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

Part 4: Acute Compression Artificially-Induced During Operation*

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IMPAIRED function of the sphincters, paralysis in the legs, and sensory losses of paresthesias appearing after operations within the chest, abdomen or pelvis, and upon structures or organs other than the spinal cord or cauda equina have, for the most part, been reported as presumed complications of spinal anesthesia. Root laceration by the puncturing needle, bleeding and meningeal reaction to blood, trophic disturbances sequential to occult actions of the anesthetic agent, subarachnoid osmotic pressure disturbances, neurolytic effects of the injected drug, infection, inadvertent introduction of chemical contaminants, and drug-produced angiopathy of the cord and roots are among the mechanisms invoked when spinal anesthesia is followed by neurological deficits.\textsuperscript{1,11-12} Acute compression of the cauda equina by artificially induced lordosis while the patient is under anesthesia must be added to this list for, in a 5-year period, I have encountered six such instances that resulted in cauda equina palsy. In each case the existence or significance of a predisposing spinal abnormality was not recognized by the anesthesiologists and surgeons. Two of these six patients had lumbosacral spondylolisthesis with intact neural arches and have been described elsewhere.\textsuperscript{10} In one of these previously described cases, the mechanism producing the harmful lordosis was undoubtedly that described by Hunter, \textit{et al.}.\textsuperscript{6} Four patients had acute lordosis-aggravated spondylotic caudal radiculopathy (SCR) due to lumbar spondylosis in developmentally shallow canals. They represent the condition which is the subject of this report.

As has been described SCR is capable of simulating many diseases and has many parallels with cervical spondylosis and its cervical myelopathy. The acute cauda equina palsy that results from forcing the back into a degree of extension or lordosis that it could not normally endure has its counterpart in cervical spondylosis. Patients with small cervical canals and a sufficient degree of arthrosis may develop an acutely paralyzing cord lesion should they experience sudden neck extension in a fall on the face, or in the hands of a cultist or careless anesthesiologist.

Case Reports

\textit{Case 1.} A 52-year-old lawyer first had low back pain in his early 30's, for a period of 20 years thereafter, he experienced many attacks of low back and leg pains. The right Achilles reflex had been absent and sensation in the back of the right thigh had been reduced for years. In April, 1964, another severe attack of back pain occurred, and sensory loss extended to the scrotum and penis.

Routine x-ray films of the chest disclosed a lesion in the left lung, and because of this the patient underwent thoracotomy while lying on his right side with his low back supported by and extended against an operating table fixture. A granulomatous rather than a neoplastic lesion was found. The patient awakened from the general anesthetic with a severe lower cauda equina lesion which, because of his generally uncomfortable state, was not fully appreciated for several days.

Myelography (his fifth) was then performed and this was followed by multiple level lumbar laminectomy.

When I first examined the patient over a month later he still had severe cauda equina deficits. The recent myelogram was diagnos-
When I saw the patient 2 weeks later, he told me of having severe back pains from the age of 9 when he fell on ice. While serving in the army in France during World War I, he had had his last severe attack during which he had to be lifted onto and off his cot for 6 weeks. In recent years he had been able to sit and drive with comfort but could not extend his back or lie supine or prone with legs extended without producing pain. Severe ascending numbness and weakness of the legs after walking two and a half blocks, followed by complete relief within 40 seconds of sitting down, had been appraised as intermittent claudication due to atherosclerosis of leg vessels. This diagnosis seemed to be confirmed by abnormalities in angiograms made before the operation which resulted in his paralysis. A urogram, made while searching for the cause of disturbed micturition, revealed that the spine was stiffened or rigid above L4-5. There were enormous spurs extending laterally and anteriorly around the L4-5 disc which retained motion (Fig. 2).

The patient agreed to another attempt at myelography provided he would not be expected to lie extended. While he lay on his side in bed with thighs and back flexed, upper lumbar puncture with a slender needle was made and Pantopaque injected. The needle was then bent over and covered with a sterile dressing so that he could be fluoroscoped lying supine with legs flexed and the buttocks supported on a padded box. This showed complete block at the L4-5 interspace which did not open up on maximum flexion of the thighs and back (Fig. 3).

Laminectomy of L-4 and 5 was then carried out with the hips and back strongly flexed to avoid further injury to the cauda equina. The L4-5 facets were huge and almost touched the intervening L-4 spinous process. The heavy yellow ligament was depressed beneath very thick laminae. An aperture in the anulus fibrosus was found, possibly related to the old history of back and leg pain. However, the spinal canal contained no herniated disc material. After laminectomy and partial medial facetectomy, the patient was able to lie extended. He could also walk erect for long distances with comfort. He has regained considerable power and sensation in his legs but sphincter malfunction persists.