Effects of Mechanical Stresses on the Spinal Cord in Cervical Spondylosis
A Study on Fresh Cadaver Material

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The brain stem and spinal cord, from a mechanical point of view, can be regarded as a single unit, securely fastened above and below. During movement, the cervical cord and its dural covering slide upwards and downwards no more than 2 or 3 mm. within the vertebral canal. Therefore, the cord adopts the length of the spinal canal. When the cervical spinal column is flexed (ventroflexed), the cervical spinal canal elongates and the cord is stretched and lengthened. Extension (dorsiflexion) of the cervical spinal column causes the cord to relax and shorten. Smith demonstrated this in the monkey, Breig and Reid in man. When the neck is flexed, the stretched cord is held against any spondylotic protrusions that may be present. During cervical extension, the flaccid cord deviates according to gravity towards the front or back of the spinal canal, depending on whether the patient is prone or supine (Fig. 1).

The present report describes the deformations of the cervical spinal cord caused by flexion and extension of the neck in subjects with and without spondylosis. Microangiography was used to see how the blood vessels of the spinal cord might be altered and also as the basis of a separate report on the blood supply of the cervical spinal cord. The pathogenesis of the myelopathy of cervical spondylosis is then discussed in relation to these findings.

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Fig. 1. Cervical air myelography with tomography in mid-sagittal plane in living patient. Spondylotic bar C4-5. A. Cervical flexion, patient supine, taut cord held against spondylotic ridge. B. Cervical extension, patient prone, slack cord falls towards front of spinal canal. X-rays taken by A. F. El-Nadi. Reprinted courtesy of Acta Radiologica, Stockholm.
Materials and Methods

The material consisted of the cervical spines and the contained spinal cord collected from 42 unselected autopsy cases. The first 40 specimens were also used in our preceding report and only additional details concerning their collection, preparation, and examination will be reported here. Cervical air myelograms were done with the neck in the flexed and extended positions to delineate the presence or absence of spondylotic bars.

Following the perfusion with barium suspension and the substitution of formalin for CSF, each cadaver was placed in the cold room overnight with the neck in a flexed, extended, or neutral position (Table 1). Subjects in the flexed and neutral positions were supine; those in the extended position were prone. Positions of flexion or extension were chosen at random in the first 20 subjects. The next 20 cadavers were placed in a position judged to take advantage of any spondylotic bars seen in the air myelo-

grams.

After at least 14 hours fixation, the cervical and upper thoracic cord, with its roots and dorsal covering, were removed. The existence and size of spondylotic bars was then assessed by inspecting and palpating the anterior wall of the spinal canal after the cord had been removed. Clips were placed on the dura at the level of spondylotic bars observed during removal of the cord.

Microradiographs were made of the whole specimens which were then sectioned and again examined radiologically. When a specimen was indented as a result of contact with a spondylotic ridge during fixation, sections were cut in a serial fashion through the groove. Twenty-six selected sections were then embedded in parafin, cut thinner, mounted, and stained as histologic slides (haematoxylin and eosin, Gomori's elastin, and van Gieson's stains).

The histologic sections and the microangio-
grams were examined and some were photographed with a microscope. The photographs were used to relate the position of the blood vessels to the outlines of the gray matter and of the whole sections.

The last 2 of the 42 specimens were handled in a different way. After perfusion with the barium suspension, laminectomy was done and the length of the anterior surface of the cervical spinal canal, from the upper border of C1 vertebral body to the lower border of C7, was measured in the positions of maximal flexion and extension. The cords were cut at the levels from which measurements had been made, removed, and x-rayed unfixed. The spinal cords were x-rayed in the shortened and elongated states, with the length of the anterior wall of the cervical spinal canal in maximal extension or flexion. To maintain these lengths, the ends of the speci-
mens were pinned through the film to a board. The x-rays were repeated with the cords in the elongated position stretched over a plastic rod, much as they might have been had they been forced against a spondylotic ridge.

Effects of Cervical Positioning

Extended Position. The cervical spinal cords retained the shape they had acquired during fixation within the cadaver. In the 11 specimens fixed with the neck in dorsal extension, the cervical cord was shortened and its relaxed roots and dentate ligaments un-
dulated as has been described previously.

TABLE 1

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Total 22 males (mean age 70 yrs.)
17 females (mean age 69 yrs.)
11 (8*) 12 (5*) 17 (13*)

* Cases with cervical spondylotic ridges.
The anterior walls of the cervical spinal canals in the last 2 cases (Nos. 41 and 42) were both 9.5 cm. long in the extended position compared to 11.0 and 11.8 cm. long in the flexed position. Microradiographs of the unfixed cervical spinal cords at these 2 lengths showed that the individual central arterial branches that ran longitudinally shortened and lengthened proportionately (Fig. 2).

The posterior half of the cord, in extension, shortens more than the anterior. Sagittal and coronal histologic sections showed that the axons of the posterior columns, in the extended cord, took a spiralled course. This was reflected in an increase in the area occupied by the posterior columns in transverse sections. They bulged backwards, accentuated the postero-lateral sulcus, and increased the A-P diameter of the cord (Fig. 3).

**Neutral Position.** The 12 cadavers fixed in what resembled the position of erect posture held the curvature of normal cervical lordosis. In all respects, their final shapes fell midway between the forms retained by those fixed in flexion and extension, so a detailed description is unnecessary.

**Flexed Position.** When the neck was flexed during fixation, the cervical cord was elongated and its roots and dentate ligaments were stretched. In 2 cases, the lateral roentgenograms of the neck showed that some lordosis was retained even in the position of maximal flexion and these specimens looked like those fixed in the neutral position.

Coronal and sagittal histologic sections showed that the axons of the posterior columns run straight when the neck is flexed. The A-P diameter of the cord was diminished but the transverse diameter did not change. The postero-lateral sulcus almost disappeared. This flattening was also apparent in the shape taken by the gray matter (Fig. 4).

**Effects of Spondylotic Bars**

Twenty-six of the first 40 subjects had one or more spondylotic ridges (Table 1). The dura reflected the shape of the protrusions but showed no other changes.

The cervical air myelograms were useful as a primary orientation but tomography, an indispensable adjunct to the procedure, was not used.

**Extended Position.** Spinal cords fixed with

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*Fig. 2. A–P microangiograms of mid-cervical spinal cord, unfixed. A. Shortened (neck extended). B. Elongated (neck flexed). The markers indicate similar segmental levels. Vessels elongate proportionately. Spec. no. 42. X4.5.*
the neck extended were deformed by the anterior wall of the cervical spinal canal on which they lay. Spondylotic bars, present in 8 of the 11 fixed in extension, produced deep grooves. The posterior surface of the cord was humped opposite these grooves (Fig. 5). No impressions were found on the posterior surface of the specimens to indicate contact with the laminae or ligamenta flava; hence there was no evidence of compression. Transverse sections looked much the same whether they were at the level of a spondylotic bar or not, except for some unevenness of the anterior surface of the cord.

Neutral Position. Specimens fixed in the neutral position were not deformed by the spondylotic ridges present in 5 of the 12 for in the supine position the cords had little or

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**Fig. 3.** Transverse sections at C7 segmental level fixed with neck extended. The postero-lateral sulcus is distinct. The A-P diameter is large. A. Van Gieson stain. Spec. no. 27. B. Microangiogram. Spec. no. 4. X9.
no contact with the anterior wall of the cervical spinal canal. A few bore postero-lateral impressions from contact with laminae during fixation.

_Flexed Position._ The specimens were grooved where the cord had been pulled taut over the spondylotic bars found in 13 of the 17 preparations fixed in cervical flexion. In extension, the bars simply displaced relaxed nervous tissue, but in flexion the cord was attenuated where it passed over a spondylotic ridge (Fig. 6). The amount of flattening varied but was greater with large protrusions than with small ones. In transverse sections, the flattening of the cord was symmetrical with midline posterior and bilateral protrusions (Fig. 7) and asymmetrical with unilateral protrusions (Fig. 8). The asymmetry was not caused by oblique sectioning for symmetry gradually returned in sections above and below. When one half of the cord was thinned in the antero-posterior diameter by a postero-lateral protrusion, there was a compensatory lateral widening of that half of the cord. The

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![Image of transverse sections at C7 segmental level fixed with neck flexed](image-url)

_Fig. 4._ Transverse sections at C7 segmental level fixed with neck flexed. Compared with Fig. 3, the area is diminished. The A–P diameter is reduced while the transverse diameter is little changed. The postero-lateral sulcus has almost disappeared. A. Van Gieson stain. B. Microangiogram. Spec. no. 18. X9.
Fig. 5. Mid-sagittal sections of cord fixed in extension. Arrows indicate sites of contact with spondylotic ridges at C4-5 and C5-6 interspaces. Grooves produced on anterior surface by spondylotic bars are associated with displacement of the relaxed cord tissue posteriorly. A. Van Gieson stain. B. Microangiogram. Spec. no. 34. X5. Many veins are filled in the microangiogram.

Fig. 6. Mid-sagittal sections of cord fixed in flexion. Arrows indicate site of contact with spondylotic ridge at C5-6 interspace. The cord is attenuated where it passes over a spondylotic ridge. The axons are not relaxed but are pulled out straight. A. Van Gieson stain. B. Microangiogram. Spec. no. 20. X5.
distortion, flattening and widening was most marked in the lateral columns and anterior horns. In these regions, the arteries, elongated by distortion of the cord, ran in a predominantly transverse direction. The flattening of the cord shortened the penetrating branches from the pial plexus to the anterior and posterior columns. Sometimes these branches had a wavy course in the transverse sections.

Filling Defects

Six of the 42 specimens (Cases 6, 10, 24, 26, 29, and 42) had a level where filling of the arteries with the barium suspension abruptly reduced or stopped (Fig. 9). In each case a spondylootic ridge, although usually a small one, had been found at the corresponding level at autopsy. One defect occurred at the C6 segmental level, the others at C7 or C8. Always, an anterior radicular artery reached the cord at the level of the defect. No filling defects occurred in the 15 subjects without spondylosis.

Case 24 had a defect involving only the side of the cord in contact with a large, nodular, postero-lateral protrusion (Fig. 9B). This was the one specimen where the defect occurred at C6. At that level, there were anterior radicular arteries on both sides and the anterior spinal artery was paired.

The spinal cords were fixed in a position of flexion in 4 of the 6 with filling defects. It was first felt that the contrast material might have been squeezed out of the segment of the cord opposite the spondylootic ridge during fixation. All the cadavers had been perfused in a supine position with their necks in a neutral position, so the cords had little or no contact with the ridges during the perfusion. Attempts were made to squeeze barium suspension from well filled regions of the 2 unfixed specimens both by stretching the cords over a rod and by manually pinching them. The contrast material could not be budged. This satisfied us that the regions of defective filling had never been filled. The anterior spinal arteries at the level of the filling defects were examined with a dissecting microscope and one with serial histologic sections. The lumina were fully patent.

Discussion

In 1924, Barré suggested that the myelopathy of cervical spondylosis (arthritis vertebrae chronic) was caused by ischemia. He noted that compression of the cervical roots in the intervertebral foramina would impair the blood supply of the cord. This theory still receives support and additional ways in which ischemia may be provoked have been proposed. So far as we know, however, no occluded radicular artery has ever been demonstrated post-mortem in a case of myelopathy due to cervical spondylosis. Pathologic studies of spinal cord lesions have supported the view that local ischemia is the final step in the pathogenesis of the disease. Precisely how and where the circulation is disturbed has remained a mystery.

Many believe that the spinal cord is damaged by compression between spondyloitic bars and the laminae and ligamenta flava. However, cases have been cited where no evidence of compression has been found on radiologic examination or at operation. The finding in the present study that the cord is grossly distorted by spondyloitic ridges when the neck is flexed, together with a consideration of the vascular anatomy, offers an alternative to the compression hypothesis.

Kahn suggested that the dentate ligaments hold the cord against spondyloitic ridges but Reid showed that in cervical flexion the contact between the cord and the protrusions is not reduced when the dentate ligaments are divided.

The arterial circulation may be diminished at any point between the vertebral arteries and the capillaries within the cord. The venous drainage system is too extensive to be deranged significantly by cervical spondylosis. The vertebral arteries may be narrowed or blocked by atheroma. They may be kinked by hypertrophic tissue about Luschka's joints. The intervertebral foramina may be narrowed and the radicular arteries forced against laterally-placed osteophytes in the foramina when the neck is flexed and the roots are stretched. The anterior radicular arteries run in a rostral direction while crossing the front of the lower cervical cord and can be indented by the same spondylootic ridges that groove the cord in cervical flexion.

Because there were no known cases of myelopathy in the present material, these possibilities could not be evaluated. The radicular and spinal arteries were never observed.
to be occluded although they were often flattened, a finding impossible to assess in post-mortem studies.

The importance of functional narrowing of the radicular arteries in spondylosis will not be known until pathological reports of cases of myelopathy include a description of the number and distribution of these arteries. If a patient with only one cervical anterior radicular artery had that artery squeezed, it would expose him to a risk which another patient with two or more arteries might escape. Many older persons have extensive spondylotic changes with no evidence of myelopathy.\textsuperscript{5,19} This suggests that a variable, such as the number and location of the radicular arteries, may be highly relevant.

The anterior spinal artery is probably well protected as it would simply be pushed deeper into the anterior median fissure by any spondylotic ridge. However, it takes a more lateral course at times, especially when it is paired, and it is then exposed to the same stress as the anterior radicular arteries crossing the front of the cord.

The filling defects encountered in 6 cases were probably artifacts but as none was found in the absence of a spondylotic bar, they merit some consideration. The lower cervical radicular arteries do not originate from the vertebral arteries, where the perfusion pressure was maintained. The contrast material apparently took the path of least resistance and flowed into the radicular artery at the level of the defect. The cords may have been in contact with the spondylotic bars during the perfusion and hence under sufficient tension to divert the flow of the solution. Pressure on the anterior spinal artery or arteries during cervical flexion may inhibit blood flow past a spondylotic ridge during life. This would only be significant in

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig7}
\caption{Transverse sections at C7 segmental level at level of a spondylotic ridge. Specimen fixed with neck flexed Ridge bilateral. Symmetrical flattening and distortion occur, most marked in the lateral columns and anterior horns. A. Van Gieson stain. B. Microangiogram. Spec. no. 12. X9.}
\end{figure}
the 10 per cent who do not have lower cervical anterior radicular arteries, for blood would usually flow in from below.

When the antero-posterior diameter of the spinal canal is narrow, the cord may be compressed when the neck is extended. It has not been proven that the cord can be damaged by had abnormalities of the spinal cord. Unless some compression occurs, the vessels within the cord are not exposed to any stress when the neck is extended, for the cord as a whole relaxes. Indeed, the position of mild extension can be recommended for achieving relaxation of an injured cord and its blood ves-

the cervical extension accompanying normal activity although the acute paraplegia that may occur in cervical spondylosis has certainly been shown to follow forced and usually sudden extension of the neck. We attach little significance to our finding that cervical extension failed to produce any evidence of compression of the cord, for the cadavers in the series were not known to have sels. Although the cords fixed in extension were grooved by the ridges over which they lay, the passive displacement of nervous tissue would be of no consequence.

The fact that laminectomy is often followed by marked improvement does not justify the assumption that compression of the cord between the ligamenta flava or laminae and a spondylotic ridge is the important mecha-
nism in the production of changes in the cord. Aboulker et al., Breig and El-Nadi, and Reid felt that the cord may be damaged by cervical flexion as well as by the compression that accompanies cervical extension. For this reason, Aboulker et al. performed an extensive laminectomy and postoperatively extended the patient’s neck so that the scar would subsequently limit flexion.

We suggest that in cervical spondylosis the arterial blood flow may be reduced in the vessels supplying the cervical cord thus setting the stage for ischemia precipitated by mechanical stresses applied to the vessels within the cord. The long penetrating branches of the lateral pial arterial plexus which supply the lateral columns are narrow. We suspect that little blood would flow through them to the capillary network when they have been lengthened and flattened by the stresses which widen the cord laterally and flatten it antero-posteriorly opposite a spondylotic ridge during cervical flexion. Nervous tissue is highly vulnerable to anoxia. The circulation would have to be shut off for only a matter of perhaps 10 minutes to cause injury. The normal lumina of these vessels could then be re-established leaving no evidence that they had ever been blocked except for the residual parenchymal damage. Moreover, the flow might simply be markedly reduced, rather than stopped, so that the effects would reflect repeated episodes of hypoxia.

The anterior columns and the outer half of the posterior columns are not destroyed even in advanced cases of myelopathy in cervical spondylosis. The posterior spinal arteries

zig-zag and are not put under tension when the cord elongates in cervical flexion. They merely straighten out a little. Their branches that penetrate the posterior columns run in an antero-posterior direction and are shortened when the cord is flattened. The concomitant longitudinal and lateral displacement of the substance of the cord would, if anything, tend to widen these arteries. Most of the arteries of the anterior columns also run in an antero-posterior direction so the stresses that flatten the cord do not narrow them.

The mechanical stresses exerted on a spinal cord flexed over a spondylotic ridge deform the nervous tissue as well as the blood vessels. The reversible symptoms and signs of the myelopathy of cervical spondylosis may not be due to circulatory changes but rather to disturbances in conduction in over-stretched or squeezed axons.

The findings led to at least one therapeutic consideration worthy of mention. Whether the cord is damaged by flexion or extension of the neck, it is apparent that prolonged maintenance of an extreme position would be more harmful than brief movements. Prolonged postures are more a characteristic of the sleeping than the awakened individual. Therefore, if a patient with signs of early spinal cord damage is given a collar to wear as conservative treatment, it seems particularly important that he wear it at night.

Summary

We have studied the shape adopted by the cervical spinal cord when the neck is flexed, extended, and in the neutral position, utilizing microangiographic and histologic methods in 42 autopsy cadavers. In flexion, the increased length of the cord was associated with a decreased antero-posterior diameter which became more marked where the cord passed over a spondylotic ridge and was then accompanied by lateral widening. The lateral columns and the anterior horns were found to be severely deformed by the mechanical stress produced by spondylotic bars during flexion. The transverse course of regional arteries makes them vulnerable to luminal narrowing when the cord is elongated or flattened. The arteries that penetrate the anterior and posterior columns from the pial plexus run in an antero-posterior direction and are, if anything, widened by the mechanical forces acting upon them. This relationship between the deformity of the spinal cord and the pathways of its intrinsic vessels might explain the pattern of spinal cord lesions that occur with cervical spondylosis.

Unless the cord is actually squeezed between a spondylotic ridge and the laminae and ligamenta flava (an occurrence we could not demonstrate but which may happen to individuals with extremely narrow spinal canals), extension of the neck induces no stresses in the presence of spondylosis.

We believe that narrowing of extra-spinal arteries by contact with spondylotic ridges and other hypertrophic connective tissue contributes to the reduction of blood supply to the cervical cord and have emphasized the possible significance of variations in the number and location of the radicular arteries.

Acknowledgment

We are indebted to Dr. Bo Törnberg, Head of the Department of Pathology, South Stockholm Hospital, for permission to prepare the spinal cord specimens in his department.

References