Intermittent Claudication from Compression of Cauda Equina by a Narrowed Spinal Canal*

A. Brish, M.D., M. A. Lerner, M.B., and J. Braham, M.D.

Departments of Neurosurgery, Radiology and Neurology, Government Hospital, Tel-Hashomer, Israel

The syndrome of intermittent claudication almost invariably is considered to be caused by vascular insufficiency of the lower limbs, so that even if oscillometric measurements are normal, patients often may be subjected to aorto-arteriography. However, claudication also has been described as resulting from diseases of the spinal cord and cauda equina. Dejerine recorded it in association with syphilitic arteritis of the cord, and Foix and Alajouanine referred to a syndrome of claudication in cases of "necrotic myelitis." (These were later considered by Wyburn-Mason to be examples of impaired blood supply associated with vascular malformations.) Verbiest, in his publications concerning the congenitally narrowed lumbar spinal canal, mentioned the occurrence of symptoms resembling intermittent claudication. Blau and Logue, described 6 cases of intermittent claudication produced by protrusion of a central disc.

Claudication resulting from degenerative disease of the lumbar spine is not an adequately recognized condition, as the histories of long duration of severe incapacitating symptoms testify. Furthermore, as well as producing radicular symptoms, as described by Epstein et al., spondylotic narrowing of the spinal canal appears to contribute to the production of claudication. Claudication is not mentioned in a review by Teng and Papatheodorou of 30 patients described as suffering from compression of the cauda equina by lumbar spondylosis. Two cases which exemplify these two contentions are therefore considered worthy of presentation.

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* All communications should be addressed to M. A. Lerner, M.B., Department of Radiology, Government Hospital, Tel-Hashomer, Israel.
gether with severe compensatory tilts at other levels. In place of the normal lordosis there was slight backward angulation at L2-L3 with prominent osteophytes and visible posterior bulging of the annulus (Fig. 1A). The 9 cc. of injected Pantopaque streamed upwards despite the semi-erect position of the patient; a standing film (Fig. 1B) demonstrated the considerable contribution to the multiple constrictions of the sac by posterior hypertrophic changes. The 24-hour erect film showed almost total block between L3-L4 and persistent loculation between the other discs (Fig. 1C).

Operation. On Dec. 20, 1962 laminectomy of L2-L5 was performed under general anesthesia. The laminae were found to be very short and thickened. On removal of the 6th lumbar lamina the dural sac bulged considerably but this decreased as unroofing proceeded. The ligamentum flavum was thickened and epidural fat was much reduced. Anteriorly, only the herniated L2-L3 disc was soft and removable; hardened bulging annuli were present at other levels. The articular processes were much thickened, contributing largely to the narrowing of the spinal canal. They also encroached upon and narrowed the intervertebral foramina; this was relieved by bilateral foraminotomies with freeing of the compressed nerve roots. The laminae produced deep impressions on the dural sac which resembled a string of beads; at the conclusion of the operation the sac was soft and pulsatile. Fig. 1D demonstrates the restoration of the smooth contours of the sac.

Course. By 6 weeks after operation the patient was able to walk as far as he wished without any discomfort. The urinary disturbances also were relieved although the reflex and sensory deficits remained unchanged. He had returned to full clerical employment.

Case 2. Y.C., a 53-year-old male, was referred from the department of vascular surgery with a 3-year history of paroxysms of pain and weakness in the legs provoked by walking. Peripheral pulses and oscillometry were reported as normal. The patient recalled a severe low-back strain 27 years previously for which he was treated by rest in bed for 9 months. Subsequently, only slight pains in the back were noted intermittently, which did not interfere with his work as a truck driver.

The present symptom-complex began while walking downhill: his legs suddenly weakened, forcing him to lean on a wall until they recovered after some minutes. This weakness began as a numb sensation in the left hallux, spreading to all the toes and to the foot, by which time foot-drop was manifest. Before he was brought to a