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Comment to “The pathogenesis of idiopathic scoliosis: uncoupled neuro-osseous growth?” by R. W. Porter

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Porter's hypothesis is: *that the longitudinal growth of the spinal cord fails to keep pace with the growth of the vertebral column, resulting in idiopathic scoliosis*. The hypothesis, based on morphological observations and a model [23, 24], is examined in his review using clinical and scientific knowledge relating to scoliosis and other conditions. Porter also evaluates what mechanisms may cause a short spinal cord.

The hypothesis is not new. Roth [26, 27, 28], a neuroradiologist, published nine papers between 1968 and 1985, seven in Czechoslovakian and German journals, in which he explained idiopathic scoliosis as a disproportion of vertebro-neural growth due to either a short spinal cord [26] or a too rapid growth spurt of the spine [27]. In his spring-string model, Roth [27] found that shortening of the spring hindered free elongation of the spring, resulting in a 'scoliotic' deformity of the model. Scheuermann's disease was also explained by Roth [27, 28] as a vertebro-neural growth disproportion. Roth's papers were scarcely quoted until 2000 [6, 14, 15].

Porter's evidence

Porter addresses the problem of relative anterior spinal overgrowth in idiopathic scoliosis, with particular reference to the axial length of the ver-

tebral canal and the vertebral bodies. His hypothesis views idiopathic scoliosis as a physical manifestation of the maladaptation of the growing immature spine to a tether created by a short spinal cord, which he supports by stating that:

1. The vertebral canal, spinal cord and foraminae tend to retain their original orientation in relation to the spinous process [24]
2. The vertebral body rotates about an axis at the site of the spinal cord [24]
3. The spinal cord is displaced in the vertebral canal towards the concavity of both single and double curves [20, 26, 27, 28]

Most recently, Porter and his colleagues [25] have found that the level of the conus in idiopathic scoliosis is not significantly different from its position in the normal population.

Porter [23] admits that a short spinal cord could be a secondary phenomenon. In this connection, it is well known in scoliosis aetiological research that, once a new finding has been established, difficulty arises in deciding whether it is related to causation or results from the scoliotic deformity [9]. Porter deals with pathomechanisms and pathogenesis, which he places in the spinal cord, but not with aetiology [32]. He does not weigh his evidence against other interpretations for the pathomechanisms of idiopathic scoliosis.

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Morphology

Porter attempts to refute his hypothesis by asking what morphological features could be expected in the light of the hypothesis of uncoupled neuro-osseous growth. He lists eight, and concludes that, while traditionally these have been explained as secondary to some unknown primary cause, disproportionate growth between the spinal cord and vertebral column is an equally plausible explanation.

Is there any evidence for a short spinal cord in subjects with idiopathic scoliosis?

As a crude indirect measure of spinal cord length, Porter uses sitting height. A review of all the published evidence [12] revealed variable sitting height in adolescent idiopathic scoliosis (AIS) subjects, with many papers reporting an increase in corrected sitting height, especially in children aged 14 years or less. In his section dealing with evidence of increased bone growth, Porter quotes evidence that spines in idiopathic scoliosis are longer than in controls. The thoracolumbar spinal length in children with juvenile idiopathic scoliosis (JIS) and AIS show no significant difference from controls, but have a tendency to be longer [33]. Hence, from both sitting height and thoracospinal length, there is no evidence for a shorter spinal cord in subjects with JIS and AIS. Floman [15], reporting on 12 males with AIS and restricted neck flexion (a paper to which Porter refers), found the magnetic resonance imaging (MRI) appearance of the entire spine was normal, and writes, '...it was not feasible to accurately measure the length of the spinal cord in relationship to the length of the vertebral column.' Nevertheless, Floman states that a short spinal cord could explain both the thoracic scoliosis and the limited neck flexion.

A critical anthropometric test of the Porter hypothesis

In most, if not all forms of scoliosis, the spinal cord will be shorter than the axial length of the vertebral bodies. Hence, measurements on the spine can never determine which came first – the short vertebral canal or the increased anterior spinal column length. The only way to assess any neuro-osseous growth disproportion is to compare spinal cord/nerve root length against the length of some extraspinal skeletal component(s).

MR imaging of the whole spine [29] makes it feasible to measure spinal cord length in AIS patients. Spinal cord length so measured could be related by linear regression analysis to the size of selected extraspinal skeletal components, such as tibial length, upper limb components, and chest size components; the results could then be compared with findings from appropriate controls. This experiment would provide a critical test of Porter's hypothesis, which it could refute. However, if the spinal cord length is found statistically to be short relative to some skeletal component(s), it would support the hypothesis. If found to be prognostic for curve progression, standards would need to be established by age, sex, and ethnicity.

Girls with AIS with right thoracic curves have been found to have a relatively small thorax and a large extra-thoracic skeleton [12]. The relatively small thorax could be secondary to the scoliotic deformity of the spine, whatever its cause, including a short spinal cord.

The skeletal deformity at the apex of the thoracic scoliotic curve: problems of interpretation

The question must be asked: Would it be possible to distinguish the effects on vertebral growth of a force, or forces, acting on the spine from different pathomechanisms? These

include a force, or forces, arising from:

1. Inside the vertebrae (spinal cord [23, 24] and theca)
2. A fixed lordotic area which, under the influence of transverse or frontal plane asymmetry, rotates to the side to give rise to a lateral curvature [22]
3. A loss of coupling between anterior spinal components and posterior components during the adolescent growth spurt [10, 42] (the posterior components could theoretically involve short ligaments and/or impaired bone growth of the vertebral arch), and
4. Outside the vertebrae (from ribs [30, 31] or muscles [3, 4, 6, 13, 14, 36, 39, 44] acting on the spine)

Porter argues in favour of (1). After examining the apical vertebrae from two museum specimens, he writes [24], 'The rotational deformity tends to be selective and anterior to the canal.' This confirms the findings of others [20, 37, 42]. He continues, 'If the deformity in scoliosis was due to imbalance secondary to neuromuscular factors, we might reasonably expect the rotational deformity to be uniform throughout the vertebra – the whole vertebra rotating as a unit – but this is not the case.' The difficulty I have with this interpretation is that the anatomy of the deformity of the apical vertebra is generally considered to be similar in neurogenic and idiopathic scolioses, although I know of no paper that has addressed this problem. Porter recognizes this dilemma in referring to the anatomical specimens that he examined as having 'probable' idiopathic scoliosis [23]. Porter does not claim that various forms of *neurogenic* scoliosis initiate scoliosis through a short spinal cord, but it could be tested critically using the experiment outlined above.

Porter assumes that the apical deformity of idiopathic scoliosis is solely one of axial vertebral rotation. Yet multiple factors, biomechanical

and biological, are thought to contribute to the deformity of the apical vertebra in structural scoliosis [2, 22, 42]. In addition to axial rotation, there may be:

1. Greater growth of thoracic concave neurocentral synchondroses in younger subjects [27, 40, 45]
2. The response of immature vertebral bodies to eccentric load transmission with torsion in the spine [2, 5, 41], created by longitudinal growth of the vertebral bodies, muscle contractions and gravity (Hueter-Volkman effect [9, 35, 42])
3. Tethering by posterior ligamentous structures of the posterior part of the spinal column [42], and
4. The stabilizing effect of the costovertebral joints [42] abetted by the rib cage and sternum, with the latter acting as the fourth column of the spine [16]. Bone drift [22, 34] is not an issue here.

Findings from measurements made on vertebrae are unlikely to distinguish between the four possibilities (1–4) above. Such vertebral findings are generally considered to show pathomechanical features consistent with a lordoscoliosis, rotation and remodelling [22, 34, 42], i.e. the three-dimensional deformity of structural scoliosis. Wever et al. [42] interpret the vertebral deformities of structural scoliosis as being caused by bone remodelling due to lateral shear forces created in the anterior spinal column driving the apical vertebra out of the midline, whereas torque forces created by posterior musculoligamentous structures attempt to minimize the deviations and rotations of the vertebrae. The rib deformities are considered to be secondary to the lateral forces induced by the scoliotic spine [42]. These pathomechanical concepts are considered to apply to structural scoliosis, be it idiopathic or neurogenic.

Based on the available morphological evidence for the apical vertebral deformity, and on the balance of prob-

abilities, I agree with Porter that his hypothesis at the present time is plausible for *idiopathic* scolioses. It is also plausible for *neurogenic* scolioses.

Thoracolumbar and lumbar structural scolioses

Porter restricts his attention to thoracic scoliosis and does not explain the idiopathic structural thoracolumbar and lumbar curves. To attribute such lumbar curves to uncoupled neuro-osseous growth, the nerve roots of the cauda equina would have to be shown to be short relative to some extraspinal skeletal component.

Curve laterality

Porter explains the common right thoracic curve on White's concept [43] that the thoracic spine is predisposed to rotate to the right. Porter does not address the predominant left curve laterality of infantile and some juvenile idiopathic thoracic scolioses. The most likely explanation is that, in such curves, the spine is predisposed to rotate to the left (hypothesis of oscillating axial torsion) [8].

Abnormalities more widespread than the spine and ribcage

Porter does not address the problem of why idiopathic scoliosis has been found to be associated with anomalies outside the spine and ribcage, including facial asymmetry, breast asymmetry, upper limb length asymmetry, complex patterns of skeletal allometry, and dermatoglyphic asymmetry [6, 8, 12, 18]. The rejoinder might be that the short spinal cord is one of the abnormalities of developmental instability [18] and the critical defect that causes the scoliosis. In this connection, there is a need for a longitudinal study using MRI to establish the post-natal growth of the spinal cord in health and in subjects with idiopathic scoliosis.

Asher's requirements for any aetiological theory of idiopathic scoliosis

Asher [1] has stated the requirements for any theory of AIS aetiology are to explain:

1. The dependence of the deformity on growth and growth rate
2. Its predilection for females
3. Its predilection for members of involved families, and
4. Its variable progression patterns

To these Goldberg [18] adds:

5. Lateralization

Porter does not address (2), (3), or (4).

Neurology

Porter lists six features, and concludes that:

1. The subtle neurological abnormalities in some children with scoliosis
2. The rarity of major neurological problems, and
3. The frequency of scoliosis with certain pathologies

are not incompatible with his hypothesis.

I find this conclusion unlikely, because, while a *normal* cord is evidently stretched without compromise during normal growth, an *abnormal* cord stretched, even gradually, would be expected to produce clinical signs of long tract (cord) and/or nerve root damage, and at times symptoms.

The absence, or rarity, of neurological deficit after correction of the deformity with brace or operation, Roth [27] argued, seems to militate against his concept; i.e. apparent lengthening of the vertebral column in treatment would subject the spinal cord and nerve roots to non-physiological stretch and damage. Porter discusses this point and concludes that, 'One might have expected a higher overall incidence if the hypothesis were correct.' I agree. It

could be argued that any distraction at surgery causing acute neurological complications might be due in some patients to a short cord. Can this be evaluated preoperatively for possible prophylaxis?

In children below the age of 12 years, several studies using MRI have revealed significant neuro-anatomical abnormalities. By definition, such children do not have idiopathic scoliosis. I agree with Porter's statement that the neurological findings he quotes are not incompatible with his hypothesis. He does not discuss evidence from somatosensory evoked potentials [11, 38]. The general view for *neurogenic* scolioses, namely that the vertebral deformity results from altered neuromuscular forces acting *outside* the spine, is more acceptable. In this connection, James [19] noted in paralytic scoliosis an unexpected and unexplained finding; namely, that as with idiopathic thoracic curves, there is a predominance of curves to the right. Porter's hypothesis would account for this finding by the predisposition of the thoracic spine and ribcage to rotate to the right. Could the shortening of the neural structures in a cranio-caudal direction (i.e. the spinal cord) not also involve a right-left asymmetry (i.e. of nerve roots)? Roth [26] addressed this point, and argued that correction of the disturbed tension equilibrium would be achieved by lateral flexion of the spine towards the side of the increased tension. Porter [24] mentions Lloyd-Roberts' suggestion [20] that progression in infantile idiopathic scoliosis occurs when the neuraxis is unable to adjust to the change in the anatomy of the vertebral column.

Uncoupled neuro-osseous growth

Roth [27] suggested that growth hormone stimulates the growth of all tissues except nervous tissue, and results in neurovertebral growth disproportion. He states that the thoracic cord grows more in length during development than the cervical or lumbosacral cord. This makes the thoracic cord more susceptible to growth insufficiency, with resulting thoracic scoliosis. In contrast, Porter suggests a different view for the site of the apical vertebra(e) of thoracic curves, namely an earlier skeletal maturity of the upper thoracic spine.

Porter addresses factors in the spinal cord that might impair its length, and also asks: Is there evidence of increased bone growth? He suggests that the spinal cord may fail to stretch in response to vertebral growth due to molecular mechanisms, and that this may result in scoliosis. He calls for a reductionist approach and suggests more research into the mechanical and cellular properties of the spinal cord in scoliosis patients. These are novel ideas, which it is hoped some researchers will evaluate.

Perspective

Currently, few would dissent from the view that the cause of idiopathic scoliosis is multifactorial [21], but whether a short spinal cord will prove to be one of those factors in none, a few or many subjects must await more research. Porter has brought the Roth concept for the causation of idiopathic scoliosis into the molecular biology of the spinal cord.

The Roth-Porter hypothesis has to be viewed in the context of other pathomechanic hypotheses for idiopathic scoliosis. The thoracospinal concept of Sevastik [30, 31] looks encouraging, with surgical potential. The possibility that AIS pathogenesis involves undetected neuromuscular dysfunction is considered likely by several workers [3, 4, 6, 11, 13, 14, 36, 39, 44], although not by all [17]. In this connection, Edgar [14] writes that neurological dysfunction in idiopathic scoliosis has moved to a centre-stage position, and now needs to be carefully considered. As idiopathic scoliosis is a final common pathway of deformity, there may be different neuromuscular mechanisms causing scoliosis in different subjects, resulting from different aetiologies and pathogeneses and different pathomechanisms in the spine. If so, this compounds the difficulties, particularly for genetic aetiological research. The spinal factors to be considered include hypokyphosis [22], osteopoenia [10], ligamentous laxity [6] and disc mechanisms. Hypokyphosis as a risk factor for thoracic curves [22] needs to be evaluated to examine whether or not it is associated with a spinal cord that is short relative to some extraspinal skeletal component(s). Neuromuscular dysfunction is considered by many to need intensive research, and this includes an evaluation of the *neuro-osseous timing of maturation* around puberty [7]. The Roth-Porter hypothesis directs attention to a local neural dysfunction within the spinal cord as a possible cause of idiopathic scoliosis. The hypothesis can be critically tested, and this needs to be done for scoliosis in all its forms – idiopathic, congenital and secondary.

References

1. Asher MA (2000) Foreword. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:14–15
2. Asher MA, Burton DC (1999) A concept of idiopathic scoliosis as imperfect torsion(s). *Clin Orthop* 364:11–25
3. Bagnall KM (1999) Debate 1. Adolescent idiopathic scoliosis: is the cause neuromuscular? The case for. In: Stokes IAF (ed) Research into spinal deformities, vol 2. IOS Press, Amsterdam, pp 91–93
4. Bagnall KM (2000) Ligaments and muscles in adolescent idiopathic scoliosis. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:447–457
5. Birchall D, Hughes DG, Hindle J, Robinson L, Williamson JB (1997) Measurement of vertebral rotation in adolescent idiopathic scoliosis using three-dimensional magnetic resonance imaging. *Spine* 22:2403–2407
6. Burwell RG, Dangerfield PH (2000) Adolescent idiopathic scoliosis: hypotheses of causation. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:319–333
7. Burwell RG, Dangerfield PH (2001) A possible neuroendocrine method for delaying the adolescent growth spurt and slowing curve progression based on the NOTOM hypothesis: potential of a medical treatment for progressive juvenile and adolescent idiopathic scoliosis. *J Bone Joint Surg Br Orthop Proc* (in press)
8. Burwell RG, Dangerfield PH, James NJ, Johnson F, Webb JK (1984) Anthropometric studies of normal and scoliotic children: axial and appendicular skeletal asymmetry, sexual dimorphisms and age-related changes. In: Jacobs RR (ed) Pathogenesis of idiopathic scoliosis. Proceedings of an International Conference. Scoliosis Research Society, Chicago, pp 27–44
9. Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (2000) Preface. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:12,13
10. Cheng J (2000) Osteopenia. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:339–348
11. Cheng JC, Guo X, Sher AH (1998) Posterior tibial nerve somatosensory cortical evoked potentials in adolescent idiopathic scoliosis. *Spine* 23:332–337
12. Cole AA, Burwell RG, Dangerfield PH, Grivas, TB, Webb JK, Moulton A (2000) Anthropometry. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:411–421
13. Dubousset J (1999) Les scolioses dites idiopathiques. Definition, pathologie, classification, etiologie. *Bull Acad Natl Med* 4:699–704
14. Edgar M (2000) Neural mechanisms in the etiology of idiopathic scoliosis. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:459–468
15. Floman Y (2000) Thoracic scoliosis accompanied by restriction of neck flexion: a distinct type of adolescent “idiopathic” scoliosis in males. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:483–488
16. Gardner ADH (2000) The significance of the sternum: the buttress of the thoracic spine. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:383–389
17. Goldberg CJ (1999) Debate 1. Adolescent idiopathic scoliosis: is the cause neuromuscular? The case against. In: Stokes IAF (ed) Research into spinal deformities, vol 2. IOS Press, Amsterdam, pp 94–97
18. Goldberg CJ (2000) Symmetry control. In: Burwell RG, Dangerfield PH Adolescent idiopathic scoliosis: hypotheses of causation. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:327,328
19. James JIP (1956) Paralytic scoliosis. *J Bone Joint Surg Br* 38:660–685
20. Lloyd-Roberts GC, Pincott JR, McMeniman P, Bayley IJL, Kendall B (1978) Progression in idiopathic scoliosis. A preliminary report of a possible mechanism. *J Bone Joint Surg Br* 60:451–460
21. Machida M (2000) Cause of idiopathic scoliosis. *Spine* 24:2576–2583
22. Millner PA, Dickson RA (1996) Idiopathic scoliosis: biomechanics and biology. *Eur Spine J* 5:362–373
23. Porter RW (2000) Idiopathic scoliosis: the relation between the vertebral canal and vertebral bodies. *Spine* 25:1360–1366
24. Porter RW (2001) Can a short spinal cord produce scoliosis? *Eur Spine J* 10:2–9
25. Porter RW, Hall-Craggs M, Walker AE, Ursu T, Edgar M (2000) The position of the cerebellar tonsils and the conus in patients with scoliosis. *J Bone Joint Surg Br* 82 [Suppl III]:286–7
26. Roth M (1968) Idiopathic scoliosis caused by a short spinal cord. *Acta Radiol Diagn* 7:257–271
27. Roth M (1981) Idiopathic scoliosis from the point of view of the neuroradiologist. *Neuroradiology* 21:133–138
28. Roth M (1981) Idiopathische Skoliose und Scheuermannsche Erkrankung: Wesensgleiche Erscheinungsformen der neuro-vertebralen Wachstumsdisproportion. *Radiol Diagn* 22:380–391
29. Schmitz A, Jaeger UE, Koenig R, Kandyba J, Wagner UA, Giesecke J, Schmitt O (2001) A new MRI technique for imaging scoliosis in the sagittal plane. *Eur Spine J* 10:114–117
30. Sevastik JA (2000) The thoracospinal concept of the etiopathogenesis of idiopathic scoliosis. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:391–400
31. Sevastik JA (2001) A new concept for the etiopathogenesis of the thoracospinal deformity of idiopathic scoliosis. First Electronic Focus Group, International Federated Body on Scoliosis Etiology (IBSE), spine92@liverpool.ac.uk
32. Sevastik JA, Stokes IAF (2000) Idiopathic scoliosis: Terminology. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches. *Spine State Art Rev* 14:299–303
33. Skoglund LB, Miller JAA (1981) The length and proportion of the thoracolumbar spine in children with idiopathic scoliosis. *Acta Orthop Scand* 52:177–185

34. Smith RM, Pool RD, Butt WP, Dickson RA (1991) The transverse plane deformity of structural scoliosis. *Spine* 16:1126–1129
35. Stokes IAF (2000) Hueter-Volkman effect. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) *Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches*. *Spine State Art Rev* 14:349–357
36. Taylor TKF (2000) The brain stem and adolescent idiopathic scoliosis: a hypothesis. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) *Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches*. *Spine State Art Rev* 14:477–481
37. Upadhyay SS, Leong JCY, Ho EKW (1992) CT measurement of vertebral rotation in 3-D scoliotic deformities. In: Dansereau J (ed) *International Symposium on 3-D Scoliotic Deformities joined with the VIIth International Symposium on Spinal Deformity and Surface Topography*. Édition de l'École Polytechnique de Montreal, Montreal, and Gustav Fischer Verlag, Stuttgart, pp 257–267
38. Uyttendaele D, Bossche LV, Rimbault M, De Muynck M, Vanderstraeten G (2000) Somatosensory evoked potentials in idiopathic scoliosis. In: Burwell RG, Dangerfield PH *Adolescent idiopathic scoliosis: hypotheses of causation*. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) *Etiology of adolescent scoliosis: current trends and relevance to new treatment approaches*. *Spine State Art Rev* 14:324, 325
39. Veldhuizen AG, Wever DJ, Webb PJ (2000) The aetiology of idiopathic scoliosis: biomechanical and neuromuscular factors. *Eur Spine J* 9:178–184
40. Vital JM, Berguieristain JL, Algara C, Villas C, Lavignolle B, Grenier N, Sénégas J (1989) The neurocentral vertebral cartilage: anatomy, physiology and physiopathology. *Surg Radiol Anat* 11:323–328
41. Wemyss-Holden SA, Butcher CCA, Burwell RG, Webb JK, Moulton A (1994) Segmental evaluation of the rotational (torsional) deformity in scoliosis: anatomical observations and surgical significance. *J Bone Joint Surg Br* 76 [Suppl I]: S9
42. Wever DJ, Veldhuizen AG, Klein JP, Webb PJ, Nijenbanning G, Cool JC, van Horn JR (1999) A biomechanical analysis of the vertebral and rib deformities in structural scoliosis. *Eur Spine J* 8:252–260
43. White A III (1971) Kinematics of the normal spine as related to scoliosis. *J Biomech* 4:405–411
44. Williamson JB (2000) Postural control. In: Burwell RG, Dangerfield PH, Lowe TG, Margulies JY (eds) *Etiology of adolescent idiopathic scoliosis: current trends and relevance to new treatment approaches*. *Spine State Art Rev* 14: 469–476
45. Yamazaki A, Mason DE, Caro PA (1998) Age at closure of the neurocentral cartilage in the thoracic spine. *J Pediatr Orthop* 18:168–172