Clinical-Radiographic Correlations in Cervical Spondylosis

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Cervical spondylosis is a progressive and disabling disease of later life characterized by a variable combination of upper extremity radiculopathy and predominantly motor myelopathy.1,4,12,14,17 The underlying pathologic process seems to begin with degeneration of cervical intervertebral discs, most commonly at the C4-5, C5-6 and C6-7 levels.2,13,17,18,25 Bony hypertrophic changes then develop as ridges or bars along the margins of the affected interspace.

How these bony-ligamentous changes produce neurologic deficits has been, and remains, a subject of debate. In all probability the observed radiculopathy results either from direct, mechanical compression of individual nerve roots within their foramina of exit or from stretching of the nerve roots secondary to abnormal angulation of vertebral bodies adjacent to degenerated interspaces,6 or to a combination of both.13,17

The mechanism of production of the myelopathy of cervical spondylosis remains more obscure. Simple mechanical compression of the spinal cord by hypertrophic bars anteriorly, or infoldings of the ligamentum flavum posteriorly,9 would be expected to produce predominantly sensory myelopathy. Kahn’s ingenious theory13 implicating the dentate ligaments as stress transmitters has been badly shaken by anatomical studies showing that the tethering effect of these ligaments is restricted to rostro-caudal and lateral displacement of the cord, with anteroposterior movement being relatively unrestricted.6,9,20,21

Considerable interest has also been devoted to the possible role played by vascular factors in the pathogenesis of the myelopathy of cervical spondylosis.2,3,7,18,22 Demonstrable thrombosis of the anterior spinal artery is rarely found,5,25 although one might expect this to be a common sequel if this vessel were compressed to a significant degree against underlying bony bars.

Breig, et al.,7 have reported a microangiographic cadaver study of the circulation of the cervical spinal cord. They were struck by the predominantly transverse course of “regional arteries” (anterior sulcal arteries) within the central spinal cord. They noted that mechanical flattening of the cord tended to narrow the lumina of these vessels through stretching. Such cord distortion or flattening could be produced by cervical flexion, and was particularly pronounced in the presence of transverse spondylotic ridges. Furthermore, clinical observations clearly document deterioration in patients with spondylotic myelopathy coincident both upon cervical flexion and extension3,10,20 while the syndrome of acute traumatic central cord injury, to which spondylotics are peculiarly susceptible, characteristically results from a hyperextension injury.1,12,19 Radiographically, also, a complete obstruction to the cephalad flow of subarachnoid contrast medium during myelography can often be demonstrated only with the neck in dorsal extension.22

Compression of radicular arteries, or veins, within the neural foramina has been a popular concept of the possible vascular pathogenesis of cervical spondylosis.2,3,16,18,23 Anatomical studies have demonstrated that the blood supply of the spinal cord is derived in greatest measure from a relatively few segmental vessels. In the cervical region two or more such dominant arteries enter the lower cervical segments in precisely the same area in which the bony-ligamentous changes of spondylosis are apt to be severe. The relatively greater sensitivity to hypoxia of anterior horn cells and posterolateral fiber columns would then explain the clinical specificity of the disease.23

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The exact mechanism of embarrassment of these radicular vessels is of itself a matter of dispute. The thesis of nerve root fibrosis, championed by Frykholm,\textsuperscript{12} is not widely accepted as a major factor in spondylotic myelopathy, though studies by Wilkinson\textsuperscript{26} and by Breig\textsuperscript{6} lend some support. In the presence of degenerative disease of intervertebral discs, presumed to be the initiating pathologic process in cervical spondylosis, some narrowing of the intervertebral foramina can usually also be demonstrated.\textsuperscript{10} The additional development of lateral bony spurs would then be expected to produce significant compression of nerve roots and their accompanying vessels.

Such bony changes should of course be readily and consistently visible by appropriate radiography. In fact, however, considerable disagreement exists as to the correlation of clinical and radiographic findings. Payne and Spillane\textsuperscript{18} have demonstrated radiographic changes typical of cervical spondylosis in patients with minimal or absent neurologic deficits. Some authors have declared the changes seen on plain roentgenograms, or even on myelography, to be of little or no value in estimating clinical severity of the disease.\textsuperscript{2,4,12,23} Nonetheless, several clinical-radiographic correlations have gained acceptance. It is generally conceded that patients with cervical spondylosis are more apt to have interspace narrowing and hypertrophic spurs and bars, though these changes may also be seen in people who do not exhibit clinical manifestations of spondylosis.\textsuperscript{17,18} In addition, patients with significant myelopathy generally have a decrease in the anteroposterior (AP) diameter of the cervical canal.\textsuperscript{6,9,13,16,18,26}

Thus, the predominant radiographic correlation of cervical spondylosis is with hypertrophic degenerative changes and decreased AP canal diameter, and would seem to support the notion of a purely mechanical compression of the spinal cord as the pathoanatomic basis of spondylotic myelopathy. That such a mechanism has been deemed unlikely in terms of the clinical features of the disease has been noted. Yet the clinically more popular notion of a vascular basis of pathogenesis has received little support from clinical-radiographic correlations. In an attempt to explore this apparent ambiguity, we have correlated the radiographic and clinical findings of a series of 54 patients with the clinically accepted diagnosis of cervical spondylosis who were seen at the Boston Veterans Administration Hospital between 1963 and 1965. Other patients, in whom plain x-rays and myelographic studies were inadequate for the purpose of this study, have been excluded.

**Methods**

A series of measurements were made at the four interspaces between C-3 and C-7, as demonstrated on plain radiographs and on cervical myelograms. Included were measurements of "pre-existing" sagittal canal diameter, height of posterior interbody spurs, "absolute" AP canal diameter ("pre-existing" sagittal diameter minus spur height), interpedicular width, width of cord shadow, and "sagittal cord diameter" (estimated by measuring from the anterior limit of the cord shadow within the column of contrast medium to the nearest point on the overlying laminae). The degree of foraminal encroachment and the degree of transverse interruption of the column of contrast medium (the severity of "myelographic bars") were estimated semiquantitatively.

**Results**

Average measurements and correlation between findings on plain x-ray films and on myelograms have been presented in a separate report.\textsuperscript{24} Of special interest were the excellent correlations found between the height of posterior interbody spurs seen on plain films and the magnitude of transverse bars demonstrated on myelography; the lack of correlation found between the magnitude of transverse myelographic bars and various measurements of spinal cord widening was equally significant. It was also demonstrated that the diminished sagittal spinal canal diameter seen in cervical spondylosis was one dimension of a general decrease in canal size affecting both sagittal and transverse diameters.

The clinical-radiographic correlations in the present study were quantitated mathematically in terms of their correlation coefficient "r" (a perfect correlation = 1.000) and its reliability factor "P". The correlation between the presence of foraminal en-