

■ Spinal Stenosis and Neurogenic Claudication

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Neurogenic claudication is diagnosed from a classical history and complementary spinal imaging. The abnormal signs may be few. It should be distinguished from intermittent claudication (peripheral vascular disease), referred pain from the back or root pain that is aggravated by walking, and psychological distress.

Pathologically, a developmentally small canal is usually affected by multiple levels of segmental degenerative change, with venous pooling in the cauda equina between two levels of low pressure stenosis. There is probably then a failure of arterial vasodilatation of the congested roots in response to exercise, with symptoms in the legs when walking.

Once established, symptoms tend neither to improve nor deteriorate. Conservative management is reasonable. Otherwise decompression at the most significant stenotic level is probably adequate to obtain a good surgical result. [Key words: claudication, decompression, spinal stenosis] *Spine* 1996;21:2046-2052

Verbiest in 1954¹² was the first to recognize that structural narrowing of the vertebral canal could compress the cauda equina and produce claudication symptoms. In the past four decades, we have developed a better understanding of its clinical presentation and its pathophysiology.

■ Clinical Presentation

History

Symptoms usually affect men aged more than 50 years. They complain of discomfort in the thighs, calves, and feet when walking. Bilateral symptoms occur with a male:female ratio of 8:1, whereas the ratio in unilateral claudication is 3:1.¹⁰ Typically, symptoms are not present at rest, but after walking a short distance patients experience weakness, tiredness, or heaviness of the legs that gradually increases and causes them to stop. The walking tolerance (when the patient stops) is usually twice the threshold distance when they first feel discomfort.

It can vary during the day or from one day to the next or even during one period of walking. When walking, the patient stoops forward, gradually reduces walking speed, and sometimes will stoop forward until he or she finally stops—the stoop test. He or she may lean on a wall or stoop to tie a shoelace, and after a few minutes the legs recover and he or she start to walk again. Some find walking downhill particularly uncomfortable but cycling may be no problem and a useful discriminator between neurogenic and intermittent claudication (peripheral vascular disease). Most patients have a long history of previous back pain.

Examination

Apart from the spinal posture, the examination is remarkable for its lack of gross abnormality. The patient may be able to flex well forward with extended knees, although lumbar extension is usually absent. These patients may have difficulty standing erect, and they adopt a "Simian stance" with hips and knees slightly flexed. If this posture is not present at rest, it tends to develop with walking; the patient gradually stooping forward until he or she stops. The center of gravity gradually moves forward. It returns after each period of rest but takes longer to return to its initial position than it takes for symptoms to settle. The lumbar spine is often tender over several segments. Straight leg raising is generally full, and there are frequently no abnormal neurologic signs. Reexamination after exercise, however, may alter the results of the neurologic examination. The peripheral circulation can be normal, but not infrequently arterial disease will coexist.

Investigation

Assessment on a treadmill will establish an objective record of walking pain, with note of the speed of walking, the distance at which symptoms develop, the distribution of discomfort, the changing posture, and the walking tolerance. The impression from a patient's history can be completely different from an objective assessment of walking. When measuring a patient's response to treatment, a treadmill is invaluable.

A plain radiograph will raise the suspicion of a shallow vertebral canal and perhaps show degenerative spondylolisthesis, which is present in half the men with bilateral claudication.⁷ Half of the patients with unilateral claudication have a structural lumbar scoliosis.

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Acknowledgment date: August 25, 1994.

First revision date: August 20, 1995.

Second revision date: January 2, 1996.

Acceptance date: May 15, 1996.

Device status category: 1.

A computed tomography scan is helpful in recording the cross-sectional area of the central canal and stenosis of the root canal, but because most patients with neurogenic claudication have multiple level pathology, a magnetic resonance imaging scan is complementary. Some clinicians prefer myelography or computed tomography myelography to demonstrate the extent of the stenosis. It usually shows multiple level stenosis with redundant nerve roots.

It is advisable to have Doppler studies of the peripheral circulation and sometimes arteriography.

Differential Diagnosis

1. Intermittent claudication from peripheral vascular disease is difficult to distinguish from neurogenic claudication by the history alone. It is not affected by posture, but this is not a very sensitive discriminator between the two types of claudication. Cerebral somatosensory-evoked potentials after walking may help to differentiate neurogenic from vascular intermittent claudication. It is confusing when spinal stenosis and peripheral vascular disease often coexist.
2. Sciatic claudication is an insufficiency of the inferior gluteal artery, producing ischemia of the sciatic nerve and claudication in a sciatic distribution. It should be considered when there is no evidence of spinal stenosis and the peripheral circulation is good.
3. Referred pain from the lower lumbar region into the buttocks and thighs can mimic neurogenic claudication when the symptoms are aggravated by walking. However, referred pain will be present in activities other than walking, and despite symptoms, walking long distance, although painful, may not be impossible. An unstable isthmic spondylolisthesis may cause referred pain into the thighs when walking, but central canal stenosis is uncommon with pars defects because the vertebra displaces forward, leaving the floating lamina behind and widening the canal.
4. Some types of root pain and multiple root pathology are made worse by walking. In Crock's patients with isolated lumbar disc resorption, 11.18% had increasing leg pain or paresthesia on walking distances up to 500 yards. Walking may aggravate a root entrapment problem, but these patients usually also have symptoms at rest.
5. Claudication pain is sometimes a symptom of distress. Abnormal behavior patterns are common in patients who have a long history of back pain, and pain in the legs when walking is not infrequently a symptom inappropriate to the underlying organic pathology in the spine. These patients usually exhibit inappropriate signs.
6. Litigation can sometimes so confuse the issue that it is not possible to decide how large the organic component is and whether the organic element of the

leg pain is neurogenic claudication, root pathology, or referred pain.

7. There are other less common causes of claudication pain, including venous claudication after thrombosis, myxedema claudication with a limited potential of muscle to increase its metabolism with exercise, multiple sclerosis, and, rarely, deep arteriovenous fistula.

Pathology

1. Spinal stenosis has unfortunately become synonymous with neurogenic claudication, but a shallow canal is only one factor in the pathology. Spinal stenosis is sometimes entirely symptomless, and stenosis is also a factor in other back pain syndromes, including disc protrusion and root entrapment from degenerative change. In addition, symptoms of claudication are unusual before the sixth decade of life, even though the vertebral canal will have been narrow for many years. Even in achondroplasia, with marked developmental stenosis, symptoms are not present in early life. The small canal is, therefore, but one factor in the pathology.
2. Degenerative soft tissue and bony pathology is invariably present in patients with neurogenic claudication. The ligamentum flavum is usually thickened or ossified. Some patients have diffuse idiopathic spinal hyperostosis (DISH) or Paget's disease.
3. Vertebral displacement with an intact neural arch will critically narrow an already small canal. Degenerative spondylolisthesis effectively reduces the canal size at the level of displacement. Although degenerative spondylolisthesis is more common in women, bilateral neurogenic claudication is more common in men, and approximately half the men with bilateral claudication have a degenerative spondylolisthesis.⁷ The rotatory effect of a degenerative lumbar scoliosis can be an important factor in unilateral claudication.
4. The neuropathology is probably the result of inadequate oxygenation or accumulation of metabolites in the cauda equina. Nerve function is probably just adequate at rest but inadequate during exercise. The ischemic effect of compression on nerve function has been studied in animal models.^{3,4} However, it is of interest that ablation of the lumbar arteries after aortic surgery is rarely followed by claudication symptoms.
5. Central stenosis at one level does not account for the symptoms. There are a number of clinical reasons why central stenosis alone does not explain the mechanism of claudication. First, a steadily progressing spinal tumor can completely block the central vertebral canal without producing claudication. Second, a large central disc protrusion can block the canal without claudication. Third, a single level stenosis from degenerative change at L3-L4 or L4-L5 may

almost occlude the dural sac and yet produce only back pain. Furthermore, imaging of asymptomatic subjects confirms that stenosis is common, and patients who present with claudication must have had symptomatic stenotic canals for many years. Again, it is surprising that in canine studies a single level experimental stenosis constricting the cauda equina by 25% did not cause neurologic deficit.³

6. Root canal stenosis does not account for the symptoms. A number of authors have thought that root canal stenosis or foraminal stenosis is responsible for claudication symptoms. However, isolated root canal stenosis may be asymptomatic or on other occasions responsible for the constant root pain of root entrapment but not claudication. If root canal pathology was important, why do patients with neurogenic claudication invariably have central canal stenosis?

7. One of the radiologic features of neurogenic claudication is the high frequency of multiple level stenosis in the central or root canals⁷ (Figure 1). The venous anatomy of the roots of the cauda equina make them vulnerable to congestion at multiple lev-

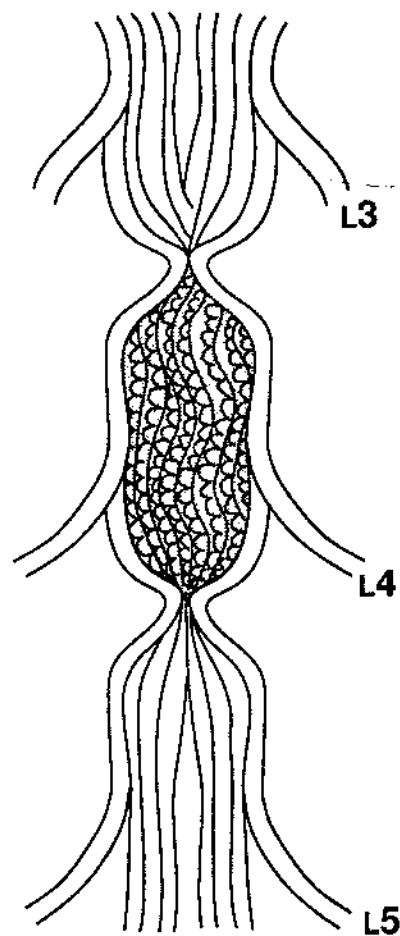


Figure 2. Diagram to show how all the roots of the cauda equina will be congested with venous blood between two levels of low-pressure occlusion of the central canal.

els. The veins of the roots (which do not anastomose between roots) generally drain distally to the foramen or, if this is occluded, proximally to the conus. A single low pressure block will affect only a small section of the root and probably not disturb conduction. However, in the presence of two low pressure blocks, there will be venous congestion in the intervening segment. The arterioles will continue to feed the segment at the higher arterial pressure, but impaired drainage will reduce the blood flow, the oxygen supply, and the nutrition, with a build-up of metabolites in the uncompressed segment between the two blocks (Figure 2).

This hypothesis is compatible with experimental studies. A single level compression of 10 mm Hg in a porcine cauda equina model had little effect on the function, but a two-level compression of 10 mm Hg caused marked reduction of blood flow by 64%,⁹ and there was significant reduction in protein transport and nerve conduction.⁴ A two-level compression below arterial pressure is also supported by the myelography studies, which show congested cauda equina in claudicating patients.⁵



Figure 1. Myelogram of a patient with symptoms of bilateral neurogenic claudication. There is a partial occlusion at L4-L5 and to a lesser degree at L3-L4. The appearance of a "canal full of roots" suggests that the vertebral canal is developmentally small.

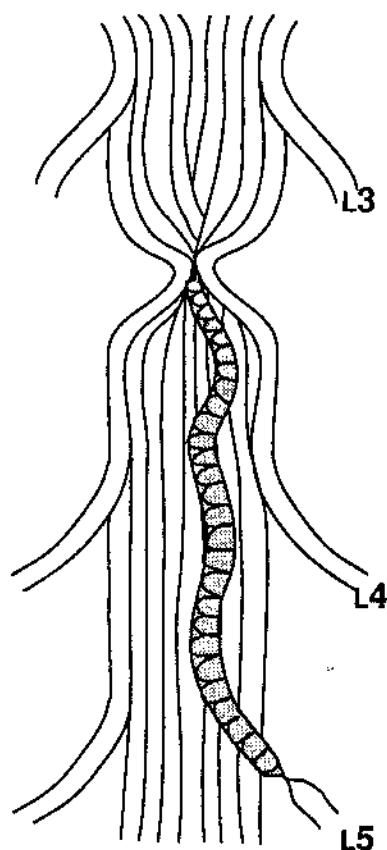


Figure 3. Diagram to show that a single level of central canal stenosis and a more distal level of root canal stenosis will cause congestion in a single nerve root.

A two-level stenosis hypothesis must include at least one level of central canal stenosis, but the second level of stenosis could either be in the central or in the root canal. Two levels of central stenosis will cause venous congestion in all the roots of the cauda equina between the two blocks. However, central stenosis at one level and bilateral root canal stenosis at the lower level will congest only two roots. Proximal central stenosis and distal unilateral root canal stenosis will produce single root congestion (Figure 3). This would explain why the symmetrical displacement in a degenerative spondylolisthesis is usually associated with bilateral and not unilateral claudication. The degenerative process is symmetrical, with central stenosis at one level and bilateral root canal stenosis at a more distal level. With degenerative lumbar scoliosis, however, the asymmetrical degenerative process is more likely to produce root claudication.

8. If venous pooling of the nerve roots of the cauda equina between two levels of low pressure stenosis is responsible for the symptoms of neurogenic claudication, one has to ask why are symptoms usually not present at rest but only when walking. One might argue that the block pressure at each level of stenosis will increase with the dynamic activity of walking.

There will be local vasodilatation of the radicular arteries in response to exercise. Exercising the single limb of a mouse will produce vasodilatation in the ipsilateral region of the spinal cord. Blood flow in the nerve root is also increased with peripheral nerve stimulation. One might expect, therefore, that the arteries of the cauda equina will dilate with exercise, and if space is already at a premium, the stenosis block pressure will rise to a critical level.

Other features associated with walking will tend to increase the block pressure. Movement in the sagittal plane alters the epidural pressure at the site of stenosis, being above normal pressure even in flexion (15–18 mm Hg) and greatly above venous pressure in extension (80–100 mm Hg).⁸ Segmental rotation associated with walking might also significantly affect the root canal where the degenerate capsule of the facet joint limits available space for the nerve root complex. In addition, the increased venous return from the exercising lower limbs will be accompanied by engorgement of the pelvic veins and Batson's venous plexus, reducing the available space for the cauda equina. Extra dural venous engorgement will then contribute to the block pressure.

There may be some patients with stenosis pressures at rest below the venous pressure, which rise above the venous pressure with exercise. However, one would expect that there will be some patients with multiple level stenosis who will have block pressures at rest above the venous pressure but not have leg symptoms at rest.

9. We have reported how there is considerable arterial vasodilatation of the porcine cauda equina to electrical stimulation and that this vasodilatation response is impaired in the presence of a two-level low pressure compression.¹ We have observed that in the response to stimulation and in the absence of any compression, there is an arterial vasodilatation producing 200% increase in the blood flow of the cauda equina, and this is maintained over 30 minutes (Figure 4, 5). However, in the presence of a double level low pressure compression, the increase in blood flow after stimulation is less pronounced and is maintained only for a few minutes (Figures 6, 7). The blood flow then rapidly falls to approximately 60% below the resting level. There is then a failure of nerve conduction.

If this is analogous to the chronic situation of multiple level spinal stenosis, it may explain the symptoms of neurogenic claudication.

A failed arterial response is compatible with the observation that patients with neurogenic claudication tend to be in the arteriosclerotic age group and that peripheral vascular disease and neurogenic claudication often coexist. Arteries that are already abnormal may be less labile, especially in the presence of venous pooling.

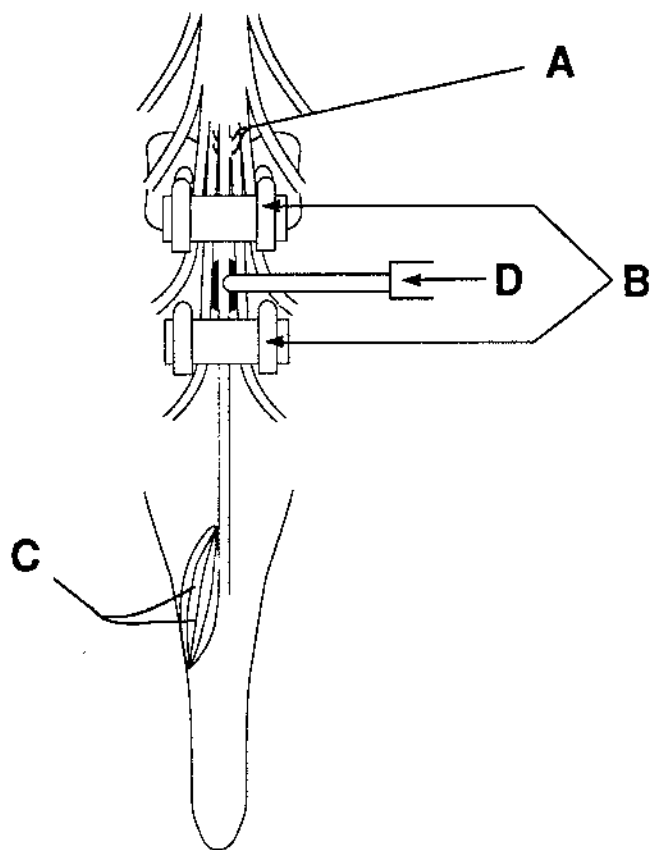


Figure 4. Diagram to show how the exposed porcine cauda equina is stimulated (A) proximal to a two-level low pressure occlusion (B), monitoring the tail muscle electromyographic activity (C) and the blood flow (D) in the cauda equina between the two levels of occlusion.

■ Management

Counseling

Patients with neurogenic claudication are either offered surgical decompression or are advised to live with their symptoms. If the disability is not too severe or if surgery is contraindicated, simple reduction of activities or alteration of lifestyle together with Back School instruc-

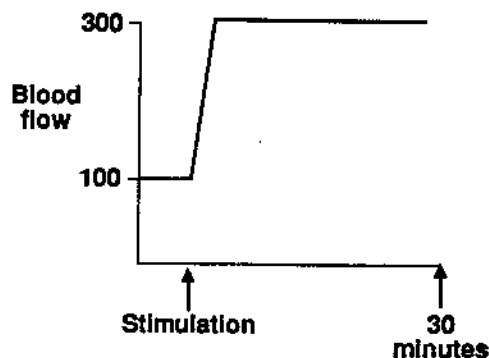


Figure 5. Without occlusion, the cauda equina blood flow increased by 200–300% when stimulated electrically. High flux and tail muscle electromyographic activity was maintained for 30 minutes.

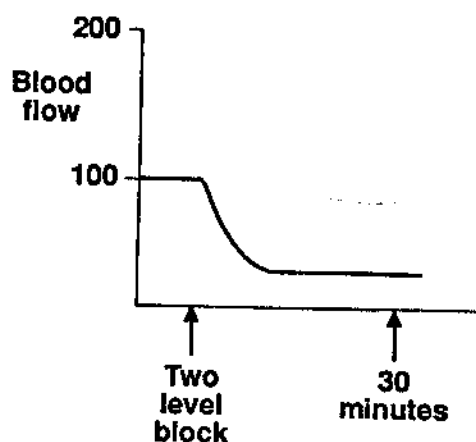


Figure 6. In the presence of a two-level compression of 10 mm Hg, blood flow fell to 35–40% resting level.

tions may enable the patients to live within their limitations. Once the syndrome is well established, however, conservative management rarely improves the quality of life.

Calcitonin

There is circumstantial evidence that calcitonin is beneficial for approximately 40% of patients with neurogenic claudication. This drug can relieve the paraparesis of patients with spinal Paget's disease.

Many patients with Paget's disease and spinal stenosis, besides losing the Paget's pain, also find that with calcitonin their walking improves suddenly and dramatically. However, stenotic patients without Paget's disease can also dramatically improve their walking distance with a short course of calcitonin.⁶ Calcitonin engenders a sense of well-being, and undoubtedly some patients experience a placebo response. Some become almost euphoric at their ability to walk unlimited distances again. The mechanism of a response is uncertain,

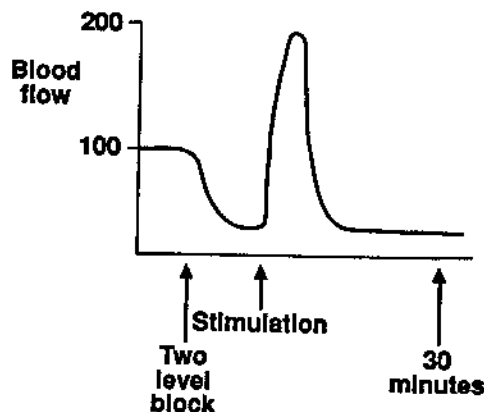


Figure 7. When a two-level compression was applied blood flow fell. The proximal cauda equina was stimulated with an increase in flux to 200% above resting level. This was only a short-lived response, with flux falling to below resting level, and with a failure of electromyographic activity in the tail muscles.

and a controlled randomized trial has not confirmed that the response is organic.

However, it is a useful first line of treatment, and responders avoid a surgical decompression.

Surgery

When recommending surgery, the clinician must adopt a different philosophy from that used for many other orthopedic problems. The patient with an arthritic hip, for example, is likely to have increasing disability, but neurogenic claudication is either present or absent. Once established, it tends not to progress, and when surgery is offered, it is for current disability and not to avoid future problems. No proof of severe deterioration is found in untreated patients, and observation for 2–3 years is an acceptable alternative to surgery. However, troublesome claudication symptoms seriously affecting lifestyle are generally relieved by surgical decompression. Most patients are immediately impressed with the improved sensation in their legs and are soon walking long distances. There may be some relapse after a laminectomy, when a membrane of fibrous tissue develops over the posterior dura, and the walking distance again becomes reduced. Some suggest that the results deteriorate with time, but others report good long-term results. Advanced age is no contraindication to the decompression, which will often improve the patient's quality of life. However, most operative series have a hard core of failures, and there are few patient characteristics that will predict outcome.¹⁰

The surgical decompression must be adequate. There are no clear guidelines to identify the significant stenotic levels. The clinical impression at the time of surgery is probably the best guide, although somatosensory-evoked potentials may have a place in determining the extent of necessary decompression. There is no longer a requirement to remove all the lamina. Retaining part of the lamina to maintain stability and reduce dead space is compatible with an adequate decompression.

If there is degenerative spondylolisthesis, it is essential not to unnecessarily increase the instability of that segment. Postoperative displacement is unusual even with wide decompression, provided there is already marked degenerative change, but one should be cautious if degeneration is minimal. The integrity of the apophysial joints should not be unduly disturbed, although the medial third of the joint can be removed and the facet undercut. It is necessary to form a decompression wide enough to ensure a completely free dura but not so wide as to produce either instability or such a shallow spinal gutter that a laminectomy membrane will soon compress the dura to a ribbon. Provided that there is not a degenerative spondylolisthesis, it is legitimate to sacrifice the major part of the apophysial joint on one side to obtain satisfactory decompression and not jeopardize stability. Decompression should be accompanied by a posterolateral spinal fusion when in a degenerative

spondylolisthesis there is minimal degenerative change and usually when stenosis complicates structural lumbar scoliosis.

A double level hypothesis has surgical implications. If the symptoms result from a two-level stenosis, it is necessary to decompress only one of the levels to relieve the claudication. However, it is inadequate to decompress only the central canal of the most stenotic segment if root canal stenosis at this level is responsible for the distal stenosis, and a significant proximal central canal stenosis is still left untreated.

In summary, the symptoms of neurogenic claudication are probably associated with developmental spinal stenosis and secondary multiple level degenerative change. At low pressures, this will produce venous pooling of one or several roots of the cauda equina. In the arteriosclerotic age group, the arterioles of the nerve root fail to maintain a vasodilatation response to exercise when there is venous engorgement. This failed arterial response is associated with failure of nerve conduction producing tiredness, weakness, heaviness, and discomfort in the lower limbs when walking. As the patients stop, nerve function temporarily recovers to permit a further period of walking.

This hypothesis explains why patients with neurogenic claudication tend to reach a plateau of disability and then not deteriorate further. They can always walk a short distance. They rarely develop serious paraparesis or "go off their feet." For this reason, although surgery will often dramatically relieve the symptoms, a conservative approach is reasonable.

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