J Child Orthop (2013) 7:17–24
DOI 10.1007/s11832-012-0460-9

CURRENT CONCEPT REVIEW

Pathogenesis and biomechanics of adolescent idiopathic scoliosis (AIS)

Fritz Hefti

Received: 15 April 2012 / Accepted: 22 April 2012 / Published online: 13 January 2013

Abstract Adolescent idiopathic scoliosis is defined as a scoliosis that starts after the age of ten and has no clear underlying disease as a reason for its development. There is, however, a disparity between the growth of the vertebral bodies anteriorly and that of the posterior elements. The vertebral bodies grow faster than the posterior elements, resulting primarily in a lordosis. The diminished dorsal growth impedes the ventrally located vertebral bodies from increasing in height, forcing them to become distorted, i.e., rotate, in order to create space for themselves. This produces a *rotational lordosis.* The idea of looking at it in this way dates back to Somerville in 1952. Many recent studies have confirmed this idea and have shown that the spinal canal is shorter than the anterior ligament of the vertebral bodies. In a mathematical model of the spine it was demonstrated that—although the vertebral column in humans is still predominantly loaded in an axial direction—certain segments of the human spine (especially the backward inclined segments) are subject to dorsally directed shear loads as well. In addition to the antero-posterior difference in growth, there is also a deformation of the vertebral bodies itself in 3-D. This is probably secondary and not primary effects, but this question is still under discussion. For the treatment of scoliosis, the biomechanical principles of axial and transverse forces are used. The combination of axial and transverse loads is most beneficial for all curves. The axial forces provide most of the corrective bending moment when deformity is severe, while the transverse loads take over the correcting function when deformity is mild. The deformity angle of 53° is the break-even point for the axial and transverse loads. In more severe curves transverse forces become less and less efficient, while axial forces rapidly gain more and more effect.

Keywords Adolescent idiopathic scoliosis · Biomechanics · Pathogenesis · Growth · Load · Rotation · Force

F. Hefti (✉)
Orthopaedic Department, University Children's Hospital, 4031 Basel, Switzerland
e-mail: fritz.hefti@ukbb.ch

Characteristics of AIS

This is by far the most common form of scoliosis and is characterized by the following features:

- AIS is defined as a scoliosis that starts after the age of ten years and has no clear underlying disease as reason for its development
- It is usually located at the *thoracic* level and at this site almost without exception involves a *right-convex* curve.
- It occurs less commonly at the thoracolumbar and lumbar levels, and such cases show a marked tendency to go out of balance. Sometimes these scolioses are not truly idiopathic, but occur secondarily to leg length discrepancies or a lumbosacral junction anomaly.
- In around 10 % of cases, adolescent scoliosis is *S* shaped, i.e., there are two primary curves: since the lumbar curve is usually more rotated than the thoracic curve, *S* shaped scolioses are less conspicuous in cosmetic respects than *C* shaped thoracic scolioses of the same severity.
- It is almost always associated with relative *lordosis* (for the thoracic level, an overall kyphotic angle of $<20°$ is considered to be relative lordosis).

- It always involves *rotation*, whereby the posterior parts of the vertebral bodies are always *rotated* towards the concave side of the curve (if this is not the case then a structural idiopathic scoliosis is not present); for a given degree of curvature, the rotation is always more pronounced at the lumbar level than the thoracic level.
- Rotation causes typical deformations of the vertebrae and the ribs.

Concepts of pathogenesis

Growth

In adolescent scoliosis there is a disparity between the growth of the vertebral bodies anteriorly and that of the posterior elements. The vertebral bodies grow faster than the posterior elements, resulting primarily in a lordosis. The diminished dorsal growth impedes the ventrally located vertebral bodies from increasing in height, forcing them to become distorted, i.e., rotate, in order to create space for themselves. This produces a *rotational lordosis.* The idea of looking at in this way dates back to Somerville [1]. In 1984 Dickson et al. [2] reported on a cadaveric, biomechanical and radiological investigation about the pathogenesis of idiopathic scoliosis and stated that biplanar asymmetry is the essential lesion. Many normal children have coronal plane asymmetry and certainly all have vertebral body asymmetry in the transverse plane, but when median plane asymmetry (flattening or more usually reversal of the normal thoracic kyphosis at the apex of the scoliosis) is superimposed during growth, a progressive idiopathic scoliosis occurs [3, 4]. These authors also postulated that idiopathic kyphoscoliosis does not exist. Median plane asymmetry is crucial for progression. Increased anterior vertebral height at the apex of the curve with posterior end-plate irregularity characterizes the median plane asymmetry and suggests that idiopathic scoliosis is the reverse of Scheuermann's disease.

Many more recent studies have confirmed this theory [5–17]. Lordosis is almost always present in adolescent scoliosis, even when the spine appears kyphotic on the X-ray in a particular projection. The cause of the unequal growth that is responsible for lordosis is unknown. The diminished growth occurs in the area of the spinal canal [14].

The axial length of the vertebral canal and the anterior aspect of the vertebral bodies were measured in 36 skeletons by Porter [14] in 2000: 8 with normal spines, 13 with kyphosis, and 15 with probable idiopathic scoliosis. The relative shortening in the scoliotic spines was correlated with the Cobb angle and the degree of rotation. No significant difference in length was found between the vertebral canal and the vertebral column in the normal spines. The kyphotic spines had canals significantly longer than the vertebral length ($P < 0.025$). The median percentage difference was 8 %. All but one of the scoliotic spines had short vertebral canals ($P < 0.01$). The degree of discrepancy was related to the Cobb angle ($r = -0.50$; $P < 0.05$), and particularly to the degree of rotation ($r = -0.88$; $P < 0.001$) (Fig. 1). The authors concluded that the findings had etiologic implications. The results are consistent with a conceivable hypothesis that in some patients with idiopathic scoliosis, there may be impaired growth in the length of the spinal cord, the posterior elements are tethered, and as the vertebral bodies continue to grow, they become lordotic and then rotate.

More recently, MRI studies helped to support this concept. In 2001 Schmitz et al. [15] examined 64 patents with idiopathic scoliosis and 27 patients without scoliosis. The MR images were made in the supine position. The sagittal Cobb angles were measured between T4–T12 and T12–L5. For the group of the thoracic and double major scoliosis, the mean sagittal Cobb angle (T4–T12) was 13° and for the group without scoliosis it was 23°. There was a negative correlation between the sagittal Cobb angles (T4–T12) and the lateral, thoracic curves. The mean sagittal Cobb angle (T12–L5) of the group with lumbar and double major curves was 35°, which was not a significant difference when compared to the 37° of the non-scoliotic group. The lordotic aspect of the thoracic deformation in scoliosis, therefore, was reliably measured, although imaging in supine position does not ideally reflect curvatures.

Guo et al. [10, 11] reported in 2003 on a study, in which whole spine MRI was undertaken on 83 girls with AIS between the age of 12 and 14 years, and Cobb's angles of between 20° and 90°, and 22 age-matched controls. Compared with the controls, the scoliotic spines had longer vertebral bodies between T1 and T12 in the anterior column and shorter pedicles with a larger interpedicular

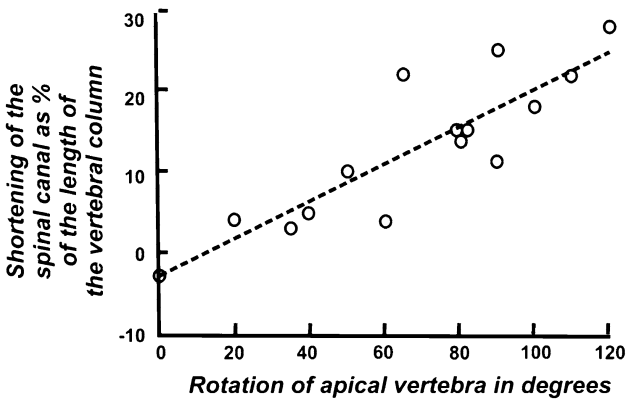

Fig. 1 Relative shortening of the spinal canal in 15 patients with AIS (redrawn after Porter [14])

distance in the posterior column. The differential growth between the anterior and the posterior elements of each thoracic vertebra in the patients with AIS was significantly different from that in the controls ($P < 0.01$). There was also a significant positive correlation between the scoliosis severity score and the ratio of differential growth between the anterior and posterior columns for each thoracic vertebra ($P < 0.01$). Compared with age-matched controls, the longitudinal growth of the vertebral bodies in patients with AIS is disproportionate and faster and mainly occurs by endochondral ossification. In contrast, the circumferential growth by membranous ossification is slower in both the vertebral bodies and pedicles.

In another, similar study Chu et al. [6] reported in 2006 on MR imaging and multiplanar reconstruction to evaluate relative length of the spinal cord to the vertebral column in AIS. They stated that there was a significant relative segmental lengthening of the spinal column at the thoracic level in AIS patients with severe curve; hence, ratios of cord to vertebral column length were significantly reduced ($P < 0.01$). This suggested a disproportional growth between the skeletal and the neural systems. Their conclusion was that the relative shortening and functional tethering of the spinal cord may play an important role in the etiopathogenesis of AIS.

Forces

Castelein et al. [18] demonstrated that the fully erect posture significantly alters spinal loading conditions compared to other primates. In a mathematical model of the spine it was shown that—although the vertebral column in humans is still predominantly loaded in an axial direction—certain segments of the human spine (especially the backward inclined segments) are subject to dorsally directed shear loads as well (Fig. 2). The facet joints play an important role in providing rotational stability to the spine and counteract ventrally directed shear loads. The vertebrae are not well designed to resist dorsally directed shear loads taking the anatomy of the facet joints, and the posterior location of the major spinal muscles and ligaments into consideration. It hypothesized that dorsally directed shear loads render the facet joints less operative in their rotational control.

A biomechanical study was recently performed in vitro by Kouwenhoven and Castelein [12] to investigate axial rotational stability of the thoracic spine under dorsally and ventrally directed shear loads (Fig. 2). The results of that study showed that at the mid and lower thoracic spine more axial vertebral rotation occurred under dorsally directed shear loads than under ventrally directed shear loads. These findings point out that—compared with the spine of other vertebrates (including bipedal animals)—the human spine,

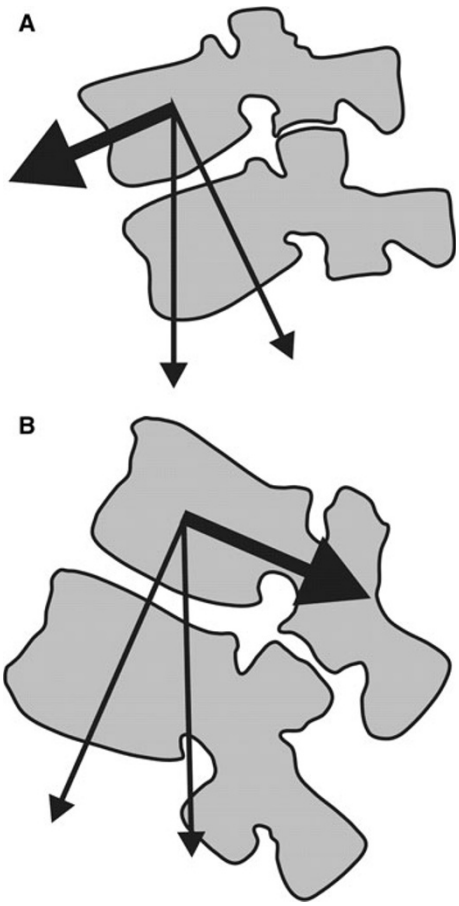

Fig. 2 Vertebrae of the human spine may be subjected to anterior (**a**) or posterior (**b**) shear loads, depending on their orientation in space (redrawn after Castelein et al. [18] and Kouwenhoven and Castelein [12])

on which these dorsal shear loads uniquely act, is a less stable construct, as far as rotation is concerned. The authors postulated that these dorsally directed shear loads can (e.g., during growth) act as an enhancer of slight preexistent vertebral rotation, whereas ventrally directed loads counteract rotation. This rotation-enhancing force working on the segments of the growing spine with backward inclination, could result in a progressive deformation of individual vertebrae because of Hueter–Volkmann's law, and ultimately lead to progressive scoliosis. This supports the findings of earlier research in which it has been demonstrated that backward inclination of vertebrae in the sagittal plane enhance progression of AIS. The concept of scoliosis as a rotatory instability of the immature spine was a consequence of this research.

In a three-dimensionally rendered CT scan study Wever et al. [19] have described the vertebral and rib deformities in idiopathic scoliosis. The observed vertebral deformities suggest that these are caused by bone remodeling due to an imbalance between forces in the anterior and posterior

spinal column [20] (Fig. 3). In this study, the authors also noted a minimal wedge deformation in the local sagittal plane in certain apical vertebrae, as mentioned by Deacon et al. [9], but it is questionable whether this deformation in the sagittal plane is a primary etiological phenomenon, as they suggest, or whether it is rather a secondary phenomenon, comparable to the other vertebral deformations. In an earlier study, these authors could not demonstrate a significant difference in the growth increments and dimensions of vertebral bodies involved in the scoliotic curve compared with the rest of the vertebral column. The growth increments were similar to those reported in the literature for normal girls. A difference in spinal flexibility could also not be established between patients with idiopathic scoliosis and controls [21].

It is conceivable that the spinal cord is protecting itself against the stretching stimulus of growth. Several investigations in recent years have reported the existence of intraspinal anomalies or neurological problems in a certain proportion of "idiopathic" scolioses. The MRI studies have shown an intraspinal syrinx in 8 % of typical idiopathic thoracic adolescent scolioses [22, 23]. Such findings seem to occur much more frequently in atypical (i.e., not right-convex and thoracic) scolioses [24]. Other investigators have found pathological somatosensory potentials in over 50 % of cases of AIS [24]. The side on which the scoliotic convexity occurs does not appear to be relevant to such findings. "Handedness" is also not responsible for the direction of the lateral curvature. The fact that idiopathic thoracic adolescent scoliosis tends to have a right-sided convex curve is rather attributable to the site of the mediastinal organs. Since the condition is not caused by the asymmetry of the muscles but rather by a (symmetrical) problem in the sagittal plane, the distorted side is essentially dependent on the anatomical configuration. As confirmation of this theory we have found an "idiopathic" left convex thoracic scoliosis in two patients with situs inversus. This has also been confirmed in the literature [25]. Thoracic scolioses that are not right convex must therefore be investigated by MRI for the possibility of intraspinal anomalies before surgery. Further tethering of the posterior elements accentuates the tendency of rotation of the spine. This has been observed after early posterior fusion in relatively young children with substantial remaining growth potential, a finding that has been called the "Crankshaft phenomena" [26, 27].

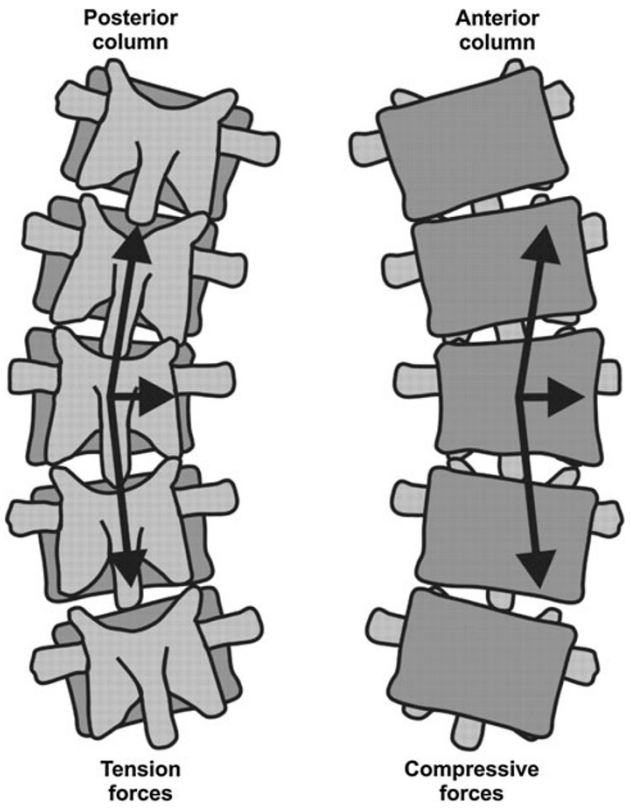

Fig. 3 Scoliotic spine, force pattern in the *anterior column* the compressive forces result in a force driving the apical vertebral body out of the midline (*right*), whereas the tension forces of the posterior column result in a force attempting to keep the posterior complex in the normal position (*left*) (redrawn after Veldhuizen et al. [21])

Deformation of the vertebral bodies

In addition to the antero-posterior difference in growth, there is also a deformation of the vertebral bodies itself in three dimensions. These are probably secondary and not primary effects. Parent et al. [28] have made a morphometric analysis of the scoliotic spine in 2002. Vertebral bodies were measured on 30 scoliotic and on 30 normal skeletons. At the apex height of the vertebral bodies, there was significant decrease at the concave side and the pedicles were smaller and shorter than on the convex side. Figure 4 illustrates the typical deformations of the apical vertebrae [19, 28, 29].

In 2002 Villemure et al. [30, 31] published an article about the simulation of progressive deformities in adolescent idiopathic scoliosis (AIS) using a biomechanical model integrating vertebral growth modulation. A finite-element-model was used. A growth disturbance perpendicular to the growth plate at the level T8 was simulated. The result was a scoliosis with a typical rotation. Similarly to clinical and experimental observations, vertebral wedging angle of the thoracic apex progressed from 2.6° to 10.7° (with growth modulation perpendicular to growth plates) and 7.8° (with additional components in the transverse plane) with curve progression. Concomitantly, vertebral rotation of the thoracic apex increased by 10° and 6°, respectively, adequately reproducing the evolution of axial rotation. The study confirmed that scoliosis progresses

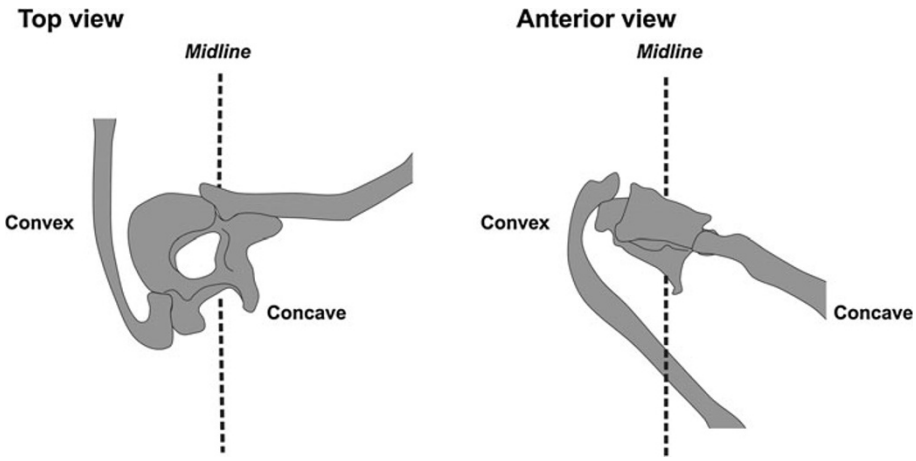

Fig. 4 Deformities within the vertebrae at the apex of a scoliotic spine, seen from the *top* (*left*) and from the *front* (*right*)

within a biomechanical process involving asymmetrical loading of the spine and vertebral growth modulation.

Biomechanics of treatment

Any treatment attempts to return the spine to a normal configuration. There are basically two types of deformation that must be taken into consideration. One is the functional curve. This is an abnormal curvature that is always present, except when some force is applied to correct it, such as active muscular strain by the patient or bending toward the convexity of the functional curve. This curve is maintained by less rigid ligaments, muscles, and gravitational forces.

The structural curve, however, is more rigid and cannot be corrected by active muscle forces. This curve usually consists of deformation within vertebrae; there is wedging and distortion of the osseous structure, and the ligamentous components of the curve are stiff. Either curve may have some component of rotation.

A variety of different techniques with correcting loads may be applied. The corrective forces vary in frequency, amplitude, duration, and mode of application. Basic mechanical principals are involved in the correction of scoliosis.

Creep and relaxation

In the treatment of scoliosis creep is an important concept. The phenomenon is due to the viscoelastic properties of the muscles, ligaments, and bones. Creep is the deformation that follows the initial loading of a material and that occurs as a function of time without further increase in load. When a force is applied to correct a spinal deformity, and the force continues to work after the initial correction, the subsequent correction that occurs over a period of time as a result of the same load is due to creep. Creep is very much dependent on stiffness. In AIS usually only four segments around the apex are stiff [32]. When a load is applied to a viscoelastic material and the deformation remains constant, the observed subsequent decrease in load with time is relaxation. There are a number of clinical examples of the use of either creep or relaxation: the use of halo femoral traction (creep); the pause of several minutes between distraction increments with a rod (relaxation); and reoperation a few months following implantation of a growing rod or a VEPTR in order to gain additional distraction (relaxation) [29].

Comparison of axial and transverse load for scoliosis correction

The scoliotic spine is multifactorial and true simulation requires a complex, 3-D mathematical model. However, White and Panjabi [29] have the merit in studying the behavior of the spine by highly simplified models in order to test a basic concept. With this model they studied the comparative efficiency of different types and combinations of loads applied to a scoliotic spine for correction. The scoliotic spine is modeled by three components: two rigid links AC and BC, connected by way of a torsional spring C (Fig. 5 left). The components lie and move in the frontal plane. The links are oriented to simulate spine deformity in Φ° as measured by the Cobb's method. The static behavior of this model is studied under two separate loading conditions, axial force and transverse forces.

The principle of axial loading in correcting a scoliotic spine is used mainly in operative treatment with distraction rods or with traction. Figure 5 (middle) shows the spine being stretched by the axial force. An axial force is applied at the two ends of the spine segment, represented by points A and B in the model, to elongate and straighten the spine. The mechanism of angular correction by elongation is not due to tensile stresses in the spine, but rather to the bending moments (stresses) created at the various disc spaces. These bending moments correct the angular deformity.

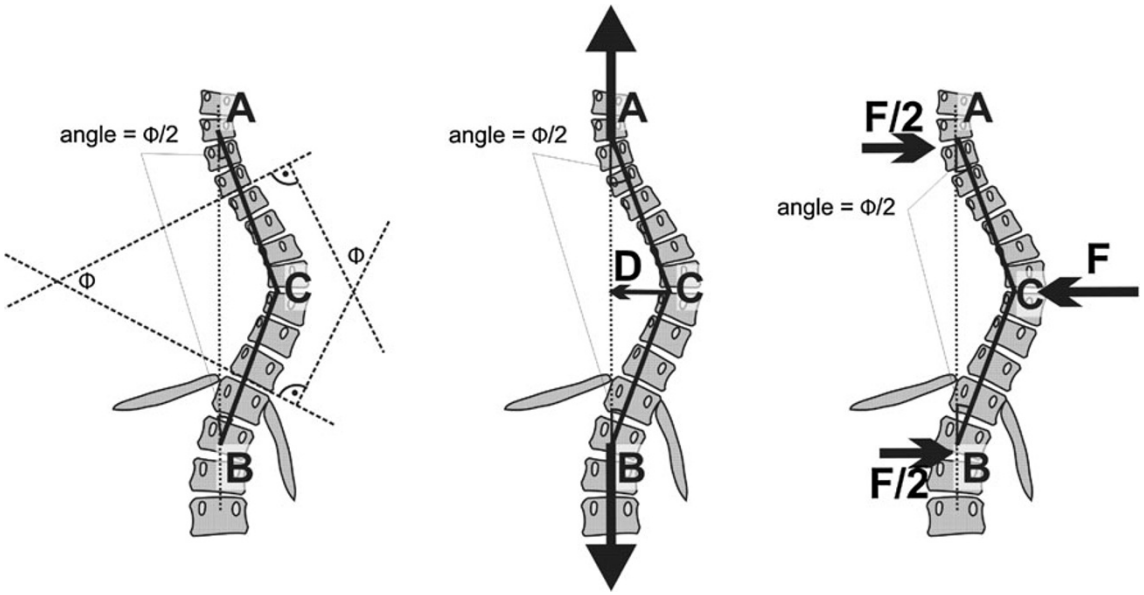

Fig. 5 *Left* a highly simplified mathematical model of the scoliotic spine is made up of 2 rigid links AC and BC connected at C by way of a torsional spring [29]. *Middle* axial load: the model is being subjected to the distraction force *F*. *Right* transverse load: the model is being subjected to three-point transverse forces *F*

Transverse loading is utilized mainly in brace treatment. Figure 5 (right) shows the spine being subjected to lateral loads. The lateral force is applied at C and reactive forces half its size, are taken up at points A and B. The angular correction is again obtained by creating corrective bending moments at the disc spaces. Simple expressions for the bending moments produced at the disc space, represented by point C in Fig. 5, for axial and transverse loads separately, may be derived.

Figure 5 (middle) reveals that the corrective bending moment at the apex of the curve is the axial force *F* multiplied by its perpendicular distance *D* to the apex of the curve. It can easily be seen that the greater the deformity, the greater is the distance *D*. In other words, the correctional ability of the force increases with the severity of the deformity.

A similar situation occurs when the spine is subjected to transverse loading. Figure 5 (right) shows that the corrective bending moment at the apex of the curve equals half of the force at the apex (the other half works on the other half of the spine) multiplied by *D*, the perpendicular distance to the apex of the curve. In contrast to the axial force the corrective bending moment for the lateral force decreases as the deformity of the spine increases.

From this discussion it becomes apparent that the combination of axial and transverse loads is most beneficial for all situations. In other words, the axial component provides most of the corrective bending moment when deformity is severe, and the transverse component takes over the correcting function when deformity is mild.

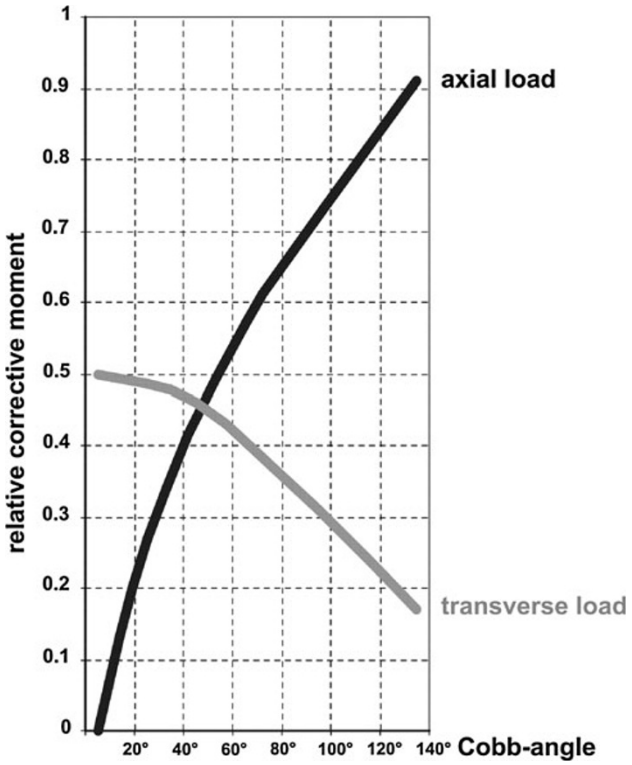

Fig. 6 A *graphic representation* of the "relative corrective moment" as a function of spine deformity in degrees (Cobb-angle). According to the theoretical model, the combined load is most efficient for any degree of scoliosis. The axial load efficiency increases with the angular deformity while the transverse load efficiency decreases. The deformity angle of 53° is the break-even point for the axial and transverse loads [29].

Comparison of the efficiency of the two loading types and their combination can be made on the basis of the corrective bending moment produced at the disc space. The greater the bending moment, the greater is the angular correction obtained. Figure 6 shows the comparative results in graphical form. The diagram shows, on the horizontal axis, the angular deformity Φ in degrees as measured by Cobb's method and, on the vertical axis, the relative corrective bending moment obtained at the apex of the curve due to any of the three load types.

Comparing the graphs of the axial load to the transverse load, it can be seen that these two curves cross at an angle Φ of 53°. Therefore, based on the analysis of this theoretical model, axial loading is more beneficial for severely deformed scoliotic spines, while transverse loading is ideal for correcting milder curves. However, comparing the graphs, it becomes clear that the combined load is the most efficient for all degrees of deformity. Use of the graphs in Fig. 6 determines the most efficient treatment for a given patient.

Conclusion

The spine is a very complex organ. Recent studies have contributed a lot to the understanding of its biomechanics and the pathomechanism of AIS. It is, however, still debated whether the concept of rotational lordosis with a discrepancy of growth between the anterior and posterior elements is the key factor or whether asymmetrical growth and muscle activities are the primary elements. Obviously the mechanism is complex and multifactorial.

Conflict of interest There are no conflict of interest or funding sources to report.

References

1. Somerville EW (1952) Rotational lordosis: the development of the single curve. J Bone Joint Surg 34-B:421–427
2. Dickson RA, Lawton JD, Archer IA, Butt WP (1984) The pathogenesis of idiopathic scoliosis. J Bone Joint Surg 66:8–15
3. Kouwenhoven JW, Vincken KL, Bartels LW, Castelein RM (2006) Analysis of preexistent vertebral rotation in the normal spine. Spine 31(13):1467–1472
4. Janssen MMA, Kouwenhoven JW, Schlösser TPC, Viergever MA, Bartels LW, Castelein RM, Vincken KL (2011) Analysis of the preexistent vertebral rotation in the normal infantile, juvenile and adolescent spine. Spine 36(7):E486–E491
5. Cheung KM, Wang T, Qiu GX, Luk KD (2008) Recent advances in the aetiology of adolescent idiopathic scoliosis. Int Orthop 32(6):729–734
6. Chu WC, Lam WW, Chan YL, Ng BK, Lam TP, Lee KM, Guo X, Cheng JC (2006) Relative shortening and functional tethering of spinal cord in adolescent idiopathic scoliosis? Study with multiplanar reformat magnetic resonance imaging and somatosensory evoked potential. Spine 31(1):E19–E25
7. Cil A, Yazici M, Uzumcugil A, Kandemir U, Alanay A, Alanay Y, Acaroglu RE, Surat A (2005) The evolution of sagittal segmental alignment of the spine during childhood. Spine 30(1):93–100
8. Cruickshank JL, Koike M, Dickson RA (1989) Curve patterns in idiopathic scoliosis. A clinical and radiographic study. J Bone Joint Surg 71:259–263
9. Deacon P, Archer IA, Dickson RA (1987) The anatomy of spinal deformity: a biomechanical analysis. Orthopedics 10:897–903
10. Guo X, Chau W-W, Chan Y-L, Cheng JC-Y (2003) Relative anterior spinal overgrowth in adolescent idiopathic scoliosis. Results of disproportionate endochondral–membranous bone growth. J Bone Joint Surg [Br] 85:1026–1031
11. Guo X, Chau W-W, Chan Y-L, Cheng J-C-Y, Burwell RG, Dangerfield PH (2005) Relative anterior spinal overgrowth in adolescent idiopathic scoliosis—result of disproportionate endochondral-membranous bone growth? Eur Spine J 14(9):862–873
12. Kouwenhoven JW, Castelein RM (2008) The pathogenesis of adolescent idiopathic scoliosis: review of the literature. Spine 33(26):2898–2908
13. Liljenqvist U, Allkemper T, Hackenberg L, Link T, Steinbeck J, Halm H (2002) Analysis of vertebral morphology in idiopathic scoliosis with use of magnetic resonance imaging and multiplanar reconstruction. J Bone Joint Surg (Am) 84:359–368
14. Porter R (2000) Idiopathic scoliosis: the relation between the vertebral canal and the vertebral bodies. Spine 25:1360–1366
15. Schmitz A, Jäger U, König R, Kandyba J, Gieske J, Schmitt O (2001) Sagittale Cobb–Winkel–Messungen bei Skoliose mittels MR-Ganzwirbelsäulenaufnahme. Z Orthop Ihre Grenzgeb 139:304–307
16. Stokes IA, Windisch L (2006) Vertebral height growth predominates over intervertebral disc height growth in adolescents with scoliosis. Spine 31(14):1600–1604
17. Wang WJ, Hung VW, Lam TP, Ng BK, Qin L, Lee KM, Qiu Y, Cheng JC, Yeung HY (2010) The association of disproportionate skeletal growth and abnormal radius dimension ratio with curve severity in adolescent idiopathic scoliosis. Eur Spine J 19(5):726–731
18. Castelein RM, van Dieen JH, Smit TH (2005) The role of dorsal shear forces in the pathogenesis of adolescent idiopathic scoliosis—a hypothesis. Med Hypotheses 65:501–508
19. Wever D, Veldhuizen A, Klein J, Webb P, Nijenbanning G, Cool J, Horn Jv (1999) A biomechanical analysis of the vertebral and rib deformities in structural scoliosis. Eur Spine J 8(4):p252–p260
20. Veldhuizen A, Wever D, Webb P (2000) The aetiology of idiopathic scoliosis: biomechanical and neuromuscular factors. Eur Spine J 9(3):p178–p184
21. Do T, Fras C, Burke S, Widmann R, Rawlins B, Boachie-Adjei O (2001) Clinical value of routine preoperative magnetic resonance imaging in adolescent idiopathic scoliosis. A prospective study of three hundred and twenty-seven patients. J Bone Joint Surg Am 83-A:577–579
22. Samuelsson L, Lindell D, Kogler H (1991) Spinal cord and brain stem anomalies in scoliosis. Acta Orthop Scand 62:403–406
23. Phillips WA, Hensinger RN, Kling TF Jr (1990) Management of scoliosis due to syringomyelia in childhood and adolescence. J Pediatr Orthop 10:351–354
24. Machida M, Dubousset J, Imamura Y, Iwaya T, Yamada T, Kimura J, Toriyama S (1994) Pathogenesis of idiopathic scoliosis. SEPs in chicken with experimentally induced scoliosis and in patients with idiopathic scoliosis. J Pediatr Orthop 14:329–335
25. Kouwenhoven JW, Bartels LW, Vincken KL, Viergever MA, Verbout AJ, Delhaas T, Castelein RM (2007) The relation between organ anatomy and pre-existent vertebral rotation in the

normal spine: magnetic resonance imaging study in humans with situs inversus totalis. Spine 32(10):1123–1128
26. Hefti FL, McMaster MJ (1983) The effect of the adolescent growth spurt on early posterior spinal fusion in infantile and juvenile idiopathic scoliosis. J Bone Joint Surg 65-A:247–254
27. Dubousset J, Herring JA, Shufflebarger HJ (1989) The crankshaft phenomenon. Pediatr Orthop 9(5):541–550
28. Parent S, Labelle H, Skalli W, Latimer B, de Guise J (2002) Morphometric analysis of anatomic scoliotic specimens. Spine 27(21):p2305–p2311
29. White AA, Panjabi MM (1990) Clinical biomechanics of the spine. JB Lippincott Co. Philadelphia, Toronto
30. Villemure I, Aubin C, Dansereau J, Labelle H (2002) Simulation of progressive deformities in adolescent idiopathic scoliosis using a biomechanical model integrating vertebral growth modulation. J Biomech Eng 124(6):p784–p790
31. Huynh AM, Aubin CE, Rajwani T, Bagnall KM, Villemure I (2007) Pedicle growth asymmetry as a cause of adolescent idiopathic scoliosis: a biomechanical study. Eur Spine J 16(4): 523–529
32. Hasler CC, Hefti F, Büchler P (2010) Coronal plane segmental flexibility in thoracic adolescent idiopathic scoliosis assessed by fulcrum-bending radiographs. Eur Spine J 19(5):732–738