Significance of the Small Lumbar Spinal Canal: Cauda Equina Compression Syndromes Due to Spondylosis

Part 1: Introduction*

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Spondyotic caudal radiculopathy (SCR), although a common ailment with important neurosurgical implications, is currently less well known and understood, even by specialists, than spondyotic cervical myelopathy with which it is identical except for the anatomical level and the neural structures involved. A predisposing feature common to both is an abnormally shallow spinal canal, probably first described at the lumbar level by Sarpyener.21

Verbiest's many descriptions20-29 of small lumbar canals, beginning in 1949, were contemporaneous with the reports of the English neurologists which established spondyotic cervical myelopathy as a neurological entity, but antedated by several years those which demonstrated a premorbid, smaller than normal cervical spinal canal as a precursor.5,14,19

Abnormally shallow lumbar spinal canals were described a decade or more before the related neurological deficits were recognized, while cervical myelopathy due to spondylosis was firmly established as a clinical entity for several years before the significance of a shallow cervical canal was discovered.

Cervical spondylosis has received increasing and deserved attention in the past 18 years.3 Discovery of the myelopathy due to spondyotic encroachments on the smaller than normal cervical canal has provided a new and rational treatment for the many lesions formerly misdiagnosed as atypical amyotrophic lateral sclerosis, primary lateral sclerosis, combined systems disease, and multiple sclerosis.

The equally important lumbar disease has not been systematically considered and is only fragmentarily described in papers dealing with one or another facet of its complex nature. It has been confused with familiar disorders known to produce low back and leg complaints such as disc herniation, arachnoiditis, intermittent claudication due to leg muscle ischemia, intraspinal tumor, post-anesthetic palsy, psychoneurosis and malingering. Many of the disappointments resulting from treatment for one or another of these disorders might be avoided if SCR is identified and appropriately treated.

Summary of Clinical Characteristics

To comprehend the variety of clinical manifestations of SCR it is important to distinguish it from spondylosis in the normal canal or ordinary disc herniation (Fig. 1). Disc herniation usually occurs at one level in a normal-sized canal and affects only one root; treatment simply involves removal of the degenerated disc fragments. Even sizable spondyotic encroachments on normal or large canals are rarely of consequence15 (Fig. 2). When, however, a canal which has always been shallow is affected by spondylosis, the arthritic encroachments at multiple levels produce multi-level interferences with cauda equina function which, unlike simple disc herniation, commonly require surgical relief at several levels. Furthermore, the spondyotic process involves more than the disc alone. The facets enlarge, the laminae become dense and heavy, and the yellow ligaments thicken and undergo other changes.17 These factors combine with the bulging disc to produce SCR. The disc abnormality is commonly the least significant of these factors. To remove it interlaminally as though it were the sole abnormality invites therapeutic failure.7

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In the neck, the length of the pedicles, the angle at which the right and left halves of each lamina meet, the size of the intervertebral foramina, and the depth of the spinal canal from the high point of the lamina to the subjacent vertebral body are easily determined on plain radiographs. The degree of variability of these structures and relations is as great in the lumbar spine as in the cervical, but is more difficult to assess. Normally, the depth of the lumbar canal is from 22 to 25 mm with some even larger. In myelograms of such deep canals, even strikingly prominent transverse ridges may have no neurological significance (Fig. 2). Certain individuals, however, have canals as shallow as 15 mm or less (Fig. 3). As Verbiest first observed, the developmentally small non-achondroplastic canal is shallow only in the A-P dimension, not from side to side. Interpedicular measurements remain normal and provide no clue to the existence of a small canal in either the lumbar or cervical regions. In patients with very small canals, little spondylosis need develop before the cauda equina begins to suffer. Such patients resemble achondroplastic dwarfs who, as a result of failure to develop pedicles of normal length, have spinal canals too shallow to support normal neurological function beyond middle life. These achondroplastic patients often become paraplegic with very little evidence of spondylosis being present.

**Myelographic Defects**

Not only does the existence of a critically shallow canal often go undetected on an ordinary radiographic examination, but it may also be missed in myelography. Since the roots are tightly packed in a subarachnoid space of reduced capacity, they are relatively fixed in position, and a myelographic needle introduced at any of the usual lower lumbar levels may produce root pain and poor fluid flow. If the myelographer ignores this clue that some pathological process has reduced or obliterated the spinal fluid compartment, the test is likely to be very disagreeable for

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**Fig. 1.** Diagram showing certain features of simple disc herniation in an otherwise normal spine (left), spondylosis in a spinal canal of normal depth (center), and severe spondylosis in a small canal (right).

**Fig. 2.** Myelogram showing large spondylotic ridges which are inconsequential because of the generous size of the spinal canal. An upper thoracic meningioma was the indication for this study. *Left:* Anteroposterior view shows striking bilateral defects at L3-4. *Right:* Lateral view shows the L3-4 ridge to encroach only trivially on the great available depth of the spinal canal. The laminal arches (L) are dimly seen far above the fluid level.