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Three-dimensional spring model: A new hypothesis of pathogenesis of adolescent idiopathic scoliosis

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SUMMARY

The pathogenesis of adolescent idiopathic scoliosis (AIS) has been the subject of many studies, but remains little understood. Previous work has shown that there is a correlation between the uncoupled spinal neuro-osseous growth and AIS. We believe that this uncoupled spinal neuro-osseous growth may also contribute to formation of normal curvature of the spine in the sagittal plane during the childhood. We speculate a three-dimensional spring model to better understand our hypothesis. The normal curvature of the spine, the uncoupled spinal neuro-osseous growth, and the overgrowth of the spine in the puberty may be the crucial factors in the pathogenesis of AIS.

Background

After more than a century of dedicated research into the origin of adolescent idiopathic scoliosis (AIS), the cause of this disorder remains unclear. Many theories exist on how a child, usually a girl, can develop normally until the onset of pubertal growth. Nonetheless, no single cause has been identified, therefore the condition is termed multifactorial. However, there are a few, well-established facts: (1) AIS is a three-dimensional deformity of the spine. (2) AIS occurs in otherwise healthy children, predominantly girls, up to their growth spurt, no visible distinction from their peers. (3) In AIS anterior parts of vertebral grow faster than the posterior parts. Growth thus plays a role in the development and progression of AIS. And there are two important hypothesis which speculate that growth may be the causative factor of AIS.

Relative anterior spinal overgrowth (RASO)

During the last 80 years anatomical studies and recently magnetic resonance imaging (MRI) have established that in structural scoliosis the anterior components of the spine are longer than the posterior elements. Somerville [1] concluded that the deformity of thoracic idiopathic scoliosis consists of lordosis, axial rotation and lateral flexion and suggested that lordosis results from failure of growth of posterior elements of a segment of the spine. This concept was further developed by Roaf [2] and then by Dickson [3,4], they ascribed pathogenetic significance to the thoracic hypokyphosis in the sagittal plane.

Spring model

Roth [5,6] speculated that idiopathic scoliosis is a disproportion of vertebra-neural growth due either to a short spinal cord or a too rapid growth spurt of the spine. In his spring model Roth found that shortening of the string hindered free elongation of the spring resulting in a scoliotic deformity of the model (Fig. 1).

> Roth wrote: The tension of the spinal nerve roots under normal conditions is distributed symmetrically on both sides of the midline, and the straight course of the spine is thus maintained. If, however, at any moment during the growth period the cord begins to lag too much behind the vertebral column, the neutral tension will be abnormally increased. Let us suppose at first that this increase in tension is asymmetrical, i.e. unilaterally higher. Correction of the disturbed tension equilibrium is then achieved by lateroflexion of the spine towards the side of increased tension. (Roth M: Idiopathic Scoliosis caused by a short spinal cord, Acta Radiol Diagn (Stockh). 1968 May;7(3):257–71, page 261, line 26–36).

Chu [7] found that in severe AIS compared with normal subjects, the vertebral column is significantly longer with no detectable change in spinal cord length. They speculate that the initiation and progression of AIS result from vertebral column overgrowth through a lordoscoliotic maladaptation of the spine to the subclinical tether of a relatively shorter spinal cord.

The hypothesis

During the lifetime, human beings have two growing spurts. The first growing spurt appears from the birth to age two with the height velocity 8–10 cm/year, while the second growing spurt appears at age 10–12 for girls and age 12–14 for boys with the

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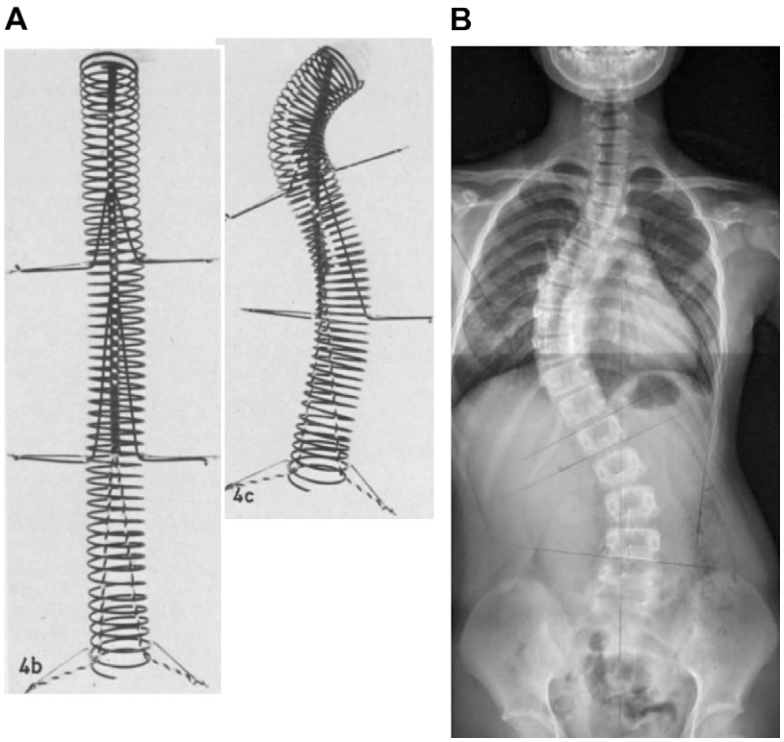

Fig. 1. (A) the spring model modified by Roth. (B) Coronal X-ray imaging of an AIS patient.

height velocity 6–8 cm/year. If growth is the causative factor of AIS, why there are so few idiopathic scoliosis patients at the first growing spurt compared with the large amount of them at the second? (Fig. 2).

Over the gestational period, the vertebral column grows disproportionately faster compared to the spinal cord [8], which results in the ascent of the spinal cord and formation of the cauda equina. The conus ascends relative to the vertebrae, and the filum elongates. Similarly, nerve roots exiting at the levels of their respective foramen must grow longer to accommodate this relative ascension of the spinal cord, thus forming the cauda equina. By 19 weeks gestation, the conus has achieved its adult level, which is typically at or near the L1/L2 interspace [9].

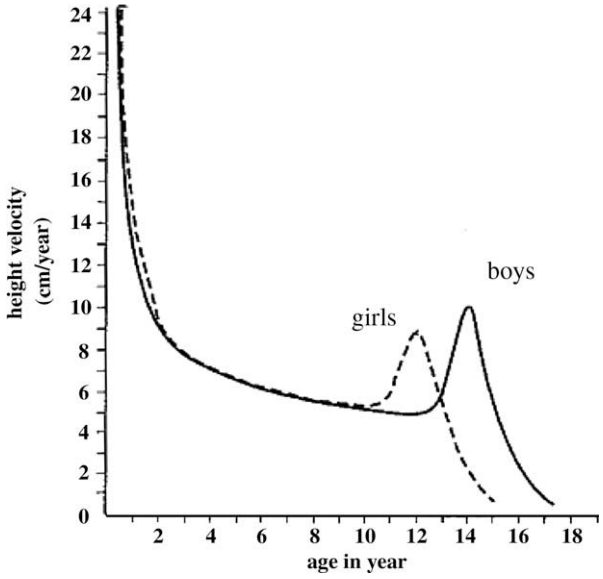

Fig. 2. Human beings have two growing spurts, but most of AIS appears at the second.

We consider that the disproportion of vertebra-neural growth not only makes the conus ascend, but also pushes the spine column change its shape. The human embryo has a very mild posterior convex curve seen on the lateral view during the prenatal period. In the newborn, a slight curvature of thoracic kyphosis and lumber lordosis appears. As the child continues to grow, the spinal curvature continues to develop into its adult configuration [10], with its primary curves of kyphosis at the thoracic and sacrococcygeal levels and secondary curves of lordosis at the cervical and lumbar levels. We use the spring and string to simulate the spine and spinal cord, and we add a magnet under the board to simulate the restriction of muscle and ligament, the shortening of the string hindered free elongation of the spring resulting in a "S" shape deformity of the model. The uncoupled spinal neuro-osseous growth may be the crucial factor which contribute to the spinal curvature development in the sagittal plane. We consider that all of the children have "scoliosis" during their childhood, but this "scoliosis" is the normal curvature of spine in sagittal plane (Fig. 3).

With the growing velocity of spine increases again at the second growing spurt, the spinal cord is relatively shorter than the vertebral column. The growing spine has to adapt this by flexing more in the sagittal plane [10]. We can simulate this situation by the spring model, but we found that with the curvature of the spring developed, the force that makes the curvature move to the other plane is increasing too. When the magnet cannot stop this force, the spring will have a scoliotic deformity by axial rotation and lateral flexion. This is our hypothesis: the spine has to adapt to the relatively shorter spinal cord by increasing its curvature on the sagittal plane. But with the curvature increases, the force that makes the curvature move to the other plane increases too. If the spine grows too rapidly, the muscle and ligament can not resist the increasing force, the scoliosis will appear (Figs. 4 and 6).

We consider that the uncoupled spinal neuro-osseous growth is the cause of normal curvature of spine in the sagittal plane at the