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 spondylosisthesis 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, et al.\textsuperscript{6} Four patients had acute lordosis-aggravated spondyloitic caudal radiculopathy (SCR) due to lumbar spondylosis in developmentally shallow canals. They represent the condition which is the subject of this report.

Received for publication September 31, 1968.

* Presented as part of a panel discussion on "Syndromes of the Small Lumbar Spinal Canal" at a meeting of the Southern Neurosurgical Society, New Orleans, Louisiana, February 17, 1968.

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

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 diagno-
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.

tic of severe spondyloitic compression of the cauda equina at multiple levels, but so was the one made in 1960 (Fig. 1). In both there were large extradural defects at L2–3, L3–4, and L4–5 with complete block at the lower level. It seemed that he had not been operated upon at the time of his 1960 study because the characteristic myelographic appearance of spondylosis in a small canal was interpreted as an extraarachnoidal Pantopaque injection.

**Case 2.** An 83-year-old man underwent sympathectomy and femoral angioplasty under general anesthesia while supine with folded sheets under his back to improve the exposure. He awakened with loss of sensation in the perineum and lower legs, paralysis below the knees, and inability to control the bowels or bladder. Disc rupture on the operating table was suspected and myelography attempted. This was aborted before the needle was even introduced because the prone position caused more pain than the patient was willing to tolerate; he persisted in turning to one side to flex his legs and back.

![Fig. 1. Case 1; myelograms made in 1960, 4 years before the development of severe cauda equina palsy during thoracotomy for a benign lesion. Left: Anteroposterior view. None of the opaque material injected at the L2–3 interspace finds its way below the L4–5 disc. At the L2–3 and L3–4 levels the spondylotic encroachments from front and back almost obliterate the subarachnoid space. Right: Lateral view. This appearance of opaque material immobilized back of several vertebral bodies is characteristic of multiple levels of spondylotic compression of the cauda equina, but is sometimes mistaken for subdural extra-arachnoidal injection of the contrast substance.](image-url)
Case 2. A 67-year-old man had a problem almost identical with that in Case 2. A low back and leg pain syndrome, aggravated by walking, and leg and pelvic traction was diagnosed as intermittent claudication due to ischemic leg muscles. After angiograms confirmed the existence of circulatory abnormalities, bilateral femoral angioplasty was carried out. When the patient regained consciousness he showed severe cauda equina deficits. These were amply explained by examination of plain films of the lumbosacral spine (Fig. 4) and a myelogram (Fig. 5). The subsequent operative findings and clinical result were the same as in Case 2.

Case 4. A 24-year-old woman had been recognized as having pseudo-pseudohypoparathyroidism since the age of 15. In August, 1967, she was admitted to a hospital for a urological operation, mentioning at the time that for 2 months she had experienced vulvar and perineal numbness. After an operation under general anesthesia with her back in lordotic posture, she had retention of urine and complaints of reduced vaginal sensation, posterior thigh hypesthesia, and "electrical" sensations in the backs of her legs. Two attempts at myelography resulted

Fig. 2. Case 2; urograms of the lumbar spine made after the patient developed cauda equina palsy while under anesthesia for arterial prosthetic surgery. Left: Anteroposterior view. Right: Lateral view. The thoracic and lumbar interspaces down to L4-5 are stiffened and L-5 appears fused to the sacrum. Only L4-5, where there are large lateral and anterior spurs, retains motion.

Fig. 3. Case 2; posteroanterior myelogram. The needle has been bent over flush with the skin and covered with a dressing. The patient is lying supine upon the needle with the knees and thighs flexed. Note the bilateral narrowing of the caudal sac by encroachment of the hypertrophic facets, yellow ligament, and disc at the site of the complete L4-5 block.
bony hard subcutaneous nodules on the extensor surface of the left forearm. There was hypesthesia in all sacral dermatomes, but tendon reflexes were active and equal. No anal reflexes were present.

Laminectomy of L-3, 4, and 5 showed that the spinal canal was almost 50% shallower than normal. Postoperatively there was some return of perineal sensation but she is still obliged to wear a Foley catheter.

This case is particularly interesting because of the existence of an abnormally shallow canal in a person who developed cauda equina compression at a relatively young age. This raises the possibility that a shallow canal may be common in pseudo-pseudohypoparathyroidism as it is in achondroplasia.

Discussion

Acute intraoperative complications such as these cannot be rare if one man has knowledge of six. However, catastrophes that may result from extending the low back under anesthesia, especially in the patient with a back complaint helped by bending forward and aggravated by standing erect or extending the back, are not mentioned in most authoritative anesthesiology texts and articles. Alexander draws attention to the fact that lordosis produced by abduction of the thighs in stirrups may cause backache, but most practicing anesthesiologists seem more familiar with the opinion of Collins'
that anesthesia impairs muscular support and maintenance of the normal spinal column configuration with resultant "flattening of the normal lordotic curve and an excessive stretch of the spinal ligaments." For operations in the supine position, he advises placing a small pillow under the lumbar area of the back.

Thinking that there must be other cases of acute SCR for which spinal anesthetic drugs received unjust blame during a variety of operations done in the supine position upon the elderly, I sent letters of inquiry to over 70 members of an anesthesiology society in my locality. However, no additional cases turned up. The difficulties in all but one of the cases I have reported occurred during operations at which one or more of these same anesthesiologists were in attendance, but in no instance were they aware of this complication having occurred.

The explanation for this may be that cauda equina malfunctions occurring during operations of considerable magnitude, especially in the elderly, are not generally recognized for 3, 4, or possibly more days by which time the anesthesiologist has made his last visit to the patient. The operating surgeon, who may expect the patient to need catheters and to complain of back and leg pain and numbness, commonly fails to relate the problem to any modifiable feature of the operation itself. Rather, he regards it as something of a mystery which he hopes urological and neurological consultants will solve for him.

As with the other types of manifestation of SCR, acute palsy due to operative positioning have undoubtedly been observed in the past and attributed erroneously to a variety of mechanisms. An interesting comparable probable case was described by Goldthwaite in 1911, whose 39-year-old patient had had 7 years of low back pain attributed to hypertrophic arthritis. Displacements of the sacroiliac joint on the right side had been diagnosed and "replaced" under anesthesia, but the pains kept returning intermittently until one day, on getting out of a tube, the patient was unable to straighten up and had intense pains in his legs. He was anesthetized with ether and the sacroiliac joint again put in place. When this failed, he was placed supine on an orthopedic frame for application of a plaster cast, and, just as his back was made lordotic or extended, he complained for a moment of intense pain after which he relaxed. In a few moments he was found to have numb legs. This deterioration progressed to complete loss of sensation, motor paralysis, and sphincter incontinence. When the patient was placed on his side in bed, neurological function returned to a considerable extent, but whenever he was placed on his back he grew worse. Finally, during an episode of supine recumbency, he lost function permanently. Thereafter he complained not only of paralysis but of explosive cutting pains in the legs, feet, and rectum. After 6 weeks of this, Harvey Cushing explored the lumbar spinal canal, and found nothing but narrowing of the bony canal at the lumbarosacral level. Gradual improvement reportedly followed.

The prevention of this lordosis-produced
catastrophe during operation depends upon realizing the potential for compression in a small canal with a spondylotic process or in spondylolisthesis with an intact neural arch. Careful attention must be given to the complaints of patients who have pain on lying supine or prone, or when attempting to extena the back. It should be better known that, in addition to leg vessel atherosclerosis, there is this other condition capable of simulating intermittent claudication by causing the legs and back to become painful, paresthetic, and weak on walking. When the history suggests the possibility of SCR, perceptive neurological examination and roentgenography of the lumbar spine with attention to the significant features mentioned above, plus myelography, should prevent most of these complications. If a patient with a spondylotic or spondylolisthetic spine is to be operated upon, the surgeon and anesthesiologist must devise ways of accomplishing the indicated procedure with the back neutral or flexed rather than lordotic, for it is the lordotic position which is intolerable to the patient with lumbar spondylolisthesis in a small canal or spondylolisthesis with an intact neural arch.

Summary

Spondylotic cauda equina radiculopathy (SCR) is a condition quite similar in its mechanical aspects to the more familiar process occurring in the cervical region. The lumbar spinal canal's diameter is reduced from the front by bulging of the discs, from the sides by encroachment of enlarged facets, and from the rear by hypertrophic and overlapping laminae depressing thick or infolded yellow ligaments. In the patient with a developmentally small spinal canal, these spondylotic enlargements produce compression of the cauda equina, the degree varying with the posture of the low back. Exaggeration of the normal lordotic curve, or even enforcement of a normal lordotic position in a patient obliged by this disease to walk stooped or bent over, increases the constrictive effect upon the cauda equina. Anesthetization of patients suffering from this condition allows them to be made lordotic in the low back without the normal chance to complain of pain; during this interval of back extension under anesthesia severe cauda equina damage may occur. Four instances of this complication due to SCR have been reported, in addition to two due to spondylolisthesis with intact neural arches previously reported. Suggestions for recognition and avoidance of the syndrome have been discussed.

References