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

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The syndrome of intermittent claudication almost invariably is considered to be caused by vascular insufficiency of the lower limbs, so that even if oscillographic 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 claudiation produced by protrusion of a central disc.

Claudiation 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 spondylisis. Two cases which exemplify these two contentions are therefore considered worthy of presentation.

Case Reports

Case 1. N.Y., a male aged 36, was referred on Dec. 12, 1962 from the department of vascular surgery. His principal complaint was of pain in the lower back radiating to the legs, accompanied by weakness and unsteadiness, and directly related to walking, or standing erect.

As a boy he had suffered physical injuries at the hands of Nazi guards at a concentration camp. In 1954, sudden low-back pain appeared while lifting a heavy weight. After 10 days of rest in bed, the residual pain was referred to both legs and subsequently relieved by an orthopedic girdle. A second similar episode occurred in 1958 when he was treated by rest in a cast.

Following a third attack in 1959, he had remained subject to paroxysms of low-back pain, associated with sensations of “electricity” and heat radiating down the posterior and lateral aspects of both legs to the toes. Transitory erection or the desire to urinate often accompanied these paroxysms. They had a constant relation to prolonged standing or walking which, if persisted in, led to unsteadiness, weakness and, finally, falling. Rest for a few minutes brought complete relief, which could be accelerated by squatting on the heels. Coughing and sneezing had no effect. During these 3 years, micturition had been difficult to initiate, was often incomplete and was terminated by dribbling. Sexual function was undisturbed.

Examination. Paravertebral lumbar spasticity was noted, especially on the right side; there was mild lumbar scoliosis to the left. Wasting of muscles in the lower limbs was not detectable. Straight raising of the leg to 70° was possible on both sides. His gait was normal. The only abnormalities of the reflexes were an impaired knee jerk and weaker plantar flexor on the right. Sensation to pin prick was impaired over both buttocks (sacral dermatomes) and over the outer aspects of both calves and feet.

Pulses of the leg and oscillography were normal. Routine laboratory findings, including serology, were normal. Lumbar puncture at L3-L4 interspace yielded normal fluid with normal manometric conditions.

Films of the spine showed an old fracture of the left half of the upper portion of L5 vertebra, to-

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Fig. 1. Case 1. (A) Lumbar spine. Loss of lordosis. Narrowed L2-L3 disc with proliferative changes in vertebral bodies. Mild spondylosis elsewhere.

(B) Myelography. Lateral film, erect. Constrictions at each discal level by protrusion of disc and hypertrophic changes in arches and ligamentum flavum. Almost total block at L4-L5.

(C) Myelography, 24-hour erect film. Some of the Pantopaque has reached the cul-de-sac. Compressed roots of cauda stand out against background of Pantopaque. Severe tilting of spine at several levels.

(D) Erect film 2 weeks postoperatively. Residual Pantopaque shows restoration of thecal contours to normal.

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
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standstill, foot-drop also appeared on the right, with numbness in the whole of the lower extremities. Burning sensations were experienced in the buttocks. This pattern of attack occurred with increasing frequency until finally he was limited to 40-60 metres on walking. Forward bending or squatting shortened the duration of the attacks. Coughing and sneezing had no effect. Latterly, lying straight, prone or supine provoked similar sensations in the legs, only avoidable by lying with knees flexed. Between paroxysms the legs felt normal.

Examination. The patient walked with a slight stoop. Mild lumbar scoliosis to the left with marked spasm of the paravertebral muscles was noted. There was slight atrophy of the left buttock, thigh and calf, with slight weakness of flexion and extension of the left toes and foot. The left ankle jerk was absent and plantar reflexes were flexor. Sensation to pinprick was diminished in the sacral distribution of the buttocks and on the outer aspect of the left calf and foot. Straight-leg raising was not restricted in either leg.

Radiologically the lumbar spine showed normal general posture; minimal forward displacement of the column was noted at the L4-L5 level. Here, and to a lesser degree at other levels, the vertebral body was compressed with eburnation and formation of spurs (Fig. 2A).

Lumbar puncture revealed clear fluid at a pressure of 150 mm. water, with free rise and fall to 300 on compression of cervical vein. Pantopaque (9 cc.) was instilled for myelography. The L3-L4 disc bulged backwards and a total hold-up occurred at the L4-L5 level though on the 24-hour film some of the residual Pantopaque was found to have flowed on. The erect films (Fig. 2B, C) showed narrowing of the entire lumbar dural sac, partly produced by indentations of the ligamenta flava. Narrowing of the spinal canal with compression of the cauda equina was diagnosed.

Operation. On Mar. 28, 1963, lower lumbar laminectomy was performed under general anesthesia. The laminae were short and thick and the ligamentum flavum was extremely thickened. Epidural fat was absent and the dura mater bulged under tension relieved only on completion of the laminectomy.

A soft small herniation of the L4-L5 disc was removed but a hard ridge at L3-L4 was not touched. Pulsation did not appear in the sac until the L3 lamina was removed.

Course. Following operation there was rapid and striking improvement. Pain disappeared and spasm of paravertebral muscles diminished considerably. Power in the left foot and toes improved. The left ankle jerk, however, remained absent and the sensory impairment persisted. One month after operation the patient could walk without restriction and without the appearance of numbness or weakness.

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Fig. 2, Case 2. (A) Slight displacement of L4 and L5. Narrowed discs and spondylotic changes at all levels.
(B) Myelography. Erect film. Pantopaque filled theca. Almost total block at L4-L5. Note indentation of column of Pantopaque opposite L5 and L4 vertebral body by hypertrophic changes in neural arch.
(C) Myelography. Anteroposterior erect film. In addition to discal lesions, roots of cauda stand out against the Pantopaque which is thinned out in the compressed thecal sac.
Discussion

This report is prompted by the paucity of publications dealing with this syndrome and because of its possible confusion with the claudication produced by lower aortic occlusive disease.\(^6\)

A review of the literature leads to the conclusion that differentiation of such claudication from that produced by compression of cauda equina is not possible on clinical grounds in patients who are found to have impaired pulses of the leg. Since both conditions occur from early middle age onwards, deficiencies of pulse may be incidental and noncausal. In such cases we should perform aortography first; if this examination excludes occlusive disease, myelography should then be carried out. When pulses of the legs are normal, as in our cases, myelography should be the first line of investigation.

The claudication of peripheral arterial obliterative disease should not cause any confusion because of the cramping muscular type of pain and changes in nutrition and color which accompany the impaired distal pulses.

The syndrome of Dejerine\(^3\) (intermittent claudication of the spinal cord) and the syndrome of Foix and Alajouanine\(^3\) (dysbasia intermittens spinalis) are associated with signs of a lesion of an upper motor neurone, because the arterial insufficiency, either from syphilitic or vascular disturbances, is above the level of the conus.

Blau and Rushworth\(^2\) demonstrated experimentally that ipsilateral segmental vasodilation in the cord occurs on exercise of a limb. They therefore go so far as to postulate that this syndrome of compression of the cauda is related to exercise by virtue of the vascular congestion, in the constricted dural sac, which occurs on exercise. One of our patients could provoke his symptoms of "claudication" by lying prone. We, therefore, favor direct neuronal compression as being responsible. As to the etiology of the narrowed canal we should not go so far as to regard every case as being predisposed to by a congenitally narrowed canal. Injury, with resultant mild deformity leading to more widespread hypertrophic changes, appears to constitute the chain of events in many cases.

Delay in diagnosis is not surprising since the relation of symptoms to exercise mimics that of pain in the ischaemic leg, whilst the usual sciatic syndrome associated with degenerative disease of the lumbar spine is lacking.

The failure of coughing or sneezing to provoke pain from the roots is explained by the extremely tight and tense dural sac one sees on exploration. It is difficult to imagine any movement of a nerve root in it, or in the compressed intervertebral foramina, except as a result of movement of the spine itself; the rise in pressure of fluid resulting from coughing or sneezing alone cannot, apparently, cause friction of roots on bony prominences and, therefore, does not produce radicular pain.

In addition to severe obstruction of the canal at a discal level, diffuse narrowing of the lumbar canal contributes largely to compression of the cauda. This was demonstrated on myelography by standing films of the Pantopaque-filled lumbar theca, and confirmed at operation, when additional laminectomies, above the level of the obstructing disc, were required to free the tense bulging and nonpulsatile lumbar sac.

Summary

Two cases are added to the literature of the syndrome of claudication caused by degenerative disease of the lumbar spine. Delay in diagnosis and appropriate treatment are liable to occur if such patients are not differentiated from those suffering from obliterative vascular disease. In addition to severe compression of the lumbar theca by discal protrusion, more extensive narrowing of the canal is held contributory, as evidenced by the myelographic and operative findings.

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References
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