Coexistent vascular and spinal claudication: a report of two cases

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Two cases of patients with evidence of both vascular and spinal intermittent claudication are presented. When patients present with low back pain and post-exercise lower extremity pain, confusion may arise with respect to the etiology of the symptoms. These cases stress the need for a rational and logical approach to the patient with intermittent claudication.

KEY WORDS: Vascular claudication, neurogenic claudication, manipulation, chiropractic.

Nous donnons ici deux cas de patients qui présentent à la fois une évidence de claudication intermittente vasculaire et vertébrale. Lorsque des patients présentent une douleur au bas du dos ainsi qu'une douleur des membres inférieurs après exercices, il peut y avoir confusion en ce qui touche l'étiologie des symptômes. Ces cas font ressortir le besoin d'une approche logique et rationelle du patient souffrant de claudication intermittente.

MOTS CLÉS: claudication vasculaire, claudication neurogénique, manipulation, chiropratique.

Introduction

"Intermittent claudication" refers to the cramping pain experienced in limbs, that is brought on by exercise and relieved by rest. This term was defined in 1858 by Charcot who ascribed the symptoms to an impairment of arterial circulation. Thus, intermittent defines the exercise – pain – rest – relief cycle and claudication defines the concomitant limping.[1] The two prerequisites for the presence of vascular intermittent claudication are the lack of oxygen and the combined muscular contraction. The resultant post-exercise pain experienced by the patient, reflects the immobility of the peripheral vascular system to satisfy the increased metabolic demands of the muscles.

However, post-exercise lower extremity pain may also be the result of neurospinal disease. In 1911, De Jerine described the symptoms of intermittent claudication of non vascular etiology.[2] Therefore, in the patient presenting with post-exercise lower extremity pain and low back pain, confusion may arise as to the etiology of the symptoms. This confusion arises from the concept that neurospinal disease could mimic the symptoms of vascular insufficiency. However, it was not until 1950, that the descriptive phase "intermittent spinal claudication" was used by Bergmark.[3] This was followed by Verbiest in 1954, who reported on the clinical symptoms of neurogenic claudication resulting from encroachment of the spinal canal by hypertrophic articular processes (spinal stenosis)[4].

More recently, Kirkaldy-Willis outlined the pathological factors contributing to low back pain including the major structures and locations of potential nerve entrapment.[5,6]

Arthrosis and subluxation of the apophyseal joints, often in conjunction with degenerative changes of the intervertebral disc, have been proposed as possible mechanisms leading to entrapment of the spinal nerve root in the narrowed lateral recess. Moir and Cassidy expanded on the pathological, clinical and manipulative considerations of lateral nerve root entrapment and the differentiation of dynamic lateral entrapment from fixed lateral entrapment.[7] This paper reviews the clinical signs and symptoms of vascular and neurogenic claudication and emphasizes the importance of physical examination in arriving at the diagnosis. Two case reports are presented that illustrate the coexistence of both types of claudication.

Case reports

Case 1:

Mrs. M.I. is a 50-year-old caucasian female who was referred to the CMCC low back pain clinic with complaints of low back and bilateral leg pain.

The low back pain had been noticed by the patient for the past ten years. It was located centrally in the lumbosacral region with no radiations into the lower extremities. The pain was described as a dull continuing ache which was worse in the morning and was eased by bending forward. Mrs. M.I. reported having to "crawl up her legs" when returning from a forward flexed position.

The leg pain had been noticed during the past two years. The patient reported stabbing and burning pain in the distal lower extremities. The leg pain was brought on by walking a few blocks and relieved by rest, at which time only a dull pain was noticed. She also complained of burning pain at night which was "dulled" by getting up from bed and walking about for several minutes. There was no significant history of trauma. However, Mrs. M.I. had undergone iliac artery bypass surgery for arterial insufficiency nine months prior to presentation.

On examination, the range of motion of the lumbar spine was painfully restricted in extension, right lateral flexion, and rotation to the left. There was tenderness to palpation and decreased joint mobility at the L4-5 vertebral level, as well as at the right sacroiliac joint with associated bilateral tightness of the erector spinae muscles. Straight leg raising was limited to 80 degrees on the right by low back pain.

Neurological examination showed a mild subjective sensory deficit to light touch over the right lateral calf. Ankle jerk and

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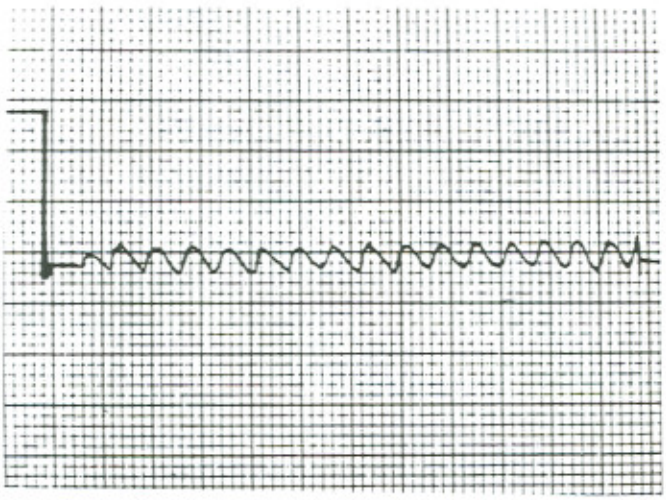


Figure 1A Plethysmograph reading of the right leg at rest, showing a stenosal pulse. Note the diminished amplitude with a slow upstroke.

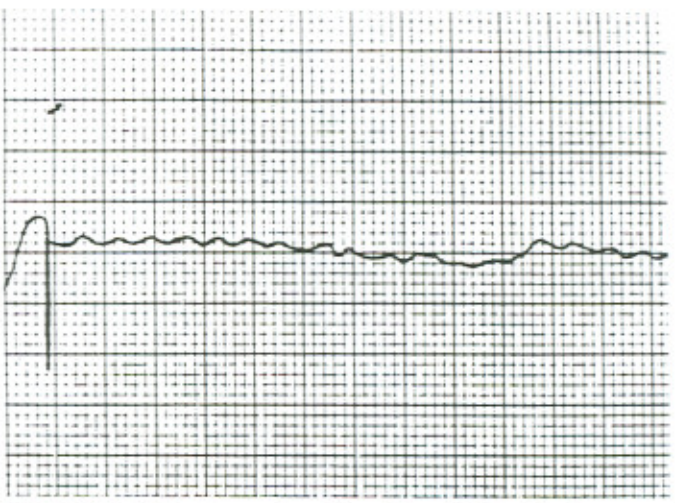


Figure 1B Plethysmograph reading of the right leg after the onset of symptoms following treadmill walking. Note the further decrease in pulse amplitude.

medial hamstring deep tendon reflexes were 1+ bilaterally. Muscle testing of the lower limbs showed tight hamstrings, quadriceps, and adductors. The abdominal muscles were found to be weak.

There was moderate swelling of the lower extremities with dryness of the overlying skin bilaterally. Both feet were cold to touch, and elevation of the legs resulted in immediate pallor of the skin which was followed by rubor on dependency.

Vascular analysis of the extremities showed an ankle to arm systolic blood pressure ratio of 0.6 (40% reduction) bilaterally. The arterial wave form from a plethysmograph appeared flatter (i.e. diminished amplitude) than normal, suggesting an increased peripheral resistance within the lower extremities (Figures 1A, 1B).

X-ray examination of the lumbar spine and pelvis revealed evidence of previous vascular surgery noted by surgical clips within the right inguinal and abdominal regions, as well as mild calcification within the aortic bifurcation (Figure 2).

A clinical diagnosis of L5 nerve root entrapment, complicated by intermittent vascular claudication of the lower extremities was made.

Mrs. M.I. underwent a five week treatment regimen consist-

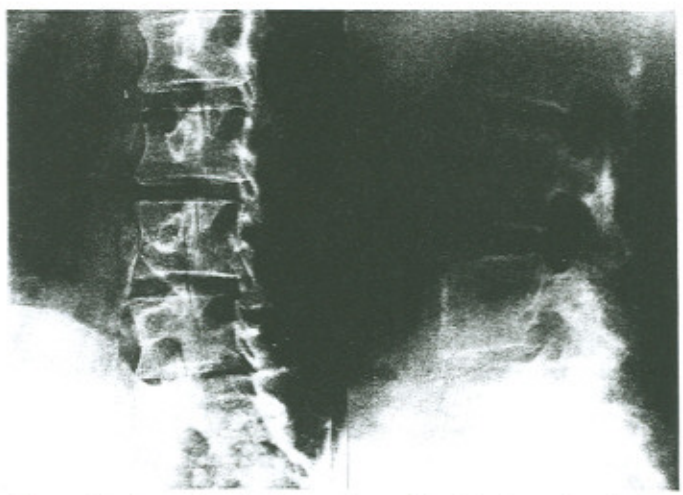


Figure 2A Lateral and oblique views of the lumbar spine showing mild degenerative changes at L5-S1, and arteriosclerotic disease of the abdominal aorta. A surgical clip is seen anterior to the L2-3 intervertebral space.

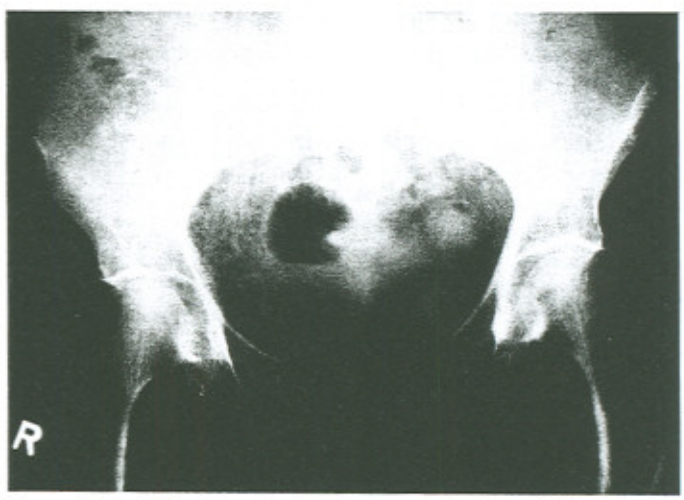


Figure 2B AP pelvis showing the surgical clips within the right inguinal and abdominal regions, as well as arteriosclerotic changes within the iliac arteries.

ing of spinal manipulative therapy directed to the right sacroiliac joint and the T11-12 and L4-5 posterior joints, soft tissue massage, resisted stretching and home exercises. After which she reported that her low back pain had considerably improved but that the leg pain had not.

Mrs. M.I. was subsequently referred for further medical evaluation of her intermittent vascular claudication. Further assessment was carried out and treatment was instituted but the patient continued to experience her leg pain.

Case 2:

Mr. S.K. is a 63-year-old male with a five year history of recurrent low back pain and bilateral posterior thigh and calf pain which developed after a lifting injury. At the time of onset he went to see a chiropractor and manipulations minimally improved his low back pain. On presentation he complained of sharp lower lumbar pain with occasional bilateral posterior thigh and calf pain. He described also a sharp burning pain and numbness of the toes and plantar aspects of both feet. Lifting and bending, as well as prolonged sitting, standing or lying in one position aggravated his pain and numbness. He was not able to walk more than three city blocks without aggravating his back pain or producing numbness in the feet. The patient was in fairly good general health, but had been a borderline diabetic taking medication for the last year and a half.

On examination the range of motion of his lumbar spine showed flexion to about the mid-tibia with markedly decreased extension by pain. There was tenderness at the L4-5 and L5-S1 posterior joints, especially on the left. Straight leg raising was to 80 degrees bilaterally with tight hamstrings. There were no nerve root tension signs. Muscle strength was graded 5/5 in the lower extremities. The patient had a subjective sensory decrease to light touch within the L4 distribution on the right side. The toes, dorsum of foot and plantar aspect of the forefoot were numb bilaterally, especially on the right foot. No deep tendon reflexes could be elicited in his lower extremities. The plantar responses were downgoing.

Vascular examination of the lower limb revealed clinically diminished dorsalis pedis and posterior tibial pulses bilaterally. Both feet showed trophic changes at the toes, that included dryness and scaliness of the skin, hair loss and nail changes. Rubor was evident bilaterally in the dependent position. Blood pressure of the lower extremities had an ankle to arm systolic blood pressure ratio of 0.25 (75% reduction) bilaterally. Plethysmographic analysis was suggestive of diminished arterial flow bilaterally (Figures 3A, 3B). X-ray examination of the lumbar spine demonstrated marked degenerative changes at the levels of L4-5 and L5-S1 (Figure 4).

A clinical diagnosis of spinal stenosis complicated by peripheral vascular disease was made and the patient was referred for further investigations. Subsequent studies included a myelogram and a CT-Scan, which were reported to show evidence of degenerative spinal stenosis at L3-4, L4-5, and at L5-S1. Angiography was performed and demonstrated blockage of the superficial femoral artery bilaterally.

From these investigations it became evident that Mr. S.K. had indeed two physical problems that may account for the lower extremity symptoms of intermittent claudication. It was decided that the spinal stenosis was the more significant problem and the patient underwent a surgical decompression procedure. At two months follow up he reported to experience occasional mild low back pain with slight numbness over the S1 dermatome on the right. His walking distance had improved considerably and he did not notice any symptoms of intermittent claudication.

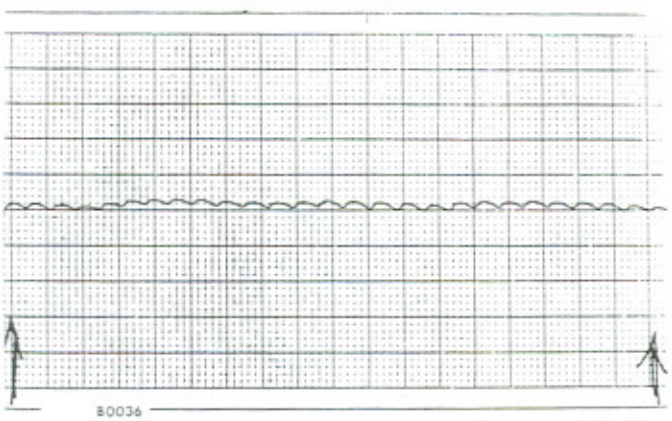


Figure 3A Plethysmograph reading at rest showing a stenosal pulse.

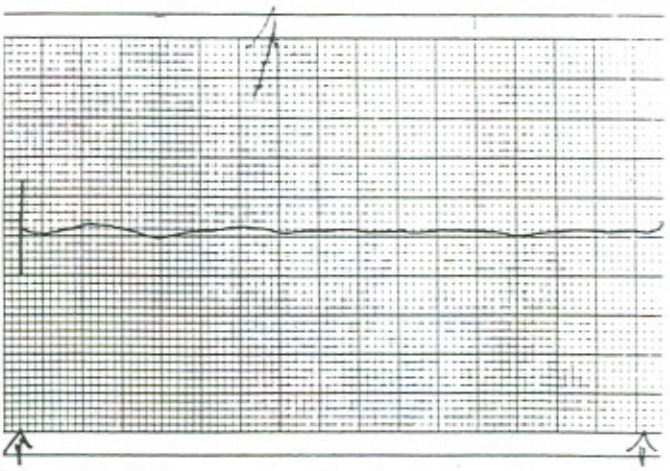
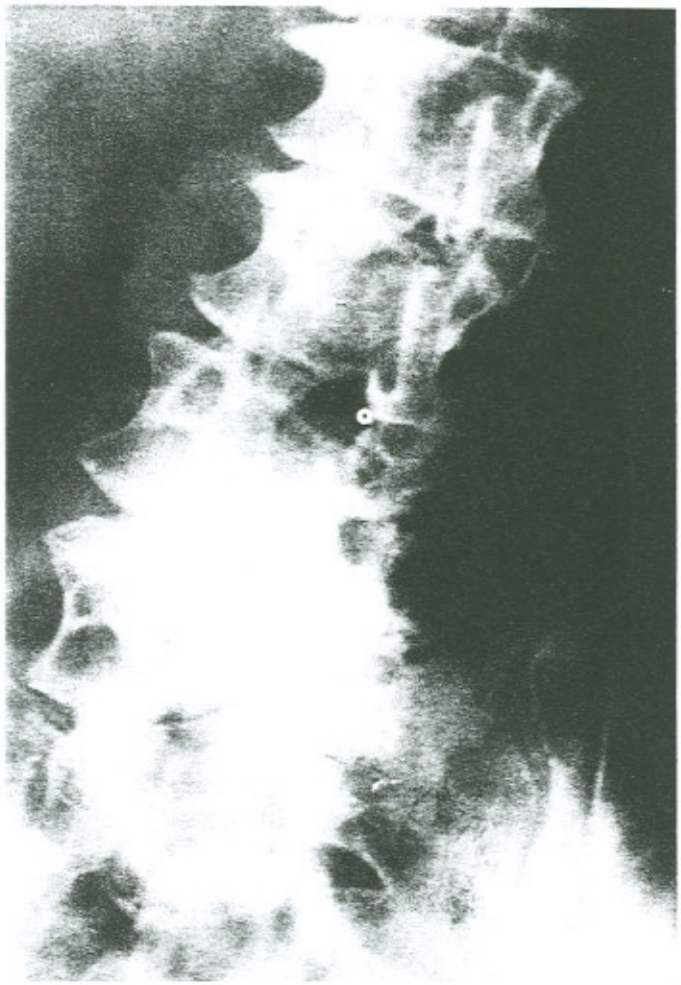


Figure 3B Plethysmograph reading after the onset of symptoms following treadmill walking.



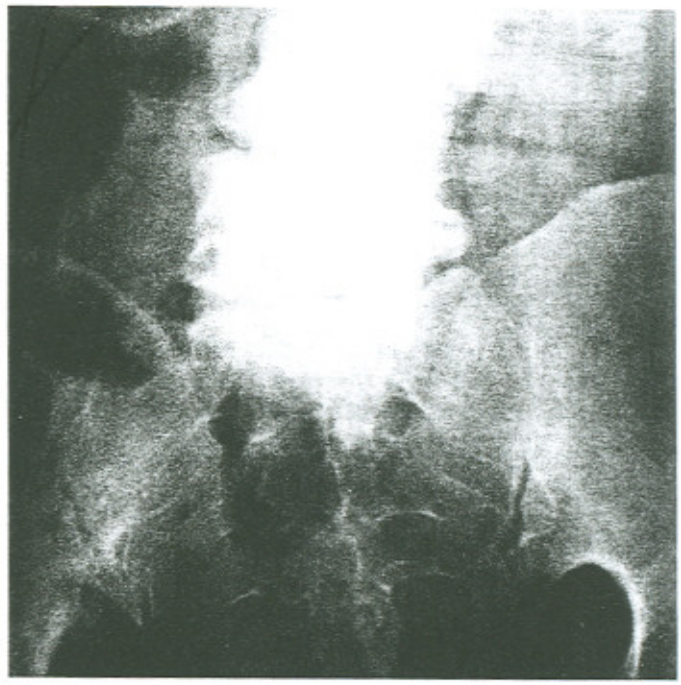


Figure 4 AP and oblique lumbar radiographs showing advanced degenerative changes at L4-5 and L5-S1.

Discussion

Intermittent vascular claudication may result from a variety of occlusive arterial diseases that are severe enough to provide an inadequate amount of blood to contracting muscles. These diseases include arteriosclerosis obliterans, thromboangiitis obliterans, thromboembolic diseases, various types of arteritis and congenital coarctation of the aorta. The most common of these causes encountered in clinical practice is arteriosclerosis obliterans.

The resultant symptoms encountered are related to the slow, insiduous development of tissue ischemia due to a deficient blood supply in the exercising muscle. The distress is described as a pain, ache, cramp, tingling paresthesia, or tired feeling that occurs on walking. Warren defined three strict criteria for the diagnosis of vascular intermittent claudication:

1 The pain must come on as a muscle cramp in thigh or calf after the patient has walked a predictable distance.
2 It must be relieved by rest in the standing position after a predictable period of time.
3 The patient must then be able to walk a similar distance again, and again obtain relief after resting the same period of time as before.[8]

Therefore, exertional leg pain not satisfying all of these criteria must be suspected to be of nonvascular origin. Sitting is not necessary to obtain relief, and in some cases the distress may be worsened by walking rapidly or uphill.

The location of the experienced leg pain and the level of arterial occlusion are closely correlated.[9] For example, aortoiliac disease frequently causes claudication in the buttocks, hips and the claves and absence of the femoral pulses. In femoropopliteal disease, the claudication is characteristically in the calf, and all of the pulses below the femoral artery are absent.

The progression of the disease is indicated by a decrease of the distance the patient can walk. The occlusive process may progress so that ischemic pain occurs at rest and is aggravated by elevation, often preventing sleep. To obtain relief, the patient will hang his foot over the side of the bed, walk about, or rest in a chair.

If the history is compatible with vascular claudication, a thorough examination of the vascular system should be performed. This includes palpation of the abdominal aorta, femoral, popliteal, posterior tibial, and dorsalis pedis pulses. Auscultation should also be done over these areas to elicit any bruits. Examination of the feet and legs should include observation of

skin color, consistency, warmth, hair growth, and capillary filling time. An advanced ischemic foot is painful, cold, and often numb. The skin may be dry and scaly with poor nail and hair growths. Physical examination will also reveal pallor of the involved foot and leg after 1 to 2 minutes of elevation, followed by rubor on dependency. Venous filling time following elevation is delayed beyond the normal limit of 15 seconds. As ischemia worsens, ulceration on the toes, heels, or occasionally of the legs may appear, especially after local trauma.

In addition to observable changes, a variety of noninvasive instruments are available to evaluate arterial insufficiency. The simplest method for estimating blood flow to the lower extremities is to measure the systolic blood pressure at the level of the ankle and compare it to the brachial systolic pressure (ankle-to-arm index). A systolic pressure in the ankle less than the systolic pressure in the arm suggests peripheral arterial disease. Therefore, the normal index is 1, while an index of 0.6 or less has been determined to be diagnostic of vascular insufficiency, and is significant enough to cause intermittent claudication (Table 1)[10].

Table 1 Comparison of ankle and arm systolic blood pressure measurements (modified after Greenfield[10]).

ANKLE TO ARM INDEX	= *Ankle systolic Bp* / Arm systolic Bp
	= 1.0 (normal)
	= 0.6 (claudication)

This index may be supplemented by measuring the postexercise ankle pressure. The normal physiologic response to moderate exercise is a slight increase in ankle pressure. The ankle pressure in patients with vascular claudication falls after a period of exercise.[10] Systolic blood pressure measurements taken at the thigh and upper calf levels give additional information about the extent and location of the occlusive disease and collateral blood flow.[9]

Complementing the pressure readings, is the analysis of the pulse volume waveforms with a plethysmograph.* The plethysmograph is a photoelectric pulse volume recording instrument that measures and graphs blood volume performance through any pulsatile artery and through the capillary beds of the fingers and toes. The use of photoelectric plethysmography provides substantial improvement in diagnostic precision, as well as a technique for objective recording of either functional or organic arterial disease.[11,12,13]

On the contrary, the nonvascular causes of exertional leg pain provide a diagnostic challenge. The etiologies of spinal or neurogenic intermittent claudication include a variety of disorders and can encompass spinal stenosis, protruding or herniated intervertebral disc, cauda equina syndrome, degenerative spondyloarthrotic changes, developmental narrowing of the spinal canal and spinal cord tumors. Lateral spinal nerve root entrapment is a relatively common lesion that can be seen either late in the unstable phase (phase II) or early in the stabilization phase (phase III) of the spinal degenerative process.[20] The pathogenesis of entrapment is due to degenerative changes affecting the posterior joints and the intervertebral disc, leading to a compromise of the lateral recess and eventually resulting in an entrapment of the spinal nerve at the site. Dynamic lateral entrapment occurs when segmental instability is superimposed on the hypertrophic changes, whereas fixed lateral entrapment is due to fibrosis and stabilization of the disc and posterior joints, creating a fixed or permanent narrowing of the lateral recess. The dynamic form is reported to respond more readily to manipulation than the more advanced or fixed variety which may necessitate surgical decompression.[7]

Lateral nerve root entrapment is characterized by a history of intermittent attacks of pain over a time span of several years. The most common site of pain is in the lumbosacral area, the buttocks, trochanteric region, and posterior thigh to knee – sometimes extending distally to the calf and the foot. Motion palpation may reveal restricted movement in the lower lumbar posterior joints and the sacroiliac joints. The neurological examination is characteristic of a single nerve root involvement and may be accompanied by signs of nerve root tension.

Routine lumbar and stress radiographs may also provide

Table 2 Comparison of neurogenic and vascular claudication.

NEUROGENIC	CLAUDICATION VS	VASCULAR
Usually male, age > 50 years		Age > 50 years
History of low back pain		No history of low back pain
Dull aching pain		Cramplike pain
Variable pain pattern post exercise		Consistent pain pattern post exercise
Aggravated by prolonged sitting or standing		Not affected by prolonged sitting or standing
Pain on back hyperextension		Not affected by back hyperextension
Relieved by bending forward, supine position, or unusual maneuvres and positioning		Relieved by standing post exercise
Ease of pain on walking uphill or riding a bicycle		Increase of pain by any exercise
Neurological deficit		Usually no neurological deficit
Possible weakness or loss of bowel and bladder function		No weakness or loss of bowel and bladder function
Normal peripheral vascular status		Abnormal peripheral vascular status

* The VDI vascular analyzer is a multi-function neurovascular instrument containing a digital photoelectric plethysmogram recording device. A doppler velocity detector and an electronic skin thermometer distributed by vascular instrumentation CO.3798 N.E.

information for the diagnosis of lateral nerve root entrapment. This includes diminished intervertebral disc height, end plate sclerosis, and other degenerative changes, as well as increased intersegmental movement.[7,20] Table 2 lists some of the more classical signs and symptoms that help to differentiate the two kinds of claudication.[1,10,14,15,16,17,18,19,21]

Conclusion

Based on the experience gained from these case reports, the need for a rational and logical approach to the patient with intermittent claudication, either vascular or neurogenic in origin, is apparent. A complete history and physical examination will generally reveal whether the problem has a vascular, a nonvascular or a combined etiology. Where a patient's symptom complex is atypical and some physical signs are equivocal, non-invasive arterial studies will aid considerably in the differential diagnosis.

However, patients with coexistent spinal and arterial disease present a diagnostic challenge. As case one demonstrates even surgery for arterial occlusive disease may not always resolve the vascular problem, and may in fact mislead the clinician to ascribe all the presenting signs and symptoms to a coexistent spinal disorder. Therefore, the clinician must keep in mind that documenting the presence of apparent spinal disease does not exclude the possibility of coexistent arterial occlusive disease, or vice versa. Both may in fact manifest symptoms simultaneously.

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