PERSPECTIVE

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Idiopathic Scoliosis as a Rotatory Decompensation of the Spine

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ABSTRACT

Many years of dedicated research into the etiology of idiopathic scoliosis have not led to one unified theory. We propose that scoliosis is a mechanical, rotatory decompensation of the human spine that starts in the transverse, or horizontal, plane. The human spine is prone to this type of decompensation because of its unique and individually different, fully upright sagittal shape with some preexistent transverse plane rotation. Spinal stability depends on the integrity of a delicate system of stabilizers, in which intervertebral disc stiffness is crucial. There are two phases in life when important changes occur in the precarious balance between spinal loading and the disc's stabilizing properties: (i) during puberty, when loads and moment arms increase rapidly, while the disc's "anchor," the ring apophysis, matures from purely cartilaginous to mineralized to ultimately fused to the vertebral body, and (ii) in older age, when the torsional stiffness of the spinal segments decreases, due to disc degeneration and subsequent laxity of the fibers of the annulus fibrosus. During these crucial periods, transverse plane vertebral rotation can increase during a relatively brief window in time, either as adolescent idiopathic or degenerative de novo scoliosis. Much more is known of the biomechanical changes that occur during disc aging and degeneration than of the changing properties of the disc during maturation. © 2020 American Society for Bone and Mineral Research (ASBMR).

KEY WORDS: BIOMECHANICS; DEFORMITY; DEGENERATIVE SCOLIOSIS; DISC; ETIOLOGY; IDIOPATHIC SCOLIOSIS; MATURATION; ROTATION

Introduction

Idiopathic scoliosis is a three-dimensional (3D) rotational deformity of the spine without obvious underlying cause that is reported to occur with a prevalence of 2% to 4% of the general population, although geographic differences have been reported.[1] Girls during the adolescent growth spurt are more often affected than boys, especially in its progressive form.[2] It carries a high burden of disease because of its lifelong course and the costs of treatment are significant; the mean hospital charge was calculated for 42 consecutive operated patients in a nonprofit setting and amounted to $126,284 (range $76,171 to $215,516).[3]

Scoliosis consists of three more or less coupled phenomena: rotation and intra-architectural torsion in the transverse plane, extension in the sagittal plane, and lateral deviation in the coronal plane. Even in the more severe forms, by far most of the deformity in all planes is located in the intervertebral disc.[4–8] There is some evidence that it may be a generic response of the spinal column to a disturbance of spinal equilibrium.[9] It leads to increased pain, truncal imbalance, disturbance of self-image, and may affect cardiopulmonary functioning in its more severe forms. It is considered a disease of the human race; it does not occur naturally in other mammalians. In zebrafish, a rotational spinal curvature can occur that shows similarities to the defining characteristics of human idiopathic scoliosis.[10,11]

Many etiological theories have been proposed, incriminating just as many of the body's organ systems.[1] There is no evidence, however, that there is a consistent underlying causative abnormality.[12] Practically all scientific knowledge is derived from established cases; very little is known of the prescoliotic spine and no mammalian animal model is known.[13] Therefore, it is practically impossible to distinguish between factors that contribute to its etiology, factors that are the result of the deformity, and simple epi-phenomena. No adequate, early preventive interventions are available to date, and treatment is aimed at the outcome of the disease process, an already relatively severe deformity.

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We propose that idiopathic scoliosis is primarily a mechanical, rotatory decompensation in the horizontal plane of that relatively unstable, prerotated human spine. Individual differences in the sagittal plane determine which type of deformity will develop. It starts with a deformation of the disc and proceeds with bony adaptations.[14–18] We also propose that degenerative de novo scoliosis has the same mechanistic background as adolescent idiopathic scoliosis.

Preexistent Rotation of the Normal, Nonscoliotic Spine

The normal spine is a slightly prerotated structure in the horizontal plane. Its rotational pattern is identical in direction to what is found in the most prevalent curve types in scoliosis.[19,20] The direction of this horizontal-plane rotation changes with age and is related to a change in the distribution of internal body mass.[19,21,22] Once the spine decompensates into scoliosis, this preexistent rotation increases, following the already built-in pattern. The occurrence of preexistent rotation in the normal spine explains the predominance of right thoracic, left lumbar curves once the spine starts to decompensate, not only in idiopathic scoliosis.[23] Gradually, irreversible changes are induced, predominantly in the shape of the discs but later also in the anatomy of the vertebrae.[8,24,25] Bone metabolism plays a role in scoliosis, through the adaptation of the shape of the vertebrae due to asymmetric growth according to Hueter-Volkmann's law as well as bone adaptation through Wolff's law.[1,26–30] The same agents that govern bone metabolism may also have a still unknown effect on the mechanical properties of the discs and other stabilizing soft structures around the spine.

Consequences of the Human Fully Upright Sagittal Spinal Profile

The introduction of dorsal shear forces

Humans have a unique and individually different sagittal spinal profile, which leads to a unique biomechanical loading.[28–31] This unique sagittal profile starts already in the shape of the human pelvis, which has developed a lordosis between the ischial and iliac bone during its evolution.[32,33] This, together with the lumbar lordosis, and the fact that humans can simultaneously extend hips and knees, has brought the human body's center of gravity more dorsal than in any other species, essentially changing the biomechanical environment (Fig. 1). All spines in nature are subject to axial compression and anterior shear, but the human spine is the only one on which a third force comes into play, that is a posteriorly directed force acting on backwardly inclined segments (Fig. 2). This force, acting on that prerotated structure, decreases the rotational stability of the involved segments.[34–37] The existence of these shear forces, both anteriorly and posteriorly directed, has been questioned because the spine is conceptualized to undergo a follower load.[38] The follower load simulates the co-contraction compression forces of muscles necessary to stabilize the spine. In this concept, forces are tangential to the spine's sagittal curvatures. Also, other studies have analyzed loads that the spine, or parts of it, undergoes.[31] This, however, is by necessity an approximation of in vivo reality. There is no doubt that, in real life, in the sagittal plane shear loads in opposing directions exist, based on a number of observations. First, in degenerative spines, it is relatively common for vertebrae to slide either anteriorly or posteriorly, dependent on their orientation in the sagittal plane (Fig. 3). The difference in the direction of shear loading can also be recognized in the orientation of the trabeculae in individual vertebrae, as was shown by Gudde[39] (Fig. 4). This demonstrates that these shear loads exist, dependent on the orientation of each vertebra in the sagittal curvature of the spine.[40–43] The resultant vector is a combination of the direction of gravity but also the continuously acting muscle tone along that S-shaped structure.

Kouwenhoven and colleagues, in an ex vivo experiment, demonstrated that, under the influence of posterior shear loading, a reduction of rotational stability of the involved spinal segment occurs.[34] The longer the backwardly inclined area of the spine is, or the higher the backward inclination angle, the more these segments are prone to develop a rotational deformity, in other words, scoliosis.[35,44] Janssen and colleagues showed in an in vivo MRI study that rotation of the spine increases in healthy, nonscoliotic young adult volunteers if they moved from a quadrupedal-like to an upright position.[44] Homminga and colleagues showed in a biomechanical modeling study that also in old age, due to disc degeneration in combination with the posterior shear loads, the rotational stability of the spinal segments is significantly reduced, leading to degenerative scoliosis along the same mechanism.[45]

Sagittal profiles in children, especially the ones that develop scoliosis, were shown to differ significantly by Abelin-Genevois in her classification system in 2018 and by Pasha in 2019.[46–49] Schlösser and colleagues found that in nonscoliotic subjects spines of girls are more backwardly inclined than the spines of boys at the moment of maximal growth velocity.[50] Brink and colleagues showed that the pelvic incidence, the determinator of sagittal spinal alignment, is significantly higher in lumbar than in thoracic scoliosis and controls.[51,52] Schlösser and colleagues showed that early lumbar scoliosis has a different sagittal profile than does early thoracic scoliosis,[53] and de Reuver and colleagues showed that degenerative de novo scoliosis has similar spino-pelvic configuration as adolescent Lenke 5 scoliosis.[54] This all points to the role of pelvic morphology and the sagittal spinal alignment in the etiology of different types of scoliosis.

Behavior of the human spine as an S-shaped elastic rod

As described above, the sagittal shape of the human spine, and its individual variations, plays a role in the reduction of rotational stability of certain vertebrae that are posteriorly inclined. This same posteriorly inclined area of the spine is also at risk of undergoing a deformation if the spine is considered according to the mechanics of a flexible rod. Pasha showed that deformation patterns of an elastic rod, when the variations in the sagittal curvatures of the spine are considered, can mimic the deformation patterns of the scoliotic spine.[55–57] For this, the spine, as a whole, was modeled as an elastic rod under bending (as a result of gravity) and torsion (as a function of trunk mass asymmetry). The mechanics of deformation in elastic rods have been studied analytically for a long time, dating back to Bernoulli and Euler in the 18th century; this was modified by Cosserat, and more recently, it was numerically solved by computer power.[58] Considering the spine as a naturally curved elastic rod under bending and torsion was thought to be suitable for studying the pathogenesis of scoliosis for three reasons:

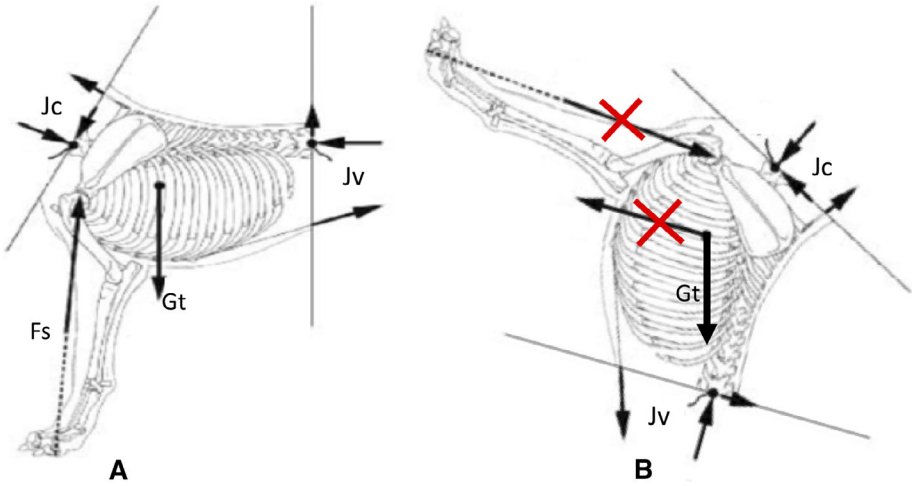

Fig 1. Simplified free body diagram of the trunk that shows the difference in loading in a quadrupedal (*A*) and human-upright position (*B*) of the same trunk, rotated to represent the human position. Jc and Jv are the reaction forces from the caudal and ventral parts of the trunk. The ground reaction of the front legs (Fs) disappears, whereas the gravity force of the trunk (Gt) changes its direction relative to the trunk. Obviously this loss of forefoot/arm ground reaction and change in gravity has consequences for the size and direction of the trunk forces Jc and Jv (3 each) in which the normal and shear forces on the spinal column are essential.

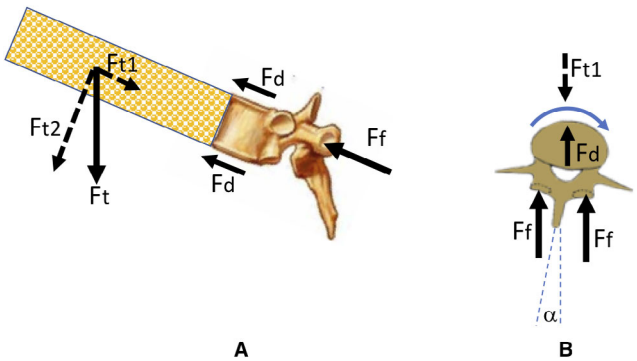

Fig 2. (*A*) Free body diagram of a slice of the trunk in the human position where the vertebra leans backwards. The gravity forces of the trunk have a component that points to the vertebra (Ft1), which should be compensated by the forces on the facet joints (Ff) and by the shear forces from the disc (Fd). (*B*) With a slightly rotated vertebra (angle a right figure), the trunk force (Ft1) further increases the preexistent rotation (blue arrow).

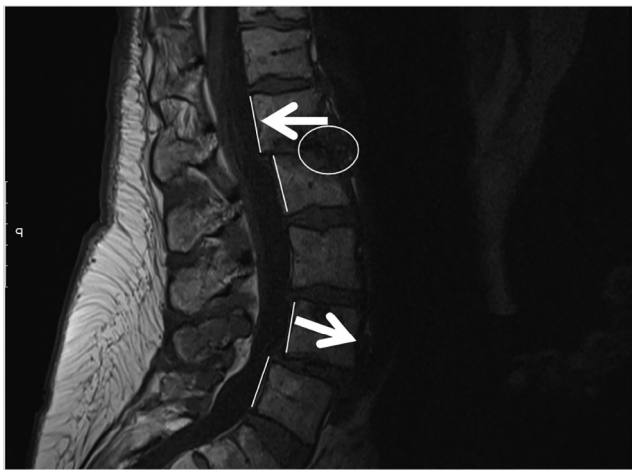

Fig 3. An MRI scan of a degenerative lumbar spine, showing at L_1 to L_2 both disc collapse (oval) as a result of the axial force and retrolisthesis (posterior arrow) as a result of the posterior force, and at L_4 to L_5 antelisthesis (anterior arrow) as a result of the anterior force.

1. the fast longitudinal bone growth at the onset of curve development and the reported slenderness of the spine in adolescent idiopathic scoliosis (AIS) patients, with a natural sagittal curvature, which satisfy the requirement of the equations for a thin rod where the rod diameter is orders of magnitude smaller than the length of the rod;[59]
2. a decrease in axial support of the spine due to rapid changes in the height, weight, and moment arm, and additional laxity due to hormonal changes around puberty that add to flexibility;[60,61]
3. the loading patterns of the spine that can be considered as bending resulting from gravity and an axial torsion representing a shift in the position of the center of mass around puberty.[22]

Relying on these assumptions, in a recent study, Pasha developed finite element (FE) models of the curved elastic rods with different geometries of their sagittal curvature. The geometry of these curvatures was derived from the main sagittal curvature of the scoliotic spines in right thoracic scoliosis.[55] These sagittal curvatures were determined from a previous statistical 3D classification that divided a cohort of 103 right thoracic AIS in five subgroups.[47] Notably, contribution of kyphosis and lordosis to the overall sagittal curvatures of the spine differed between these

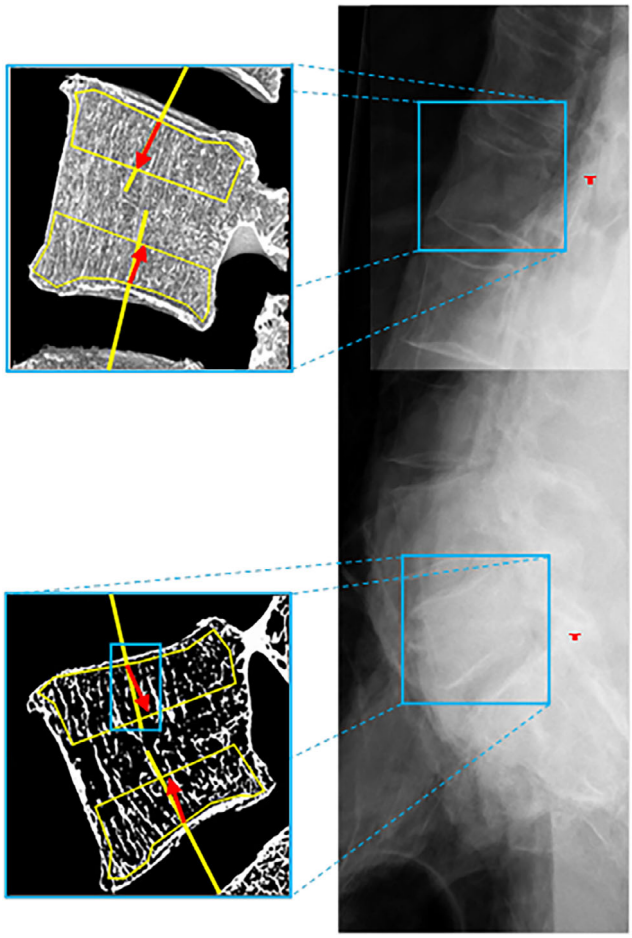

Fig 4. Two vertebral bodies (L_1 and L_5) that underwent 3D micro-CT analysis and a precise calculation of the main trabecular orientation. The calculated principal trabecular orientation is shown as a red arrow, whereas the yellow line shows the normal of the proximal and distal surface of the vertebral body. The calculations were performed on the trabeculae in the vertebral bodies from cadaver specimen in the upper 1/3 and lower 1/3 of the vertebral bodies' volume. There is a clear difference between the calculated main orientation of the trabeculae and the surface normal in particular for L_5 (lower panel). It is hypothesized that the trabecular orientation is in the main loading direction that is a consequence of both the normal (compressive) force and the shear forces. With higher shear forces, the deviation between the trabecular orientation and the surface normal is larger (L_5). With permission from Gudde and colleagues (master thesis, TU Delft, 2018).

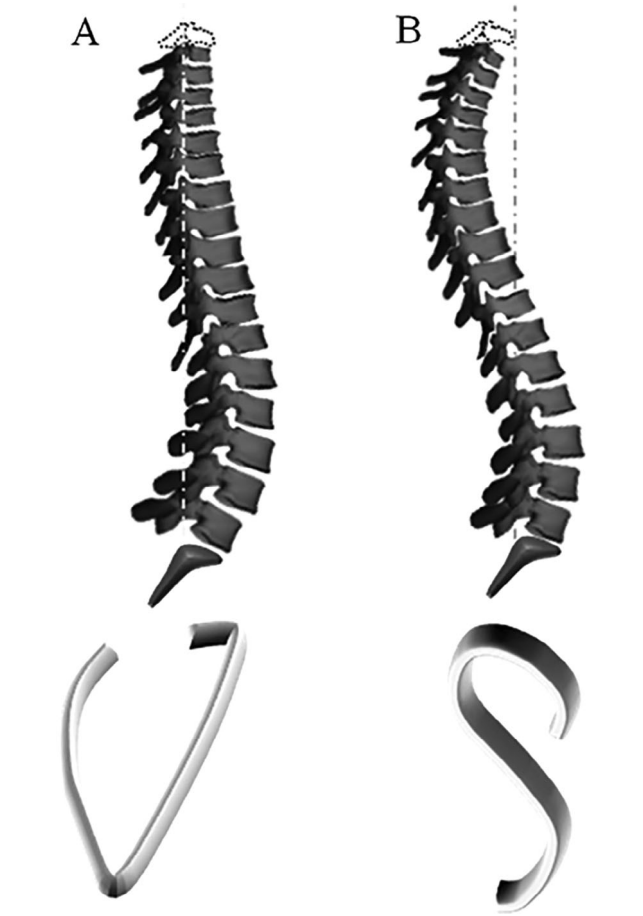

Fig 5. The two characteristic subtypes of sagittal profiles: (*A*) a long lordosis and (*B*) relatively longer kyphosis compared with lordosis. The axial projection of each sagittal profile (loop for type *A* and lemniscate for type *B*) is shown.

different subtypes. The two dominant main sagittal curvatures in this cohort were a subset of patients with a spine that was mainly lordotic and a second subset that was slightly more kyphotic than lordotic.[47] These two different sagittal profiles were deformed under gravity and torsion in the FE model, and yielded the same deformation pattern also in the horizontal plane (one a loop shape, the other a lemniscate shape), as was observed in the real spines with the same sagittal curvature characteristics (Fig. 5).

The prerequisite of deformation in this model was a slight torsion that initiates the twist. Evidence for such a twist as a prerequisite has been demonstrated in the literature.[19–22] The mechanics of deformation of the rods can explicitly show that the moments acting on the rod change as a function of the geometrical curvature of the rod. This explains different patterns of the coronal plane spinal deformity development and the relationship between the initial geometry of the spine that is sagittal profile and the deformation patterns.[62] As the continuous deformation of the rod changes its geometry, the forces that cause the geometrical deformation evolve as well. The direction and magnitude of the bending moment varies as a function of the curvature and the twist in the centerline of the spine that is caused by the initial, preexistent torsion. It can be concluded that, for the spines with a long lordotic curve, a majority of the moments, determined by the geometry of the curve, act in the same direction, causing a long section to rotate in one direction, resulting in a loop-shaped axial projection. When the length and curvature of the kyphotic and lordotic sections of the spine are more similar in magnitude, however, the two curves bend off-plane in the direction of the governing moments, thus forming a torsion in the spine with, in the transverse projection, a lemniscate shape (twisted loop) (Fig. 5). A similar top-down projection of the scoliotic spine was also observed by Dubousset.[15] This mechanism can explain the initiation of curve development on pure mechanical grounds. The deformation concept explained

here is not based on the Euler's critical load as used before to describe stability in ligamentous spines[63–65] but is an elastic reversible deformation that depends on the magnitude of the loading and flexibility of the rod. As such, variations in properties of the bone and soft tissues will have its impact on the ultimate behavior per individual. The proposed model also does not consider the role of the stabilizer mechanisms of the spines such as muscles or discs,[66] as the main purpose of the model was to relate the deformity patterns to the variations in the sagittal curvature of the spine. The progression of the curve can be explained by the effects of a vicious cycle, initially in the soft tissue, but at a later stage also in the bony architecture by Hueter Volkmann's principle during growth.[67–69]

Growth

Idiopathic scoliosis develops often during the period of rapid pubertal growth, which has led to the assumption that growth is abnormal in scoliosis.[70,71] Both body height and body mass increase dramatically during adolescence; this may suggest mechanisms of altered growth of the spine to play a role in the initiation of idiopathic scoliosis.

Growth, however, is not a prerequisite for scoliosis to develop in a previously straight spine in an otherwise healthy person. Scoliosis occurs predominantly during two vital phases of life, in which important changes occur:

1. Puberty, when loading of the spine increases rapidly because of the growth spurt, leading to an increase in both body mass and moment arm. This increasing load puts high demands on the stabilizers of the spine, predominantly the intervertebral discs that are in the course of their own process of maturation. Usually these processes are in balance and no deformity ensues, but in a minority the maturation of the stabilizing structures may lag behind the sudden increase in mechanical demands.
2. Older age, when disc degeneration initially leads to a decrease in the disc's rotational stiffness under more or less constant loading, allowing the inherent tendency to rotate to finally become manifest in what is called degenerative scoliosis.[45,72]

Of course, many underlying factors may play a role in whether the spine will decompensate; genetics, proprioception, metabolomics, and even inflammatory processes may all influence at a certain point the biomechanical behavior of the tissues concerned and thereby the rotational stability of the spine in the horizontal plane. Whether that will result in a scoliotic deformity depends on a number of these factors coinciding in time, in what can be called a "perfect storm."

We showed that altered growth predominantly plays a role in the progression of scoliosis once the process has already started, not in its initiation phase.[73]

So-Called Relative Anterior Spinal Overgrowth

For many decades, it has been known that in scoliosis, the anterior part of the spine is longer than the posterior part; that is, the global thoracic kyphosis is transformed into a rotated apical lordosis.[74–77] This has led to the assumption of causality; relative anterior spinal overgrowth (RASO) was considered a generalized bony overgrowth disorder of the anterior spine as part of AIS etiology.[78,79] However, a number of more recent observations contradict this etiological model. Additional anterior length in AIS was not observed in the junctional zones between the curves, thus not fitting the theory of a more generalized growth disturbance.[4] Furthermore, this anterior lengthening was observed significantly more in the intervertebral discs than in the vertebral bodies. The bony morphology did not differ much from nonscoliotic controls.[5,6] Lastly, similar length discrepancies were also observed in other types of scoliosis.[4–7,9]

Low Bone Mass and Abnormal Bone Qualities

A number of recent studies have demonstrated a correlation between a low bone mineral density (BMD) and the occurrence of adolescent idiopathic scoliosis.[1,26–29,80,81] This low BMD seemed to be unrelated to the effect of bracing.[82] It appeared to be a generalized phenomenon, and it was observed not only in the hip or spine but also in the tibia, radius, and os calcis and involved both cortical and cancellous bone. The low BMD was associated with abnormal bone qualities affecting the volumetric BMD, bone microarchitecture, and lower bone mechanical strength that could persist beyond skeletal maturity and was shown to have prognostic significance on curve progression.[83–85] This points at a disturbed balance between bone resorption and formation, that is, osteoblast and osteoclast activity. Osteocytes in scoliosis patients were shown to have a different shape and are clustered in an abnormal manner.[86]

This process is under the influence of a number of hormones, such as melatonin, leptin, and estrogen, and corresponding pathways that have been described to be disturbed in scoliosis.[87] Little is known of the effect of all agents that are related to bone metabolism on the stabilizing soft structures surrounding the vertebral column such as the intervertebral discs.

Discussion

This article addresses idiopathic scoliosis as a mechanical, rotational decompensation of the human spine, based on its unique and individually variable sagittal profile.

The nonscoliotic human spine is a prerotated and prebent structure on which, because of its sagittal alignment, three forces act: axial compression, anterior shear, and dorsal shear (Fig. 3). This last force is unique for the human race and was shown to render backwardly inclined segments rotationally unstable.[34] The importance of these backwarldy inclined vertebrae in the development of different curves was demonstrated in a number of studies.[1,34–37,44,45,50,52–54]

Pasha, using a different approach, also showed that the more backwardly inclined vertebrae are most prone to start a decompensation into a scoliotic deformation. She showed that, because of the characteristics of the human spine in the sagittal plane, its mechanical behavior can be compared with that of a prebent S-shaped elastic rod.[55] Differences in sagittal profile determine which vertebrae will be at risk to start the deformation and thus which curve type will ensue.

This scoliotic decompensation is not exclusive to the period of rapid growth. In older age, de novo curves may arise in backwardly inclined areas of the spine in previously healthy persons. We propose that both adolescent and degenerative scoliosis are different ends of the same spectrum, both at ages when the balance between the deformation-inducing forces and the spine's stabilizers is disturbed. In adolescence, a sudden increase in loading of the spine during a vulnerable period when, in some,

the discs and other passive stabilizers may not have matured mechanically may disturb that delicate balance in 2% to 4% of the population. Subsequently, when the disc degenerates in older age, it loses its torsional stiffness and the deformity that becomes manifest is called degenerative scoliosis.[45,72] It has been shown that bone quality plays an important role in the development of a scoliosis, through the adaptation of the bony vertebral shape, but possibly also through still unknown interaction between bone and surrounding soft tissues.[1,26–29,80,81,86]

Scoliosis has a chance to develop during a relatively small window of opportunity. In adolescents, it will have no chance to develop once the disc has matured, and in old age, once the disc reaches the phase of ankylosis, it will no longer allow a scoliosis to develop either. Both adolescent idiopathic and degenerative scoliosis are a severe burden to the patient and society, treatment is very costly, and the results, although better than ever, are far from perfect. The challenge for the near future is to recognize those at risk for developing either deformity and to protect the spine from going into this rotatory decompensation during this limited window in time by external means but, if necessary, with temporary and motion-preserving implants.

Disclosures

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Peer Review

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