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,2 and will not be considered further.

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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.13 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-
eral 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 dimensions50) 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,19 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

Breig5 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 claudication, symptoms correlate almost perfectly with the lumbar lordotic posture and occur independently of muscular contraction. Ischemia of
the involved nerve roots may occur as a passive event secondary to mechanical compression if, as proposed by Denny-Brown and Brenner,10 paralysis produced by a tourniquet bears close resemblance to the effects of prolonged local pressure.

Ischemic Cauda Equina Claudication. Case 2 in Evans' discussion18 is a typical example of ischemic cauda equina claudication. This patient developed cramp-like pain in the anterolateral aspect of both legs while walking, and the discomfort diminished when he stood still. Exercise of his gastrocnemius and soleus muscles on a foot ergometer reproduced his symptomatic pain, not in the maximally exercised muscles but over the fifth lumbar dermatome. Evans could not induce pain in patients with ischemic cauda equina claudication by having them stand for prolonged periods, and in two patients when pain was produced by ergometric exercise of the gastrocnemius muscle while lying in bed, neither the severity nor the time of onset was affected in the slightest by increasing or decreasing lumbar lordosis by means of pillows. The onset of symptoms was directly related to the rate and duration of exercise.

In one patient, Evans determined the "claudication time" by arranging a fixed rate of work on a foot ergometer, and then measured the time to claudication while the patient breathed air, pure oxygen, and a mixture of nitrogen and 12% oxygen. Claudication time varied directly with oxygen tension, and on this basis Evans postulated a relative ischemia of active cauda equina roots during exercise. Blau and Logue25 also favored an ischemic basis for cauda equina claudication because the symptoms could be abolished rapidly and because sensation was impaired before motor function.82

In the mouse, Blau and Rushworth4 discovered that exercising a single hindlimb produced local increased vascularity in the corresponding segment of the spinal cord. Vessels of the spinal nerve roots became prominent, often dilating widely in exercised animals. Tureta and Hodes85 confirmed these observations. Blau and Logue8 suggested that the symptoms of cauda equina claudication were produced by an increase in pressure on abnormally confined nerve roots due to vasodilatation and a probably negligible increase in volume of the nerve fibers. Evans17 disputed this hypothesis because he could not cause induced pain to radiate into an inactive limb by exercising the opposite one, an event that he reasoned should occur according to the mechanism proposed by Blau and Logue.

Working with rats, Cranefield and colleagues5 determined the rate of oxygen uptake in peripheral nerves. Oxygen uptake reached a higher steady level when the nerve was conducting impulses, and the levels paralleled the frequency of stimulation. The rate of oxygen up-take in excised sympathetic ganglia also increases during repetitive stimulation, as shown by Larrabee.81 These two studies establish the increased oxygen demand of conducting peripheral neural tissue.

Gilliat and Wilson23,24 studied the effect of a pneumatic arm tourniquet in patients with various disorders involving sensory nerves in the upper limb. In patients with carpal tunnel syndrome, elevation of the pneumatic tourniquet above arterial pressure produced an early onset of paresthesias and sensory loss on the involved side when compared to the onset of sensory disturbances in control limbs.23 Gilliat and Wilson24 later extended their study to patients with lesions of nerve roots and peripheral nerves proximal to the tourniquet and obtained identical results. They suggested that 30% to 40% of nerve fibers must cease to conduct before sensory loss becomes apparent. They further suggested that an occult lesion may become manifest with ischemia, that is, that a sensory deficit at any level of the nervous system may be exaggerated by the addition of an ischemic peripheral nerve block, the peripheral block by itself being insufficient to produce sensory loss.22

Circulatory Factors. Cauda equina roots derive their blood supply from anterior and posterior radicular arteries that course centrally along the nerve roots.25 These arteries supply their respective nerve roots only, and contribute nothing to the circulation of the spinal cord. They are end arteries and possess no effective anastomotic connections with surface vessels of the spinal cord. Occlusion of a radicular artery at any point from its origin to its termination near the spinal cord leads to ischemia of the portion of the nerve root proximal to the occlusion.
Because lumbar spondylosis usually spares the highest lumbar spinal segments, the great anterior medullary artery (which enters most often at the first or second lumbar segment on the left)\(^5\) is involved only rarely. Reports of ischemic cauda equina claudication give us no reason to suspect involvement of the great anterior medullary artery, whereas impairment of radicular arterial flow at and above areas of caudal root compression would seem consistent with clinical observations and experimental studies cited above.

Although compression of cauda equina roots forms the pathological basis for both postural and ischemic claudication, observations strongly suggest that compression may affect predominantly either nerve roots or their vascular supply.\(^6\) While the concept of two separate forms of cauda equina claudication may serve no practical purpose from the standpoint of treatment, separation of mechanical and vascular factors may provide insight into the more complicated problem of cervical spondylotic myelopathy and radiculopathy. The difficulty of separating postural from vascular factors is illustrated in a patient (described by Blau and Logue)\(^7\) who developed symptoms after standing, but whose peripheral pulses in the legs were just palpable.

**Intermittent Claudication Due to Ischemic Muscle**

True intermittent claudication constitutes one of the most specific symptoms in medicine.\(^1,2,6\) This angina of contracting somatic muscle is due almost always to chronic occlusive arterial disease, although on occasion it may be caused by systemic anoxia or anemia. Symptoms are brought on only by continuous exercise and are promptly relieved by discontinuance of that exercise.\(^2\) Rarely, a patient with aorto-iliac occlusive disease may develop distress in the lower back, buttocks, and thighs when standing for prolonged periods, presumably because of the active muscular contractions needed to maintain balance. Relief does not depend on cessation of weight bearing or to a change in the position of the affected part. The pain, seldom severe, is usually preceded by slight fatigue. The character of the pain is in the nature of cramping, tightness, or tiredness in

the exercised muscles. If the patient continues to walk, the muscles gradually become stiff and tight, and use of the limb becomes impossible. The pain may be either unilateral or bilateral, and may occur anywhere from the lumbar region to the feet. Gifford and Hurst\(^6\) have correlated the location of pain with the level of obstruction.

Aorto-iliac occlusion is the vascular problem most likely to be confused with cauda equina claudication. The earliest and most common complaint of patients with aortic occlusion is pain in the hips, thighs, and back,\(^6\) and in practice, the syndrome described by Leriche is encountered only occasionally.\(^1\) In contrast to peripheral arterial obliterative disease, which usually leads to early recognized changes in skin color with impaired distal pulses, occlusive disease of the aorta and iliac vessels rarely produces nutritional manifestations. Although peripheral pulses are typically normal or near-normal with proximal arterial obstruction, they may become diminished after walking. Except in rare instances, aorto-iliac occlusion causes diminution of the femoral pulse and often produces an audible murmur over the occluded vessel. Vascular disease may produce uncomfortable dysthesias in the feet and, on occasion, neurological signs such as wasting and hypotonia.

**Case Reports**

Two examples of the postural variety of intermittent claudication related to the cauda equina illustrate typical symptoms, their relief by operation, and the vulnerability of cauda equina roots to minor intrusions into a developmentally small spinal canal.

**Case 1.** A 38-year-old man was seen in June, 1967, because of intermittent numbness and weakness of both legs for the previous 6 years. Symptoms occurred during either walking or quiet standing, and squatting or simple flexing of the thighs brought relief. The patient reported that after he had walked a short distance both legs “went to sleep,” and he had to stop because of progressive numbness and “loss of control of moving.” Symptoms had gradually increased during the preceding 6 months, and at the time of admission he could walk only one-
half a city block or stand quietly for only 10 to 15 minutes before they appeared. By contrast, he could swim without difficulty, and he volunteered that swimming relaxed him. He denied injury to his back.

**Examination.** The spine was normal except for mild thoracic scoliosis. Spinal movements were unrestricted and painless. Motor and sensory examinations of the legs including straight leg raising to 90° bilaterally were normal. Pulses in both legs were strong, and lumbosacral spine films were normal. A provisional diagnosis of “intermittent claudication of the cauda equina” was made.

Myelography performed by a lumbar puncture at L3-L4 revealed a complete extradural block at the lower border of the L-4 vertebral body.

**Operation.** Bilateral laminectomy was performed, during which the surgeon was impressed by the small size of the spinal canal. The dural sac was dorsally displaced by a firm protruding mass composed of intervertebral disc and bone, and a free disc fragment was discovered in the narrow lateral recess on the right.

**Postoperative course.** The patient was completely relieved of symptoms and returned to work 2 months later.

**Comment.** This patient developed symptoms of cauda equina dysfunction after quiet standing, and obtained relief by acutely flexing the lumbar spine, either by squatting or by lying with thighs flexed. He was not examined when symptoms were present, but his history indicated that he developed painless weakness which temporarily prevented further activity. In this case disc protrusion of only mild degree compromised a developmentally small spinal canal.

**Case 2.** A 52-year-old woman was first seen in November, 1966, because of pain of 18 years' duration in her back and buttocks and more recent intermittent numbness of both legs. Symptoms were first noted after an automobile accident in which she sustained fractures of several lumbar spinous processes and the left femur. During the 5 years prior to examination, quiet standing and walking had brought on numbness and paresthesias beginning in the buttocks and spreading distally into both feet. More recently, she had become unable to walk for more than 50 feet before symptoms caused her to stop. She could obtain relief by sitting or lying down.

**Examination.** Lumbar spinal movements were normal. Findings on neurological examination were normal except for absence of both ankle jerks. Dorsalis pedis pulses were strong. Plain spine films disclosed anterior displacement of the L-4 vertebral body, narrowing of the L-4 and L-5 interspaces, and a healed fracture of the L-3 transverse process.

A myelogram revealed a small spinal canal with a near-complete block at L4-L5.

**Operation.** Bilateral laminectomy was difficult because of greatly thickened and bulbous articular facets. The spinal canal was extremely narrow in both sagittal and transverse dimensions. The annulus fibrosis at the narrowed L4-L5 interspace was folded slightly but contributed insignificantly to spinal narrowing. The L5-S1 disc was flat.

**Postoperative course.** One month after operation all symptoms had disappeared, and the patient had returned to work part-time.

**Comment.** This patient had a basically small lumbar spinal canal which was further narrowed by traumatic spondylolisthesis. She described a typical proximal-to-distal sensory march after standing or walking a short distance. The dull aching quality of the discomfort in her buttocks and thighs was more akin to the pain of muscular ischemia than to radicular pain.

**Discussion**

Table 1 shows the major features that permit separation of aorto-iliac arterial obstruction from intermittent claudication of the cauda equina. Cauda equina claudication is subdivided into postural and ischemic types; their cardinal differences are indicated.

Despite the many similarities between vascular and neurogenic claudication, several features afford reliable clues for a differential diagnosis. If a patient develops symptoms while standing quietly, the cause is almost certainly postural claudication caused by changes in the cauda equina. The pain of arterial insufficiency is confined to contract-
ing muscles whereas the discomfort of cauda equina involvement is distributed over the lower lumbar and sacral nerve roots. The discomfort of "postural cauda equina claudication" is relieved merely by a change of posture, whereas the discomfort of either arterial insufficiency or the ischemic cauda equina syndrome is unaffected by body position and persists as long as exercise continues. Examination of femoral and distal pulses both before and immediately after exercise to the point of pain should identify the patient with arterial insufficiency. The appearance or exaggeration of motor and sensory deficits characterizes the patient with cauda equina claudication. When a patient with arterial insufficiency walks to the point of pain, exercised muscles will become stiff. This should not lead to confusion with the weakness and sensory loss induced in the patient with cauda equina involvement.

In the patient with cauda equina involvement, lumbar puncture is often difficult to perform, and may reveal partial or complete spinal block with an increase in the amount of cerebrospinal fluid protein.

Plain x-ray films of the lumbar spine offer limited information. Calcification in the aortic and iliac vessels often occurs as an asymptomatic and incidental finding in the older age group. Degenerative, traumatic, and developmental abnormalities of the lumbar spine may suggest narrowing or encroachment on the spinal canal, but measurements, even with the aid of laminography, provide only supporting evidence rather than proof of cauda equina involvement.14, 17

Aortography and myelography constitute the definitive diagnostic tests. The patient with normal pulses in the legs before and after exercise almost certainly does not have primary arterial insufficiency, and myelography should be selected as the initial definitive diagnostic test. In some instances, such as in the patients reported by Blau and Logue3 and by Verbiest,37 vascular and neurogenic claudication may coexist, and both aortography and myelography must then be performed.

Spanos and Andrew44 reported three cases as examples of intermittent claudication attributable to lateral lumbar disc protrusion.

<table>
<thead>
<tr>
<th>Findings</th>
<th>Arterial Insufficiency</th>
<th>&quot;Cauda Equina Claudication&quot;</th>
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<tbody>
<tr>
<td>Pain induced by location</td>
<td>characteristic muscular contraction</td>
<td>may be absent muscular contraction</td>
</tr>
<tr>
<td>&quot;march&quot; character relief by</td>
<td>usually cramping, dull rest (standing)</td>
<td>varies, often dysesthetic rest (standing)</td>
</tr>
<tr>
<td>Motor Deficit</td>
<td>rare; during walking exercised muscles cramp and become tight</td>
<td>mild, variable; during walking appearance or exaggeration of motor and sensory deficits</td>
</tr>
<tr>
<td>Sensory Deficit</td>
<td>rare</td>
<td>mild, variable</td>
</tr>
<tr>
<td>Lasègue Sign</td>
<td>normal</td>
<td>frequently normal</td>
</tr>
<tr>
<td>Lumbar Puncture</td>
<td>normal</td>
<td>may be difficult, demonstrate block, CSF protein elevated</td>
</tr>
<tr>
<td>Pulse</td>
<td>either femoral or distal pulse decreased; peripheral pulse may decrease during walking</td>
<td>normal; peripheral pulse unaffected during walking</td>
</tr>
<tr>
<td>Arterial Murmur</td>
<td>proximal (aorto-iliac)</td>
<td>absent</td>
</tr>
<tr>
<td>Plain X-Ray Films</td>
<td>arterial calcification</td>
<td>abnormal lumbar spine (osteophytes, short pedicles)</td>
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<tr>
<td>Aortography</td>
<td>diagnostic</td>
<td>normal</td>
</tr>
<tr>
<td>Myelography</td>
<td>normal</td>
<td>diagnostic</td>
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TABLE I
Differential diagnostic findings in intermittent claudication
They proposed that walking and weight-bearing produced pressure on the intervertebral disc and caused gradual herniation of soft material. They also thought that each step was a movement equivalent to straight leg raising to 40°. By our criteria, their cases do not qualify as intermittent claudication but rather represent ruptured lumbar discs.

Thus, there are two forms of intermittent claudication related to the cauda equina. The anatomical requirement for both conditions is a constricted lumbar spinal canal with compression of the cauda equina roots at one or more levels. In the postural form, the appearance or aggravation of signs and symptoms follows as an added increment of compression produced by hyperextension of the lumbar spine. If ischemia has any role in this variety of cauda equina involvement, it is passive or secondary. In the less common ischemic form of cauda equina claudication, occult compression of cauda equina roots also exists.

The appearance of signs and symptoms during exercise of the lower limbs can be explained by the following series of events: 1) the transmission of neural impulses through the cauda equina roots leads to hyperemia and dilatation of radicular vessels, which not only increases the degree of compression on nerve roots and radicular vessels, but also impairs blood flow through radicular arteries at points of constriction; 2) actively conducting nerve roots demand an increased supply of oxygen which puts them in competition with blood flow to actively contracting muscles; and 3) the relative ischemia of conducting cauda equina roots may be additive to occult mechanical impairment of conductive through these roots at one or more sites of compression.

The treatment of cauda equina claudication, whether ischemic or postural, differs in no way from the treatment of spondylotic cauda equina radiculopathy of other varieties. Treatment involves wide laminectomy and partial facetectomy well above and below the stenotic areas delineated by myelography.

**Summary**

Two varieties of claudication related to the cauda equina (cauda equina claudication), postural and ischemic, have been described. In both varieties the cauda equina roots are compressed within a developmentally narrow lumbar spinal canal, and the onset of symptoms can be related either to additional mechanical (postural) narrowing of the canal, or to exercise-induced ischemic radiculopathy. Aorto-iliac arterial insufficiency offers the main differential diagnostic problem. After myelographic demonstration of cauda equina compression, laminectomy can be expected to relieve the symptoms of this type of claudication.

**References**


