocity. Spine (Phila Pa 1976) 1997;22:1352-6.
34. Tanner J, Whitehouse RH, Takaishi M. Standards from birth

to maturity for height, weight, height velocity, and weight velocity: British children, 1965. Parts 1 and II. Arch Dis Child 1966;41:454-71.
35. Noonan KJ, Weinstein SL, Jacobson WC, Dolan LA. Use of Milwaukee brace for progressive idiopathic scoliosis. J Bone Joint Surg Am 1996;78:557-67.
36. Bunnell WP. The natural history of idiopathic scoliosis before skeletal maturity. Spine (Phila Pa 1976)1986;11:773-6.
37. Lonstein JE, Carlson JM. The prediction of curve progression in untreated idiopathic scoliosis during growth. J Bone Joint Surg Am 1984;66:1060-71.
38. Weinstein SL, Ponseti IV. Curve progression in idiopathic scoliosis: Long term followup. J Bone Joint Surg Am 1983;65:447-55.
39. Edgar MA, Mehta MH. Long term followup of fused and unfused idiopathic scoliosis. J Bone Joint Surg Br 1988;70:712-6.
40. Dickson JH, Mirkovic S, Noble PC, Nalty T, Erwin WD. Results of operative treatment of idiopathic scoliosis in adults. J Bone Joint Surg Am 1995;77:513-23.
41. Weinstein SL, Zavala DC, Ponseti IV. Idiopathic scoliosis: Long term followup and prognosis in untreated patients. J Bone Joint Surg Am 1981;63:702-12.
42. Aaro S, Ohlund C. Scoliosis and pulmonary functions. Spine (Phila Pa 1976) 1984;9:220-2.
43. Winter RB, Lovell WW, Moe JH. Excessive thoracic lordosis and loss of pulmonary function in patients with idiopathic scoliosis. J Bone Joint Surg Am 1975;57:972-7.
44. Hilibrand AS, Tannenbaum DA, Graziano GP, Loder RT, Hensinger RN. The sagittal alignment of the cervical spine in adolescent idiopathic scoliosis. J Pediatr Orthop 1995;15:627-32.
45. Visscher W, Lonstein JE, Hoffman DA, Mandel JS, Harris BS 3rd. Reproductive outcomes in scoliosis patients. Spine (Phila Pa 1976) 1988;13:1096-8.
46. King HA, Moe JH, Bradford DS, Winter RB. The selection of fusion levels in thoracic idiopathic scoliosis. J Bone Joint Surg Am 1983;65:1302-13.
47. Nash CL Jr, Moe JH. A study of vertebral rotation. J Bone Joint Surg Am 1969;51:223-9.
48. Potter BK, Rosner MK, Lehman RA Jr, Polly DW Jr, Schroeder TM, Kuklo TR. Reliability of end, neutral and stable vertebrae identification in adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2005;30:1658-63.
49. Lenke LG, Betz RR, Harms J, Bridwell KH, Clements DH, Lowe TG, *et al*. Adolescent idiopathic scoliosis: A new classification system to determine the extent of spinal arthrodesis. J Bone Joint Surg Am 2001;83:1169-81.
50. Hamzaoglu A, Talu U, Tezer M, Mirzanli C, Domanic U, Goksan SB. Assessment of curve flexibility in adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2005;30:1637-42.
51. Watanabe K, Kawakami N, Nishiwaki Y, Goto M, Tsuji T, Obara T, *et al*. Traction versus supine side-bending radiographs in determining flexibility: What factors influence these techniques? Spine (Phila Pa 1976) 2007;32:2604-9.
52. Cummings RJ, Loveless EA, Campbell J, Samelson S, Mazur JM. Interobserver reliability and intraobserver reproducibility of the system of King *et al*. for the classification of adolescent idiopathic scoliosis. J Bone Joint Surg Am 1998;80: 1107-11.
53. Lenke LG, Betz RR, Bridwell KH, Clements DH, Harms J, Lowe TG, *et al*. Intraobserver and interobserver reliability of the classification of thoracic adolescent idiopathic scoliosis. J Bone Joint Surg Am 1998;80:1097-106.
54. Stone B, Beekman C, Hall V, Guess V, Brooks HL. The effect of an exercise program on change in curve in adolescents with minimal idiopathic scoliosis. Phys Ther 1979;59:759-63.
55. Negrini S, Minozzi S, Bettany-Saltikov J, Zaina F, Chokalingam N, Grivas TB, *et al*. Braces for idiopathic scoliosis in adolescents. Spine (Phila Pa 1976) 2010;35:1285-93.
56. Watts HG, Hall JE, Stanish W. The Boston brace system for the treatment of low thoracic and lumbar scoliosis by use of girdle without superstructure. Clin Orthop Relat Res 1977;126:87-92.
57. Emans JB, Kaelin A, Bancel P, Hall JE, Miller ME. The Boston bracing system for idiopathic scoliosis: Followup results in 295 patients. Spine (Phila Pa 1976) 1986;8:792-801.
58. Nachemson AL, Peterson LE. Effectiveness of treatment with a brace in girls who have adolescent idiopathic scoliosis. A prospective, controlled study based on data from the Brace Study of the Scoliosis Research Society. J Bone Joint Surg Am 1995;77:815-22.
59. Wiley JW, Thomson JD, Mitchell TM, Smith BG, Banta JV. Effectiveness of the boston brace in treatment of large curves in adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2000;25:2326-32.
60. Moe JH, Kettleson DN. Idiopathic scoliosis: Analysis of curve patterns and the preliminary results of Milwaukee brace treatment in one hundred sixty nine patients. J Bone Joint Surg Am 1970;52:1509-33.
61. Greene NE. Part time bracing of Adolescent idiopathic scoliosis. J Bone Joint Surg Am 1986;68:738-74.
62. Lonstein JE, Winter RB. The Milwuakee brace for the treatment of adolescent idiopathic scoliosis: A review of 1020 patients. J Bone Joint Surg Am 1994;76:1207-21.
63. Rowe DE, Bernstein SM, Riddick MF, Adler F, Emans JB, Gardner-Bonneau D. A meta-analysis of the efficacy of nonoperative treatments for idiopathic scoliosis. J Bone Joint Surg Am 1997;79:664-74.
64. Danielsson AJ, Hasserius R, Ohlin A, Nachemson AL. A prospective study of brace treatment versus observation alone in adolescent idiopathic scoliosis: A followup mean of 16 years after maturity. Spine (Phila Pa 1976) 2007;32:2198-207.
65. Montgomery F, Willner S. Screening for idiopathic scoliosis: Comparison of 90 cases shows less surgery by early diagnosis. Acta Orthop Scand 1993;64:456-8.
66. Dolan LA, Weinstein SL. Surgical rates after observation and bracing for adolescent idiopathic scoliosis: An evidence based review. Spine (Phila Pa 1976)2007;32:S91-100.
67. Janicki JA, Poe-Koechert C, Armstrong DG, Thompson GH. A comparison of the thoracolumbosacral orthoses and providence orthosis in the treatment of adolescent idiopathic scoliosis: Results using the new SRS inclusion and assessment criteria for bracing studies. J Pediatr Orthop 2007;27:369-74.
68. Diab M, Sharkey M, Emans J, Lenke L, Oswald T, Sucato D. Preoperative bracing affects postoperative outcome of posterior spine fusion with instrumentation for adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2010;35:1876-9.
69. Berg U, Aaro S. Long term effects of Boston brace treatment on renal fuction in patients with idiopathic scoliosis. Clin Orthop Relat Res 1983;180:169-72.
70. Matsunaga S, Hayashi K, Narua T. Psychologic management of brace therapy for patients with idiopathic scoliosis. Spine (Phila Pa 1976) 2005;30:547-50.
71. Katsaris G, Loukos A, Valavanis J. The immediate effect of a Boston brace on lung volumes and pulmonary compliance in

mild adolescent idiopathic scoliosis. Eur Spine J1999;8:2-7.
72. Snyder BD, Katz DA, Myers ER, Breitenbach MA, Emans JB. Bone density accumulation is not affected by brace treatment of idiopathic scoliosis in adolescent girls. J Pediatr Orthop 2005;25:423-8.
73. Sponseller PD. Bracing for adolescent idiopathic scoliosis in practice today. J Pediatr Orthop 2011;31:S53-60.
74. Ogilvie JW, Nelson LM, Chettiar R, Smith-Berry T, Ward K. Predicting brace-resistant adolescent idiopathic scoliosis. Abstracts of the 43rd Scoliosis Research Society (SRS) Annual Meeting and Conference; 2008. Available from: http://www.srs.org/professionals/meetings/am08/doc/oral-abstracts.pdf.
75. Richards BS, Bernstein RM, D'Amato CR, Thompson GH. Standardization of criteria for adolescent idiopathic scoliosis brace studies; SRS Committee onBracing and Nonoperative Management. Spine (Phila Pa 1976) 2005;30:2068-75.
76. Hibbs RA. A report of 59 cases of scoliosis treated by the fusion operation. J Bone Joint Surg 1924;6:3-37.
77. Albee FH. Transplantation of a portion of tibia into the spine for Pott's disease. A preliminary report. JAMA 1911;57:885-6.
78. Moe JH. A critical analysis of methods of fusion for scoliosis: An evaluation in two hundred and sixty six patients. J Bone Joint Surg Am 1958;40-A: 529-54.
79. Dodd CA, Fergusson CM, Freedman L, Houghton GR, Thomas D. Allograft versus autograft bone in scoliosis surgery. J Bone Joint Surg Br 1988;70:431-4.
80. Blanco JS, Sears CJ. Allograft bone use during instrumentation and fusion in the treatment of adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 1997;22:1338-42.
81. Betz RR, Petrizzo AM, Kernea PJ, Falatyn SP, Clements DH, Huss GK. Allograft versus no graft with posterior multisegmented hook systems for the treatment of idiopathic scoliosis. Spine (Phila Pa 1976) 2006;31:121-7.
82. Brandoff JF, Silber JS, Vaccaro AR. Contemporary alternatives to synthetic bone grafts for spine surgery. Am J Orthop (Belle Mead NJ) 2008;37:410-4.
83. Harrington PR. Correction and internal fixation by spine instrumentation. J Bone Joint Surg Am 1962;44-A: 591-610.
84. Tambornino JM, Armbrust EN, Moe JH. Harrington instrumentation in correction of scoliosis. A comparison with cast correction. J Bone Joint Surg Am 1964;46:313-21.
85. Danielsson AJ, Nachemson AL. Radiologic findings and curve progression 22 years after treatment for adolescent idiopathic scoliosis: Comparison of brace and surgical treatment with matching control group of straight individuals. Spine (Phila Pa 1976) 2001;26:516-25.
86. Aaro S, Ohlen G. The effect of Harrington instrumentation on the sagittal configuration and mobility of the spine in scoliosis. Spine (Phila Pa 1976) 1983;8:570-5.
87. Casey MP, Asher MA, Jacobs RR, Orrick JM. The effect of Harrington rod contouring on lumbar lordosis. Spine (Phila Pa 1976) 1987;12:750-3.
88. Luque ER. The anatomic basis and development of segmental spinal instrumentation. Spine (Phila Pa 1976) 1982;7:256-9.
89. Allen BL Jr, Ferguson RL. The Galveston technique of pelvic fixation with L-rod instrumentation of the spine. Spine (Phila Pa 1976) 1984;9:388-94.
90. Allen BL Jr, Ferguson RL. L-rod instrumentation for scoliosis in cerebral palsy. J Pediatr Orthop1982;2:87-96.
91. Rinsky LA, Gamble JG, Bleck EE. Segmental instrumentation without fusion in children with progressive scoliosis. J Pediatr Orthop 1985;5:687-90.
92. Wilberg RG, Thompson GH, Shaffer JW, Brown RH, Nash CL Jr. Postoperative neurological deficits in segmental spinal instrumentation. J Bone Joint Surg Am 1984;66:1178-87.
93. Winter RB, Anderson MB. Spinal arthrodesis for spinal deformity using posterior instrumentation and sublaminar wire. A preliminary report of 100 consecutive cases. Int Orthop 1985;9:239-45.
94. Wenger DR, Carollo JJ, Wilkerson JA Jr, Wauters K, Herring JA. Laboratory testing of segmental spinal instrumentation versus traditional Harrington instrumentation for scoliosis treatment. Spine (Phila Pa 1976) 1982;7:265-9.
95. Thometz JG, Emans JB. A comparison between spinous process and sublaminar wiring combined with Harrington distraction instrumentation in the management of adolescent idiopathic scoliosis. J Pediatr Orthop1988;8:129-32.
96. Cotrel Y, Dubousset J, Guilloumat M. New universal instrumentation in spinal surgery. Clin Orthop Relat Res 1988;227:10-23.
97. Lee SM, Suk SI, Chung ER. Direct vertebral rotation: A new technique of three dimensional deformity correction with segmental pedicle screw fixation in adolescent idiopathic scoliosis. Spine (Phila Pa 1976) 2004;29:343-9.
98. Thompson JP, Transfeldt EE, Bradford DS, Ogilvie JW, Boachie-Adjei O. Decompensation after Cotrel-Dubousset instrumentation of idiopathic scoliosis. Spine (Phila Pa 1976) 1990;15:927-31.
99. Been HD, Kalkman CJ, Traast HS, Ongerboer de Visser BW. Neurological injury after insertion of laminar hooks during Cotrel-Dubousset instrumentation. Spine (Phila Pa 1976) 1994;19:1402-5.
100. Delorme S, Labelle H, Aubin CE, de Guise JA, Rivard CH, Poitras B, *et al*. A three-dimensional radiographic comparison of Cotrel-Dobousset and Colorado instrumentation for the correction of idiopathic scoliosis. Spine (Phila Pa 1976) 2000;25:205-10.
101. Asher M, Lai SM, Burton D, Manna B, Cooper A. Safety and efficacy of Isola instrumentation and arthrodesis for adolescent idiopathicscoliosis. Spine 2004;29:2013-21.
102. Luk KD, Lu DS, Cheung KM, Wong YW. A prospective comparison of the coronal deformity correction in thoracic scoliosis using four different instrumentations with fulcrum bending radiographs. Spine (Phila Pa 1976) 2004;29:2013-23.
103. Remes V, Helenius I, Schlenzka D, Yrjonen M, Ylikoski M, Poussa M. Cotrel-Dubousset (CD) or Universal Spine System (USS) instrumentation in adolescent idiopathicscoliosis. Spine (Phila Pa 1976) 2004;29:2024-30.

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