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Can a short spinal cord produce scoliosis?

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Abstract Some patients with scoliosis have a relatively short vertebral canal. This poses the question of whether a short spinal cord may sometimes cause scoliosis. The present paper presents two observations that may support this concept. It presents a scoliosis model demonstrating what effect a short, unforgiving spinal cord might have on the spinal column. The model uses two flexible parallel tubes with the facility to tighten one. It demonstrates that a short, unforgiving spinal cord could produce the abnormal rotatory anatomy observed at the apex in scoliosis, with first lordosis, then lateral deviation and finally a rotation of the vertebral column, with the rotation occurring between the canal and the vertebral body, around the axis of the cord. The anatomy of the apical vertebra is described from two museum

specimens, a computed tomography (CT) myelogram and seven magnetic resonance imaging (MRI) studies. The study confirms that the vertebral canal and the intervertebral foraminae retain their original orientation. The spinal cord is eccentric in the canal towards the concavity of the curve; the major component of rotation occurs anterior to the vertebral canal and the axis of this rotation seems to be at the site of the spinal cord. These observations do not establish that a short spinal cord will result in scoliosis, but the results are compatible with this hypothesis, and that impairment of spinal cord growth factors may sometimes be responsible for scoliosis.

Keywords Scoliosis · Spinal cord · Growth factors · Anatomical model

Introduction

In scoliosis, there is a differential of growth between the posterior and anterior spine. The spinous processes and the spinal cord in the vertebral canal tend to remain towards the mid-line whilst the vertebral bodies rotate towards the convexity of the curve [5, 15, 34]. The vertebral canal is therefore shorter than the anterior vertebral column.

This growth differential was recognised in 1927 by Heuer [16], who concluded that isolated overgrowth of the anterior spinal elements might be a primary cause of

scoliosis. Somerville [30], on the other hand, suggested that there may be inhibition of posterior growth by tether of muscles or ligaments.

Roth in 1968, proposed an asymmetrical posterior tether of spinal nerve roots on the concavity of the curve as a possible cause of scoliosis [29], and a decade later Lloyd Roberts et al. suggested that unilateral root traction might cause imbalance and explain progression of the deformity [22]. There is a resurgence of interest in a neurological mechanism for scoliosis, because some patients with scoliosis have changes in vestibular function, proprioception, vibratory response and abnormal posterior column function [4, 6, 9]. The focus is on the mid brain and

spinal cord, where primary neurological pathologies might cause a functional asymmetry in balance [4], and consequently scoliosis.

More recently failure of spinal cord growth has been proposed as an initial pathology [28], this being responsible for both the vestibular changes and also for the relative shortening of the posterior bony elements, whilst the anterior column continues to grow and deform. This paper presents two further observations that might support the concept that a short spinal cord could result in scoliosis. The first is a simple model. The second refers to the anatomy of the apical vertebrae.

Materials and methods

A model

A model was designed to demonstrate what effects an unforgiving, short spinal cord might have on the anterior vertebral column. There were certain requirements:

1. The anterior vertebral column should be flexible in three planes of rotation.
2. The vertebral canal should be segmental, and initially parallel to the anterior vertebral column.
3. There should be freedom of rotatory movement in the transverse plane between the vertebral canal and the anterior vertebral column. This is because, in idiopathic scoliosis, it is mainly the vertebral bodies that rotate and not the posterior elements.
4. The spinal cord within the vertebral canal should be flexible.

The model was constructed using easily available materials (Fig. 1).

1. The anterior vertebral column was represented by a foam insulation tube 42 mm×1 m. It was flexible in the coronal and sagittal plane, and because of a longitudinal incision in the tube, it also could rotate (Fig. 2).
2. The vertebral canal was represented by eight metal key rings 23 mm in diameter.

Fig.1 Materials used for the model: 1 m flexible pipe-insulation tube, 1.2 m length of electric cable, eight retainer clips, and eight 18-mm metal key rings

Fig.2 Photographs of the flexible pipe-insulation tube with retainer clips and metal key rings, showing **A** the longitudinal incision throughout the length of the tube, and **B** that the tube can not only bend, but it can rotate

Fig.3 The model constructed with the electric cable through the key rings. These were loosely attached to the flexible pipe-insulation tube by retainer clips

Fig.4 Photograph of the same specimen from above, showing the cranial aspect of the upper vertebra (T8), with the long pedicle on the concavity, part of which forms the base of the canal. The vertebral body is rotated in relation to the vertebral canal (shown diagrammatically in Fig.6)

Fig.5 Photograph of specimen of right sided scoliosis from the Museum of the Royal College of Surgeons of Edinburgh (GC 986), showing the cranial aspect of the apical vertebra, T8. The vertebral body is rotated in relation to the vertebral canal

3. Freedom of rotation of the flexible insulation tube on the key rings was provided by loosely attaching the rings to the tube with eight self-retaining plastic clips (Fig. 2).
4. The spinal cord was represented by a 1.2 m length of 6-mm plastic-covered flexible electric cable.

The end of the cable (representing the spinal cord) was fixed by a tight insulation clip to the end of the insulation tube. The cable was threaded through the key rings (representing the vertebral canal), and the rings were aligned so that they and the cable were parallel to the insulation tube (Fig. 3). The cable was then gradually tightened and the deformity of the system observed.

An anatomical study of the apical vertebra

Two skeletal specimens were examined, one from the Huntarian Collection of the Royal College of Surgeons of England (no. 439) and one from the Museum of the Royal College of Surgeons of Edinburgh (GC 986). The specimens were very similar, being three articulated vertebrae – T8, 9 and 10 in each case – from the apex of longer scoliosis curves. The first was from a left-sided curve (Fig. 4) and the second was right-sided (Fig. 5). Both were recorded as idiopathic scoliosis, though the diagnosis could not be confirmed. There were no signs of infection, but there may have been an unrecorded neurological pathology. The dry specimens were in good condition, having been preserved in a wax-sealed jars. There were minor erosions.

In the first specimen (RCSEng 439), the mean of three measurements was recorded with dividers at the five sites shown in Fig. 6.

1. The height of the vertebral body anteriorly in the mid sagittal plane taking into account the rotation of the vertebral body
2. The posterior height of the vertebral body where the mid sagittal plane of the vertebral body transected the base of the vertebral canal

Fig.6 Diagrammatic representation of T8 from the cranial aspect, showing the rotation of the vertebral body towards the convexity of the curve. The anterior height in the mid sagittal plane, taking into account the rotation of the vertebral body (a), the posterior height at the base of the canal (b), the height of the left lateral border (c), the transverse diameter (d) and the antero-posterior diameter (e) were measured

3. The height of the left and right lateral borders of the vertebral body
4. The transverse diameter of the vertebral body
5. The antero-posterior diameter of the vertebral body

The vertebra with the greatest deformity was identified as the apical vertebra.

The clinical measurement of transverse plane deformity is controversial. Whilst traditional methods measure the rotation of the posterior elements [13,18], the major degree of rotation is in the vertebral body [15,34], and this is difficult to measure on computed tomography (CT) scan. However, the vertebral body rotation is more easily identified in a skeletal specimen, because the vertebral end plate is preserved. The end plate retains its oval shape as it rotates with the vertebral body (Fig.4). Figure 7 therefore shows how the angles of rotation between the base of the vertebral canal and the vertebral body were measured in the specimen using a protractor.

Magnetic resonance imaging (MRI) scans of seven patients with idiopathic scoliosis and one CT myelogram were also examined, recording the anatomy of the apical vertebrae and particularly the position of the spinal cord in the transverse plane.

Fig.7 Diagram to showing the angle of vertebral body rotation in relation to the base of the vertebral canal (see Fig.4)

Results

The model

Initially, as the cable was tightened, the insulation tube developed a gentle curve with the concavity towards the cable – a slight lordosis in the sagittal plane. With further tightening, the cable deviated laterally to one side of the key rings, whilst the flexible tube deviated slightly to the opposite side – lateral deviation in the coronal plane. Gradually, a rotatory deformity began to develop between the key rings and the flexible tube, the cable being the axis of rotation. The tube continued to rotate with maximum rotation at the apex of the curve. The cable and key rings tended to stay towards the mid-line, whilst the flexible tube buckled into a three-dimensional scoliotic deformity (Fig. 8).

The anatomy of the apical vertebra

The measurements of the three articulated vertebrae from the first museum specimen with scoliosis (RCS Eng 439) are recorded in Table 1. The greatest deformity was at T8. The three vertebral bodies were lordotic in the sagittal plane, the anterior vertebral height being greater than the posterior height. Each of the three vertebrae were wedged laterally (in the coronal plane) by angles of 15°, 11° and 9° respectively.

The vertebral canal and the intervertebral foraminae tended to retain their original orientation in relation to the spinous processes. However, the vertebral bodies were rotated in relation to the canal (Fig.4, Fig.5). The base of the spinous processes were just lateral to the posterior apex of the canal. The right side of the canal on the con-

Fig.8 Tightening of the cable caused the flexible tube to rotate into a scoliosis deformity, whilst the cable within the key rings tended to remain towards the mid-line

Table 1 Measurements of the vertebral bodies of the museum specimen RCSEng 439: the anterior and posterior height in the mid sagittal plane, and right and left lateral height in the coronal

plane, taking into account the rotation of the vertebral bodies (see Fig. 6), and the antero-posterior (AP) and transverse (TS) diameters

	Height of the vertebral body (mm)				Diameter of the vertebral body (mm)	
	Anterior	Posterior	Left lateral	Right lateral	AP	TS
T8	21	18	23	12	20	35
T9	23	21	23	15	23	36
T10	24	22	22	19	25	38

cavity of the curve was formed by an acute angle creating a lateral recess. The left lateral boundary of the canal on the convexity of the curve was much less acute. The right pedicle on the concavity of the curve formed the flat base of the vertebral canal. This pedicle was longer and its base larger and more robust than the shorter pedicle on the convex side, suggesting that an overgrowth at the concave neurocentral synchondrosis was contributing to the rotational deformity of the vertebral body. The vertebral body of T8 was rotated 68° compared with the vertebral canal, and T10 was rotated 46° .

The left intervertebral foraminae on the convexity of the curve were much closer to the lateral border of the rotated vertebral bodies [(c) in Fig. 6] than were the right foraminae to the right lateral border – 7, 9 and 10 mm on the convexity of the curve compared with 22, 23 and 24 mm on the concavity.

The anatomy of the second museum specimen (GC 986) was almost identical, being a mirror image of the first specimen. The vertebral bodies were lordotic and wedged laterally. The shape of the vertebral canal and the pedicles was similar. The foraminae on the concave side were also

further from the lateral border of the vertebral bodies than were the foraminae on the convexity. The apical vertebra was T8, and the vertebral body was rotated 56° when compared with the vertebral canal.

Transverse MRI scans of the apex of the curve were examined in seven patients with idiopathic scoliosis; five had single thoracic or thoracolumbar curves and two had double major curves. At the apex of the curves, they showed identical features to the museum specimens. In each patient the spinous processes and the vertebral canal tended to remain in the mid-line, and the major rotation was anterior to the vertebral canal. The spinal cord was eccentric in the canal, being close to the lateral recess on the concavity of the curve. In the two patients with double major curves, the cord was close to the concavity of each curve in turn.

A pre-operative CT myelogram in a 13-year-old premenarchal girl, who had a 75° single right-sided scoliosis, showed the same features as seen in the MRI scans (Fig. 9). The vertebral canal tended to maintain its original orientation in the plane of the patient's body, with the base of the canal remaining in the coronal plane. The vertebral

Fig. 9 Computed tomography (CT) myelogram at the apex of the curve in a 13-year-old girl with a right-sided idiopathic scoliosis and a Cobb angle of 75° (acknowledgements to Mr J. Dove)

body was rotated 36° from the coronal plane, towards the convexity of the curve. The spinal cord was in the mid-line, anterior to the spinous process, but eccentric in the canal. The cord and the dural sac were closely applied to the lateral recess of the vertebral canal on the concavity of the curve, following the shortest distance. There was very little extra-dural space on this side of the canal.

The eccentric position of the spinal cord and the dural sac identified in the CT myelogram and MRI scans was superimposed on the diagram of the museum specimen (Fig. 7). The vertebral body rotation was then about an axis of the spinal cord in the lateral recess of the vertebral canal.

Discussion

In a previous paper by the present author, describing the degree of relative shortening of the vertebral canal in skeletons with scoliosis, a short spinal cord was suggested as a possible primary mechanism for some patients with scoliosis [28]. The paper acknowledged that a short posterior element may be part of the overall deformity and secondary to other factors, but it suggested that a short cord should be considered as a possible primary mechanism.

In this paper, a simple model demonstrates what effects would be expected if the spinal cord were relatively short, tight and unforgiving. In the model, shortening of the cable initially caused a slight lordosis. This was followed by a degree of lateral deviation as the cable slipped to the lateral side of the rings. With increasing tension on the cable the anterior flexible tube rotated – the only way in which a relative advantage could be conferred on the tight cable. The rotation of the anterior column was about the axis of the tight cord.

The deformity produced in the model exactly matched the deformity in scoliosis, where there is first a straightening out of the kyphosis [8], a lateral deviation and then the vertebrae rotate towards the convexity of the curve. A simple model has obvious limitations, and an electrical cable is only representative of a soft spinal cord if the spinal cord is physiologically less forgiving than the bony vertebrae. The scoliosis demonstrated in this model does not, of course, confirm that a tight spinal cord is the cause of scoliosis. However, it does show that if there is tight posterior tether within the vertebral canal, with continued bone growth the vertebrae will rotate into a scoliotic deformity. It is a reasonable hypothesis that if the spinal cord is relatively short and physiologically it resists stretching, the vertebrae may deform and rotate in order to protect the cord.

The second part of the paper examines the anatomy of the apical vertebrae. Adams [1] thought that, in scoliosis, the apical vertebrae turned uniformly about the point of the spinous processes as if they were solidly held there by a powerful ligament. Others, however, have shown that

the rotatory deformity is relative: there is only minimal deformity of the posterior elements, whilst deformity of the anterior column is much more marked [15,22]. Stagnara [31] compared the rotation of the apical vertebra to a delivery tricycle, where the delivery man sits on the tricycle seat and the rotation of the delivery box takes place in front of him. His illustration shows a relatively greater torsion anteriorly. Wever and colleagues demonstrated that the posterior complex of the vertebrae attempts to retain its original position whilst there is an increasing torsion of the vertebral body [34].

This present study of the apical vertebrae confirms that the vertebral canal retains its original position and the rotation of the vertebral body occurs anterior to the vertebral canal. Furthermore, the rotation seems to be about an axis towards the lateral side of the canal on the concavity of the curve – at the site of the spinal cord. 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 rotational deformity tends to be selective and anterior to the canal.

The eccentric position of the spinal cord in the canal, lying towards the concavity of the curve, has been previously described [22,23], and it is again confirmed in the MRI scans and the CT myelogram. The cord's position suggests that it is not a disinterested component sharing in a general rotational deformity, otherwise it would follow the vertebral rotation towards the convexity of the curve. Instead, the cord remains towards the mid-line of the patient and the vertebral bodies seem to rotate about the cord's axis. In addition, the spinal cord itself does not rotate with the vertebral bodies; spinal angiography shows the anterior spinal artery lying in the position of the mid-line of the spinal cord [17,22].

The intervertebral foraminae also tend to retain their former position in relation to the canal, and they do not rotate with the vertebral bodies. On the concave side of the curve they are much further from the lateral border of the vertebral body than on the convex side, suggesting that the intraspinal roots as well as the cord do not rotate.

These anatomical features, although not conclusive, lend support to a short spinal cord being a primary factor in scoliosis – the canal and foraminae tend to retain their original position, the spinal cord is eccentric within the canal towards the concavity of the curve, the major component of rotation is anterior to the vertebral canal, and the vertebral body rotation seems to be around the axis of the cord.

It might be argued that the cord's position is secondary, and that it always takes the shortest route – that it is only a passive component in the overall deformity. The cord does take the shortest path and it seems to retain its original orientation, but it is not only the cord but also the canal that maintains its original position. What if the cord

determines the path itself – the position and shape of the posterior elements?

To think of the soft spinal cord having a powerful influence on the surrounding bone is not intuitive. However, we have shown previously, when studying the growth of the fetal vertebral canal, that the canal size and growth is closely related to the size of the spinal cord [33]. The fetal spinal cord, with its meninges, seems to have a strong influence on the surrounding bone, probably influencing it epigenetically, as the brain determines the size of the skull. In older children also, the bony vertebral canal adapts to accommodate intramedullary tumours. It is not being suggested that the cord is a mechanical tether, but we should not be surprised if failure of spinal cord growth, with continued bone growth, resulted in spinal deformity.

The spinal cord is not the only structure within the vertebral canal that could be responsible. A tight posterior longitudinal ligament might also result in the same deformity. However, scoliosis has not been reported in patients with ossification of the posterior longitudinal ligament, and a tight ligamentum nuchae causing an extension contracture of the neck was not associated with scoliosis [7].

It is not suggested that a short spinal cord is responsible for all types of scoliosis. It could not explain the adolescent curve that continues to progress in adult life, nor the adult “nouveau” scoliosis. However, it deserves consideration as a possible mechanism in some young patients. There are then some questions to ask if we believe that a short spinal cord is sometimes responsible for scoliosis.

Does it help us to understand the relationship between scoliosis and proximal cord lesions [2, 20,24], for example the Chiari I malformation? A recent study showed that in 27% of patients with severe idiopathic scoliosis, a tongue of cerebellum and medulla oblongata herniated into the cervical canal, often with abnormal somatosensory evoked potential (SEP) findings [6]. *Could a tight cord be responsible for the Chiari formation, for the subtle neurological changes and also the scoliosis?*

Up to 85% of children with syringomyelia will have scoliosis, and some of these also have a Chiari malformation [12, 14, 24,35]. These children have an unexplained high risk of neurological complications during surgical correction [26,25]. *Could syringomyelia and cord tumours sometimes impair cord growth?* In Tachdjian and Matson’s large series [32] of 115 children with spinal cord tumours, scoliosis was present in 31 patients (27%). These various neuro-pathologies have been found in between 19% and 26% of scoliotic children undergoing routine MRI [10,21]. *Is it possible that some of these pathologies*

are associated with a short spinal cord, which then causes spinal deformity?

Distal lesions of the spinal cord [19] with a low conus from a tethered filum or diastematomyelia can be associated with scoliosis, but in contrast to idiopathic scoliosis, these patients often have associated neurological problems. It could be argued that if the spinal cord is tight in patients with idiopathic scoliosis, some of them would present with an upper motor neurone lesion. On the other hand, scoliosis may be a mechanism to avoid pathological cord tension and to protect the cord and brainstem from neurological damage. In addition, there may be a difference between a short spinal cord present from birth and one arising at the beginning of the adolescent growth spurt. We have yet to identify the position of the conus in patients with idiopathic scoliosis.

Why is the range of neck flexion sometimes reduced in patients with scoliosis [11]? Flexion of the neck stretches the spinal cord [3], and a stiff neck in some patients with scoliosis could be explained by an already tight cord. *Is neck flexion more restricted after correction?*

Can the difference between large, sweeping paralytic curves and more discrete curves in idiopathic scoliosis be explained by some of the latter having a short cord?

If the spinal cord is short in scoliosis, we might speculate about the causation of uncoupling of neuro-osseous growth. *Is there a balance between growth hormone and a spinal cord growth factor? Are there mechanisms which turn on and off spinal cord growth factors?* Pinealectomy in 3-day-old chickens routinely results in scoliosis [27]. *Could factors from the pineal gland (in the posterior part of the third ventricle) affect cord growth and normally be in balance with growth hormone from the pituitary (in the anterior part of the third ventricle)?*

Although some of these answers are speculative, the questions need to be asked.

Conclusions

The model, the anatomical study, and the clinical observations described in this paper do not establish that a short cord causes scoliosis. However, they are compatible with the hypothesis, and uncoupling of neuro-osseous growth deserves investigation as a possible mechanism for scoliosis.

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