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The development of adolescent idiopathic scoliosis

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Abstract There are many conflicting actiological theories for adolescent idiopathic scoliosis. We present a simple new model of scoliosis and a mechanism by which it is initiated and progresses. This mechanism provides a final common pathway for the multiple aetiological factors. A simple model of the spine, incorporating its fundamental mechanical features, was constructed. The model consisted of interconnected anterior compression and posterior tension columns. It allowed normal spinal movements, with flexion limited by the posterior column and rotation centred around the anterior column. It also allowed deformities to develop. The ends of the model were fixed in the position of the vertebrae they represented. Overgrowth of the anterior column relative to the posterior column caused the model to take up the shape of an idiopathic scoliosis. The greater the overgrowth, the more marked the deformity. Normally anterior and posterior column growth are coupled. During the growth spurt the thoracic kyphosis flattens indicating that anterior growth temporarily exceeds posterior growth. If this overgrowth is marked a scoliosis will develop, as demonstrated by the model. Once this occurs the coupling is lost, anterior growth further outstrips posterior growth and the deformity progresses. Not all scolioses worsen, as the tendency to progress is balanced by neuromuscular factors and remodelling. Factors that increase the growth rate, induce asymmetry or decrease the inherent stability of the spine all encourage the development and progression of a scoliosis. This explains the complex biomechanics of scoliosis and provides a final common pathway by which the multiple aetiological factors can induce idiopathic scoliosis. It has important implications for the understanding and treatment of this condition.

Key words Idiopathic scoliosis · Model · Aetiology

Introduction

Adolescent idiopathic scoliosis is a complex three-dimensional deformity that usually occurs in the thoracic spine [10]. Its aetiology is multifactorial and poorly understood, with many conflicting theories [16, 18, 20, 29]. There is little that can be offered in the way of treatment for minor curves. For major or rapidly progressive curves the treatment is surgical, the aim being to correct the deformity as much as possible, and to prevent it from recurring by fusion. This is a major undertaking for what may just be a cosmetic problem that develops during growth. An ideal treatment would be one in which the biomechanical environment was altered, encouraging the deformity to decrease spontaneously. This would use rather than destroy the potential for growth and remodelling. New types of treatment based on this rationale cannot be developed un-

til there is a greater understanding of the aetiology and biomechanics of this condition.

Idiopathic scoliosis is a deformity that has components in all planes [10]. The "coronal plane" deformity, demonstrated by AP radiographs, consists of lateral curvature or buckling. In the centre of the curve the vertebrae are rotated so that their spinous processes lie on the concave side of the curve. Although this is known as the "transverse plane" deformity, the vertebrae are rotated in the oblique plane in which they lie rather than in the true transverse plane. There is also a complex deformity in the sagittal plane. In the true sagittal plane the spine may appear kyphotic [27]. However, radiographs of the curve apex taken lateral to the rotated vertebrae (and perpendicular to the plan d'election of Stagnara) show that the normal thoracic kyphosis is reduced (hypokyphosis) or even reversed to a lordosis [11, 24]. This hypokyphosis or lordosis is known as the "sagittal plane" deformity, even though it is not actually in the sagittal plane, as the vertebral column has rotated out of the sagittal plane. The presence of the lordosis has been confirmed by direct measurement of cadaveric scoliotic spines, as the anterior column is longer than the posterior column [8, 28]. Above and below the primary curve are compensatory curves. The vertebrae at the end of these curves, the neutral vertebrae, have a normal alignment as if there were no scoliosis in between, if the scoliosis is balanced [10]. In particular, the angle between the upper thoracic vertebrae and the lower thoracic vertebrae measured in the true sagittal plane is the same in normal spines and spines with thoracic scolioses [26].

From a mechanical point of view the spine consists of two columns that are connected at regular intervals. The anterior column consists of the vertebral bodies and intervertebral discs, and resists compression [21]. The posterior column consists of ligaments and therefore resists tension. The facet joints control the movements that occur between vertebrae. In the thoracic spine the facet joint surfaces are arranged so that vertebrae can rotate around the axis of the anterior column [46], whereas in the lumbar spine rotation is prevented as the centre of the facet joints do not coincide with the centre of the discs [46]. The facet joints also allow lateral bending and flexion/extension [21]. In vivo and in isolated spines flexion is limited by the posterior ligaments [34, 44]. Of these, the supraspinous ligament is probably the most important ligament for preventing extreme flexion, because of its strength and long lever arm [19]. The total range of flexion/extension in isolated thoracic spines is about 70°, which is considerably more than the total range in vivo [15, 45]. Therefore, in vivo extension must be limited by a structure extrinsic to the spine. This structure is likely to be the rib cage [2].

Numerous models of scoliosis have been described. These range from simple two-dimensional sketches representing the deformity in the coronal plane [22] to complex three-dimensional computer reconstructions [13]. The more tangible models include those in which the spine has been considered to be like a triangular prism [7, 8], a strip of cardboard [28] or rubber [4], a column of wooden blocks [3], two columns of corks [17], a flexible rubber tube with elastic bands attached posteriorly [30], a spinal column with a postero-lateral wire [14] and a column of wooden blocks connected to two longitudinal posterior wires [37]. None of these incorporated all the fundamental mechanical features of normal and scoliotic spines described above. We present a simple new model that incorporates these basic constraints and seems to explain the biomechanics of idiopathic scoliosis. We also present a possible mechanism by which a scoliosis is initiated and progresses, and which provides a final common pathway for the multiple aetiological factors.

Model

In the model the anterior compression column of the spine was represented by a flexible plastic rod, which could both bend and twist. Small amounts of bending or twisting equate with the normal movements that occur at the intervertebral disc, whereas large distortions of the rod equate with the deformities that may develop in both disc and vertebral bodies under prolonged abnormal loads [42]. Rigid struts were attached to the plastic rod at regular intervals, these represented the posterior vertebral elements. The posterior tension column, typified by the supraspinous ligaments, was represented by a string attached to the end of each posterior strut. The model allowed rotation around the axis of the rod as well as flexion/extension and lateral bending. Flexion was limited by the posterior string. The only external constraint applied to the model was that the two ends of the rod were held rigidly at a constant angle to each other, with the upper end flexed relative to the lower. This was to reproduce the situation found in vivo, where the relationship of the vertebrae at the top and bottom of the scoliosis is the same as it is in normal controls [26], as a result of the extrinsic constraints such as the rib cage [2]. When movement was investigated the constraints at the end of the rod were relaxed to allow a small amount of motion.

In a normal thoracic spine the anterior column is shorter than the posterior column. A model set up in this manner took up the shape of a normal spine (Fig. 1). The model allowed normal flexion/extension, lateral bending and rotation without showing any tendency to buckle.

In idiopathic scoliosis the anterior column is longer in relation to the posterior column than it is in normal spines. To reproduce this, the model was constructed with the anterior and posterior column the same length. The model then, quite spontaneously, took up the three-dimensional shape of an idiopathic scoliosis (Fig. 2): it buckled and rotated, with the posterior elements directed towards the

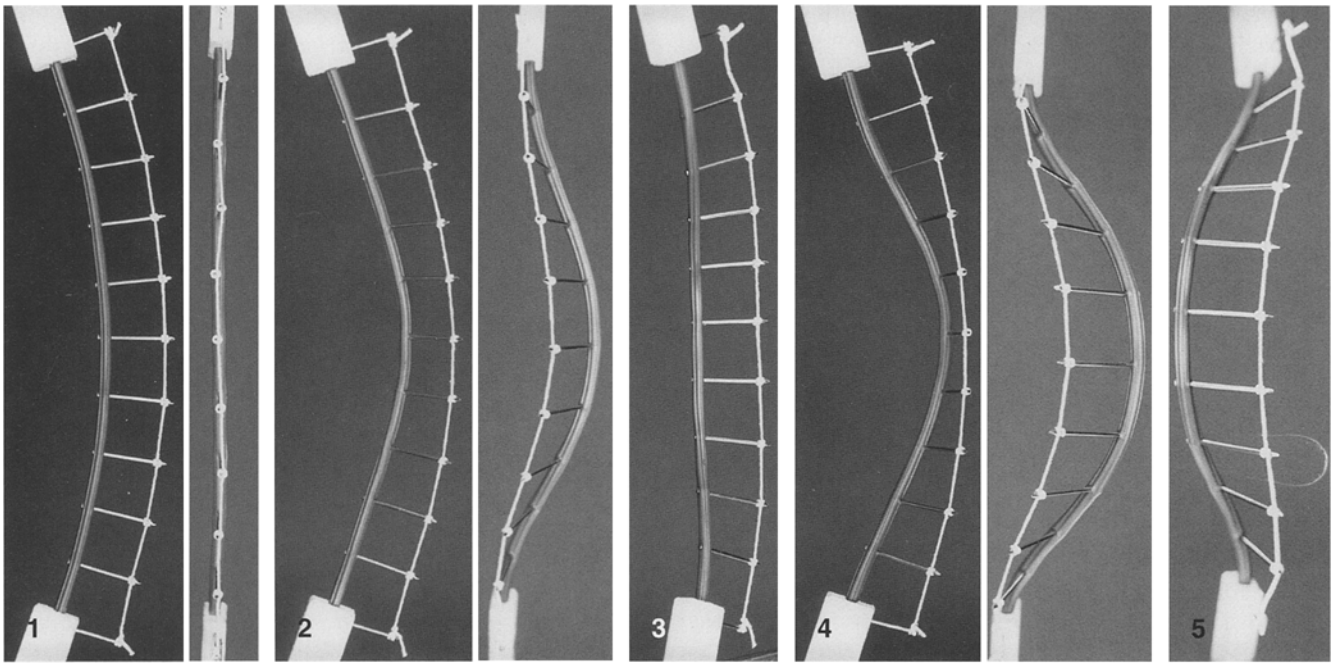

Fig. 1 Lateral and PA views of the model, constructed with the anterior column shorter than the posterior column

Fig. 2 Lateral and PA views of the model, constructed with the anterior and posterior columns the same length

Fig. 3 View perpendicular to the apex. Model constructed with the anterior and posterior columns the same length

Fig. 4 Lateral and PA views of the model, constructed with the anterior column longer than the posterior column

Fig. 5 View perpendicular to the apex. Model constructed with the anterior column longer than the posterior column

concavity. On a true lateral view of the apex the spine was seen to be hypokyphotic (Fig. 3). Compensatory curves developed above and below the primary curve. A slight increase in flexion of the whole spine increased the rotatory deformity of the apical vertebrae. As vertebral rotation is related to the size of the rib hump, this finding is similar to the (disputed) clinical observation that the rib hump becomes more marked in flexion [11, 40, 41].

As a scoliosis deteriorates, the anterior column becomes progressively longer than the posterior column. A model was constructed with the anterior column longer than the posterior. This was found to take up the shape of a gross scoliosis (Fig. 4). When the apical vertebrae were viewed from their true lateral the kyphosis was found to have reversed and the spine was now lordotic (Fig. 5). However, when the whole model was viewed from its true lateral the kyphosis of the thoracic spine was still present (Fig. 4). This paradox is characteristic of idiopathic thoracic scoliosis, and is caused by the marked rotation of the apical vertebrae.

A model of the lumbar spine was also constructed. The lumbar spine is usually lordotic and is not directly connected to the rib cage, so the extrinsic constraints that limit spinal extension in the lumbar spine have much less effect than those in the thoracic spine. The lumbar spine can flex until it is straight, so the posterior elements, which resist flexion, must be the same length as the anterior elements. The model demonstrated that for a primary lumbar scoliosis to occur, there must be marked relative overgrowth of the anterior column combined with abnormally restricted extension. This combination is less likely to occur than is the small amount of anterior overgrowth associated with a thoracic scoliosis. This may explain why idiopathic scoliosis is much more common in the thoracic than the lumbar spine [10]. The facet joints in the normal lumbar spine do not allow rotation [46]. However, in lumbar scolioses there is considerable rotation [30]. For this to occur there must be deformity of disc and joint [30]. This may be another reason why lumbar curves are not as common as thoracic ones.

Mechanism

Fusion of the epiphyseal growth plates in the limbs stops longitudinal growth of both the bones and associated soft tissues [25]. In contrast, progressive distraction causes tension and growth in soft tissues [43]. It seems likely, therefore, that growth at the growth plate causes elongation of the limb, and by generating tension in the soft tissues, causes soft tissue growth. By analogy, it is likely that longitudinal growth of the spine is generated by the growth plates of the vertebral bodies, and this causes tension and thus growth in the posterior ligaments. Growth plate growth is influenced by the load across the growth

plate, so it is likely that the tension in the posterior column will tend to constrain the growth of the vertebral bodies [23, 42, 49]. Under normal circumstances the rate of growth of the anterior column will be in equilibrium with the posterior column. If this equilibrium is lost, and the growth in the anterior column outstrips the growth in the posterior column, then a scoliosis will develop as demonstrated by the model.

During the adolescent growth spurt the thoracic kyphosis flattens slightly, suggesting that the growth in the anterior column does marginally outstrip the posterior column [48]. Fortunately, in most instances the posterior column soon catches up and the thoracic kyphosis returns to normal. A few adolescents will have particularly rapid anterior column growth, and in these the equilibrium between anterior and posterior column growth can become so upset that a scoliosis will develop. The spine is likely to buckle to the right because of vertebral asymmetry caused by the aorta [11]. A column that has buckled loses much of its ability to support load. The tension and rate of growth in the posterior column will therefore decrease. Similarly, the constraint that the posterior elements put on the longitudinal growth of the vertebral bodies falls, so the rate of growth of the vertebral bodies increases. The loss of equilibrium in growth between the anterior column (accelerated by loss of load) and the posterior column (slowed by loss of stretching force) will cause a progressive increase in the scoliosis. This effect will be most pronounced at the apex of the curve, and this is indeed where the most marked lordosis is found [6]. Once an appreciable curve has developed the loads on each side of the vertebral bodies and intervertebral discs become very unequal. This causes asymmetrical growth of both discs and vertebrae. The vertebrae become wedged and the scoliosis becomes structural [38].

Fortunately, not all mild scolioses progress. Numerous muscles and ligaments support the spine and tend to prevent buckling. It has been demonstrated in an animal model that vertebral remodelling and bone drift tends to bring the vertebral bodies back towards the mid-line [36]. Only if the rate of buckling is faster than the remodelling will the scoliosis deteriorate. Once started, the deterioration will progress as the spine becomes increasingly unstable.

Discussion

Aetiology

Although the aetiology of adolescent idiopathic scoliosis is multifactorial, it is likely that most cases develop by a final common pathway [12, 20]. The difference between the models of scoliotic and normal spines is the relative length of anterior and posterior columns. This difference is not necessarily the final common pathway, but may be secondary to primary events that are extrinsic to the spine. However, in growing animals an identical deformity to idiopathic scoliosis can be produced by tethering the posterior column [35, 37]. In contrast, if other possible pathways, like abnormal rib growth or neuromuscular problems, are reproduced in animals the resulting deformities are not always identical to idiopathic scoliosis, as there may be little or no rotation or the rotation may be in the wrong direction [1, 5, 9, 31–33]. We therefore believe that the relative difference in length between anterior and posterior column is the final common pathway to scoliosis.

Adolescents with a particularly high growth rate during the growth spurt, for genetic or other reasons, are particularly likely to start on the pathway to scoliosis, because they are likely to have a relatively large amount of anterior overgrowth. This may be the reason why patients with idiopathic scoliosis tend to be taller than age-matched controls [47]. Any factors that decrease the inherent stability of the spine, and thus decrease its resistance to buckling, increase the likelihood of a scoliosis developing. These factors include neuromuscular and ligamentous problems, and metabolic and chemical abnormalities [20]. Similarly, any factors that tend to induce asymmetry, for example altered rib growth, also predispose to scoliosis [32].

Models

A model of idiopathic scoliosis should incorporate the fundamental mechanical features of normal and scoliotic spines. It should also accurately represent the deformity in three dimensions. Above all, a model only acquires real value when it is so simple and logically complete that it can be represented mathematically. It can then be validated, as it can be studied analytically [39]. We believe that our model, unlike previous models, fulfils all these criteria and provides a three-dimensional biomechanical explanation of idiopathic scoliosis.

The new model differs from previous models in various ways. The most important features present in the new model that are not all present in other models are as follows: the model consists of separate single symmetrical tension and compression columns; the upper and lower parts of the model are fixed in the position of the vertebrae they represent; the deformity is generated by a generalised imbalance of growth between anterior and posterior columns, rather than a localised abnormality.

Most models represent some of the features of scoliosis. For example, when strips of rubber [4] or cardboard [28], or columns of corks [17] are flexed they buckle and rotate with the posterior elements directed towards the concavity; however, when extended slightly they buckle and rotate, with the posterior elements directed towards the convexity. This type of rotation does not occur clinically, nor does it occur with the new model or with the

model of Somerville [37], as in both of these the posterior columns only resist tension. Somerville's model has two parallel posterior tension columns, suggesting that the facet joints or their capsules limit flexion. In Somerville's model a scoliosis is generated by a two-level structural lordosis, which rotates when the spine is flexed. It is difficult to imagine how the final common pathway of the various factors causing idiopathic scoliosis is the generation of such a localised structural lordosis. Although such a lesion, which for example may occur after radiotherapy, undoubtedly can cause a scoliosis, our model suggests that it is more generalised anterior overgrowth that initiates the scoliosis, and once established the forces causing abnormal bone growth are maximal at the apex so that this becomes the site of the greatest lordosis [8].

The triangular prism model of scoliosis was first described by Deane and Duthie [8] and then further developed by Dickson [7, 10, 11]. Essentially, a triangular prism is more likely to buckle if flexed towards rather than away from the apex of the prism, and when it buckles it rotates. It is suggested, without any biomechanical evidence, that vertebrae in the thoracic spine behave like prisms with their apexes anteriorly, whereas vertebrae in the lumbar and cervical spine behave like prisms with their apexes posteriorly. Therefore, when flexed, buckling is more likely to occur in the thoracic rather than lumbar or cervical regions. However, the model also predicts that when the spine is extended buckling will occur in the lumbar spine. This buckling would be associated with rotation in the opposite direction to that which occurs clinically. The model has been developed to include the principles that it is a short lordotic segment that causes a scoliosis (discussed above) and that a lordosis is less rotationally stable than a kyphosis. Cadaver studies [15] and our studies with models (unpublished) do not support the assertion that a lordotic thoracic spine is less rotationally stable than a normal spine.

Scolioses are generated in different ways in different models. In some the deformity is generated by vertical [14] or lateral [30] loads. This type of loading tends to cause small amounts of rotation. In the majority of models the deformity is generated by flexing the model. This flexion is not fundamental to idiopathic scoliosis clinically. In the new model the ends are constrained in the positions that occur both in normal spines and scoliosis and the deformity is generated by unequal growth in the anterior and posterior columns. Although rigid constraints are not present in vivo, there are undoubtedly some constraints that tend to keep the head above the pelvis [37].

Posterior tether

A scoliosis was generated because the anterior column was relatively long compared with the posterior column. It is therefore similar to the posterior tethering that is thought by some authors to be the cause of scoliosis [28, 37]. It is, however, fundamentally different from the experimental model of a posterior tether developed by Jarvis [14], which induced 2° of rotation in a human cadaveric spine. In this model a vertical load was applied to the spine to induce lateral bending. The posterior tether, which was offset laterally, prevented the posterior elements from being deflected as far laterally as the anterior elements, and thus induced rotation. With the new model, a vertical load is not necessary for a scoliosis to occur, although it does make the scoliosis worse. The posterior tether has been dismissed by some authors, although the reason for this is not always clear [30]. Stokes and Gardner-Morse [39] showed with finite element analysis that a postero-lateral tether could not induce a scoliosis. Even though this analysis did not represent our model in a number of ways, including the lack of symmetry and different end constraints, we believe the reason why it failed to generate a scoliosis was that it did not adequately take into account the asymmetrical growth and deformity that is induced in both bone and cartilage by abnormal loading [42], because this is not well understood.

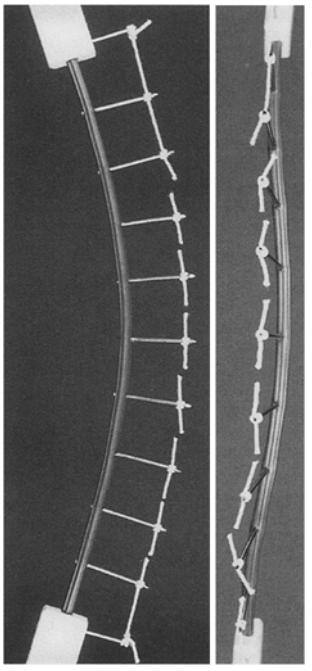

Fig. 6 Lateral and PA views of the model, constructed as in Fig. 2 with the anterior and posterior columns the same length, after division of the posterior elements

Developments

The model provides a simple explanation of idiopathic scoliosis. More sophisticated versions incorporating the basic features of the current model could be used to test and improve spinal instrumentation. Using the model, new treatment approaches could also be developed. Instead of destroying the potential for growth and remodelling by fu-

sion, these new approaches would alter the mechanical environment of the spine, allowing normal growth and remodelling to decrease the deformity spontaneously. The forces that drive the deformity would ideally be reversed by spinal instrumentation. Details of these forces would be obtained by mathematical analysis of the model. As the forces are complex, the treatment would probably also be complex, but would include division of the posterior tension elements (Figs. 2, 6).

Conclusions

The model incorporates the basic constraints of the spine and provides a simple three-dimensional biomechanical explanation of idiopathic scoliosis. It demonstrates that slight generalised anterior overgrowth of a mechanically normal symmetrical spine is enough to precipitate the deformity. Once initiated, anterior and posterior column growth become uncoupled and the overgrowth increases, resulting in a tendency for the deformity to progress. Normally during the growth spurt there is anterior overgrowth. If this is particularly marked or if there is an abnormality in the factors that give inherent stability to the spine than a scoliosis will develop. This therefore provides the final common pathway by which the multiple aetiological factors can contribute to an adolescent idiopathic scoliosis.

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