"Bicycle test" of van Gelderen in diagnosis of intermittent cauda equina compression syndrome

Case report

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The authors describe a simple clinical adjunct to the routine neurological examination of patients with intermittent cauda equina compression syndrome. The "bicycle test" helps exclude intermittent claudication due to vascular insufficiency and frequently confirms the relationship of posture to radicular pain.

KEY WORDS • **"bicycle test"** • **spinal canal stenosis** • **intermittent cauda equina compression** • **pseudoclaudication**

THE pain of both intermittent claudication and so-called "pseudoclaudication" is often brought on by walking. Rest brings prompt relief from pain of muscle ischemia, but not necessarily from pain of radicular origin. We believe van Gelderen[16] was the first to make this observation. In 1948, he described two patients whose legs became painful, weak, and numb when they walked for 10 to 15 minutes. Both men also experienced the same pain when they stood erect, and were relieved of it when they sat down. One of the men played tennis, and rode his bicycle without pain. Since cycling was painless, van Gelderen reasoned that ischemia was an unlikely cause. He came to the conclusion that lordotic posture, rather than exercise, caused their pain.

We have performed this "bicycle test" as an aid to diagnosis in 10 patients, where the clinical diagnosis was in some doubt. In the patient to be described, an endeavor was made to correlate the myelographic abnormality with the clinical symptoms in greater detail than usual.

Case Report

This 68-year-old man was referred to us because of increasingly disabling buttock and lower-extremity pain. Often his legs would tingle and then become numb. These symptoms were invariably produced by walking but sometimes also when standing still. The patient himself attributed the pain to "poor circulation" because his feet felt cold. He had no low-back pain but did experience midline sacral discomfort when the extremity pain was most intense. The left leg was more painful than the right. On several occasions the pain was so intense he had to sit down on the floor to avoid a fall.

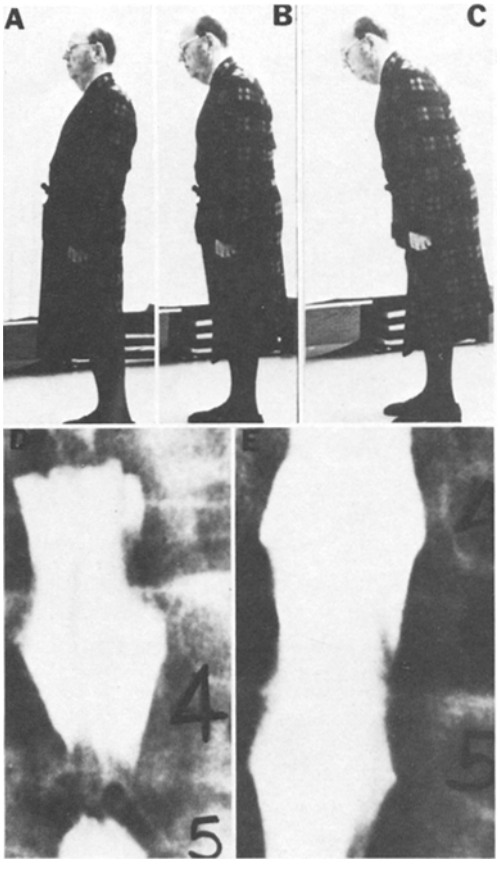

FIG. 1. With the patient standing upright or with slight hyperextension of the low back (A and B), high-grade partial obstruction was present myelographically (D). As he stooped forward (C), free flow of contrast material was established (E). The lateral views were identical to those with the patient sitting on an exercise cycle as illustrated in Fig. 2. The encroachment on the common dural sac was primarily from the apophyseal joints laterally, to some extent from the lamina and ligamentum flavum dorsally, and from the osteophytic ridges ventrally. This patient described pain when there was a myelographic block, and became symptom-free as he stooped forward, thus relieving the obstruction to flow of contrast material.

Examination. He had a minimal restriction of lumbar range of motion. There was no spinous process tenderness, but percussion of the lumbosacral musculature produced ipsilateral aching buttock pain. The sciatic nerve trunks were not tender. There was no muscle weakness or sensory deficit. The patellar reflexes were present; both ankle jerks were absent. Straight leg raising was unrestricted bilaterally to 80°. The dorsalis pedis pulses were not palpable and the posterior tibial pulses were faint. Femoral artery pulsations were also reduced and a faint bruit was detected on the right side. There was slight calcification of the abdominal aorta.

Walking upright caused the patient bilateral buttock pain (Fig. 1 A). Standing still, but remaining upright, brought no relief but as he bent forward his discomfort subsided (Fig. 1 C). In this posture, the myelographic deformity was less marked (Fig. 1 E).

In the physical therapy department, the patient was seated on an exercise cycle and asked to pedal against resistance (Fig. 2 A). The activity was begun with him leaning backward to accentuate the lumbar lordosis. He described buttock and posterior thigh pain almost immediately. As the pain increased, he developed tingling in the lower extremities, particularly the left foot. He was asked to lean forward and grasp the handlebars but continue to exercise (Fig. 2 B), and when he did this the pain decreased; as he crouched forward (Fig. 2 C) all discomfort subsided within a short time, although the pedaling was never interrupted. When the patient sat upright, the pain once again recurred and persisted as he walked back to his room. When he was lying in bed in a semi-Fowler position, he was again free of pain.

The electromyogram was normal and spinal fluid protein was 60 mg%. X-ray films revealed lumbar spondylosis with particularly prominent spur formation at the fourth intervertebral level. Myelography was performed with the patient assuming the various postures described above to correlate the symptoms with the myelographic abnormality.

Operation. Since the pain pattern was most reminiscent of a fifth lumbar radiculitis and the myelographic deformity was most impressive opposite the L4–5 intervertebral level, the fourth and fifth laminae were bilaterally excised. A concomitant medial facetectomy helped free the nerve roots from lateral entrapment. The ventral osteophytic ridges were not removed, and no fusion was performed.

Postoperative Course. The postoperative convalescence was uneventful. He was discharged 2 weeks later with some low-back discomfort, but free from the disabling lower-extremity and gluteal pain.

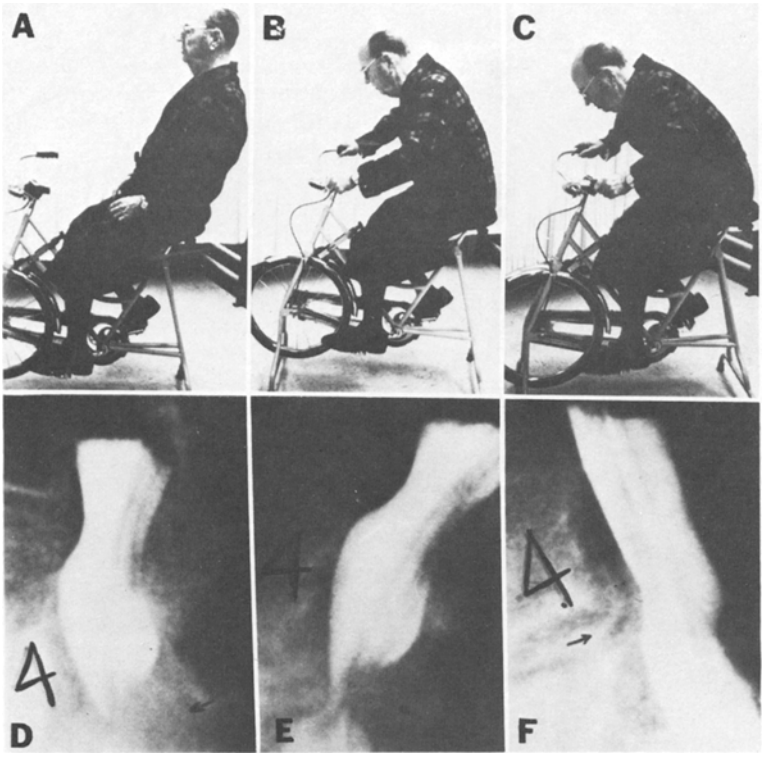

FIG. 2. The "bicycle test." With the patient seated and leaning backward he is asked to begin the exercise (A). The lateral myelographic views in a comparable position revealed a complete block to Pantopaque flow (D). Within a minute this patient experienced leg pain. He was then asked to continue pedaling the cycle but grasp the handlebars (B). The myelographic appearance in this position is depicted in (E). The pain became less intense. With the patient crouched over the handlebars (C) the pain subsided and the myelographic block was completely relieved (F). Slight subluxation was produced.

Discussion

A painful limp is the most obvious manifestation of muscle ischemia. To describe it, Charcot[2] coined the term "intermittent claudication." This was derived from the Latin root "claudicatio," which means to limp or be lame. With time, claudication and ischemia became essentially synonymous in the minds of physicians. As other causes of limping were added, the taxonomy of lameness became complex from a differential diagnostic standpoint. Compression of the cauda equina also produced a painful limp. This limp was superficially indistinguishable from the gait disorder seen in patients suffering from intermittent claudication. Thus terms such as neurogenic intermittent claudication,[6] intermittent claudication of the cauda equina,[1] intermittent ischemia of the cauda equina,[8] pseudoclaudication,[9] and others[4,5,17] have been used to describe this caudal compression syndrome.

Intermittent cauda equina compression, particularly due to lumbar spondylosis, is becoming recognized more frequently, especially in elderly patients. Since the vascular status of the lower extremities in these patients is frequently impaired, the differentiation between ischemic and radicular pain may be difficult. Concomitant diabetic or nutritional neuropathy is also not uncommon and may account for the frequently unobtainable Achilles reflex. Nor can one rely on the myelographic examination to prove or disprove radicular compression since Hitzelberger and Witten[7] have shown that gross myelographic deformity may be present in totally asymptomatic patients. It is also an accepted fact that traumatized nerve roots are exquisitely painful.[10,11] Under these circumstances, a study of the relationship of

pain to posture appears imperative to arrive at a correct diagnosis of intermittent cauda equina compression.

It occurred to one of us (PD) that van Gelderen's observations[16] could be easily adapted as a useful diagnostic test that would differentiate ischemic from radicular pain, and at the same time reveal the relationship of pain to posture. It is now generally accepted that laminal hypertrophy,[3] ligamentum flavum thickening[14,15] or buckling, and zygoapophyseal joint encroachment[17] are common causes of cauda equina compression. The fact that this compression is intermittent and related to posture has been myelographically corroborated by Schreiber and Kulik,[12] Yamada, *et al.*,[18] and Snyder, *et al.*,[13] to cite only a few.

Had the pain been due to muscle ischemia it would have increased as the cycling exercise continued. Instead, with the patient crouched forward, it subsided because the neural compression had been relieved. This simple test thus helps exclude intermittent claudication while at the same time confirming the presence of intermittent cauda equina compression.

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References

1. Blau JN, Logue V: Intermittent claudication of the cauda equina: an unusual syndrome resulting from central protrusion of a lumbar intervertebral disc. **Lancet 1**:1081–1086, 1961
2. Charcot JMC: Sur la claudication intermittente observée dans un cas d'oblitération complète de l'une des artères iliaques primitives. **Comptes Rendu Soc Biol 10**: 225–238, 1858
3. Cooke TD, Lehman PO: Intermittent claudication of neurogenic origin. **Can J Surg 11**:151–159, 1968
4. Ehni G: Spondylotic cauda equina radiculopathy. **Tex J Med 61**:746–752, 1965
5. Epstein JA, Epstein BS, Lavine L: Nerve root compression associated with narrowing of the lumbar spinal canal. **J Neurol Neurosurg Psychiatry 25**:165–176, 1962
6. Evans JG: Neurogenic intermittent claudication. **Br Med J 2**:985–987, 1964
7. Hitzelberger WE, Witten RM: Abnormal myelograms in asymptomatic patients. **J Neurosurg 28**:204–206, 1968
8. Joffe R, Appleby A, Arjona V: Intermittent ischemia of the cauda equina due to stenosis of the lumbar canal. **J Neurol Neurosurg Psychiatry 29**:315–318, 1966
9. Kavanaugh GJ, Svien HJ, Holman CB, et al: "Pseudoclaudication" syndrome produced by compression of the cauda equina. **JAMA 206**:2472–2481, 1968
10. Murphey F: Sources and patterns of pain in disc disease. **Clin Neurosurg 15**:343–351, 1968
11. Norlen G: On the value of the neurological symptoms in sciatica for the localization of a lumbar disc herniation; contribution to the problem of surgical treatment of sciatica. **Acta Chir Scand (Suppl) 95**:1–95, 1944
12. Schreiber RR, Kulik WJ: Personal communication, June, 1970
13. Snyder EN Jr, Mulfinger GL, Lambert RW: Claudication caused by compression of the cauda equina. **Am J Surg 130**:172–177, 1975
14. Spurling RG, Mayfield FD, Rogers JB: Hypertrophy of the ligamenta flava as a cause of low back pain. **JAMA 109**:928–933, 1937
15. Towne EB, Reichert FL: Compression of the lumbosacral roots of the spinal cord by thickened ligamenta flava. **Ann Surg 94**: 327–336, 1931
16. van Gelderen C: Ein orthotisches (lordotisches) Kaudasyndrom. **Acta Psychiatr Neurol 23**:57–68, 1948
17. Verbiest H: Neurogenic intermittent claudication in cases with absolute and relative stenosis of lumbar vertebral canal (A.S.L.C. and R.S.L.C.), in cases with narrow lumbar intervertebral foramina, and in cases with both entities. **Clin Neurosurg 20**:204–214, 1973
18. Yamada H, Ohya M, Okada T, et al: Intermittent cauda equina compression due to narrow spinal canal. **J Neurosurg 37**:83–88, 1972

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