Neurogenic Positional Pedal Neuritis Common Pedal Manifestations of Spinal Stenosis

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Neurogenic positional pedal neuritis is a presentation of neuritic symptoms in one or both feet usually affected by body position, specifically, the position of the spine. Its etiology is similar to that of neurogenic-induced claudication caused by spinal stenosis in that the symptoms are caused by compression or irritation of nerves of the lower lumbosacral spine, usually the fifth lumbar and first sacral nerve roots. Burning, stabbing, a cold feeling, aching, numbness, paresthesia, or a weak or tired feeling of the feet (during some part of the disease process) depend on spinal position and may occur during standing, walking, or even lying in bed. Symptoms may be severe and are often eliminated by lumbosacral spine flexion, such as by walking with wheeled support such as a grocery cart or walker; less frequently by negative-heel shoe modification, which can change the position of the lumbosacral spine in stance; or by alteration of sleeping position. This condition, which can include loss of protective sensation, is often misdiagnosed as neuropathy (especially in diabetic patients) or less frequently as biomechanical in origin. In diabetic patients, this condition is frequently the cause of failure of monochromatic infrared energy therapy for diabetic peripheral neuropathy. Treatment is aimed at reducing the spinal nerve or nerve root irritation. Clear definition of the pedal symptoms of spinal nerve compression within a single diagnostic category should facilitate identification and treatment. (J Am Podiatr Med Assoc 93(3): 174-184, 2003)

Lumbar spinal stenosis is a condition involving pressure on the central spinal cord, a spinal nerve within the spinal canal, or the nerve root exiting the spinal canal that can cause a variety of symptoms in the lower extremities. It has long been recognized, and has been recently documented, that body position (stance) and spinal position (extension) affect the pressure. Flexion of the spine causes an increase in the diameter of both the central canal and intervertebral foramen, and extension has been shown to decrease the diameter.¹ Axial loading, as is seen with weightbearing, can decrease the diameter of the central canal and cause narrowing of the lateral recess.²

Prevalence is not well established, but spinal stenosis has been reported to occur in 1.7% to 8% of the general population.³ The symptoms of spinal stenosis typically develop in the fifth or sixth decade of life

*South Florida Foot Center, 2900 N Military Trail, Ste 230, Boca Raton, FL 33431. and vary greatly depending on many factors, including the exact location and manner of compression and the severity of pressure on the nerve. Patients may have symptoms in the extremities in the absence of significant back pain. Common symptoms, often affected by the position of the lumbosacral spine, include numbness, paresthesia, pain, burning, and muscle weakness and aching, and they can affect part or all of the foot or leg, depending on the factors described previously.³

The common and near pathognomonic symptom of spinal stenosis is neurogenic-induced claudication. In this condition, walking or even standing results in leg aching or cramping that is similar to that of vascular-induced claudication. The difference is that to obtain relief the patient with neurogenic-induced claudication must sit, lie down, or at least flex the lumbosacral spine,³ whereas with vascular-induced claudication the patient can stand erect and still have relief of the leg symptoms. Patients with spinal stenosis may walk better when pushing a wheeled support such as a walker or a grocery cart.⁴ These two types of claudication are compared in Table 1.⁵

Clinicians should be aware that the nerve root distribution is different regarding the dermatomal and myotomal patterns. Dermatomal innervation involves primarily the second and third lumbar nerves and the second sacral nerve (L2, L3, and S2) for symptoms in the thighs and the fourth and fifth lumbar nerves and the second sacral nerve (L4, L5, and S2) for symptoms in the legs. Myotomal innervation for the many muscle groups of the lower extremities involves the L2 through S2 nerves. Muscles in the anterior thigh are primarily innervated by the L2 through L4 nerve roots, and those in the posterior thigh are primarily innervated by the L5 through S2 nerve roots. Muscles of the anterior and posterior leg are innervated by the L4 through S2 nerve roots. Intrinsic muscles of the foot are innervated primarily by the S1 through S3 nerve roots.⁶ However, innervation is anatomically inconsistent, resulting in inconsistent location of the perception of symptoms with compression, and the previous descriptions are only guidelines.

Spinal stenosis and related conditions can, in the same manner, cause symptoms in the feet if compression involves nerve fibers innervating the foot, primarily the L5 and S1 nerve roots. Patients may feel symptoms—including burning, aching, paresthesia, or a feeling of weakness—immediately on standing, after several minutes of standing, or after walking either short or long distances, depending on the amount of compression present and the sensitivity of the nerve. As with neurogenic-induced claudication, neurogenic positional pedal neuritis symptoms are often reduced or eliminated by changing the position (flexion) of the lumbosacral spine. These patients usually can walk much more comfortably (and in many cases indefinitely) when pushing a grocery cart (if they are taller than approximately 5 feet 2 inches to 5 feet 5 inches and thus need to flex) or on a treadmill if they walk while holding the handles, thus flexing the lumbosacral spine. Numbness may be severe, with an absence of protective sensation that may involve the course of one spinal nerve or the entire foot, ankle, or leg, mimicking peripheral neuropathy. Presentations that warrant consideration of the diagnosis of neurogenic positional pedal neuritis are summarized in Table 2.

Diagnosis

The diagnosis of neurogenic positional pedal neuritis is usually made by recognition of symptoms consistent with the diagnosis combined with magnetic resonance imaging (MRI) or computed tomography (CT) showing nerve compression in the area corresponding to extremity symptoms. An article explaining many of the conditions that could result in spinal nerve compression has been previously published.⁵ An MR image with contrast is ordered if the patient has had previous surgery in the involved area, to identify potential scarring. A CT scan is ordered for patients with a pacemaker or other metallic device precluding MRI. Elimination of all pedal symptoms with an epidural injection is believed to confirm the diagnosis. Partial improvement of symptoms with epidural injection may be seen with a concomitant lowerextremity condition (including diabetic neuropathy) that was perceived as more painful in the presence of stenosis, indicating a synergistic or hyperalgesic condition. Failure to improve with an accurate epidural injection does not negate the diagnosis, as some pa-

Evaluation	Vascular-Induced Claudication	Neurogenic-Induced Claudication		
Claudication distance	Consistent	Often inconsistent		
Pain relief	Standing and resting	Sitting, lying down, or flexion of the lumbosacral spine ^a		
Walking up slope	Pain	May have less pain ^a		
Walking down slope	Pain	May have more pain ^a		
Riding a bicycle	Pain	May have no pain ^a		
Walking on a treadmill	Pain	May have no pain (if leaning on bar or handles) ^a		
Pushing a cart or walker	Pain	May have no pain (if height is appropriate) ^a		
Circulation	Severe deficit	Irrelevant		
Back pain	Irrelevant	Frequent, not always		
Night pain	Severe cases	Frequent, and often affected by sleep position ^a		

	Table 1, Com	parison of Vascular-I	Induced and Neuroo	genic-Induced	Claudicatior
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Source: Modified with permission from Goldman et al.5

^a Symptoms of neurogenic-induced claudication may be reduced with activities associated with flexion of the spine.

 Table 2. Presentations Suggesting Neurogenic Positional

 Pedal Neuritis as a Cause of Symptoms

- Neuritic foot symptoms while standing or walking that are relieved by sitting or flexion of the low back.^a
- Improved standing or walking ability with use of a grocery cart, wheeled walker, or treadmill.^a
- Neuritic symptoms at night relieved by sleeping in a recliner (or in a similar flexion position) or by sitting on the edge of the bed with legs hanging over the side.^a
- 4. Asymmetrical presentation of discomfort of peripheral neuropathy symptoms of the foot.
- Loss of protective sensation that is severe (above the ankle), asymmetrical (medial *versus* lateral), or atypical (greater proximally than distally) in one or both feet.
- Foot (and leg) neuritic symptoms may be sensitive to heel height, being worse when wearing high-heeled or standard shoes than in flat shoes or barefoot. Less frequently, patients may be more comfortable in highheeled shoes.
- 7. In patients who are more comfortable in flat shoes or barefoot, symptoms may be relieved by wearing a negative-heel shoe—this may be tested by adding ¹/₄ to ³/₄ inch of felt to the distal two-thirds of the sole of the shoe.^a
- 8. Hyperalgesic perception of pain coming from foot pathology such as hyperkeratosis, arthritis, nail pathology, and neuropathy.
- 9. Absence of another identifiable cause of neuritic symptoms in the foot.

^aSymptoms of neurogenic positional pedal neuritis may be reduced by flexion of the lumbosacral spine.

tients have such a severe pathologic condition that only surgical decompression may relieve symptoms.

The diagnosis is also supported by identifying the listed symptoms, with elimination of the painful symptom by behavioral changes such as sleeping in a recliner, walking with a walker, or walking with a negative-heel shoe causing lumbosacral flexion (often used in patients unable to undergo epidural injections and who chose not to undergo MRI or CT).

The MRI report does not always exactly correspond to the expected level of compression because of anatomical inconsistency and variations in myotomal and dermatomal patterns that can result in symptoms being present in nonclassic patterns. It has been documented that eccentric compression can cause contralateral and ipsilateral symptoms.⁷ Proximal stenosis can affect and therefore cause symptoms involving distal nerves.⁸ As a result, it is strongly recommended that anatomical findings and symptoms be discussed in detail with an expert before ruling out spinal compression as the etiology if the clinical picture is strongly suggestive of it.

Clinical examination of the foot and leg in patients with spinal stenosis may or may not reveal a presentation consistent with peripheral neuropathy. Patients may have normal, diminished, or absent sensation to light touch, temperature, or vibratory stimulus³ or have symptoms that follow the course of one or many nerve roots.^{3, 9} A recent report¹⁰ indicated that 81% of patients with spinal stenosis studied had absent or decreased neurosensory responses (pinprick, vibration, or tendon reflexes).

Many patients with spinal stenosis cared for in the author's practice have had normal tactile sensitivity identified with Semmes-Weinstein monofilaments. Other patients have diminished or absent sensation to 5.07 or 6.45 Semmes-Weinstein monofilament that may involve a single dermatomal area or, in the case of multiple nerve or nerve root involvement, present as a stocking distribution, as would be seen with diabetic peripheral neuropathy. Patients with spinal stenosis are more likely to have severe absence of protective sensation in the entire foot and at times the ankle and leg, even in cases of recent onset, than patients with diabetic peripheral neuropathy. Asymmetry of loss of protective sensation (medial versus lateral, right versus left) or an atypical pattern (with greater sensory loss at proximal tested sites than distally) that would not be consistent with a distal symmetrical polyneuropathy has frequently been observed. However, these patterns also have been seen, although much less frequently, in patients with diabetic peripheral neuropathy. The author uses a loss of protective sensation (LOPS) scale to clarify the presentation of hypoesthesia in patients (Fig. 1).

Some patients with stenosis have a significant discrepancy between the amount of numbness reported (on a linear scale from 0 [normal] to 10 [severe]) and the actual loss of protective sensation measured with Semmes-Weinstein monofilaments, claiming significant loss of sensation when none is documented by Semmes-Weinstein monofilament testing. In addition, some patients have demonstrated a hyperpathic condition involving hyperalgesia (perception of a painful stimulus as more painful than it would usually be perceived as), with elimination of this severe pain either by treating the foot condition (such as trimming a corn or injecting a painful joint) or by successful epidural injection.

Patients with spinal stenosis are reported to have increased sensitivity on palpation of the sciatic nerve in the area of the sciatic notch, and they may have more pain on increased extension (lordosis) of the lumbosacral spine.³ The author observes that patients with spinal stenosis are likely to have extremity nerve sensitivity to palpation, not a pattern observed frequently with peripheral neuropathy alone. Just as tenderness on palpation of one or more intermetatarsal



Figure 1. Loss of protective sensation scale record.

spaces can be seen with Morton's neuroma and the proximal nerve compression syndrome of tarsal tunnel syndrome, so is it that tenderness on palpation of the intermetatarsal nerves, posterior tibial nerve, tibial nerve (medial approach, midcalf), or femoral nerve (medial approach, midthigh), which could be caused by a local nerve irritation or compression, can also be found in the proximal nerve compression syndrome of spinal stenosis. Also, patients with an asymmetrical presentation of pain are more likely to have symptoms caused by nerve compression rather than by neuropathy, although mononeuritis is a recognized pattern in diabetic peripheral neuropathy.⁹ Observations of the extent or symmetry of loss of protective sensation, the symmetry of neuritic symptoms, an augmented perception of pain from other pathologic findings, and patterns of nerve hypersensitivity to pressure are strongly suggestive but not necessarily diagnostic of nerve compression such as spinal stenosis rather than neuropathy being the cause of symptoms.

Identification of the symptoms of either neurogenic-induced claudication or neurogenic positional pedal neuritis has been facilitated by the development of a questionnaire for patients with neuropathy that includes the Grocery Cart Test (Fig. 2) and the symptoms described in Tables 1 and 2. Patients were asked to relate their symptoms to activity level or body position. A linear pain scale from 0 (no symptoms) to 10 (severe symptoms) was used to subjectively quantify numbness, paresthesia, and pain. Patients obtaining this questionnaire before their initial visit were asked to perform the Grocery Cart Test, and all patients with limitation of walking distance were asked to do so after the initial visit.

The Grocery Cart Test was developed as a screening tool for spinal stenosis. Patients are directed to test their walking distance in a controlled environment, ie, a grocery store. After resting for 5 min in the store to establish a baseline, the patients walk without stopping and note the duration and aisle number in which they notice increased symptoms in the feet or legs. They also note if and when the symptoms are severe enough to make them feel that they must stop walking or sit. The following day, the patients repeat the test while pushing a grocery cart.

The author observed that patients with vascular or nonspinal orthopedic pathologic conditions received inconsistent help from pushing a grocery cart, with most increasing walking time by a few minutes or not at all. Patients with spinal stenosis usually increased walking time and distance dramatically (>75%) and

Peripheral Neuropathy versus Spinal Stenosis Questionnaire Name	
To what do you attribute your neuropathy?	
How long have you had this condition? Symptoms of neuropathy?	
1. How far can you walk before your feet or legs tire? Less than 1 block 1-2 blocks 3-5 blocks 1/2-1 mile Over 1 mile Stopped by symptoms in: Feet Legs Thighs Back Other	
2. To get relief of foot or leg ache/tiredness when you walk do you usually need to: Stand and rest Lean against something Sit Lie down Other	
3. Do you usually find it much easier to walk or stand using a: Grocery cart? Yes No Treadmill? Yes No	
 4. How long do you usually stand before feeling greater symptoms? Not affected by standing Less than 5 minutes Less than 15 minutes Over 15 minutes 5. Are you most comfortable standing: Barefoot Very flat shoes Standard shoes High-heeled shoes No difference 	
6. If you have limitations standing or walking, how long has that been a problem?	
7. OVERALL how would you describe these symptoms in your FEET?	
Numbress $10 \ 9 \ 8 \ 7 \ 6 \ 5 \ 4 \ 3 \ 2 \ 1 \ 0$	
Severe Moderate Mild None	
$\operatorname{Paresthesia}$ (tingling, pins & needles) 10 9 8 7 6 5 4 3 2 1 0	
Severe Moderate Mild None	
Discomfort (burning, pain) 10 9 8 7 6 5 4 3 2 1 0 Severe Moderate Mild None	
 Are symptoms at (circle all that apply): Top of foot Bottom of foot Front of leg Back of leg 9. Do you have significant symptoms of neuropathy in your hands? Severe Moderate Mild None 10. Are symptoms often worse at night in bed? Yes No Are they relieved by sleeping in a recliner? Y By sleeping with a pillow under your knees? Yes No Other 	es No
The Grocery Cart Test Very important, please do this if	
Please do this test if you have limitations in walking because of your feet or legs. Choose a store with numbered aisles and a place to sit in the front. You will need to do this on two separate d each day you will follow an identical protocol, with one major difference. On the first day you will wa no grocery cart, and on the second test day you will use a cart. If you do not receive improvement with ing a cart and you are less than 5 feet 5 inches tall, we will provide you with a wheeled walker a for your height so that you may again test yourself.	grocery ays. On lk with <i>h push-</i> <i>djusted</i>
Enter the store. Rest on a chair for 5 minutes. Starting at the side of the store where the numbers begin Record the time at which you start. Walk until you feel your legs begin to tire, and note where you are aisle) and what the time duration was. Continue walking until you feel you must stop or sit to get rel record the aisle and amount of time since you began. On the second test day, do the identical walk bu pushing a grocery cart.	ı, walk. (which lef, and lt while
For consistency, do not stop at all , even to look at any items or to pick them up, even when using a	cart.
Day 1 (no cart) Day 2 (with cart) Day 3 (with our (if under a	valker) 5′ 5″)
11me (# of minutes) until tired	
Time until you feel you must stop or sit	
Row at which you feel you must stop or sit	



in many cases could walk without any limitation. Patients shorter than approximately 5 feet 2 inches to 5 feet 5 inches with spinal stenosis did not benefit as consistently from using the grocery cart, as they did not necessarily have to bend over to push it. For patients shorter than 5 feet 5 inches who do not benefit from the grocery cart, or for those who did not do the Grocery Cart Test, an adjustable wheeled walker is provided to allow patients to test themselves. The author could not locate a three-wheeled walker (standard minimum height 34 inches) with handles low enough to facilitate lumbosacral flexion for patients shorter than approximately 5 feet 2 inches, so one was cut down 4 inches to accommodate shorter patients.

The author had several patients who initially reported that they could walk much better while pushing a grocery cart but who after doing the test as described reported little improvement, no improvement, or worsening while pushing a cart. Those with arterial-induced claudication seemed to have a false impression of walking better with a cart because the normal pattern of grocery shopping involves periods of stopping. Pushing the cart without stopping, as the test directs, did not give the legs a chance to rest and recover, so there was no significant increase in walking time or distance. A few patients with arteriosclerosis obliterans could not walk as far in this manner, probably because of the increased strain of pushing the cart. It seemed clear from these test results that these patients would not get worthwhile improvement by using the wheeled walker.

The Grocery Cart Test became a successful guide to convince appropriate patients to accept the use of a walker as part of their lifestyle. Identification of neurogenic-induced claudication or neurogenic positional pedal neuritis with relief of symptoms by using a grocery cart enabled many patients to recognize the effect of spinal mechanics on their ability to be active. Many patients who had previously been severely limited in walking distance then became willing to use a wheeled walker, thus increasing their exercise and freedom of movement. Many could then also walk for exercise using a treadmill, especially if an elevation was used that induced leaning forward on the rail, flexing the spine.

Choice of the walker is important. The traditional walker, with two small fixed wheels, is often harder to use on uneven ground. It requires lifting to allow the patient to turn, and thus may require the patient to extend, negating the goal of lumbosacral flexion. This may also exacerbate local symptoms in patients with lumbosacral pathology. The author orders a three- or four-wheeled walker, with large wheels that are freely moveable in the front, allowing the patient to turn without lifting. Patients able to walk a few blocks with this type of wheeled support are usually prescribed a three-wheeled walker. Those whose walking duration is improved but still significantly limited are prescribed a four-wheeled walker with a dropdown seat that allows them to sit wherever necessary.

The author had four patients with diabetes, loss of protective sensation, and neither neurogenic-induced claudication nor neurogenic positional pedal neuritis who did not respond to monochromatic infrared therapy and were subsequently identified by MRI as having moderate or severe spinal stenosis. Although the presence of either neurogenic-induced claudication or neurogenic positional pedal neuritis is strongly suggestive of spinal stenosis, their absence does not preclude spinal stenosis as a diagnosis. In addition, as the constriction causing spinal stenosis can worsen with time, noting a past history of symptoms affected by spinal position should also be a stimulus to consider spinal stenosis as a cause of symptoms.

Shoe heel height has been documented as having an effect on spine position,^{11, 12} which the author postulates could affect nerve compression. On questioning, many patients with neurogenic positional pedal neuritis or neurogenic-induced claudication report being very sensitive to heel height, often feeling more comfortable barefoot or in flat shoes than in highheeled or even walking shoes, blaming the shoes for being too tight, too heavy, or poorly fitted. (Less frequently, patients with spinal stenosis noted less discomfort in high-heeled shoes.) The author experimented with eliminating the heel height (or creating a negative heel) by adding 1/4 to 3/4 inch of adhesive felt to the distal two-thirds of a walking shoe or sneaker (the Felt Test) and found that some patients noted immediate improvement in the ability to tolerate standard shoes, stand, or walk (Fig. 3). Patients are warned that the felt can make the shoes slippery and to exercise caution. The negative heel, which in some patients results in a conscious backward tilt of the legs and pelvis, seems to induce flexion of the lumbosacral spine in order for the patient not to lean or fall backward. (The change in stance can be experienced by standing on a board or magazine.) Patients noting clinical relief with this temporary modification over a few days, including reduced burning or pain or improved ability to stand or walk, had a neutral or 1/8- to ³/₈-inch negative-heel modification done by a pedorthist (Fig. 4). The midfoot elevation above the height of the heel ends with a thruster sole modification at the level of the ball of the foot to facilitate rectus gait. Experimenting with different felt thicknesses during the test and close follow-up after shoe modification are recommended. Stretching exercises should be



Figure 3. Felt applied to test for shoe modification.

done first in patients with significant equinus. This condition may account for the preference of some patients for negative-heel earth shoes to standard shoes. About 20% of patients tested in this manner noted significant improvement in standing or walking capability. Further investigation is necessary.

Recognition of the effect of sleeping position on symptoms can suggest spinal involvement. Some patients with neurogenic positional pedal neuritis note exacerbation of symptoms in bed at night. This can confuse the diagnosis because it is reported that peripheral neuropathy symptoms are often worse at night. However, the author finds that with neurogenic positional pedal neuritis, it is the sleeping position that causes the symptoms, not the time of day, and these patients may note significant (up to 100%) relief of nocturnal symptoms by sleeping in a recliner or with a pillow under the knees, causing flexion of the lumbosacral spine. In addition, it is reported that paraspinal venous congestion in the presence of spinal stenosis can cause neuritic symptoms in the feet and legs (Vesper's curse) while sleeping, and this can be relieved by changes in sleep position, such as sleeping in a more vertical position.¹³ For many years,



Figure 4. Professional shoe modification.

obtaining relief from foot or leg pain at night by sitting up in bed and dangling the feet was believed to suggest arterial insufficiency. According to these observations, it may be caused by spinal stenosis.

Neurologic testing may be inconclusive in these patients. Changes seen with diabetic peripheral neuropathy⁹ and lumbosacral radiculopathy³ are not always clear and may be seen in patients without symptoms. Many patients seen in the author's practice had diabetic peripheral neuropathy and spinal stenosis and have nerve testing changes suggestive of one or both problems. The author has also seen many patients with neurogenic positional pedal neuritis misdiagnosed as having peripheral neuropathy despite a lack of nerve conduction velocity or electromyographic changes supporting that diagnosis. The author believes that a combination of approaches including a thorough history and physical examination, therapeutic approaches as described previously, and confirmatory spinal images is usually adequate for recognition of this condition. In unclear cases, neurology or physiatry consultation, including nerve testing, may clarify the cause of symptoms.

Differential Diagnosis

Some of the signs and symptoms of neurogenic positional pedal neuritis are similar to those observed in peripheral neuropathy such as diabetic peripheral neuropathy, and the aching feeling can be mistaken for biomechanically induced discomfort. Burning or aching pain of the foot without an apparent mechanical or vascular etiology is often recognized to be neurologic in origin. Patients with such pain, even without significant evidence on nerve testing, are often classified as having neuropathy.

Neurogenic positional pedal neuritis is common and is frequently misdiagnosed, especially in diabetic

patients, who the author believes are frequently labeled as having neuropathy without full evaluation for other possible disorders. It is also documented that many more diabetic patients have neurologic changes on nerve conduction studies indicative of neuropathy than have clinical symptoms of neuropathy.⁹ The author has cared for many patients previously diagnosed firmly as having either hypoesthetic or painful diabetic neuropathy that improved dramatically after identification and treatment of spinal stenosis. Some patients had elimination of all neuropathic symptoms, suggesting that diabetic peripheral neuropathy was not clinically present, and many had both neurogenic positional pedal neuritis and diabetic peripheral neuropathy. The author has also cared for many patients misdiagnosed as having painful idiopathic peripheral neuropathy who had classic neurogenic positional pedal neuritis that responded to the approaches described. The author, therefore, believes that patients with painful peripheral neuropathy, with or without diabetes, should be evaluated for neurogenic positional pedal neuritis using the simple techniques described here.

The author has also had several patients who believed the problem was mechanical and who were seeking yet another pair of orthoses. Some patients reported that all shoes were uncomfortable or too heavy, despite the good-quality shoes and orthoses that they had used in the past. Some were diabetic patients who found that none of the shoes or inserts that they had received through the Diabetic Shoe Bill were as comfortable as their flat, inexpensive shoes. The Felt Test and subsequent neutral- or negativeheel modification has allowed several patients to wear the good-quality shoes they could not have otherwise worn. The author, therefore, believes that this pattern-of lack of tolerance for apparently appropriate shoes or inserts-should also stimulate evaluation for neurogenic positional pedal neuritis.

Differential diagnosis also includes common podiatric conditions such as tarsal tunnel syndrome, Morton's neuroma, plantar fasciitis, arthritis, metatarsalgia, tendinitis, and many other conditions that could cause foot pain. Appropriate evaluation for this condition must also include expert evaluation for these and other causes of foot pain.

The presence of other pathologic findings should not eliminate consideration of neurogenic positional pedal neuritis caused by spinal stenosis. Most of the author's patients with neurogenic positional pedal neuritis have had mechanical or systemic conditions that led to previous misdiagnosis. Patients with pain out of proportion to the physical presentation, those who do not respond as expected to treatment, and those with an unknown cause of foot pain should be considered for a diagnosis of neurogenic positional pedal neuritis.

Monochromatic infrared therapy¹⁴ has recently been reported to be helpful in restoring sensation in patients with diabetic peripheral neuropathy. The suggested mechanism of therapy is the induced release of nitric oxide from red blood cells, resulting in increased local circulation, tissue perfusion, and nutrition of the nerve, improving the function of the nerve. The author has used this therapy for 16 months in diabetic patients with classic distal symmetrical polyneuropathy with a high frequency of success in improving tactile sensitivity and reducing the pain and paresthesia associated with this form of neuropathy. This therapy has not proved at all helpful in patients with spinal stenosis or neurogenic positional pedal neuritis in the absence of diabetic peripheral neuropathy, although it has proved helpful in many patients with both diabetic peripheral neuropathy and neurogenic positional pedal neuritis. Failure of monochromatic infrared therapy to provide relief of symptoms of diabetic peripheral neuropathy should be stimulus to investigate the possibility of other diagnoses, with the most likely cause being neurogenic positional pedal neuritis, in the author's experience. Further research and documentation on this subject is needed.

Clinicians should be aware that competence at reading spinal CT scans and MR images is not uniform. A recent study¹⁵ concluded that the inconsistency of evaluation of CT scans by spinal surgeons was enough to render it an unreliable test. Thus a positive CT report can confirm the physical findings consistent with the diagnosis, but a negative report does not negate the diagnosis. The author has had several patients whose symptoms were classic but who had negative findings reported on either CT or MRI. Review of the images was done by a musculoskeletal radiologist specializing in spine imaging, who then identified moderate or even severe deformity that had been missed by a general radiologist. If the referring physician is confident of the possibility of neurogenic positional pedal neuritis being the cause of symptoms, it is recommended that an expert either read the images or review tests read as negative.

Treatment

Standard treatment for spinal stenosis and related conditions is usually managed by a physiatrist, a pain specialist, an orthopedist, or a spine surgeon. Overall treatment, the details of which are beyond the scope of this article, can include use of medications such as tricyclic antidepressants or gabapentin for neuritic pain, physical therapy to reduce inflammation in the back or to strengthen the abdominal muscles, bracing, epidural injections, and surgery. The author's ancillary office recommendations also include use of a walker, use of a modified shoe, and modification of sleeping position.

Epidural injection is an art that is inconsistently practiced. A study¹⁶ reported in 1991 found that approximately 35% of all central epidurals placed without fluoroscopic guidance did not reach the designated area. In addition, constriction caused by lateral and not central stenosis, as is often the case, may require injections directed at the foramen, which is best done under fluoroscopy. In the opinion of the author, referral to a specialist experienced in this technique and with access to the necessary equipment will result in a greater likelihood of successful injection.

Case Studies

Case 1

A 75-year-old man presented with chronic aching in his right foot, diagnosed as plantar fasciitis, that had bothered him for many years. Bilateral pes planus was present, and he had had several pairs of custommade and over-the-counter orthotic devices over the years, all of which were uncomfortable and provided no help. A vague ache and tired feeling when standing or walking was present in the right foot with all shoes, but no discomfort was present when standing or walking barefoot. Podiatric examination showed an absence of pain on palpation or range of motion of the structures of the foot. There was a bilaterally symmetrical moderate pes planus, with no metatarsus adductus or equinus. A low-Dye strap provided minimal improvement. At a subsequent visit, recognizing the possibility of neurogenic positional pedal neuritis, the author added 1/4 inch of felt to the front of his shoe to eliminate the effect of the heel. This eliminated all pain when standing and walking. A previously ordered MR image revealed arthritis and a bulging disk combining to cause right-sided L5-S1 foraminal stenosis. The patient began wearing only flat boat shoes, and all foot pain was eliminated. He was referred to a physiatrist for overall management.

Case 2

A 67-year-old man was referred by his endocrinologist for treatment of painful feet diagnosed as diabetic peripheral neuropathy. He had a 5-year history of diabetes mellitus controlled with insulin therapy. Pain was present constantly but was much worse when standing or walking. He could walk only 1 to 2 blocks before sitting to get relief of foot and leg pain. Lying down worsened the foot symptoms. He could walk much better in a grocery store while pushing a cart.

Physical examination showed palpable pulses, warm feet, and normal hair growth. The patient had significant pain on palpation of the posterior tibial, tibial, and femoral nerves. Semmes-Weinstein monofilament testing showed a loss of protective sensation score of 8 bilaterally, with an inability to feel the 6.45 Semmes-Weinstein monofilament in the toes, ball of the foot, and distal arch bilaterally; an inability to feel the 5.07 Semmes-Weinstein monofilament in the proximal arch and malleoli; and the ability to feel the 5.07 Semmes-Weinstein monofilament above the ankle. The initial diagnosis included spinal stenosis and diabetic peripheral neuropathy. Magnetic resonance imaging showed L5-S1 bilateral foraminal stenosis and L4-5 central canal and foraminal stenosis. A medical condition precluded the use of epidural injections.

The patient was treated with 12 monochromatic infrared therapy sessions, and he reported elimination of 80% of his pain. He had good restoration of protective sensation (loss of protective sensation score of 2 bilaterally) and was able to feel the 6.45 Semmes-Weinstein monofilament in the toes and ball of the foot and the 5.07 Semmes-Weinstein monofilament throughout the remainder of the foot. Persistent symptoms included a tired and achy feeling in his feet and legs when standing or walking longer than 5 min. He could not wear his diabetic shoes because they felt too heavy. A 1/4-inch lift added to the sole of his diabetic shoes did not help; a ¹/₂-inch lift added to the sole of his shoes resulted in elimination of the heavy, tired feeling of his feet and reduced his neurogenic-induced claudication. Professional shoe modification to create a ¹/₄-inch negative heel was done to the previously uncomfortable diabetic shoes, which made them guite comfortable. Two months later, the patient still had no foot pain and had improved his walking distance from 1 to 2 blocks to 5 to 10 blocks.

Case 3

An 80-year-old man presented with a chief complaint of severe pain on the bottom of his feet of 5 years' duration. Non-insulin-dependent diabetes had been present for 10 years. Mild pain was constantly present, with either standing or walking making it severe within a few minutes. The patient had severe limitation of activity because of the foot pain. He reported

being able to walk much better while pushing a grocery cart. He had obtained a monochromatic infrared therapy home unit (Anodyne Therapy System, Medassist, Tampa, Florida) and had used it daily on each foot for 2 months with no improvement. He reported moderate numbness of his feet, but Semmes-Weinstein monofilament testing revealed a loss of protective sensation score of 0 bilaterally, with sensitivity to the 5.07 Semmes-Weinstein monofilament throughout all tested areas in the foot. The initial diagnosis was neurogenic positional pedal neuritis caused by spinal stenosis. A negative-heel shoe modification was tried in the office. An MRI scan demonstrated L4-5 central canal stenosis and L4-5 and L5-S1 foraminal stenosis. The patient returned to the office and reported that the negative heel eliminated about 75% of the burning pain, now involving only the ball of the foot. He performed the Grocery Cart Test (in other shoes) and increased his walking time from 5 min to more than 30 min by pushing a cart. He was advised to use a walker to allow increased activity, to have his shoes professionally modified, and to see a physiatrist for further management.

Case 4

A 72-year-old woman was referred by her internist for evaluation. She had been diagnosed years before as having peripheral neuropathy despite negative nuclear venographic or electromyographic testing. Administration of amitriptyline and gabapentin had been attempted without improvement. Pain was localized to the feet, dorsally more than plantarly, and was constantly present at a low level, but it was much worse when standing or walking for just 1 to 2 min. She could walk much better while pushing a grocery cart. Symptoms were worse in bed at night but were much better when she slept in a recliner. Semmes-Weinstein monofilament testing revealed a loss of protective sensation score of 4, with an inability to feel the 6.45 Semmes-Weinstein monofilament in the toes or ball of the foot but sensitivity to the 5.07 Semmes-Weinstein monofilament in the proximal and distal arch. Pain was present on palpation of the femoral, tibial, and posterior tibial nerves bilaterally and in all intermetatarsal spaces. The initial diagnosis was neurogenic positional pedal neuritis. With the felt shoe modification, she could stand and walk in the office for several minutes with only mild exacerbation of pain. An MRI scan showed L4-5 and L5-S1 stenosis. By using a four-wheeled walker with a seat and the modified shoes, she had moderate improvement in symptoms and the ability to walk. She was referred to a spine surgeon who performed three

transforaminal injections under fluoroscopy, which she reported as relieving 80% of residual symptoms. She then had minimal discomfort and could walk extensively with the modified shoe and walker without significant pain. Recurrence developed slowly over 2 months. Spinal decompression surgery was then performed; 6 months after surgery she had good relief of all foot pain and limitation of walking.

Case 5

A 67-year-old woman presented for evaluation of peripheral neuropathy, which she had been diagnosed as having for many years despite negative findings on nuclear venographic and electromyographic testing. She could stand or walk for only 5 to 10 min before burning in her feet and aching in her legs forced her to sit. She also felt a burning pain in her feet at night in bed. She had a history of back pain, with laminectomy performed three times between 1967 and 1984. Her medical history was otherwise noncontributory. Semmes-Weinstein monofilament testing showed a loss of protective sensation score of 2 bilaterally, with loss of sensitivity to the 5.07 Semmes-Weinstein monofilament in the toes and balls of the feet bilaterally and sensitivity to the Semmes-Weinstein monofilament proximally. An MRI scan showed moderate L5-S1 foraminal stenosis. She was given a ³/₈-inch felt addition to her sneakers, which resulted in dramatic improvement of symptoms. Professional modification of walking shoes with a ¹/₄-inch negative heel with a thruster sole eliminated almost all symptoms. She could stand and walk for 4 to 6 hours each day before a mild, diffuse burning sensation developed in her feet. The burning sensation at night also resolved. She has refused any other treatment. Eight months after shoe modification she reported occasional burning with extensive walking only.

Case 6

A 78-year-old man presented for treatment of constant neuropathic pain of the dorsal and plantar aspects of both feet, with severe claudication symptoms in the legs with limited walking. He had been diagnosed as having diabetes 2 years earlier, and he had a history of two back surgeries that reduced his back pain but did not relieve his extremity symptoms, which had worsened over the previous 2 years. IA-5 and L5-S1 foraminal stenosis was still present. Epidural injections provided excellent but temporary relief of his foot and leg pain. He had a history of walking much better when pushing a grocery cart, but this was no longer the case, as extremity pain

and congestive heart failure limited his activity level severely. He reported numbress as being moderately severe (8 of 10) and pain as severe (10 of 10). Semmes-Weinstein monofilament testing revealed a loss of protective sensation score of 1A on the right and 6A on the left, with asymmetrical loss of sensation, and greater sensory loss proximally in the foot than distally was noted bilaterally. The initial diagnosis was neurogenic positional pedal neuritis with a possible contribution of diabetic peripheral neuropathy to symptoms. Six monochromatic infrared therapy sessions provided no relief of symptoms or improvement of protective sensation of the left foot. A 3/8-inch negative-heel modification provided moderate relief of burning pain, but within 2 days symptoms returned to their initial level, and subsequent attempts at shoe modification did not help. The author loaned him a walker, but he reported that this improved his standing and walking capability by only a few minutes. He was prescribed a four-wheeled walker with a dropdown seat, but he instead chose to get a motorized wheelchair. He was referred for the epidural injections that had previously provided excellent but temporary help, understanding that this could destabilize blood glucose management.

Conclusion

The newly defined syndrome of neurogenic positional pedal neuritis is a common cause of symptoms in the feet, including burning, numbress, paresthesia, hyperalgesia, an achy or weak feeling, and other neuritic symptoms. It is often misdiagnosed as diabetic peripheral neuropathy, idiopathic peripheral neuropathy, or a biomechanical pathologic disorder. The cause of neurogenic positional pedal neuritis is compression or irritation of nerves of the lower lumbosacral spine, primarily the L5 and S1 nerve roots, and it may accompany neurogenic-induced claudication caused by pressure on other lumbosacral nerves. A key to recognition is the appreciation of the effect of spinal mechanics and body position on the symptoms, which is facilitated by the questionnaire with the Grocery Cart Test presented here. The presence or exacerbation of symptoms with changes in spinal position, such as with standing, walking, or lying in bed, should stimulate investigation of this diagnosis. Confirmation of the diagnosis may be made by MRI or CT or by elimination of symptoms by reducing the nerve root irritation. Standard treatments include physical therapy, medications, epidural injections, bracing, and surgery. Mechanical treatment, such as use of a wheeled walker or a modified shoe, may reduce pain and facilitate an increase in activity. Alteration in sleep position may reduce or eliminate nocturnal symptoms. Identification and treatment of this common condition will greatly improve the quality of life for many patients.

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