Case Report

# A Nonsurgical Treatment Approach for Patients With Lumbar Spinal Stenosis

The purpose of this case report is to describe a physical therapy approach to the evaluation, treatment, and outcome assessment of two patients diagnosed with lumbar spinal stenosis. Evaluation consisted of assessment of neurological status, spinal range of motion, and lower-extremity muscle force production and flexibility; administration of the Modified Oswestry Low Back Pain Questionnaire and the Roland-Morris Disability Questionnaire; assessment of pain using a visual analog scale; and performance of a two-stage treadmill test. The treatment program was designed to treat the impairments, and harness-supported treadmill ambulation (unloading) was used to address the limitation in ambulation identified by the treadmill test. Outcome assessment included measuring changes in the status of the impairments and assessing responses to the disability questionnaires and performance of the two-stage treadmill test. Improvements were noted on all outcome measures for both patients after 6 weeks of physical therapy and at the 4-week follow-up examination. Larger case series and randomized trials with long-term follow-ups are recommended. [Fritz JM, Erhard RE, Vignovic M. A nonsurgical treatment approach for patients with lumbar spinal stenosis. *Phys Ther.* 1997;77:962–973.]

**Key Words:** *Rehabilitation, Spinal stenosis, Treadmill, Unloading.*

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**Lumbar spinal stenosis can be considered as a pathology with both structural and movement-associated components.**

Lumbar spinal stenosis (LSS) is defined as a narrowing of the spinal canal, nerve root canals, or vertebral foramina.[1] It is the most common preoperative diagnosis in persons over age 65 years who are undergoing lumbar spinal surgery.[2] Annually, in the United States, approximately 1 in 1,000 individuals over the age of 65 years undergoes a laminectomy for lumbar spinal stenosis, with costs estimated to be $1 billion.[3] Lumbar spinal stenosis is congenital or acquired.[1,4–6] Acquired forms of LSS are further classified as degenerative, spondylolisthetic, iatrogenic (postsurgical), posttraumatic, or combined.[1,5,7] Lumbar spinal stenosis also is classified as central, lateral, or combined. Central stenosis involves narrowing of the central spinal canal. Lateral stenosis affects the nerve root canal.[1,8]

Degenerative changes are the most common cause of LSS.[6,9] The degenerative changes leading to LSS are believed to be progressive, but the rate of deterioration is not considered to be linear and factors influencing the progression of changes have not been identified.[10,11] Nevertheless, many researchers believe that the prognosis for persons with LSS is poor and that surgical intervention is the most viable treatment option.[12,13] Indications for surgery for LSS are poorly defined, and controversy exists as to the optimal surgical procedures.[2,14–16] Surgery for LSS is associated with increased rates of mortality and morbidity,[17] and reoperation rates are reported to be as high as 21%.[2,15] A trial of nonsurgical therapy, including anti-inflammatory medications, corsets, epidural steroid injections, and physical therapy, is frequently recommended,[5,18–20] but specific conservative treatment regimens have not been defined.

Patients with LSS are most often at least 50 years of age with prolonged histories of low back pain and recent onset of unilateral or bilateral lower-extremity pain.[2,5,8,21] The symptoms, which are posture-dependent, are worsened with extension of the lumbar spine or weight-bearing postures of the spine and decreased with flexion or non–weight-bearing postures of the spine.[4,22,23] Neurological deficits are reported in about 50% of cases.[2,8] Acute cauda equina syndrome, although rare, has been reported.[2,8] Neurogenic claudication, defined as pain, paresthesia, and cramping of the lower extremities brought on by walking and relieved by sitting,[24] frequently accompanies LSS.[2,5,8] Progressive reduction of walking tolerance due to neurogenic claudication is common and is con-

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sidered by some authors [5,25] to be an indication for surgical intervention.

Acquired LSS has been attributed to structural narrowing of the spinal canal by one or more of the following conditions: facet-joint arthrosis and hypertrophy,[9,22,26] thickening and bulging of the ligamentum flavum,[4,27,28] outward bulging of the intervertebral disk,[29] and anterior displacement of the superior articulating process of the vertebral body due to lumbar spinal instability.[4,28,29] Symptomatic LSS cannot be attributed solely to structural narrowing of the canal dimensions, however, as evidenced by the high prevalence of narrowing seen on magnetic resonance imaging (MRI) scans or myelograms in individuals who have no symptoms[30,31] and by the poor correlations found between the severity of findings from imaging studies and the symptoms of patients with symptomatic LSS.[8,21] In addition to structural encroachment with movement (extension), narrowing of the spinal canal can occur. The cross-sectional area of the lumbar spinal canal and lateral recesses has been shown to increase with spinal flexion and to decrease with extension.[4,28,32] In a normal spine, the cross-sectional area is reduced by 9% during extension, but the reduction increases to 67% with severe stenosis.[32] Penning[4] has described the interplay between structural narrowing and changes that occur with movement as the "rule of progressive narrowing," which states that the more the canal is structurally narrowed by a stenosing process, the more it will be narrowed with extension. In addition to lumbar extension, loading of the spine through the compressive force associated with a weight-bearing posture reduces the cross-sectional area of the spinal canal.[4,28] Schonstrom et al[28] found that compressive loading had a slightly greater effect on decreasing the dimensions of the canal than did lumbar extension.

The movement-associated component of the stenosing process makes the symptoms of LSS posture-dependent, worsening with extension or compressive loading of the spine and improving with flexion or unloading of the spine.[4,22,23] Ambulation is an activity that involves both extension and compressive loading of the spine, and therefore it is frequently limited in patients with symptomatic LSS.[2,5,9] Limitations in ambulation can come from a variety of sources such as vascular claudication, lumbar disk herniation, or degenerative changes in the joints of the lower extremities. The differential diagnosis of whether the limitation is due to LSS can be difficult. A two-stage treadmill test, making use of the posture-dependent nature of the symptoms of LSS, is currently being investigated as a clinical tool to assist in the differential diagnosis of limited ambulation.[33] Walking on an inclined treadmill increases spinal flexion, and this increased spinal flexion should improve ambulation in persons with LSS.[33] Theoretically, the flexion should not improve the ambulation of patients with limitations due to causes other than LSS. Preliminary findings with the use of a two-stage treadmill test appear to support the hypothesis that patients with LSS will demonstrate one or all of the following findings: increased ambulation time on inclined treadmill tests, earlier onset of pain during the level ambulation stage versus the inclined ambulation stage, or prolonged recovery time after the level ambulation stage.[33]

The use of harness-supported treadmill ambulation, or unloading, has been advocated for use in patients with amputations,[34,35] foot injuries,[36] and herniated lumbar intervertebral disks,[37,38] but its application for patients with LSS has not been reported. Unloading involves the use of a traction harness and the application of a vertical traction force while the patient ambulates on a treadmill. The traction force is intended to reduce the gravitational force on the spine. This reduction in the compressive loading on the spine during ambulation may be useful in the treatment of patients with LSS.

A variety of measurements can be used to assess treatment of patients with low back pain, including those with LSS. In a recent meta-analysis of studies of the surgical treatment of patients with LSS, there was criticism of authors for generalizing outcomes into broad categories instead of looking at outcomes at multiple levels of a disability model.[2] Nagi[39] presented a scheme that defines four dimensions of disablement that need to be considered in a comprehensive assessment of treatment outcomes: (1) *active pathology*, or interruption of normal processes and the organism's inability to regain a normal state, (2) *impairment*, or loss or abnormality of an anatomical, physiological, or emotional nature, (3) *functional limitation*, or restriction of performance of the individual, and (4) *disability*, or restriction of an individual's ability to perform socially defined roles.

The purpose of our case report is to describe an approach to the physical therapy evaluation of two patients diagnosed with LSS. A treatment approach based on the evaluation results of each patient is described, including the use of harness-supported treadmill ambulation. Outcome measurements for different levels associated with the disablement process are presented for each patient.

## Case Description

### Patients

The two patients selected for this case report had pathology and clinical presentations consistent with a diagnosis of LSS. Patient data are summarized in Table 1.

**Table 1.**
Patient Characteristics

| | Patient 1 | Patient 2 |
|---|---|---|
| Age (y) | 58 | 76 |
| Gender | Female | Male |
| Height (cm) | 152 | 190 |
| Weight (kg) | 55 | 99 |
| Medical history | 9 y after kidney transplantation<br>2 y after left tibial plateau fracture<br>Non–insulin-dependent diabetes mellitus<br>Hypertension | Left-knee osteoarthritis<br>Hypertension |
| Medication | Immunosuppressive medication, prednisone, Tylenol® with codeine[a] | Altace®[b] |
| Diagnostic imaging results[c] | Right-facet osteoarthritis at L3-4, L4-5, L5-S1<br>Degenerative disk disease at L3-4, L5-S1<br>Mild central stenosis at L2-3<br>Moderate central stenosis at L3-4, L4-5<br>Central disk herniations at L3-4, L5-S1 | Mild central stenosis at L2-3<br>Severe central stenosis at L3-4, L4-5<br>Right lateral stenosis at L4-5 |

[a] McNeil Pharmaceutical, 1000 US Rte 202, PO Box 300, Raritan, NJ 08869.
[b] Hoechst-Roussel Pharmaceuticals Inc, Rte 202–206, PO Box 2500, Somerville, NJ 08876.
[c] Patient 1: radiographs and magnetic resonance imaging scans; patient 2: radiographs and computed tomography scans.

Patient 1 was a 58-year-old woman with a 10-year history of low back pain and a 6-month history of right leg pain exacerbated by walking. Onset of the lower-extremity symptoms was gradual; no spinal trauma was reported. Anteroposterior and lateral radiographs and MRI scans obtained 2 months prior to physical therapy showed degenerative changes of the facets and intervertebral disks at the L3-4 and L5-S1 levels (Fig. 1) in addition to multilevel central stenosis.

Patient 2 was a 76-year-old man with a 25-year history of low back pain and a 7-month history of left anterior leg pain exacerbated by walking. No spinal trauma was reported. This patient had undergone anteroposterior and lateral radiography and a computed tomography scan 2 weeks prior to referral for physical therapy that showed multilevel central stenosis and lateral stenosis of the right L4-5 intervertebral foramen (Fig. 2).

### *Initial Physical Therapy Evaluation*

During the initial physical therapy evaluation, the patients completed medical history questionnaires, visual analog pain scales, the Modified Oswestry Low Back Pain Questionnaire,[40] and the Roland-Morris Disability Questionnaire.[41] The Modified Oswestry Low Back Pain Questionnaire[40] covers 10 areas of daily living and expresses the degree of disability as a percentage. The Roland-Morris Disability Questionnaire[41] contains 24 items selected from the 136-item Sickness Impact Profile[42] and reports a score from 0 to 24, with a score of 24 reflecting the greatest limitation. Scores on both questionnaires have shown Pearson correlation coefficients between .76 and .99, and the construct validity of

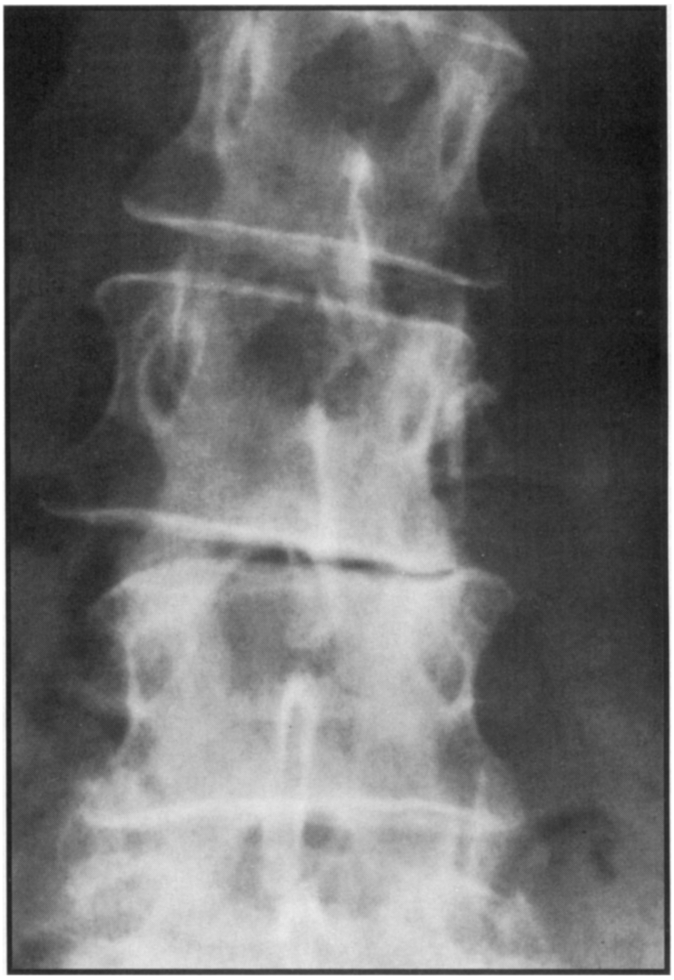

**Figure 1.**
Anteroposterior radiograph of L-3 to L-5 levels in patient 1, showing lateral displacement of L-3 on the body of L-4 and advanced degenerative changes at the L-3 and L-4 levels.

**Figure 2.**
Computed tomography scan at the L-4 and L-5 levels in patient 2, showing moderate to severe central stenosis, as evidenced by the narrowed black triangular area in the center of the spinal canal. Facet-joint osteoarthritis is also present.

the two questionnaires is supported through correlations with variables such as pain, spinal mobility, and psychological and patient satisfaction measures.[40,41,43,44] Studies of the reliability and validity of existing scales have been conducted on patients with low back pain without regard to diagnosis. The performance of these instruments in a subgroup of patients with LSS is not known. For the visual analog pain scale, we asked the patients to rate their level of pain on a scale of 0 to 10, with 0 representing no pain and 10 representing extreme pain. Visual analog pain scales have been shown to yield reliable measurements.[45]

After the patients completed the questionnaires and visual analog pain scale, a physical examination was performed. In patients with symptoms extending below the knee, we conduct a neurological examination, which includes the measurement of lower-extremity reflexes, sensation and muscle force production testing, and assessment of straight leg raise. The Achilles and patellar tendon reflexes are tested. Manual muscle testing of the lower extremities is performed to identify any myotomal pattern of weakness. Sensation is assessed by light touch and pinprick over each dermatome of the lower extremity. We consider the straight leg raise test to be positive for irritation of the sciatic nerve when motion is limited to less than 70 degrees and produces radicular pain.[46]

Assessment of the bony landmarks of the pelvis was then done while the patients were in a standing position. We palpated the posterior superior iliac spines (PSISs), anterior superior iliac spines (ASISs), and iliac crests bilaterally and compared the relative heights of the left and right landmarks. Consistently higher landmarks on one side may indicate a leg-length discrepancy,[47] whereas an inconsistent pattern (eg, a high right ASIS with a high left PSIS) may indicate another condition, one probably associated with the sacroiliac region.[48] If we suspect a leg-length discrepancy, we palpate the bony landmarks with the patient in a sitting position. In the presence of a leg-length discrepancy, we believe that the landmarks should appear to be level with the patient in a sitting position. If a sacroiliac joint-related problem is suspected, tests are performed as described elsewhere.[48] When a composite of several positive tests is used to define the presence of sacroiliac joint dysfunction, acceptable intertester reliability has been shown.[49] We tested active spinal range of motion with the patients in a standing position. The patients bent to the left and right sides, flexed, and extended. Measurements were taken with a gravity goniometer,* which has been shown to yield reliable measurements of spinal range of motion.[50] We recorded the patients' reports of changes in symptoms with movement as well as the range of motion. In persons with low back and lower-extremity pain, according to McKenzie,[51] symptoms may (1) peripheralize (paresthesia is produced or the pain or paresthesia moves distally from the lumbar spine), (2) centralize (paresthesia or pain is eliminated or moves from the periphery toward the lumbar spine), or (3) be unchanged with movements.

The symptoms of a degenerative hip joint and LSS are similar, and the two conditions can occur simultaneously.[52] We believed that an examination of the hip was therefore necessary to rule out potential involvement of the hip joint in these two patients. The examination consisted of the Patrick and scour tests.[52] The Patrick test is performed with the patient positioned supine with the hips and knees extended. The knee of the tested extremity is placed over the opposite knee, bringing the tested hip into flexion, abduction, and lateral (external) rotation. Pressure is applied to the medial aspect of the knee on the tested side. Anterior groin or thigh pain is considered a positive finding for hip joint dysfunction. A loss of motion of one hip joint relative to the opposite side has been correlated with radiographic evidence of osteoarthritis of the hip joint.[54] Production of pain in the low back or buttock is indicative of lumbar spine or sacroiliac joint involvement.[55] The scour test is performed by compressing the hip joint while moving from the position of flexion, medial (internal) rotation, and adduction into extension, lateral rotation, and abduction. Production of pain or crepitation is a positive test for hip joint dysfunction.[53] If a positive test for hip joint

* Vigor Equipment Inc, 4915 Advance Way, Stevensville, MI 49127.

**Table 2.**
Results of Self-Report Measures Administered at Initial Evaluation, After 6 Weeks of Physical Therapy, and at 4-Week Follow-up

| | Patient 1 | | | Patient 2 | | |
|---|---|---|---|---|---|---|
| **Measure** | **Initial Evaluation** | **After 6 Weeks of Physical Therapy** | **4-Week Follow-up** | **Initial Evaluation** | **After 6 Weeks of Physical Therapy** | **4-Week Follow-up** |
| Modified Low Back Pain Oswestry Questionnaire[40] (%) | 48 | 16 | 12 | 53 | 0 | 0 |
| Roland-Morris Disability Questionnaire[4] | 17 | 2 | 3 | 19 | 1 | 1 |
| Visual analog pain scale | 6/10 | 1/10 | 1/10 | 5/10 | 0/10 | 0/10 |

dysfunction is found, a more specific evaluation of the hip joint is indicated to determine the nature of the dysfunction.

In patients with chronic low back pain, such as those with LSS, we include an examination for signs of physical impairments such as weakness or lack of flexibility.[48] Manual muscle testing and flexibility testing of the lower extremities were performed as described by Kendall and McCreary.[56] In patients with chronic low back pain, the gluteal muscles tend to become weak, whereas the hip flexors and hamstring muscles may become shortened.[57,58] Assessment of these muscle groups is therefore appropriate in patients with chronic low back pain. Flexibility of the one- and two-joint hip flexors was assessed with the Thomas test.[56] We consider one-joint hip flexors to be shortened when the tested hip is unable to fully extend while the pelvis maintains a posteriorly tilted position.[56] We consider two-joint hip flexors to be shortened if the knee is unable to reach 80 degrees of flexion without increasing hip flexion.[56] Hamstring muscle length was assessed by measuring the angle of hip flexion obtained during a straight-leg-raising test with the opposite leg extended and the pelvis posteriorly tilted. Kendall and McCreary[56] defined a positive test as less than 80 degrees of hip flexion. Gluteus maximus muscle force production was determined by resisting hip extension with the patient positioned prone and the knee fully flexed. The gluteus medius muscle was tested with the patient positioned side lying and the hip in abduction with slight hip extension and lateral rotation. The motion of abduction is resisted.[56]

We have been investigating the ability of a two-stage treadmill test to discriminate between patients with and without LSS.[33] The two patients described in this case report agreed to participate in that study. This test is performed by having the patient ambulate on a level treadmill and a treadmill with a 15-degree incline. As part of the research protocol, the order of incline is determined randomly. For patients undergoing repeat testing following intervention, the same order of testing is used for the follow-up test. The patients were asked to walk at a comfortable pace without using handrails. The walking time until the symptoms of low back or lower-extremity pain increased over the level recorded before the test began, and the maximal walking time, limited by either fatigue or symptoms, were recorded. Patients walked for a maximum of 15 minutes then sat, and the time required for symptoms to return to the pretreadmill walking level was recorded. The patients rested for a total of 15 minutes, and the test was repeated using the second treadmill position.

### *Outcome Measures*

Measures of treatment outcomes for these two patients included the visual analog scale, the Modified Oswestry Low Back Pain Questionnaire,[40] the Roland-Morris Disability Questionnaire,[41] impairments identified at the initial evaluation, and the two-stage treadmill test. All outcome measures were assessed at the initial evaluation and at completion of physical therapy. Except for the measure of impairments, the measures were assessed again 4 weeks later.

### *Findings of the Initial Physical Therapy Evaluation*

The results of the self-report questionnaires administered at the initial evaluation are presented in Table 2, and the results of the physical examination are shown in Table 3. Patient 1 had a leg-length discrepancy, with a long right leg. Both patients exhibited a peripheralization of symptoms with lumbar extension, as expected in patients with LSS. Patient 1 had positive findings on neurological assessment in the form of reflex, sensory, and motor changes, as well as a positive straight-leg-raising test. The results of the two-stage treadmill test for patient 1 (Tab. 4) showed a longer walking time on the inclined treadmill, an earlier onset of symptoms on the level treadmill, and a longer recovery after level treadmill ambulation. The two-stage treadmill test results for patient 2 showed an earlier onset of symptoms and a longer recovery time with level treadmill ambulation than with inclined treadmill ambulation.

**Table 3.**
Physical Examination Findings for Both Patients

| Procedure | Patient 1 Initial Evaluation | Patient 1 After 6 Weeks of Physical Therapy | Patient 2 Initial Evaluation | Patient 2 After 6 Weeks of Physical Therapy |
|---|---|---|---|---|
| Pelvic landmark palpation | Landmarks consistently high on the right | Level with 1.27-cm (1.5-in) heel lift in the left shoe | Level | Level |
| Single movement testing and symptom response | | | | |
| Flexion | 33° unchanged | 38° unchanged | 31° centralized | 43° unchanged |
| Extension | 6° peripheralized | 9° unchanged | 17° peripheralized | 25° unchanged |
| Left side bending | 16° unchanged | 20° unchanged | 10° unchanged | 16° unchanged |
| Right side bending | 11° unchanged | 19° unchanged | 9° unchanged | 12° unchanged |
| Neurological assessment | Left L-3, L-4 weakness<br>Right L-5 sensory deficit<br>Diminished left quadriceps femoris muscle reflex | Left L-3, L-4 weakness<br>Diminished left quadriceps femoris muscle reflex | No deficits noted | No deficits noted |
| Straight leg raise | | | | |
| Left | 60° negative | 63° negative | 74° negative | 75° negative |
| Right | 44° positive | 59° negative | 70° negative | 70° negative |
| Hip joint clearing | Negative | | Negative | |
| Manual muscle testing | | | | |
| Gluteus medius | Left 2/5 Right 3/5 | Left 3+/5 Right 4/5 | Left 5/5 Right 5/5 | Left 5/5 Right 5/5 |
| Gluteus maximus | Left 4/5 Right 4/5 | Left 4/5 Right 4/5 | N/A[a] | N/A |
| Quadriceps femoris | Left 4/5 Right 5/5 | Left 4/5 Right 5/5 | Left 4/5 Right 5/5 | Left 5/5 Right 5/5 |
| Hamstring | Left 5/5 Right 5/5 | Left 5/5 Right 5/5 | Left 5/5 Right 5/5 | Left 5/5 Right 5/5 |
| Tibialis anterior | Left 4/5 Right 5/5 | Left 4/5 Right 5/5 | Left 5/5 Right 5/5 | Left 5/5 Right 5/5 |
| Flexibility testing | | | | |
| One-joint hip flexors | Left + Right + | Left + Right + | Left − Right − | N/A |
| Two-joint hip flexors | Left + Right + | Left + Right + | Left − Right − | N/A |
| Hamstring muscles | Left + Right N/A | Left + Right + | Left + Right + | Left + Right + |

[a] N/A=not assessed.

**Table 4.**
Results of Two-Stage Treadmill Test for Patient 1 at Initial Evaluation, After 6 Weeks of Physical Therapy, and at 4-Week Follow-up

| Measure | Inclined Treadmill Initial Evaluation | Inclined Treadmill After 6 Weeks of Physical Therapy | Inclined Treadmill 4-Week Follow-up | Level Treadmill Initial Evaluation | Level Treadmill After 6 Weeks of Physical Therapy | Level Treadmill 4-Week Follow-up |
|---|---|---|---|---|---|---|
| Walking speed (mph) | 0.7 | 0.8 | 0.8 | 0.7 | 0.8 | 0.8 |
| Time to increase in symptoms (min) | 4¼ | No increase noted | No increase noted | 2½ | No increase noted | No increase noted |
| Maximum walking time (min) | 7⅙ | 15 | 15 | 4½ | 15 | 15 |
| Symptoms at completion | Low back pain, fatigue | Fatigue | Fatigue | Low back pain, left calf pain | None noted | None noted |
| Recovery time (min) | 3⅓ | N/A[a] | N/A | 4⅙ | N/A | N/A |

[a] N/A=not assessed.

*Treatment Plan*

Both patients received physical therapy for LSS over a 6-week period. Patient 1 was seen for eight visits and patient 2 was seen for 11 visits during that period. The treatment approach had two components: an exercise program and a program of harness-supported treadmill. In addition, patient 1 received a 1.27-cm (0.5-in) heel lift in the left shoe to correct a leg-length discrepancy of 1.27 cm. The exercise program was designed to address impairments identified at the initial evaluation. The impairments to be addressed were decreased lower-extremity muscle force production, flexibility, and peripheralization of symptoms with lumbar extension. Spinal flexion increases the spinal canal dimensions.[4,28,32] We believed, therefore, that flexion exercises may help to decrease symptoms.

Flexion exercises including posterior pelvic tilts, quadruped spinal flexion, and single-knee-to-chest exercises were performed by patient 1. Patient 2 performed quadruped spinal flexion. Patient 2 tolerated treadmill exercise better than did patient 1 initially. Treatment for patient 2, therefore, was focused more on treadmill training than on flexion exercises. All flexion exercises were performed in gravity-eliminated postures to avoid the compressive loading associated with weight bearing that reduces the dimensions of the spinal canal.[28] Both patients performed flexion exercises three to four times per day, performing 10 repetitions of each exercise.

Flexibility deficits were addressed with hamstring muscle stretching performed by extending the knee while positioned supine with the hip flexed to 90 degrees. This position was held for 30 seconds and repeated five times. Hip flexor stretching was performed by maintaining a posterior pelvic tilt while in a half-kneeling posture. The muscle force production deficits identified in patient 1 were addressed with lower-extremity strengthening exercises focusing on the gluteus medius muscle and consisting of hip abduction in a standing position with the pelvis maintained in the horizontal plane, progressing to single-leg standing and then to lateral step-ups while maintaining a horizontal pelvis. Both patients performed mini-squats for general lower-extremity strengthening. Mini-squats were formed in the standing position by slowly flexing the knees to approximately 45 degrees and then returning to the extended position. Patient 2 was progressed to straight leg raises in flexion, extension, abduction, and adduction and terminal knee extension exercises to address the quadriceps femoris muscle weakness.

Flexion exercises were performed as a single set of 10 repetitions; flexibility exercises were performed as a single set of 5 repetitions consisting of 30-second stretches; and strengthening exercises were performed in sets of 10 repetitions, beginning with a single set and progressing to three sets, as tolerated by the patient. Each physical therapy session lasted approximately 1 hour. Both patients reported that they performed their exercises once or twice daily at home.

Each patient's initial performance on the two-stage treadmill test showed limitations in the ability to ambulate on level surfaces without symptoms of low back or lower-extremity pain. These limitations were addressed by using harness-supported treadmill ambulation in which a vertical traction force can be applied to reduce the compressive loading on the spine and allow for

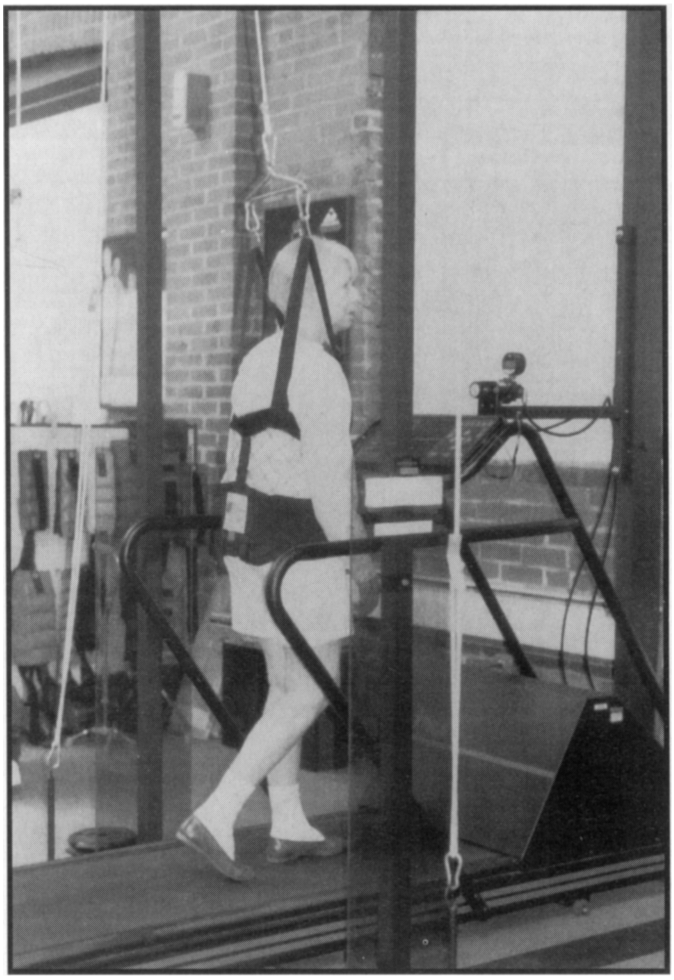

**Figure 3.**
Patient performing harness-supported treadmill ambulation. The traction harness provides the unloading force.

pain-free gait training. This ambulation can be performed in a pool using the buoyancy of the water, as was initially done for patient 1, or using a treadmill and traction harness* to provide a traction force to partially unload the spine of the compressive forces associated with weight-bearing postures (Fig. 3). Sufficient traction was applied to completely relieve the patient's symptoms of low back and lower-extremity pain during ambulation. The progression of harness-supported treadmill ambulation for both patients is shown in Table 5. Patient 1's ambulation was begun in the pool because this is considered to be a less demanding activity. This patient was soon able to progress to harness-supported treadmill ambulation.

## Treatment Outcome

At the completion of 6 weeks of treatment (both patients were referred for 6 weeks of treatment and then returned to their physician), the patients' impairments were reassessed (Tab. 3) and the self-report measures (Tab. 2) and the two-stage treadmill test (Tabs. 4, 6) were repeated. Improvements were noted in some of the

**Table 5.**
Progression of Harness-Supported Treadmill Ambulation During Physical Therapy Sessions

| Session No. | Patient 1 | Patient 2 |
|---|---|---|
| 1 | Pool walking × 10 min | Treadmill unloading: 2.0 mph<br>22 lb of traction<br>30 min of ambulation |
| 2 | Pool walking × 15 min | Treadmill unloading: 2.0 mph<br>22 lb of traction<br>30 min of ambulation |
| 3 | Treadmill unloading: 0.7 mph<br>40 lb of traction<br>Two sets of 10 min of ambulation | Treadmill unloading: 2.0 mph<br>22 lb of traction<br>30 min of ambulation |
| 4 | Treadmill unloading: 0.8 mph<br>37 lb of traction<br>Two sets of 15 min of ambulation | Treadmill unloading: 2.0 mph<br>20 lb of traction<br>35 min of ambulation |
| 5 | Treadmill not functioning<br>No gait training performed | Treadmill unloading: 2.0 mph<br>20 lb of traction<br>35 min of ambulation |
| 6 | Treadmill unloading: 0.8 mph<br>24 lb of traction<br>Two sets of 20 min of ambulation | Treadmill unloading: 2.0 mph<br>15 lb of traction<br>40 min of ambulation |
| 7 | Treadmill unloading: 0.8 mph<br>18 lb of traction<br>Two sets of 20 min of ambulation | Treadmill unloading: 2.0 mph<br>15 lb of traction<br>45 min of ambulation |
| 8 | Treadmill: 0.8 mph<br>No traction used<br>Two sets of 15 min of ambulation | Treadmill: 2.0 mph<br>No traction used<br>30 min of ambulation |
| 9 | N/A[a] | Treadmill 2.5 mph<br>No traction used<br>30 min of ambulation |
| 10 | N/A | Treadmill: 2.5 mph<br>No traction used<br>30 min of ambulation |
| 11 | N/A | Treadmill: 2.5 mph<br>No traction used<br>30 min of ambulation |

[a] N/A=not assessed.

impairment measures. Both patients showed improvements in lumbar range of motion. Patient 1 showed improvements in neurological status and sensation as well as improvement in performance of the straight-leg-raising test. Improvements in muscle force production were noted, particularly in the gluteus medius muscle of patient 1 and the quadriceps femoris muscle of patient 2. It must be remembered that the reliability of grading manual muscle testing is known to be poor,[59] and the improvements noted could represent measurement error. Substantial improvements were found on the self-report outcome measures (Tab. 2) and the two-stage treadmill test (Tabs. 4, 6). Both patients were able to ambulate the full 15 minutes during the 6-week reassessment. Both patients were instructed to continue their home exercise program daily after discharge from physical therapy and to perform at least 15 to 20 minutes of symptom-free walking daily. If symptoms occurred, the patients were instructed to stop walking and sit until the symptoms diminished.

Both patients returned for a follow-up assessment 4 weeks after discharge from physical therapy. The self-report measures (Tab. 2) and the two-stage treadmill test (Tabs. 4, 6) were readministered. The results indicated that the improvements in limitations and disability noted at the conclusion of physical therapy were maintained over a 4-week period following discharge. Both patients reported doing their home exercise programs, and neither patient reported using any pain medication following discharge.

## Discussion

The structural component of LSS is identified by imaging techniques such as radiography, MRI, and computed tomography. Treatment of patients with LSS has focused on addressing the structural component through surgical procedures such decompression-laminectomy and lumbar fusion.[6,12,25] The movement-associated component of LSS results from the changes in the spinal canal dimensions with lumbar motion or with compressive loading.[4,23,28,32] Failure to consider the movement-associated component of LSS may partially explain the relatively high incidence of structural changes on imaging studies of individuals without symptoms of LSS[30,31] and the poor correlation between the degree of structural changes seen on imaging and the severity of symptoms.[8,21] Treatment of the movement-associated component through appropriate exercise programs has received little attention.

We believe that findings from the patient's history and clinical examination must correlate with the structural findings before a diagnosis of LSS can be made.[8,21,52] Physical therapy, in our opinion, also must be based on the patient's signs and symptoms and not on a structural diagnosis, even when such a diagnosis exists.[48] In addition, the severity of the structural pathology seen on diagnostic imaging studies in patients with LSS has been shown to correlate poorly with the severity of symptoms and limitations.[8,21]

**Table 6.**
Results of Two-Stage Treadmill Test for Patient 2 at Initial Evaluation, After 6 Weeks of Physical Therapy, and at 4-Week Follow-up

| | Inclined Treadmill | | | Level Treadmill | | |
|---|---|---|---|---|---|---|
| Measure | Initial Evaluation | After 6 Weeks of Physical Therapy | 4-Week Follow-up | Initial Evaluation | After 6 Weeks of Physical Therapy | 4-Week Follow-up |
| Walking speed (mph) | 1.5 | 2.5 | 2.5 | 1.5 | 2.5 | 2.5 |
| Time to increase in symptoms (min) | 2¼ | No increase noted | No increase noted | 1⅚ | No increase noted | No increase noted |
| Maximum walking time (min) | 5⅙ | 15 | 15 | 5¼ | 15 | 15 |
| Symptoms at completion | Left anterior leg pain | None noted | None noted | Left anterior leg pain | None noted | None noted |
| Recovery time (min) | 4 | N/A[a] | N/A | 4⅓ | N/A | N/A |

[a] N/A=not assessed.

The patients in this case report had moderate to severe pathological changes (Figs. 1, 2), yet they responded positively to physical therapy. Both patients demonstrated peripheralization of symptoms with extension, and they reported claudication-like symptoms, which were confirmed by the two-stage treadmill test. The treadmill test also confirmed the posture-dependent nature of the patients' symptoms, with less severe symptoms noted during inclined walking with the spine in more flexion. Delitto et al[48] classified these signs and symptoms as a flexion syndrome, and they recommended the use of flexion exercises as treatment. We also use harness-supported treadmill ambulation for patients with leg pain brought on by walking and relieved with sitting. This type of ambulation provides a functional rehabilitation tool that addresses a common limitation for patients with LSS.[35,36] The amount of unloading force is monitored and progressed until unloading force is no longer required to relieve pain during ambulation, as was demonstrated by the patients in this case report.

We believe that a comprehensive assessment of patients with low back pain should include outcome measures that capture the multidimensional nature of pain and the degree of disablement due to low back pain.[60,61] For the patients we described, we assessed each dimension, with the exception of the active pathology. Measurements of spinal range of motion, neurological status, lower-extremity muscle force production, and flexibility were used as outcomes in the assessment of impairments. Limited ambulation is a frequent limitation among patients with LSS and was noted for both patients described in this case report. The use of a treadmill test to approximate ambulation tolerance has been recommended as an outcome measure for patients with spinal disorders.[62,63] The two-stage treadmill test, therefore, serves as both a clinical diagnostic tool and an outcome measure for these patients. Disability was assessed by the Modified Oswestry and Roland-Morris questionnaires. For the patients described in this case report, improvements were found at the completion of treatment, as indicated by the results from both disability questionnaires (Tab. 2). These improvements coincided with improvements in the total walking time during the treadmill test (Tabs. 4, 6). Both patients were able to more than double their total walking time.

The purpose of our case report was to describe an approach to the evaluation, treatment, and outcome assessment for patients with LSS. No experimental evidence is offered. The tendency for low back pain to improve over time must be considered. In addition, the patients were treated with a program of treadmill ambulation, and the improvements noted on the two-stage treadmill test could represent task-specific improvements. In our view, experimental studies can be performed only after an approach to evaluation, treatment, and outcome assessment has been defined for the population being studied. This case report of two patients with short-term follow-up needs to be followed by reports describing larger series of patients with LSS treated with this approach with longer follow-up periods. If the treatment approach we are recommending produces favorable long-term outcomes in larger series of patients, then a randomized clinical trial would be warranted to compare this approach with the present "standard of care," which consists of the use of medications or nonspecific exercises.[15] Only a randomized clinical trial could produce experimental evidence for the efficacy of the treatment approach we suggest.

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## References


1 Arnoldi CC, Brodsky AE, Cachoix J, et al. Lumbar spinal stenosis and nerve root entrapment: syndromes, definition, and classification. *Clin Orthop.* 1976;115:4–5.

2 Turner JA, Ersek M, Herron L, et al. Surgery for lumbar spinal stenosis: an attempted meta-analysis of the literature. *Spine.* 1992;17: 1–8.

3 Graves EJ. Detailed diagnoses and procedures: National Hospital Discharge Survey 1987; National Center for Health Statistics. *Vital Health Stat.* 1989;13(100):151, 194–195.

4 Penning L. Functional pathology of lumbar spinal stenosis. *Clin Biomech.* 1992;7:3–17.

5 Onel D, Sari H, Donmez C. Lumbar spinal stenosis—clinical/radiologic therapeutic evaluation in 145 patients: conservative treatment or surgical intervention? *Spine.* 1993;18:291–298.

6 Getty CJM. Lumbar spine stenosis: the clinical significance and results of operation. *J Bone Joint Surg [Br].* 1980;62:481–485.

7 Epstein BS, Epstein JA, Jones MD. Lumbar spinal stenosis. *Radiol Clin North Am.* 1977;15:261–274.

8 Amundsen T, Weber H, Lilleas F, et al. Lumbar spinal stenosis: clinical and radiologic features. *Spine.* 1995;20:1178–1186.

9 Herzog RJ, Kaiser JA, Saal JA, et al. The importance of posterior epidural fat pad in lumbar central canal stenosis. *Spine.* 1991;16:s227–s233.

10 Cassidy JD, Wedge JH. The epidemiology and natural history of low back pain and spinal degeneration. In: Kirkaldy-Willis WH, ed. *Managing Low Back Pain.* New York, NY: Churchill Livingstone Inc; 1988: 3–13.

11 Johnsson KE, Rosen I, Uden A. The natural course of lumbar spinal stenosis. *Clin Orthop.* 1992;279:82–86.

12 Sanderson PL, Wood PLR. Surgery for lumbar spinal stenosis in old people. *J Bone Joint Surg [Br].* 1993;75:393–397.

13 Wiltse LL, Kirkaldy-Willis WH, McIvor GWD. The treatment of spinal stenosis. *Clin Orthop.* 1976;115:83–91.

14 Tuite GF, Stern JD, Doran SE, et al. Outcome after laminectomy for lumbar spinal stenosis, part 1: clinical correlations. *J Neurosurg.* 1994; 81:699–706.

15 Bigos S, Bowyer O, Braen G, et al. *Acute Low Back Problems in Adults.* Rockville, Md: Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services; 1994. Clinical Practice Guideline No. 14, AHCPR Publication No. 95–0642.

16 Deyo RA, Ciol MA, Cherkin DC, et al. Lumbar spinal fusion: a cohort study of complications, reoperations, and resource use in the medicare population. *Spine.* 1993;18:1463–1470.

17 Deyo RA, Cherkin DC, Loeser JD, et al. Morbidity and mortality in association with operations on the lumbar spine. *J Bone Joint Surg [Am].* 1992;74:536–543.

18 Katz JN, Lipson SJ, Larson MG, et al. The outcome of decompressive laminectomy for degenerative lumbar spinal stenosis. *J Bone Joint Surg [Am].* 1991;73:809–816.

19 Herkowitz HN, Kurz LT. Degenerative lumbar spondylolisthesis with spinal stenosis. *J Bone Joint Surg [Am].* 1991;73:802–808.

20 Grob D, Humke T, Dvorak J. Degenerative lumbar spinal stenosis. *J Bone Joint Surg [Am].* 1995;77:1036–1041.

21 Katz JN, Dalgas M, Stucki G, et al. Degenerative lumbar spinal stenosis: diagnostic value of the history and physical examination. *Arthritis Rheum.* 1995;38:1236–1241.

22 Penning L, Wilmink JT. Posture-dependent bilateral compression of L4 or L5 nerve roots in facet hypertrophy: a dynamic CT-myelographic study. *Spine.* 1987;12:488–500.

23 Takahashi K, Miyazaki T, Takino T, et al. Epidural pressure measurements: relationship between epidural pressure and posture in patients with lumbar spinal stenosis. *Spine.* 1995;20:650–653.

24 Porter RW. Pathophysiology of neurogenic claudication. In: Wiesel SW, Weinstein JN, Herkowitz H, et al, eds. *The Lumbar Spine.* Philadelphia, Pa: WB Saunders Co; 1996:717–723.

25 Herno A, Airaksinen O, Saari T. Long-term results of surgical treatment of lumbar spinal stenosis. *Spine.* 1993;18:1471–1474.

26 Rausching W. Normal and pathologic anatomy of the lumbar root canals. *Spine.* 1987;12:1010–1019.

27 Postacchini F, Gumina S, Cinotti G, et al. Ligamenta flava in lumbar disc herniation and spinal stenosis. *Spine.* 1994;19:917–922.

28 Schonstrom N, Lindahl S, Willen J, et al. Dynamic changes in the dimensions of the lumbar spinal canal: an experimental study in vitro. *J Orthop Res.* 1989;7:115–121.

29 Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, et al. Pathology and pathogenesis of lumbar spondylosis and stenosis. *Spine.* 1978;3:319–328.

30 Hitselberger WE, Witten RM. Abnormal myelograms in asymptomatic patients. *J Neurosurg.* 1968;28:204–206.

31 Boden SD, Davis DO, Dina TS, et al. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. *J Bone Joint Surg [Am].* 1990;72:403–408.

32 Sortland O, Magnaes B, Hauge T. Functional myelography with metrizamide in the diagnosis of lumbar spinal stenosis. *Acta Radiol.* 1977;355(suppl):42–54.

33 Fritz JM, Erhard RE, Delitto A, Nowakowski PE. Preliminary results of the use of a two-stage treadmill test as a clinical diagnostic tool in the differential diagnosis of lumbar spinal stenosis. *J Spinal Disord.* In press.

34 Hunter D, Cole ES, Murray JM, et al. Energy expenditure of below-knee amputees during harness-supported treadmill ambulation. *J Orthop Sports Phys Ther.* 1995;21:268–276.

35 Pillar T, Dickstein R, Smolinski Z. Walking reeducation with partial relief of body weight in rehabilitation of patients with locomotor disabilities. *J Rehabil Res Dev.* 1991;28:47–52.

36 Kelsey DD, Tyson E. A new method of training for the lower extremity using unloading. *J Orthop Sports Phys Ther.* 1994;19:218–223.

37 Simpson S, Bettis B, Herbertson J. Unloaded treadmill training therapy for lumbar disc herniation injury. *Journal of Athletic Training.* 1996;31:57–60.

38 Olson J, Svendsen B. Medical exercise therapy: an adjunct to orthopaedic manual therapy. *Orthopedic Practice.* 1992;4:7–10.

39 Nagi SZ. Some conceptual issues in disability and rehabilitation. In: Sussman MB, ed. *Sociology and Rehabilitation.* Washington, DC: American Sociological Association; 1965:100–113.

40 Hudson-Cook N, Tomes-Nicholson K, Breen A. A revised Oswestry disability questionnaire. In: Roland MO, Jenner JR, eds. *Back Pain: New Approaches to Rehabilitation and Education.* New York, NY: Manchester University Press; 1989:187–204.

41 Roland M, Morris R. A study of the natural history of back pain, part I: the development of a reliable and sensitive measure of disability in low-back pain. *Spine.* 1983;8:141–144.

42 Bergner M, Babbitt RA, Carter WB, et al. The Sickness Impact Profile: development and final revision of a health status measure. *Med Care.* 1981;19:787–805.

43 Deyo RA. Comparative validity of the Sickness Impact Profile and shorter scales for functional assessment in low-back pain. *Spine.* 1986; 11:951–954.

44 Kopec JA, Esdaile JM. Functional disability scales for back pain. *Spine.* 1995;20:1943–1949.

45 Downie WW, Leatham PA, Rhind VM, et al. Studies in pain rating scales. *Ann Rheum Dis.* 1978;37:382–284.

46 Supik LF, Broom MJ. Sciatic tension signs and lumbar disc herniation. *Spine.* 1994;19:1066–1069.

47 Porterfield JA, DeRosa C. The sacroiliac joint. In: Gould JA, ed. *Orthopedic and Sports Physical Therapy.* 2nd ed. St Louis, Mo: CV Mosby Co; 1990:564–573.

48 Delitto A, Erhard RE, Bowling RW. A treatment-based classification approach to low back syndrome: identifying and staging patients for conservative treatment. *Phys Ther.* 1995;75:470–489.

49 Cibulka MT, Delitto A, Koldehoff RM. Changes in innominate tilt after manipulation of the sacroiliac joint in patients with low back pain: an experimental study. *Phys Ther.* 1988;68:1359–1363.

50 Fitzgerald GK, Wynveen KJ, Rheault W, et al. Objective assessment with establishment of normal values for spinal range of motion. *Phys Ther.* 1983;63:1776–1781.

51 McKenzie RA. *The Lumbar Spine: Mechanical Diagnosis and Therapy.* Waikanae, New Zealand: Spinal Publications Ltd; 1989:85–93.

52 McNamara MJ, Barrett KG, Christie MJ, et al. Lumbar spinal stenosis and lower extremity arthroplasty. *J Arthroplasty.* 1993;8:273–277.

53 Woerman AL. Evaluation and treatment of dysfunction in the lumbar-pelvic-hip complex. In: Donatelli R, Wooden MJ, eds. *Orthopaedic Physical Therapy.* 2nd ed. New York, NY: Churchill Livingstone Inc; 1994:481–560.

54 Theiler R, Stucki G, Schutz G, et al. Parametric and nonparametric measures in the assessment of knee and hip osteoarthritis: interobserver reliability and correlation with radiology. *Osteoarthritis and Cartilage.* 1996;4:35–42.

55 Hoppenfeld S. *Physical Examination of the Spine and Extremities.* Norwalk, Conn: Appleton-Century-Crofts; 1976.

56 Kendall FP, McCreary EK. *Muscles: Testing and Function.* 3rd ed. Baltimore, Md: Williams & Wilkins; 1983.

57 Bullock-Saxton JE, Janda V, Bullock MI. Reflex activation of gluteal muscles in walking: an approach to restoration of muscle function in patients with low-back pain. *Spine.* 1993;18:704–708.

58 Janda V. Muscle weakness and inhibition (pseudoparesis) in back pain syndromes. In: Grieve GP, ed. *Modern Manual Therapy of the Vertebral Column.* New York, NY: Churchill Livingstone Inc; 1986:197–201.

59 Frese E, Brown M, Norton BJ. Clinical reliability of manual muscle testing: middle trapezius and gluteus medius muscles. *Phys Ther.* 1987;67:1072–1076.

60 Delitto A. Are measures of function and disability important in low back care? *Phys Ther.* 1994;74:452–462.

61 Deyo RA, Andersson G, Bombardier C, et al. Outcome measures for studying patients with low back pain. *Spine.* 1994;19:S2032–S2036.

62 Deen HG, Zimmerman RS, Lyons MK, et al. Measurement of exercise tolerance on the treadmill in patients with symptomatic lumbar spinal stenosis: a useful indicator of functional status and surgical outcome. *J Neurosurg.* 1995;83:27–30.

63 Tokuhashi Y, Matsuzaki H, Sano S. Evaluation of clinical lumbar instability using the treadmill. *Spine.* 1993;18:2321–2324.