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

Part 3: Intermittent Claudication*

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After recognizing the condition in carriage horses, Charcot,2¹ in 1858, gave the first adequate description of intermittent claudication. In its original meaning, the term intermittent claudication means the painful contraction of anoxic muscle, but because in the literal sense it means periodic limping, the term has also been applied to disorders of two neural structures, the spinal cord and the cauda equina. Dejerine,2 believing that exercise could produce symptoms on the basis of spinal cord ischemia, reported three cases of “intermittent claudication of the spinal cord.” Many reports of intermittent claudication of the cauda equina have appeared,3-6,7,13,15,16,18,19,29,33,36,37 The three varieties of intermittent claudication all produce signs and symptoms that appear after the patient has walked a predictable distance, and they disappear when he has rested. The symptoms cause the patient to claudicate, that is, to limp.

This paper is concerned primarily with intermittent claudication due to spondylotic changes affecting the cauda equina. Intermittent claudication due to ischemic muscle, the commonest of the three conditions, will be discussed only in relation to differential diagnosis of cauda equina claudication. Intermittent claudication due to lesions of the spinal cord is an exceedingly rare disorder,2² and will not be considered further.

Received for publication October 17, 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.
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Syndromes of Intermittent Claudication of the Cauda Equina

The syndrome of “intermittent claudication of the cauda equina” derives its name from the relationship between the times of appearance and disappearance of the symptoms. Once induced, either by activity or by posture, symptoms disappear within a matter of seconds or at most a few minutes after the precipitating activity has been discontinued.¹³ For a particular patient, the activity leading to symptoms and the period of rest required for their relief have a predictably quantitative relationship. Except in rare instances sensory symptoms precede motor manifestations in the patient with cauda equina claudication, and characteristically the patient stops the pertinent activity before weakness develops. However, if the patient continues his activity beyond the appearance of discomfort or paresthesias, the legs may become weak to the point of collapse. Examination at this time may disclose sensory deficits and loss of reflexes.

Symptoms. Intermittent claudication due to lesions affecting the cauda equina is characterized by predominantly sensory symptoms quantitatively related to exercise. The pain has distinctly paresthetic qualities and, even when severe, seldom simulates classical sciatica. The pain has been described as numbness, coldness, or burning; less often it has a distinctly cramping quality.¹⁵ Coughing and sneezing rarely provoke these symptoms. Some patients deny pain altogether and complain only of numbness and paresthesias. Sensory symptoms beginning in the lumbar region and buttocks may remain confined to these areas, but often descend in the distribution of the lower lumbar and sa-
crical dermatomes. In other patients sensory symptoms begin in the feet and ascend to the buttocks. This sensory “march” is common.

Each patient can relate his particular pattern of symptoms to specific activities. Almost all patients voluntarily report pain on walking. Careful questioning will reveal that many patients experience identical symptoms with the adoption of certain body positions, such as standing immobile. The significance of different factors in the provocation of symptoms will be considered in the section on pathogenesis.

*Signs.* Examination of the patient at rest usually discloses few neurological abnormalities, and this disparity between symptoms and signs has diagnostic importance. Although muscular atrophy may be pronounced, minor degrees of weakness and reflex asymmetry occur more commonly. Sensory abnormalities may be found in lower lumbar and sacral dermatomes, but in many cases are absent. Seldom are spinal movements restricted significantly, and the straight leg raising maneuver is typically normal or nearly so. Repetition of the neurological examination after the patient has been asked to continue walking until the symptoms appear may be highly informative, because motor and sensory disturbances, absent at rest, may then become apparent.5,18

**Pathogenesis of Cauda Equina Claudication**

There is a near-perfect correlation between this syndrome and sagittal narrowing of the lumbar spinal canal secondary to enlarged apophyseal points, shortened pedicles, thickened ligamenta flava, and posterior protrusion of either bulging intervertebral discs or marginal osteophytes.7,13,17,36 Compression of cauda equina roots from other causes (extradural or intradural neoplasms, and massive disc protrusions in spinal canals of normal dimensions39) rarely produces an intermittent claudication syndrome.

All patients with intermittent claudication of the cauda equina develop symptoms while walking, yet close questioning allows their separation into two groups. In the larger group, symptoms also appear during any activity or body position that involves extension of the lumbar spine, and for convenience this group may be termed “postural.” In the second and smaller group of patients, symptoms appear only after exercise of the affected extremities. For reasons given below, symptoms related to exertion rather than to posture are believed to be caused by arterial insufficiency of cauda equina roots, and this group will be termed “ischemic.”

**Postural Cauda Equina Claudication.** In these patients the appearance of symptoms is clearly related to lordotic posture. Symptoms appear not only after walking, but also after the patient stands quietly or kneels with his back extended. Depending upon his spinal posture, symptoms may or may not appear when the patient reclines. Symptoms are not precipitated by activities such as riding a bicycle or playing tennis,10 since neither of these activities involves continuous hyperextension of the lumbar spine. Symptoms are characteristically relieved when the patient bends forward or squats.8 Most of the cases reported by Verbiest fall into this group,36,37 as does Duvoisin and Yahr’s Case 3, an achondroplastic dwarf.12

Breig8 has shown that extension of the lumbar spine causes protrusion of the intervertebral disc(s) with displacement of cauda equina roots in a dorsal direction. Extension also increases the cross-sectional diameter of the cauda equina roots by decreasing the length of the spinal canal to which the roots accommodate by shortening. Ehni13 has shown the effects of the lordotic position in the course of myelography: extension produced total block, and flexion permitted the contrast medium to pass through the blocked area. With the spine immobilized, leg movements such as the Lasègue maneuver produce no movement of the cauda equina roots.5

Although weight-bearing in the upright position produces slight bulging of the lumbar intervertebral discs, this factor probably plays an insignificant role in the production of symptoms of postural cauda equina claudication. That weight-bearing is coincidental to the lordotic posture of standing can be shown by the occurrence of symptoms in the recumbent position. Brish, et al.’s second patient could lie neither prone nor supine with any comfort unless he flexed his knees.6 In patients with postural intermittent claudiation, symptoms correlate almost perfectly with the lumbar lordotic posture and occur independently of muscular contraction. Ischemia of