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Cord Shape and Measurements in Cervical Spondylotic Myelopathy and Radiculopathy

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A combined clinical myelographic and computed myelographic study was performed in 30 patients with cervical spondylotic myelopathy; computed myelography was also performed in 16 control patients. Good correlation was found between degree of deformity of cross-sectional shapes of the cord and the outcome of surgery, and measurements of anteroposterior diameter and area of the cord seemed helpful in this preliminary study. There was also reasonably good correlation between the clinical features when grouped according to specific tract involvement, and the pattern of deformity shown by the abnormal cross-sectional cord shapes.

Cervical spondylotic myelopathy with or without radiculopathy still presents diagnostic and therapeutic problems for several reasons. Cervical spondylosis is common and causes narrowing of the spinal canal in 75% of persons over age 50 years, half of whom have cord signs [1]. Moreover, this condition exhibits clinical features shared by a wide variety of cervical cord diseases. In one series initially diagnosed as spondylotic myelopathy, as many as 17% of the patients were later shown to have other diseases such as multiple sclerosis [2]. Thus it may be difficult to decide whether a myelographic abnormality is producing cord signs. It can also be difficult to determine the degree of cord deformity produced by a combination of osteophytes, disk protrusion, ligamentum flavum infolding, and canal narrowing. The matter is further complicated by the fact that the mechanisms leading to myelopathy in cervical spondylosis are not fully understood.

We designed this study to assess the significance specifically of cord shape and measurements in relation to clinical features in spondylotic myelopathy, using computed tomography (CT) with intrathecal contrast enhancement.

Materials and Methods

Thirty patients (22 men, eight women; mean age 51.1 years, age range 31–71 years) with clinical features of cervical spondylotic myelopathy and/or radiculopathy who underwent myelography were studied prospectively using metrizamide CT. The clinical findings were classified under particular tract dysfunction into three groups. Group 1 had anterolateral column involvement. Symptoms included upper motor neuron signs, disturbance of bladder control, impairment of pain and temperature sensations, and paresthesias. Group 2 had posterior and posterolateral column lesions. Symptoms here included impairment of joint position, light touch and vibratory sensations, two-point discrimination, and ataxia. Group 3 had radiculopathy, and presented with segmental sensory disturbance and diminished reflexes or wasting and weakness in the upper limbs, though the last feature could also indicate anterior horn cell damage. CT and operative findings were compared in those who underwent surgery, and the subsequent course of all patients was followed for 6–14 months. Cervical CT scans to serve as controls were also obtained in 16 patients (nine men, seven women; mean age 42.6 years, age range 31–70 years) who underwent metrizamide lumbar myelography for suspected lumbar disk disease and in whom detailed neurologic examination revealed no evidence of spinal cord dysfunction.

Localization of levels and horizontal positioning of the spine were achieved by screening with markers and then reproducing the position on the scanner couch to obtain sections perpendicular to the long axis of the cervical canal. The scans were obtained within 4 hr of completion of myelography with an EMI 5005 machine using single sections 5 mm thick at intervertebral levels C2–C3 to C7–T1, plus overlapping or consecutive sections at the affected levels in order to cover the adjacent areas. Scans were imaged with the appropriate window center settings ranging from +40 to +200 EMI units, depending on the attenuation of the metrizamide and the cord as described by Seibert et al. [3]. Window width settings were 100–400 EMI units.

Cord shape was assessed visually and deformity classified into four categories, with arbitrary assessments of degree of severity. Category A (fig. 1) defined a cord cross-sectional shape, the main characteristic of which was a concave anterior surface. There were three degrees of severity: A1, minimal deformity of the cord (amounting to a reduction in sagittal diameter of less than 25%); A2, moderate deformity (sagittal diameter reduced by less than 50%); and A3, severe deformity (sagittal diameter reduced by more than 50%). Category B (fig. 2) defined a cord shape showing lateralized deformity. Two arbitrary grades were assigned: in B1 the rounded configuration of the lateral surface of the cord was preserved; in B2 it became pointed, with more severe deformity. Category C (fig. 3) defined a cord shape showing bilateral lateralized deformity resulting in an angular rather than a smoothly convex anterior surface. In the C1 subcategory the posterior surface retained its smooth convexity, but in C2 it became angular due to posterolateral flattening. Category D (fig. 4) defined cords in which the main characteristic was flattening of the entire anterior surface. In the D1 subcategory the posterior surface was normal, and in D2 it was angular. In 10 cases the subarachnoid space was incompletely outlined or obliterated in one or two slices of the most affected level. The cord shape adjacent to these slices was used for classification.

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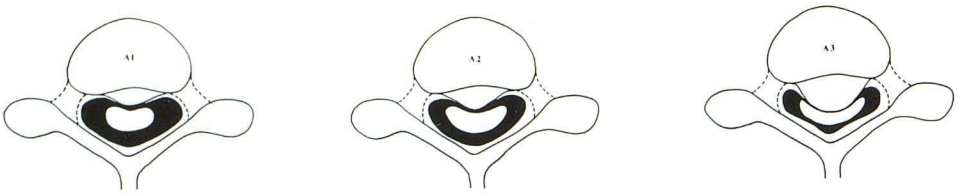
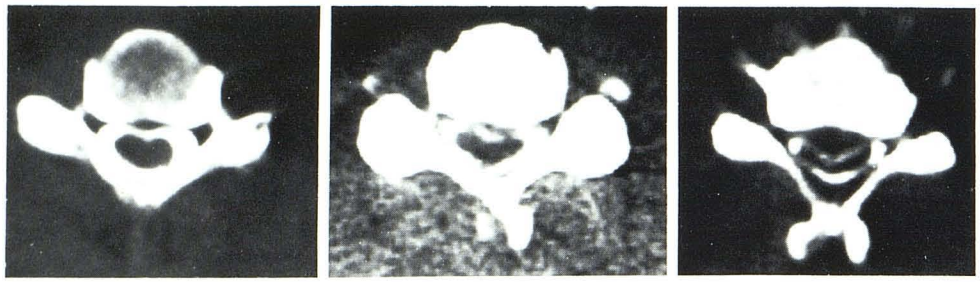


Fig. 1.—Line drawings and scans of A-shape cervical spondylotic myelopathy. Cord is deformed centrally by osteophyte or disk protrusion. A1 = mild, with slightly increased anterior concavity of cord; A2 = moderate cord deformity; A3 = marked cord deformity.



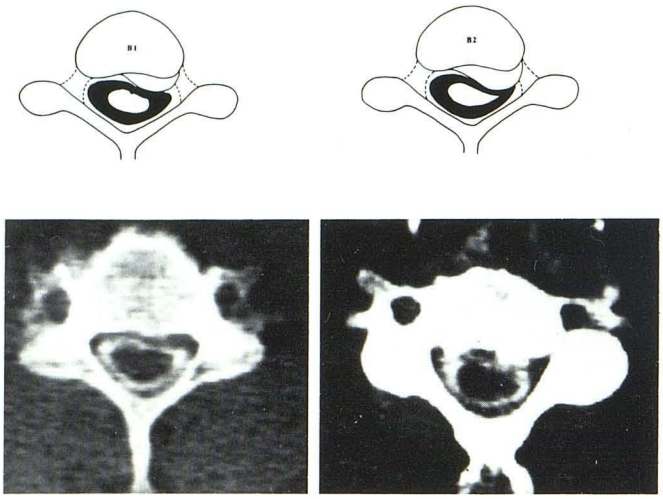


Fig. 2.—B shape. Anterolateral deformity on one side. B1 = mild, lateral funiculus rounded; B2 = severe, lateral funiculus pointed.

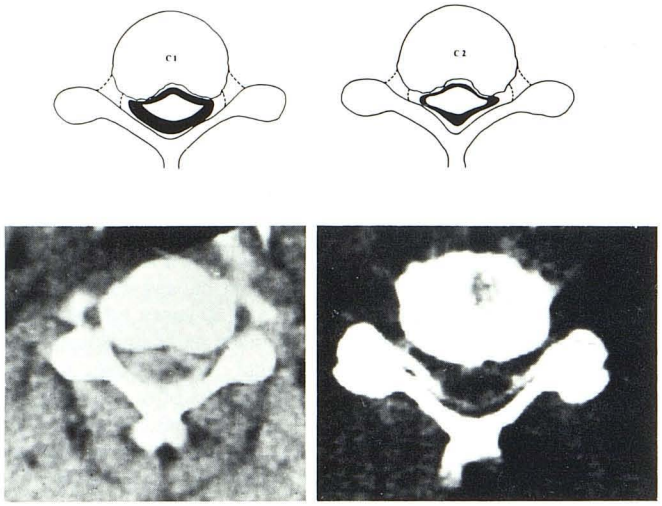


Fig. 3.—C shape. Bilateral deformity. C1 = anteriorly only; C2 = both anteriorly and posteriorly.

The abnormal cross-sectional cord shapes correlated closely with abnormalities of the bony canal and extradural soft tissues as illustrated in the figures. The dimensions of the spinal canal sometimes varied considerably between flexion and extension; however, a detailed comparison of measurements of canal and cord is not possible in this preliminary report.

In 23 of the 30 cases, abnormal cord shapes were present at more than one level. In these cases all severely affected levels were considered. The clinical features were then correlated with the cord shapes. In the 21 operated cases, success or failure of surgery was considered to indicate whether deformities at the operated and adjacent levels were significant.

Measurements of the cord were also made at each intervertebral level. These included direct anteroposterior diameter (APD) and transverse diameter (TD) measurements to the nearest 0.5 mm, and area (a) and circumference (c) using the MOP Image Analyzer (Komtron Messgeräte, Eching, W. Germany). The ratio APD to TD and circularity, as defined by the formula $4\pi\ (a/c^2)$, were calculated. The control values at each level were compared with those of the abnormal cords in the myelopathy cases. The mean APD and area at the 23 levels of surgery and the nine nonoperated but deformed levels were also calculated and compared in the 19 patients who had significant improvement after surgery.

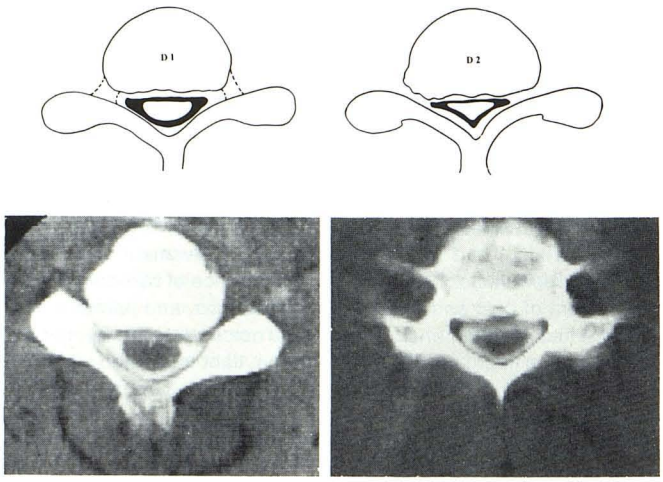


Fig. 4.—D shape. Diffuse distortion, often in association with a markedly narrow canal. D1 = anteriorly only; D2 = anteriorly and posteriorly.

TABLE 1: **Control Values of Cervical Cord Measurements**

Level	Mean (SD, SEM)					
	Anteroposterior diameter (mm)	Transverse diameter (mm)	Ratio APD/TD	Area (mm^2)	Circumference (mm)	Circularity ($4\pi a/c^2$)
C2–C3	8.0 (0.5,0.2)	12.5 (1.2,0.3)	0.65 (0.08,0.02)	85.8 (9.2,2.7)	34.5 (2.2,0.6)	0.90 (0.05,0.01)
C3–C4	7.7 (0.5,0.1)	13.4 (0.9,0.2)	0.58 (0.07,0.02)	86.5 (10.6,2.7)	35.3 (2.3,0.6)	0.86 (0.05,0.01)
C4–C5	7.2 (0.8,0.2)	13.6 (1.1,0.3)	0.53 (0.08,0.02)	86.6 (14.3,3.6)	36.1 (3.0,0.8)	0.83 (0.06,0.01)
C5–C6	7.2 (0.6,0.2)	13.4 (1.2,0.3)	0.54 (0.06,0.01)	84.4 (14.6,3.6)	35.6 (3.4,0.8)	0.83 (0.06,0.02)
C6–C7	7.0 (0.6,0.2)	12.2 (1.3,0.3)	0.58 (0.09,0.02)	73.8 (12.7,3.2)	32.4 (3.2,0.8)	0.88 (0.05,0.01)
C7–T1	7.0 (0.7,0.2)	10.3 (1.5,0.4)	0.69 (0.11,0.03)	62.6 (13.9,3.7)	29.1 (3.6,1.0)	0.92 (0.04,0.01)

Note.—APD = anteroposterior diameter; TD = transverse diameter; a = area; c = circumference. There were 16 cases: nine men and seven women. Mean age was 42.6 years (SD, 10.7; range, 31–70). There was no significant difference in cord size in relation to gender and age.

TABLE 2: **Measurements in Abnormal Cord Shapes**

Shape	Mean (SD, SEM)					
	Anteroposterior diameter (mm)	Transverse diameter (mm)	Ratio APD/TD	Area (mm^2)	Circumference (mm)	Circularity ($4\pi a/c^2$)
A1	6.4 (0.7,0.2)*	13.2 (0.8,0.2)	0.48 (0.05,0.01)	75.1 (11.8,3.0)†	34.6 (2.4,0.6)	0.79 (0.07,0.02)
A2	5.2 (0.8,0.2)*	13.3 (1.2,0.3)	0.40 (0.07,0.02)	58.8 (12.0,3.0)*	33.6 (2.8,0.7)	0.65 (0.09,0.02)
A3	3.3 (0.7,0.3)*	13.5 (0.5,0.2)	0.24 (0.05,0.02)	40.6 (9.4,3.8)*	32.6 (1.5,0.6)	0.48 (0.12,0.05)
B1	6.9 (0.7,0.2)†	13.6 (0.8,0.2)	0.51 (0.05,0.02)	80.4 (11.6,3.7)†	34.6 (3.0,0.9)	0.85 (0.10,0.03)
B2	6.5	15.0	0.44	78.9	36.4	0.75
C1	5.6	11.5	0.49	44.8	26.6	0.79
C2	4.2	13.8	0.31	46.3	28.8	0.70
D1	6.3 (1.3,0.7)†	13.1 (1.0,0.9)	0.48 (0.06,0.03)	65.4 (23.2,11.6)†	32.8 (5.0,2.5)	0.73 (0.09,0.04)
D2	5.2 (0.7,0.3)*	12.9 (1.5,0.6)	0.41 (0.06,0.02)	53.1 (17.3,7.1)*	30.8 (5.3,2.2)	0.68 (0.06,0.02)

Note.—Measurements represent average values from C3–C4 to C6–C7 levels. APD = anteroposterior diameter; TD = transverse diameter; a = area; c = circumference. SD and SEM were not calculated if there were only one or two cases. There were 30 cases: 22 men and eight women. Mean age was 51.1 years (SD 10.3; range 31–71).

* $p = 0.005$.

† $p = 0.05$.

Results

Visual Assessment of Cross-Sectional Shapes

Spinal cords of A shape were seen in 22 patients. Because six of these had considerable deformity of the cord at a further level, only 16 were suitable for analysis of the clinical features associated with the A shape. Anterolateral column involvement was present in nearly all cases: corticospinal tract in 15, bladder control in five, spinothalamic tract in 13, and anterior horn cell in two. Posterior column involvement occurred in 10, and root lesions in four. In 30% the lower limbs were solely or most severely involved; in the others both upper and lower limbs were similarly affected. Both sides were involved symmetrically in 75%. Although bladder dysfunction and the more severe grades of motor disability were only seen where A2 and A3 shapes were present, two cases with an A1 shape at a single level had mild but definite cord signs that improved after anterior spinal fusion. Fourteen patients were operated on by the anterior approach, and all were considerably improved. In one other case laminectomy was performed for a three level abnormality, shape C2 at C3–C4, D2 at C4–C5, and A1 at C5–C6, with only slight improvement. Six patients were treated by immobilization with a cervical collar. Two were slightly and one moderately improved, but three were unchanged.

Seven of the eight patients with B cord shape had relevant symptomatology. On the deformed side of the cord, all had root involvement and five had corticospinal tract signs in the legs. Three cases had wasting of both hands. There were no instances of bladder dysfunction or spinothalamic and posterior column involvement. Two cases underwent surgery. In one recovery was complete, but the other relapsed after initial improvement. The others were managed conservatively, and all but one had some improvement.

One C1 shape was seen, but because an A3 deformity was present at another level it was not possible to isolate the clinical features due to the C1 deformity. The C2 shape occurred in two patients. Corticospinal, spinothalamic tracts, and posterior columns were involved in both upper and lower limbs symmetrically, bladder control was lost, and anterior horn cell damage and sensory root involvement was present in one. Surgery resulted in slight improvement in one, and moderate improvement in the other.

In seven of the 14 patients in the D shape group, the deformity was isolated and suitable for analysis. Anterolateral column, corticospinal, and spinothalamic tracts were involved in all, bladder control in four, anterior horn cell damage in one, and posterior columns in five. The lower limbs only were affected in two, and in all cases involvement was symmetric. Six patients were treated surgically. Five had moderate and one slight improvement. One patient treated conservatively had moderate improvement.

Radiculopathy also occurred at four levels in patients with normal cord shape and the absence of any associated cord deformity.

An ovoid shape of the spinal cord was usually seen at all levels of the control group, with the anterior surface of the cord being less convex than the posterior. However, three patients did show an abnormal shape (A1) in this group.

Cord Measurements

The control and abnormal cord shape values are presented in tables 1 and 2, respectively. The TD and circumference values varied surprisingly little with the degree of deformity, whereas the APD and area values varied significantly. Therefore, we considered the statistical significance of the APD and area only. The mean APD of the 23 operated levels was 4.8 mm (SD 1.50, SEM 0.52) and the mean area 51.3 mm^2 (SD 17.8, SEM 3.9). Corresponding values for the nine nonoperated levels were 5.5 mm (SD 0.94, SEM 0.31) and 60 mm^2 (SD 12.2, SEM 4.06). The difference was not significant ($p > 0.1$).

Discussion

Spinal cord pathology in this condition has been described in over 50 cases [4–7]. Cord indentation corresponding to spondylotic protrusions is a constant feature. Histologic changes are maximum in these sites. These include gray-matter destruction with neuronal

loss and sometimes ischemic changes, white-matter destruction with irregular areas of myelin pallor or necrosis, lateral and posterior column degeneration, and often widespread proliferation of hyalinized small blood vessels. However, the pathogenesis of these cord lesions is still debated. Major postulations are compression, ischemia, and recurrent trauma.

Compression, due to a constitutionally narrow canal and superimposed spondylotic changes, is widely accepted as the most important factor. Pallis et al. [1] noted the association of cord signs and acquired canal narrowing due to posterior osteophytes and subluxation. Payne and Spillane [8] and Burrows [9] demonstrated that patients with spondylotic myelopathy had constitutionally narrower canals than the general population, and the importance of this factor has been confirmed in other studies [7, 10]. Ono et al. [6] found good correlation between deformity as measured by APD/TD on spinal cords at autopsy and the region of most severe intrinsic cord damage, and concluded that compression was the only pathogenetic mechanism.

The distribution of ischemic lesions in autopsy material [4] has suggested that diminution of blood supply could be the dominant factor in cervical spondylotic myelopathy and radiculopathy. However, anterior spinal artery occlusion is seldom observed [5, 6], and the course of myelopathy and the response to surgery are not influenced by concomitant generalized vascular disease [9]. Fibrosis in intervertebral foramina reducing flow through radiculomedullary arteries [11] fails to explain why radiculopathy is frequently unassociated with myelopathy. It is likely, however, that ischemia is the final common pathway for cord damage, a conclusion supported by experimental compression of the cervical cord of dogs, in which reduced blood flow was found at the site of maximum tolerable compression [12].

Adams [13] emphasizes the dynamic aspects of the spinal canal and its contents and believes that myelopathy is caused by recurrent trauma to the cord, either by further compromise of an already narrow spinal canal during extension of the neck or by stretching of a tensed cord against posterior osteophytes during flexion of the neck.

In the present study there was good correlation between the degree of cord deformity and the severity of symptomatology. Cases with A1 and B1 shapes had relatively few or mild cord signs, while in those at the other end of the spectrum (with obliteration of the subarachnoid space) cord signs were numerous and severe.

The A shapes were usually associated with central disk protrusions, the B shapes with lateral protrusion or osteophytes, and the C and D shapes with irregular osteophytes and/or a bulging annulus. The contribution of posterior compression by lamina or infolded ligamentum flavum could seldom be determined directly on the scans, which were obtained with the neck slightly flexed, a position in which these structures usually do not encroach on the spinal canal. However, the posterolateral flattenings seen in cords of C2 and D2 shape were apparently produced by compression from these structures. In most cases it was clear that the predominant deforming force had acted on the anterior surface of the cord, where deformity was most severe and clinical features of anterolateral column dysfunction were most frequent.

The decision for surgery in our cases was made primarily on the basis of myelography. In most, the levels of significant compression diagnosed by myelography and CT were identical, and the results of surgery at those levels were good. However, one case had three abnormal levels on CT (C3–C4 and C4–C5 with A2; C5–C6 with C2). An anterior operation was performed at C5–C6, but, because only transient improvement resulted, C4–C5 and finally C3–C4 levels were operated on. It was only after the third operation that sustained improvement was achieved. The cord shapes had not changed at the unoperated levels on scans obtained between operations, indicating that no new complicating factor such as subluxation had been introduced. Moreover, rescanning of the operated levels in this and another case showed that the cord was much less deformed. This example suggests that CT predicts more accurately than does conventional myelography the degree of cord deformity at which symptoms responding to surgery occur. However, there were eight patients in whom CT had shown minor or moderate cord compression at levels other than those at which surgery was eventually performed, and who improved enough so that no further surgery was contemplated. Also, patients with minor or moderate cord compression who were managed conservatively tended to show improvement, though to a lesser extent than those receiving surgery. In the controls there were three A1 cords similar to those in some myelopathy patients with cord signs that responded to surgery, though the width of the subarachnoid space was narrower in the symptomatic cases. Thus it is not possible to predict precisely from cross-sectional cord shape alone the surgical significance of mild and some moderate compressive deformities. Still, all the severe and most of the moderate deformities were significant and made substantial improvement after surgery.

The tables show that APD and area are the most sensitive measurements of cord compression, and in this preliminary study appear helpful for predicting the outcome of surgery. Furthermore, although Crandell and Gregorious [14] found no correlation between the type of cord syndrome and the radiographic features, grouping the clinical features according to specific root and column involvement correlates reasonably with the cross-sectional shapes of the cord, and may prove helpful in determining whether a particular deformity is responsible for the clinical presentation.

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