Pathogenesis of cervical spondylotic myelopathy

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Abstract

Objective—To determine whether either of two mechanical theories predicts the topographic pattern of neuropathology in cervical spondylotic myelopathy (CSM). The compression theory states that the spinal cord is compressed between a spondylotic bar anteriorly and the ligamenta flava posteriorly. The dentate tension theory states that the spinal cord is pulled laterally by the dentate ligaments, which are tensed by an anterior spondylotic bar.

Methods—The spinal cord cross section, at the level of a spondylotic bar, is modelled as a circular disc subject to forces applied at its circumference. These forces differ for the two theories. From the pattern of forces at the circumference the distribution of shear stresses in the interior of the disc—that is, over the transverse section of the spinal cord—is calculated. With the assumption that highly stressed areas are most subject to damage, the stress pattern predicted by each theory can be compared to the topographic neuropathology of CSM.

Results—The predicted stress pattern of the dentate tension theory corresponds to the reported neuropathology, whereas the predicted stress pattern of the compression theory does not.

Conclusions—The results strongly favour the theory that CSM is caused by tensile stresses transmitted to the spinal cord from the dura via the dentate ligaments. A spondylotic bar can increase dentate tension by displacing the spinal cord dorsally, while the dural attachments of the dentate, anchored by the dural root sleeves and dural ligaments, are displaced less. The spondylotic bar may also increase dentate tension by interfering locally with dural stretch during neck flexion, the resultant increase in dural stress being transmitted to the spinal cord via the dentate ligaments.

Flexion of the neck increases dural tension and should be avoided in the conservative treatment of CSM. Both anterior and posterior extradural surgical operations can diminish dentate tension, which may explain their usefulness in CSM.

The generality of these results must be tempered by the simplifying assumptions required for the mathematical model.

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The pathogenesis of cervical spondylotic myelopathy (CSM) is still not firmly established, even though CSM is a very common and well known condition. Several theories of pathogenesis have been proposed, but there has been no conclusive demonstration that any one theory explains the reported neuropathology better than the others. The purpose of this paper is to review the various theories of pathogenesis, to determine what pathology each predicts, and to decide which theory, if any, best explains the neuropathological findings.

The oldest[1] and still most often cited theory is that CSM is caused by mechanical compression. It is thought that the spinal cord is compressed between a spondylotic bar anteriorly[1] and the ligamenta flava posteriorly.[2] The spinal cord is most vulnerable to such compression during extension of the neck, when the ligamenta flava bulge into the spinal canal,[3] decreasing its anteroposterior depth, while the anteroposterior dimension of the spinal cord itself increases.[4] People with congenitally narrow spinal canals are more vulnerable to this pinching of the spinal cord between an anterior spondylotic bar and the posterior ligamenta flava.

A second mechanical theory[5] is that CSM is caused by tensile stresses transmitted to the spinal cord from the dentate ligaments, which attach the lateral pia to the lateral dura. The spondylotic bar displaces the spinal cord posteriorly, but this displacement is resisted by the dentate ligaments. The dural attachments of the dentate ligaments provide a fixed point, so that dentate tension can increase when the spinal cord is displaced posteriorly. The dural attachments do not move because the dural sac is constrained by the dural root sleeves, which are held fixed in the neural foramina. The spinal cord is most vulnerable during flexion of the neck, when the dura is unfolded, and the nerve roots and dentate ligaments are relatively taut.[4]

The major non-mechanical theories are vascular. They have arisen not because of evidence of vascular occlusion in any neuropathological study of CSM, but rather because of the assumed inability of the mechanical theories to predict the pathological lesions. For example, Greenfield,[6] while acknowledging a role for mechanical damage, thought that ischaemia was needed to account for the lesions in the ventral portions of the

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Figure 1 Topography of neuropathology of CSM. The top row shows myelin stained cross sections of the spinal cord at the level of a spondylotic bar from two representative cases studied at postmortem. On the left is case 4 of Brain et al[9] and on the right is case 3 of Mair and Druckman.[7] On the bottom is a schematic diagram adapted from Ogino et al[13] with permission. The dark shading indicates the most vulnerable areas, affected in even mild cases, the intermediate shading designates moderately vulnerable regions, and the light shading indicates areas damaged only in severe cases. The unshaded areas are spared.
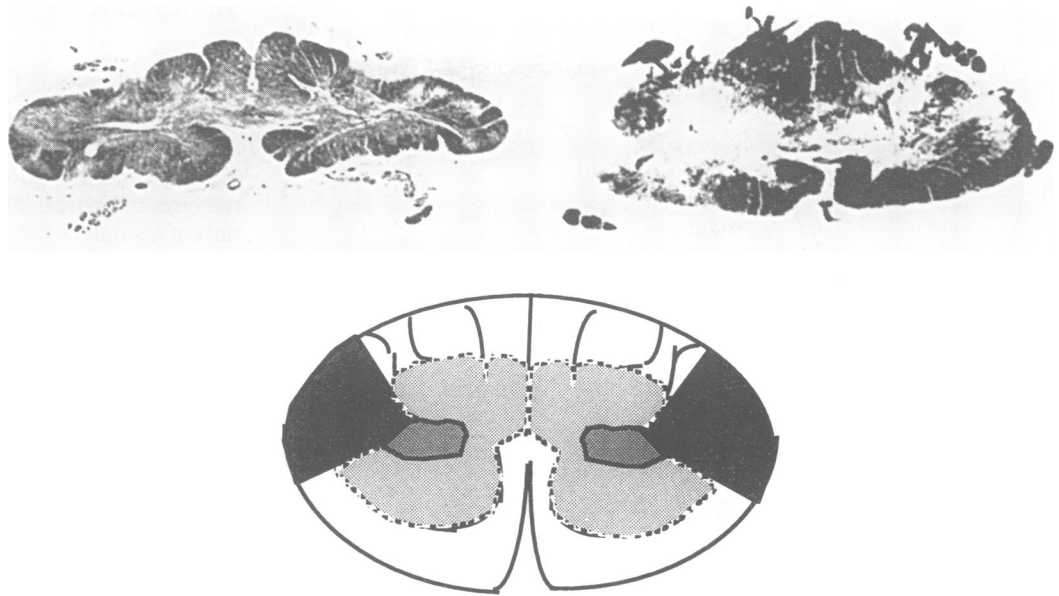
posterior columns that are often present in severe cases. Both arterial and venous ischaemic mechanisms have been discussed. Mair and Druckman[7] suggested that compression of the anterior spinal artery and its branches in the spinal cord caused CSM. Taylor[8] considered that compression of the radicular arteries in the intervertebral foramina caused CSM. Brain *et al*[9] thought that compression of veins on the anterior aspect of the spinal cord by a spondylotic bar was important.

Despite the lack of consensus regarding pathogenesis there is little disagreement about the neuropathological findings (fig 1) in CSM.[7 9-13] The spinal cord at the level of the spondylotic bar is flattened in the anteroposterior dimension but not in the transverse dimension. The damage is most severe in cross sections at the level of the spondylotic bar. There the lateral columns are the most vulnerable, and in them the involved areas are often wedge shaped with the apex medial and the base lateral. In more severe cases the damage extends further medially to involve the intermediate portions of the grey matter and the ventral portions of the posterior columns. Microscopically, there is demyelination and gliosis of the affected white matter and loss of nerve cells and gliosis in the affected grey matter. Above and below the level of the spondylotic bar the damage becomes progressively less severe, and Wallerian degeneration is seen in the posterior columns rostral to the level of compression and in the lateral columns caudal to it.

I have developed a model of the spinal cord that allows calculation of the pattern of stresses in the cross section of the cord at the level of an anterior spondylotic bar. The purpose of this paper is to present these calculations and to compare the predicted stresses with the topography of the neuropathological damage. In this manner it should be possible to decide whether one, both, or none of the mechanical theories should be accepted.

Methods

The spinal cord at the level of a spondylotic bar is modelled as a circular disc subject to forces applied at its circumference. The location and nature of these forces differ in the two mechanical theories. Both have in common an anterior compressive force produced by the spondylotic bar. The first theory (fig 2A) postulates bilateral posteriorly applied compressive forces directed anteriorly and slightly medially, produced by the ligamenta flava. The second theory (fig 2B) postulates laterally applied tensile forces directed laterally and slightly anteriorly, produced by the dentate ligaments. These two different patterns of applied force will produce two different distributions of stress in the spinal cord cross section.

The stress at any point in the disc can be resolved into two parts: a uniform pressure or tension and a pure shear. Because nervous tissue is relatively incompressible, little harm is caused by changes in the uniform pressure.[14] It is the shear stress, which causes distortion or deformation of nerve tissue, that results in tissue damage.[15] In an isotropic medium the distribution of damage should correspond to the topographic distribution of the shear stresses. Each mechanical theory predicts a specific distribution of shear stresses over the involved cross section of the spinal cord. A comparison of the predicted distributions with the neuropathological data may therefore be a test of the theory's validity.

The stresses are calculated as follows (fig 2C):

(1) A coordinate system is established with the origin at the centre of the circular spinal cord cross section. The *x* axis is positive anteriorly, and the *y* axis is positive to the right.

(2) The locations of the forces at the circumference are specified in terms of their *x-y* coordinates. The direction of each force is designated by the angle it makes with the *x* axis. The magnitudes of the forces are constrained by the requirements of mechanical equilibrium. Specifically:
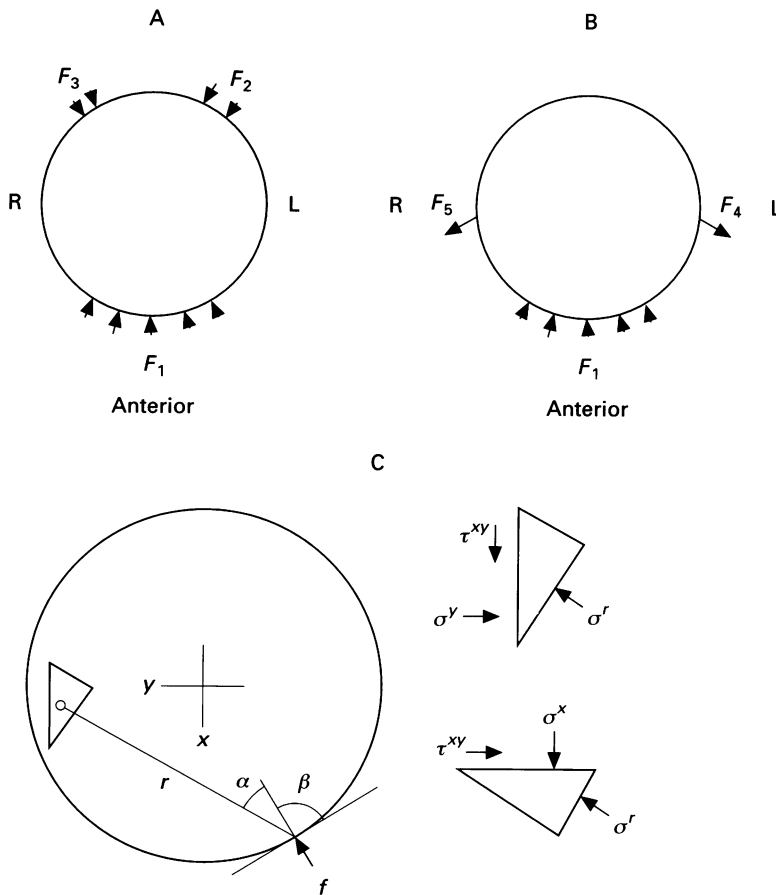
Figure 2 *(A) The compressive model and (B) the dentate tension model of CSM. F_i is the force exerted by the spondylotic bar, F_2 and F_3 are the forces exerted by the ligamenta flava, and F_4 and F_5 are the forces exerted by the dentate ligaments. R = right, L = left, Ant = anterior. (C) Variables used in calculating stresses. f represents one element of the distributed force F_1, r is the radial distance from the point of application of f to the point (x, y), represented by the small circle, α is the angle between r and the direction of f, and β is the angle between the direction of f and the tangent to the disc at the point of application of f. The small triangle surrounding (x, y), with one side parallel to r, a second side perpendicular to r, and a third side parallel to the x axis, is used to calculate σ^y and τ^{xy} from σ^r and is reproduced with these stresses illustrated at the upper right. Another triangle, with the third side parallel to the y axis, is used to calculate σ^x and is shown at the lower right.*

(*a*) In both models (fig 2A, B) the disc force, F_1, is applied anteriorly, uniformly distributed over an arc length of 60° centred on the x axis. It is directed posteriorly, perpendicular to the tangent to the circumference at each point of the arc. Its magnitude is given an arbitrary value 1.

(*b*) In the first model (fig 2A) the ligamentum flavum forces F_2 and F_3 are applied posterolaterally, the locations of the two forces being symmetric with respect to the x axis. Thus F_2 is centred 30° to the left and F_3 30° to the right of the axis. Each force is uniformly distributed over an arc length of 30°. The forces are directed anteromedially at an angle of 30° to the x axis. The magnitude of each force is constrained by the requirement of physical equilibrium to be:

$$F_2 = F_3 = \tfrac{1}{2}\cos 30$$

(*c*) In the second model (fig 2B) the dentate ligament forces F_4 and F_5 are applied laterally. Each is centred 15° anterior to the y axis and is distributed uniformly over a narrow arc of 4°. The forces are directed laterally and slightly anteriorly at an angle of 60° with the x axis.

The magnitude of each force is constrained to be:

$$F_4 = F_5 = \tfrac{1}{2}\cos 60$$

(3) At a given interior point (x, y) of the spinal cross section the stress is determined by summing the contributions of the three forces at the circumference. To determine the contribution of a single force, the arc length over which it is distributed is divided into a large number N of small, equal segments. The force at each element, of magnitude

$$f_i = \frac{F_i}{N} \quad (i = 1 \text{ to } 5)$$

is considered to be applied at the midpoint of the element. These elements are represented schematically by the arrowheads in fig 2, but for the calculations presented here each F_i is divided into 200 elements—that is, N = 200.

The contribution at (x, y) from the jth element (j = 1 to N) of the ith force is determined according to Timoshenko.[16] It consists (fig 2C) of the sum of a purely radial stress $\sigma^r_{i,j}$ and a uniform stress $p_{i,j}$ with magnitudes

$$\sigma^r_{i,j} = \frac{-2f_i\cos\alpha_{i,j}}{\pi r_{i,j}}$$

$$p_{i,j} = \frac{f_i\sin\beta_{i,j}}{\pi d}$$

where $r_{i,j}$ is the length of the ray from the point of application of the jth element of the ith force to the point (x, y), $\alpha_{i,j}$ is the angle between the direction of the jth element of the ith force and the ray of length $r_{i,j}$, d is the diameter of the spinal cord, and $\beta_{i,j}$ is the angle between the direction of the jth element of the ith force and the tangent to the circle at the point of application of the force.

Because the radial stresses at (x, y) from the different elements of a force differ in direction, they cannot be summed (integrated) directly. They must first be transformed into stresses referable to a common set of directions. By the laws of statics each radial stress σ^r can be transformed into a normal stress σ^x in the x direction, a normal stress σ^y in the y direction, and a shear stress parallel to the coordinate axes in the x-y plane τ^{xy} (fig 2C) according to the formulae:

$$\sigma^x_{i,j} = \sigma^r_{i,j}\cos^2\alpha_{i,j}$$
$$\sigma^y_{i,j} = \sigma^r_{i,j}\sin^2\alpha_{i,j}$$
$$\tau^{xy}_{i,j} = -\sigma^r_{i,j}\sin\alpha_{i,j}\cos\alpha_{i,j}$$

For the ith force, the normal stress in the x direction, σ^x_i, the normal stress in the y direction, σ^y_i, and the shear parallel to the axes in the x-y plane, τ^{xy}_i, at the point (x, y), can now be obtained by summation over the index j.

$$\sigma^x_i = \sum_{j=1}^{N}\sigma^x_{i,j} + \sum_{j=1}^{N}p_{i,j}$$

$$\sigma^y_i = \sum_{j=1}^{N}\sigma^y_{i,j} + \sum_{j=1}^{N}p_{i,j}$$

$$\tau^{xy}_i = \sum_{j=1}^{N}\tau^{xy}_{i,j}$$

Summation over the index i combines the

forces to yield the total stress σ^x, σ^y, τ^{xy} for the point (x, y). Thus for the first model,

$$\sigma^x = \sigma^x_1 + \sigma^x_2 + \sigma^x_3$$
$$\sigma^y = \sigma^y_1 + \sigma^y_2 + \sigma^y_3$$
$$\tau^{xy} = \tau^{xy}_1 + \tau^{xy}_2 + \tau^{xy}_3$$

and for the second model,

$$\sigma^x = \sigma^x_1 + \sigma^x_4 + \sigma^x_5$$
$$\sigma^y = \sigma^y_1 + \sigma^y_4 + \sigma^y_5$$
$$\tau^{xy} = \tau^{xy}_1 + \tau^{xy}_4 + \tau^{xy}_5$$

Finally, the maximum shear stress τ_{max} at the point (x, y) can be calculated:

$$\tau_{max} = \sqrt{\left(\frac{\sigma^x - \sigma^y}{2}\right)^2 + \tau^{xy2}}$$

(4) τ_{max} is determined for each point of the circular cross section. τ_{max} can then be plotted across the disc by converting the value at each point to a value on a grey scale to facilitate comparison with the neuropathological data.

The above calculations were performed and the graphs plotted on a Macintosh Quadra 800 computer using the Matlab software system.[17]

Results

Figure 3A shows the topography of maximum shear predicted by the compressive theory. The greatest stresses cut two linear swaths that run predominantly in an anteroposterior direction, from the ligamenta flava posterolaterally to the spondylotic bar anteriorly. To either side—the central portion of the cord medially and the lateral columns laterally—the stress is intermediate in magnitude. Of note, the most lateral portions of the lateral columns are relatively free of stress.

Figure 3B shows the topography of maximum shear predicted by the theory of dentate ligament tension. The greatest stresses lie along a transverse line extending from one dentate ligament to the other. Along this line the stresses are considerably greater laterally than medially. Thus the lateral columns are subjected to the most stress, especially their most lateral portions, whereas the intermediate portions of the grey matter and the ventral

portions of the posterior columns are also stressed, but to a lesser degree. The anterior columns and the dorsal portions of the posterior columns are relatively unstressed.

Discussion

COMPARISON OF THE MECHANICAL THEORIES WITH NEUROPATHOLOGY

It is evident that there is a remarkable correspondence between the topography of the neuropathology of CSM (fig 1) and the spatial distribution of stresses predicted by the dentate tension hypothesis (fig 3B). By contrast, there is very little correspondence with the stresses predicted by the compression hypothesis (fig 3A). All neuropathological studies agree that the lateral columns are most vulnerable to damage, especially the most lateral portions. In the dentate tension model the lateral portions of the lateral columns are the most highly stressed regions, whereas in the compression model these regions sustain relatively little stress. As the disease process becomes more severe, the neuropathology extends medially to involve the spinal grey matter and the ventral portions of the posterior columns. In both models these regions are subject to intermediate levels of stress. The neuropathological studies also agree that even in advanced CSM the anterior columns and the posterior portions of the posterior columns remain relatively free of disease. In the dentate tension model these areas are relatively free of stress, whereas in the compression model the anterior columns are subject to considerable stress.

THE DENTATE TENSION HYPOTHESIS

The present model of increased dentate tension was designed to test the hypothesis of Kahn,[5] in which the primary pathogenetic event is stretching of the dentate ligaments as a result of posterior displacement of the spinal cord by a spondylotic bar. The stretching occurs because the dural attachment of the ligaments remains fixed, anchored by the dural root sleeves which resist displacement of the dural sac.

Several experimental studies, in both human

Figure 3 Plot of calculated maximal shear stresses at each point of the cross section of the spinal cord model. Lighter shading signifies greater stress. (A) The compressive model; (B) the dentate tension model. R = right; L = left.
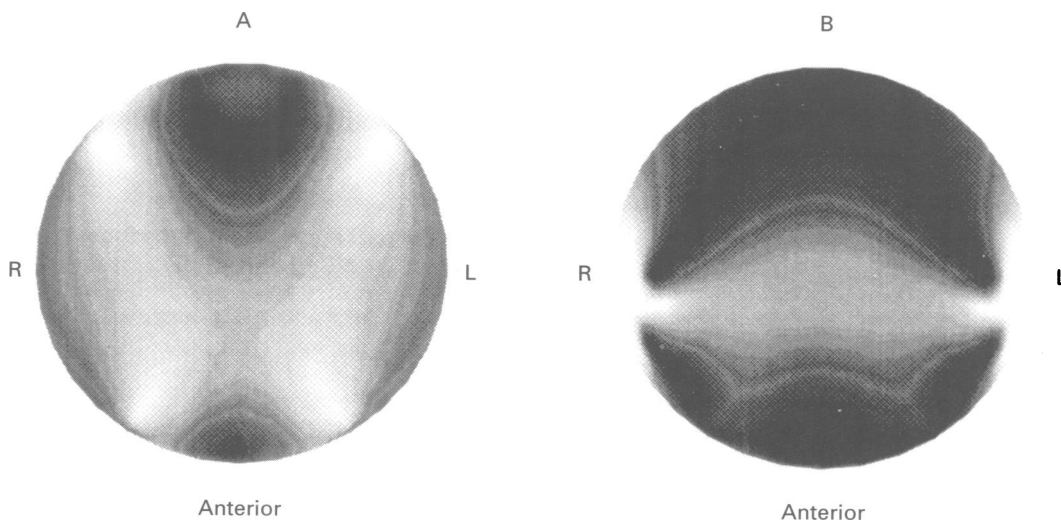
cadavers and in animals, have considered the questions of whether the dentate ligaments constrain posterior displacement of the spinal cord within the dural sac[18-20] and whether the nerve root sleeves anchor the dural tube to provide a fixed point for the dentate ligaments to act.[19 21] The results of these studies can be synthesised and understood only by taking into account the degree of flexion-extension of the spine. In extension the dura, the dural nerve root sleeves, and the dentate ligaments are slack. The root sleeves do not hold the dura forward, and the dentate ligaments do not prevent posterior displacement of the spinal cord within the dural sac. In flexion, however, the spinal canal lengthens and the dura is stretched. The dural root sleeves may also become taut and anchor the dural tube. Dural tension is transmitted to the dentate ligaments, which stretch the spinal cord axially and resist posterior displacement of the cord within the dural tube.

There is another means by which dentate ligament tension can be increased, to which the results of our model also apply. Any increase in longitudinal tension in the dura is transmitted to the spinal cord as an increase in both longitudinal and radial tension, because the dentate fibres run mediolaterally as well as rostrocaudally. Tencer *et al*[21] found that although the elastic properties of the dura were uniform throughout its length, the degree of dural stretch during flexion of the spine was much greater in the cervical than in the thoracic and lumbar regions. This difference in longitudinal stretch was independent of any tethering effects of the dural root sleeves or ligaments, because it persisted when the nerve roots and ligaments were cut. Tencer *et al* speculated that friction between the dura and the anterior surface of the thoracic spinal canal may interfere with stretching of the dura and increase the tension more rostrally. Reid[19] suggested that similar frictional effects may apply to flexion in the presence of a localised spondylotic protrusion. Adams and Logue[22] showed a localised increase in dural stretch adjacent to a point of dural fixation in human cadavers. The increased dural tension could be transmitted to the spinal cord through the dentate ligaments. Thus the primary pathogenetic event need not be direct pressure of the spondylotic bar on the spinal cord, but rather interference with dural stretch during flexion.

Either of these mechanisms of dentate ligament stress can explain two additional neuropathological features of CSM. Firstly, the pronounced flattening of the spinal cord in the anteroposterior dimension at the level of the spondylotic bar is probably produced by the transmission of increased lateral and longitudinal tension from the dentate ligaments to the spinal cord. Because nervous tissue is relatively incompressible the resulting longitudinal and transverse elongations will be accompanied by a compensatory contraction in the remaining anteroposterior dimension. Secondly, thickening and fibrosis have been described in the dentate ligaments, in the dura, and in the dural root sleeves.[10 11 23] Such fibrosis probably represents a physiological response to chronically raised levels of stress in these tissues.[20]

VASCULAR THEORIES

The close correspondence between the predicted pattern of shear stress and the topography of the spinal cord pathology eliminates the need to postulate a separate vascular hypothesis. Ironically, Kahn,[5] who first postulated the dentate tension hypothesis, gave impetus to non-mechanical hypotheses by presenting an erroneous stress analysis, in which a main "pressure wave" involved the anterior columns in addition to other pressure waves involving the lateral columns. It is highly likely that this error was caused by failing to represent the force exerted by the disc as distributed over the anterior part of the circumference of the cord and instead representing the force as localised to a single point or a highly concentrated region. When the force is represented correctly as distributed, the anterior pressure wave disappears and the anterior columns remain unstressed, corresponding to their being spared neuropathologically.

There is little or no supportive evidence for most of the vascular hypotheses. Neuropathology studies have rarely if ever reported occluded vessels. The restricted longitudinal distribution of the spinal cord damage is evidence against occlusion of a vertebral artery or one of its major radicular vessels. The topography of the damage in the cross section at the level of the spondylosis is evidence against occlusion of the anterior spinal artery, because the anterior columns are spared. Neither is the topography of damage consistent with the hypothesis of venous congestion leading to infarction. The slowly progressive evolution of CSM, often over many years, is also difficult to explain on a vascular basis, as is the not infrequent improvement after decompressive surgery.

A mechanical hypothesis, however, can be consistent with one type of vascular theory. Shear stresses cause tissue damage by distorting tissue. Thus small intramedullary vessels can be damaged along with neurons, nerve cell processes, and neuroglia. Breig *et al*[24] have shown that transversely running blood vessels become elongated as the spinal cord is stretched transversely with resulting narrowing of the vessels' cross section. Such narrowing might cause reduction in blood flow. The resulting ischaemia could contribute to the neuropathology.

ASSUMPTIONS AND LIMITATIONS OF THE MODEL

The model that we have employed involves several assumptions and simplifications. Firstly, the equations that we have used are for a homogeneous and isotropic substance, and the spinal cord is neither homogeneous nor isotropic. The main source of inhomogeneity is that the grey matter is slightly less rigid than the white matter. We therefore expect that the model underestimates the extent of damage to the border between grey matter and white matter, where stresses are accentuated because

of the inhomogeneity.[25] The anisotropy of the spinal cord is not a major problem because it results primarily from the craniocaudal orientation of the long white matter tracts. My model, however, applies only to a single cross section, in which, on a macroscopic level, the spinal cord is more nearly isotropic. Secondly, I have used equations for plane stress and strain, which assume that adjacent segments of the spinal cord exert no shear stress on the segment in question, but rather exert, at most, a uniform tension or compression. This is likely not to be the case. Further, I have modelled the cervical spinal cord cross section as a circle whereas it is more elliptical than circular. Both of these simplifications mean that I cannot draw precise quantitative conclusions from the data. However, I have not attempted to do so. Rather, I have looked for qualitative patterns and have shown a major difference between the predictions of the two hypotheses. There is no reason to think that this difference would be significantly altered by these simplifying assumptions. Finally, I have assumed that the spinal cord is an elastic structure whereas it is viscoelastic[26]—that is, it is subject to plastic deformation. In a condition of chronic stresses such as CSM the viscous behaviour is evidenced by the permanent flattening and widening of the spinal cord even after it is removed at necropsy and thus freed of stress. In such viscoelastic substances the immediate response to stresses of modest degree is elastic in nature, and viscous flow then occurs to alleviate the stress if it persists. The viscous flow dampens the peaks of shear stress but does not alter its overall topographic pattern.[25] Thus there is no reason to think that the viscous behaviour of the spinal cord would alter the substantial qualitative difference between the two mechanical theories.

THERAPEUTIC IMPLICATIONS

If the dentate tension hypothesis is correct there are several implications with regard to the treatment of CSM. As flexion increases tension in the dura, dentate ligaments, and spinal cord, CSM can be treated conservatively by a restraining collar limiting mobility of the cervical spine. In particular, flexion of the cervical spine should be avoided. Improvement has been reported in nearly half of patients treated with a cervical collar.[27-29]

In patients not responsive to conservative treatment removal of the spondylotic bar through an anterior surgical approach may be successful by removing the source of posterior pressure on the spinal cord and the source of interference with stretching of the dura during flexion. Fixation of the vertebrae bordering the spondylotic bar with a bone graft may also help by reducing spinal mobility. The surgery fails in about 25% of cases,[29] possibly because postoperative adhesions between the anterior dura and the anterior wall of the spinal canal may cause increased tension in the dura during neck flexion.[22]

Surgical laminectomy alone would seem unhelpful because the problem is not one of compression by the posterior elements of the spine. Yet the reported benefits of laminectomy are not clearly worse than those of anterior surgery.[29 30] There are several possible explanations for the beneficial effects of laminectomy. Firstly, facetectomy is often performed with laminectomy and seems to improve outcome.[31 32] Facetectomy releases the tethering of the dural sac by the dural root sleeves in the foramina, removing the fixed point against which the dentate ligaments can be stretched. It has also been suggested[33] that facetectomy reduces the support of the posterior elements of the spine, thus tipping the vertebrae into extension, releasing tension in the dura and the dentate ligaments. However, similar benefit can result from extensive laminectomy without facetectomy.[34 35] After extensive unroofing of the cervical spinal canal the posterior dura bulges dorsally into the site of decompression, and gas myelography shows that the cervical spinal cord has a milder lordotic curve and courses more posteriorly within the dural sac, no longer in contact with the spondylotic bar.[29 36] Because the anterior dura is anchored to the anterior wall of the spinal canal, dorsal migration of the posterior dura must be accompanied by a decrease in the transverse dimension of the dural sac if intradural volume remains constant. This decrease in width will relax the dentate ligaments. However, the posterior bulging of the dural tube may also reflect increased filling with CSF.[33] Even so, dorsal migration of the posterior dura will displace the dural attachments of the dentate ligaments somewhat posteriorly. The posterior dura will have a straighter course and may need to develop less tension during neck flexion. In addition the extensive posterior scar that eventually develops may serve to limit flexion of the cervical spine and thus any remaining stresses on the dura. Like the anterior approach, extensive laminectomy fails in a minority of cases, possibly because the posterior scar may tighten, compress the dura during neck flexion,[37] and increase the tension in the dentate ligaments.

Although the most direct surgical approach to relieving excessive tension in the dentate ligaments may be to open the dura and to section the ligaments, intradural operations result in more extensive adhesions, often involving the spinal cord itself.[38] Several studies have failed to show an advantage to intradural section of the dentate ligaments,[31 36 39] and the procedure has largely been abandoned. It seems that it is possible to relax the excessive tension in the dentate ligaments with either an anterior or a posterior approach that remains extradural.

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