RHEUMATIC DISEASE CLINICS OF NORTH AMERICA

Diagnostic Issues

DAVID E. TRENTHAM, MD, GUEST EDITOR

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Predictors of disease activity severity and outcome have both been
reviewed. While data to date usually examine one predictor at a time,
it is likely that a combination of genetic factors, joint tenderness/
swelling, rheumatoid factor positivity, presence of erosions early in
disease, and gender can be combined to predict disease activity/
severity. The precise "mix" and contribution of these factors, how-
ever, still needs to be determined. Longer term functional outcome
can best be predicted by accounting for baseline functional disabil-
ity, disease severity per se, and psychologic variables; again, the
precise variables of most importance still need some research. Fi-
nally, mortality appears to be predicted by functional factors, but
medications, social factors, and age also contribute to mortality.

Arthroscopy can serve as an important diagnostic role in the arthritis
patient, with indications for its performance based on specific clini-
cal scenarios that commonly involve issues of diagnostic uncertainty
or therapeutic frustration. Office based "needle" arthroscopy, per-
formed mainly on the knee, is capable of identifying abnormalities
of cartilage, menisci, and synovium and directing further therapeutic
endeavors. Research applications of diagnostic arthroscopy include

identification of patient subgroups for therapeutic trials, and serial visual and biopsy assessment of treatment effects on the pathologic features of target tissue as identified by the arthroscope.

Magnetic resonance (MR) imaging has revolutionized the assessment of pathology involving the musculoskeletal system. The soft tissue contrast, superb resolution, multiplanar acquisition potential, and the ability to monitor physiologic processes combine the best features of other imaging modalities. The sensitivity and specificity of MR imaging for a wide range of disease processes matches or supersedes conventional radiology, nuclear medicine, and clinical examination. This article provides a brief overview of the use of MR imaging for some of the more common clinical situations confronting the rheumatologist.

In early Lyme disease, the presence of erythema migrans, the geographic location of the patient, and the time of year are critical in assessing the likelihood of Lyme disease. In late Lyme disease, the issues are different. The clinician must recognize characteristic, objective disease manifestations and avoid the diagnosis in individuals with "chronic fatigue" alone. Serologic testing is useful because few, if any, individuals with late Lyme disease will be seronegative.

Genetically determined HLA antigens have been associated with many rheumatic diseases. Currently, the clinical usefulness of HLA typing for diagnosis in most of these disorders is limited. Typing for HLA-B27 may be used as both sensitive and specific tests for atypical spondyloarthropathies, especially in children, in most populations. DNA oligotyping for HLA-DRB1 alleles associated with adult RA may prove to be useful in predicting disease severity. Similarly, the classification of the various subsets of juvenile rheumatoid arthritis may be aided by determining HLA-DRB1 and DPB1 alleles. HLA typing in other HLA-associated rheumatic diseases, including the connective tissue diseases and systemic vasculitides, are discussed.

The criteria for the diagnosis of Sjögren's syndrome remain controversial, leading to confusion in clinical practice and in research pub-

lications. Dryness of eyes and mouth are nonspecific symptoms that are influenced by sex, age, medications, anxiety, and systemic autoimmune diseases. Even using stringent criteria for Sjögren's syndrome, the group of patients currently designated as Sjögren's syndrome at the author's clinic is heterogeneous in their clinical and laboratory features. The key issue will be to establish a diagnostic criteria that identifies a subgroup(s) of patients that share a common etiopathogenesis and response to treatment.

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Diagnosis of Lumbar Spinal Stenosis 471
Jeffrey N. Katz, Marianne Dalgas, Gerold Stucki,
and Stephen J. Lipson

Lumbar spinal stenosis is a clinical-anatomic syndrome. Radiographic evidence of cauda equina compression is necessary but not sufficient to establish the diagnosis. Patients must have a clinical syndrome consisting of back and lower extremity discomfort exacerbated by lumbar extension or relieved by flexion, or evidence of lower extremity neurologic deficits. Symptomatic lumbar spinal stenosis may arise from a variety of specific etiologies and frequently coexists with other pain syndromes.

DIAGNOSIS OF LUMBAR SPINAL STENOSIS

Jeffrey N. Katz, MD, MS, Marianne Dalgas, MD, Gerold Stucki, MD, and Stephen J. Lipson, MD

Degenerative lumbar spinal stenosis is a common source of back and lower extremity pain in the elderly and leads to substantial functional disability. Approximately one in every 1000 individuals over the age of 65 undergoes laminectomy annually,[7] primarily for degenerative lumbar spinal stenosis. The annual inpatient expense for surgically treated patients with spinal stenosis approaches $1 billion, and considerable additional expense is incurred by outpatient treatment and indirect costs. Although its incidence is not known, lumbar spinal stenosis has been diagnosed increasingly in the last two decades because of ready availability of computed tomography and magnetic resonance imaging, as well as heightened physician awareness.

Accurate diagnosis of spinal stenosis is critical to appropriate selection of therapy. Unfortunately, critical literature on the diagnosis of spinal stenosis is sparse and limited by the absence of criteria for the integrated clinical-anatomic syndrome of spinal stenosis. This review begins by proposing diagnostic criteria for the clinical-anatomic syndrome of spinal stenosis and then evaluates existing literature on the role of the history, physi-

From the Department of Rheumatology and Immunology (JNK) and the Robert B. Brigham Multipurpose Arthritis and Musculoskeletal Disease Center (JNK, GS), Brigham and Women's Hospital; the Department of Orthopedic Surgery, Beth Israel Hospital (SJL); and Harvard Medical School (JNK, SJL), Boston, Massachusetts; and the Department of Rheumatology, Community Hospital, Holbaek, Denmark (MD)

cal examination, and diagnostic imaging studies in the diagnosis of spinal stenosis.

DEFINITION

Discussion of the diagnosis of spinal stenosis must begin with an acceptable case definition. Classic myelographic studies by Verbiest,[33,34] Arnoldi et al,[1] and others defined lumbar spinal stenosis as diminution in the diameter of the bony spinal canal. This concept proved valuable in understanding congenital spinal stenosis, which is characterized by diminished cross-sectional area of the bony spinal canal.[33,34] Studies by Schonstrom, Spengler, and others,[25] utilizing computed tomography, demonstrated that in degenerative spinal stenosis the bony dimensions of the spinal canal are generally normal, but the cross-sectional area of the cauda equina is reduced. Both osseous and soft tissues, including osteophytes, ligamentum flavum, and intervertebral disk, compress the cauda equina.

These definitions are limited because they are entirely anatomic. Extensive literature on the herniated lumbar disk syndrome teaches that in the absence of compatible clinical findings, anatomic studies are frequently misleading.[4,29] Imaging results must be viewed in the context of a clinical syndrome. In fact, abnormal findings on imaging studies are commonly seen in asymptomatic individuals.[36] Thus, an integrated clinical-anatomic definition of spinal stenosis is needed. We propose (Table 1) that the criteria for degenerative lumbar spinal stenosis consist of both (1) anatomic evidence (on imaging studies) of cauda equina or nerve root encroachment by a combination of soft-tissue and osseous lesions, and (2) the clinical syndrome of neurogenic claudication or evidence of chronic nerve root compression or both.

Neurogenic claudication is the characteristic clinical syndrome observed in patients with spinal stenosis. The most literal and narrow definition of "neurogenic claudication" consists of calf pain with walking as is observed in patients with vascular claudication. Up to 15% of patients with classic symptomatic spinal stenosis, however, have discomfort restricted to the thighs, without radiation to the calves or feet.[8] The critical feature of neurogenic claudication is exacerbation of pain with lumbar extension and

Table 1. DEFINITION OF THE CLINICAL-ANATOMIC SYNDROME OF SPINAL STENOSIS

Patients must meet both of the following criteria:
  (1) Evidence on spine imaging studies (computed tomography, magnetic resonance imaging, or myelography) of impingement of the cauda equina and/or exiting nerve roots; and
  (2) One or both of the following clinical syndromes:
     (a) Neurogenic claudication: Calf and/or thigh discomfort (pain, numbness, or paresthesia) that is exacerbated by lumbar extension (including prolonged standing and walking), and relieved with lumbar flexion.
     (b) Chronic nerve root compression: Radicular or polyradicular abnormalities in lower extremity reflexes, muscle strengh, or sensation (temperature, light touch, or vibration), that cannot be explained by the presence of a generalized peripheral neuropathy.

relief with lumbar flexion. These characteristic lumbar mechanics reflect the increase in cross-sectional area of the spinal canal with lumbar flexion, and reduction in area with extension. Thus, lumbar extension increases nerve root compression, producing an acute neural ischemic syndrome, manifest as neurogenic claudication. In addition to pain, patients frequently experience numbness, paresthesia, and weakness that may be exacerbated by lumbar extension. Thus, we propose a more inclusive definition of neurogenic claudication: thigh or calf discomfort (pain, numbness, paresthesia, or weakness) or both that is exacerbated by lumbar extension (including prolonged standing and walking) and relieved with flexion.

The other clinical syndrome observed in association with spinal stenosis is chronic nerve root compression. This generally occurs relatively late in the course of disease and in some patients does not occur at all. The syndrome of chronic nerve root compression is manifest subjectively by weakness and balance disturbance and objectively by abnormalities in reflexes, lower extremity muscle strength, and sensibility. Although the weakness, balance disturbance, and physical findings may worsen with lumbar extension,[28] they also persist in flexion, reflecting chronic nerve injury. We have observed that fewer than 5% of patients have anatomic evidence of lumbar spinal stenosis in association with chronic nerve root compression, but in the absence of neurogenic claudication.

DIAGNOSIS OF THE CLINICAL-ANATOMIC SYNDROME OF SPINAL STENOSIS: LITERATURE REVIEW

The value of specific historical and physical examination findings in the diagnosis of spinal stenosis has not been studied. The lack of critical research in this area is probably due to the relatively recent, widespread recognition of spinal stenosis and the lack of a standard conceptual and operational definition of symptomatic spinal stenosis. Existing data on diagnostic tests in spinal stenosis are derived primarily from case series, mostly surgical. This introduces serious limitations and biases.[24] Surgical patients tend to have more advanced disease and thus more striking symptoms and physical examination abnormalities than do patients managed nonoperatively. Thus, the sensitivity of clinical findings may be overestimated. Also, surgical studies provide no information on patients without spinal stenosis, and therefore they do not allow estimation of specificity. Critical study is needed on the diagnostic value of historical and physical examination findings in patients with and without the clinical-anatomic syndrome of spinal stenosis.

In the absence of such a study, we will present data on clinical findings in spinal stenosis from three sources: review of major case series,* a recent meta-analysis of surgical cases by Turner et al,[32] and previously unpub-

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* References 5, 6, 8, 9, 11, 13, 15, 16, 18, 23, 26, 28, 30, 31, 33, 34

Table 2. PRESENCE OF SELECT HISTORICAL FINDINGS IN PATIENTS WITH SPINAL STENOSIS

Histoical Findings	Literature Survey*	Meta-analysis	Brigham
Female	23%–70%	44%	62%
Mean age (years)	52–67	54	69
Duration of symptoms (months)	6–360	51	29
Subjective weakness	43%–72%	44%	87%
Neurogenic claudication	38%–100%	62%	96%
Discomfort with standing	65%–94%	NA	NA
Low back pain	65%–100%	87%	97%
Leg pain	68%–100%	84%	93%
Bilateral leg symptoms	41%–55%	NA	NA
Numbness or paresthesia	26%–100%	51%	76%
Bowel or bladder symptoms	4%–29%	11%	NA
Balance disturbance	NA	NA	66%

* Ranges exclude the lowest and highest reported means (unless these values are reported by more than one study)

NA = not available

lished data from our own prospective series[17] of operative and nonoperative patients with degenerative lumbar spinal stenosis.

HISTORICAL FINDINGS

Table 2 summarizes the frequency of historical findings in the three sources of data. The age distribution depends on the spectrum of patients studied. Patients with congenital spinal stenosis generally become symptomatic in the third through fifth decades, whereas patients with degenerative stenosis become symptomatic in the sixth through eighth decades. Thus, in both our retrospective[18] and prospective series[17] of patients with degenerative spinal stenosis, the mean age was 69, as compared with 54 in the meta-analysis,[32] which included spinal stenosis of all origins. Most series report a slight male predominance, 56% in the meta-analysis, whereas our current[17] and previous[18] series had over 60% female. The reasons for this discrepancy are not clear but may relate to the higher risk of degenerative spondylolisthesis—present in one third of our patients—in females.[10] Duration of symptoms is highly variable in these series; patients typically endure insidious progression of central low back pain for years before the onset of neurogenic claudication.

The most useful historical clue to the diagnosis of spinal stenosis is neurogenic claudication. As noted in the preceding discussion of the definition of spinal stenosis, the phenomenon of neurogenic claudication has variable manifestations. Common complaints include calf or thigh pain (or both) with lumbar extension, pain with prolonged standing or walking, which is relieved with sitting, and a tendency to walk with a stooped posture and to lean on carts while shopping. Patients who cannot walk five blocks may be able to bicycle 5 miles. These complaints share the essential lumbar

mechanics of neurogenic claudication—increased lower extremity symptoms with lumbar extension and relief with flexion.

Most patients have a combination of low back pain and leg pain.[8] The low back pain is generally not due to cauda equina compression, but rather to facet arthropathy, disk degeneration, and involvement of ligaments and other soft tissues. The central low back pain is important to distinguish from neurogenic claudication because it is less responsive to surgery and should be treated with a comprehensive rehabilitative approach. Leg pain is often bilateral and distributed more diffusely than the discrete dermatomal localization of sciatic pain observed in herniated disk syndromes. The more diffuse distribution is due to involvement of multiple rather than single nerve roots, the ischemic rather than acute inflammatory origin of the radiculopathy, and distal referral of nonradicular, mechanical pain. Thus, in patients with symptomatic lumbar spinal stenosis, it is common to observe three or four different syndromes including neurogenic claudication, chronic nerve root compression, central low back pain, and nonradicular referred lower extremity pain.

About three fourths of patients with the clinical-anatomic syndrome of spinal stenosis report numbness and paresthesia, and over 80% report subjective weakness. Balance disturbance is rarely commented upon by other authors, but it is reported by two thirds of patients in our prospective series. The balance disturbance is correlated with deficits in vibration sensibility, suggesting it arises from compression of large proprioceptive fibers, producing a pseudocerebrellar syndrome.[19] Bowel and bladder symptoms suggest cauda equina syndrome and are reported in 11% of patients summarized by Turner et al[32] but in none in our series. Bowel and bladder dysfunction is a late finding and will likely become less frequent as early recognition of this disorder increases. Weakness, paresthesia, balance disturbance, and bladder dysfunction are common in the elderly and arise from a variety of conditions. These symptoms should be attributed to spinal stenosis cautiously.

PHYSICAL FINDINGS

Table 3 presents the frequency of specific physical examination findings in the cohorts noted previously. Range of motion of the lumbar spine is seldom reported but provides useful diagnostic information. Flexion is generally somewhat limited because of advanced degenerative changes, but it typically does not reproduce radicular pain. Lumbar extension, on the other hand, is typically quite limited, with flexion contractures noted in advanced cases. Furthermore, extension commonly reproduces back and lower extremity pain. In fact, provocation of lower extremity discomfort on lumbar extension can be viewed as the physical examination equivalent of neurogenic claudication. Often, patients must maintain lumbar extension for 30 to 60 seconds to reproduce radicular pain. Although the diagnostic value of this maneuver has not been evaluated critically, we believe prolonged lum-

Table 3. PRESENCE OF SELECT PHYSICAL FINDINGS IN PATIENTS WITH SPINAL STENOSIS

Physical Finding	Literature Review	Meta-analysis	Brigham
Limited lumbar extension	66%–100%	NA	NA
Straight leg raise	10%–90%	49%	NA
Absent knee reflexes	18%–50%	24%	24%
Absent ankle reflexes	50%–68%	58%	71%
Muscle weakness	18%–52%	51%	29%*
Sensory deficit	32%–58%	52%	12%†
Vibratory sensation	NA	NA	88%‡

* Extensor hallucis longus
† Medial foot pinprick—diminished or absent
‡ Absent or diminished at medial foot
NA = not available

bar extension is the most informative physical examination finding in documenting the clinical syndrome of spinal stenosis.

The straight leg raising maneuver is positive in about half of reported patients but is also detected in about 90% of patients with documented disk herniations.[2] The presence of a positive straight-leg-raising test in patients with spinal stenosis may reflect concomitant disk herniation, hamstring tightness, or radiation of central low back pain.

Absence of deep tendon reflexes is also common but nonspecific. We have found that reflexes are diminished less commonly at the knee than the ankle in patients with spinal stenosis, and that decreased knee reflexes correlate better with functional loss. Objective evidence of weakness in the lower extremity varies widely among series, with a mean of 51% in the meta-analysis of Turner et al.[32] The extensor hallicus longus, which is innervated solely by L5, is most commonly involved. Sensory deficits also vary widely, occurring in 58% of patients in the meta-analysis. Vibratory sensation is not reported upon in most series, but it was diminished or absent in the medial foot in over 80% of patients in our series and was associated with self-reported disability, suggesting that vibratory sensation may be the most sensitive neurologic test for spinal stenosis.[19] Older patients with spinal stenosis frequently have coexistent medical conditions, such as diabetes mellitus, which are associated with peripheral neuropathies; thus, these physical findings are nonspecific and should be interpreted carefully. Peripheral pulses are diminished in vascular claudication but may also be reduced fortuitously in patients with neurogenic claudication, because peripheral vascular disease is prevalent in the elderly.

Importantly, historical and physical findings with greatest diagnostic value may not correlate with pain or functional loss. Patients in our series are bothered most by back pain, leg pain, and reduced walking capacity, in that order. Numbness, tingling, and weakness are useful diagnostically, because they indicate nerve root involvement, but they are less bothersome to patients. Thus, once the diagnosis is established, therapy should be di-

rected at the manifestations that bother patients most, not necessarily those that were critical in making the diagnosis.

RADIOLOGIC FINDINGS

Lateral plain films may reveal short pedicles in congenital and developmental stenosis. In general, however, plain radiographs do not reveal the cauda equina and therefore provide no direct evidence of lumbar spinal stenosis. The plain films, however, do show the extent of disk degeneration and facet joint arthropathy, two key elements of the pathogenesis of degenerative lumbar spinal stenosis. They also reveal degenerative spondylolisthesis and scoliosis, which may develop as a consequence of the degenerative process and attendant instability and lead to cauda equina compression.

Direct radiologic evidence of spinal stenosis is obtained from imaging studies including myelography, computed tomography (CT), and magnetic resonance (MR) imaging. Myelography, generally regarded as the gold standard for imaging the cauda equina,[28] provides excellent visualization of dural sac compression. The technique is invasive, however, and may be complicated by reactions to contrast material, headaches, and rarely, arachnoiditis and infection.

CT is noninvasive, it is relatively inexpensive, and it provides excellent bony detail. Because degenerative lumbar spinal stenosis often occurs at several lumbar segments, the spine should be scanned from L2 to S1. Also, because the degenerative changes occur at the level of the disk space and facet joints, but not the pedicles and vertebral body, the scans should focus primarily upon the disk space and facet level. CT images of the soft tissues such as the cauda equina and ligamentum flavum are of variable quality and utility. The resolution of soft tissues may be enhanced by the addition of intrathecal contrast material. In fact, the postmyelogram CT is perhaps the most informative study for spinal stenosis, but it involves the expense and risks of myelography.

Schonstrom and others[25] showed that the bony diameter of the spinal canal on CT scan correlated poorly with surgically confirmed diagnoses of degenerative spinal stenosis, whereas the cross-sectional area of the dural sac correlates extremely well. Of 24 patients with surgically confirmed spinal stenosis studied by these authors with myelography and CT, all 24 had stenosis detected on preoperative myelograms, 22 had CT scan evidence of reduced diameter of the dural sac, and only 5 of 24 had reduced cross-sectional area of the bony canal. This observation supports the argument that soft tissues (disk, ligamentum flavum, posterior longitudinal ligament) and not the bony elements of the spinal canal encroach upon the cauda equina in degenerative spinal stenosis.

The CT scan is sensitive for the anatomic diagnosis of spinal stenosis, but somewhat nonspecific. Weisel et al[36] found that 50% of asymptomatic patients over 40 years of age had significant abnormalities on CT scan,

including disk herniation in 27%, spinal stenosis in 3.4%, and facet arthropathy in 10%. The frequency of asymptomatic CT evidence of anatomic spinal stenosis would likely be higher in an older population. The poor specificity of imaging studies reinforces the argument that radiographic findings are meaningful diagnostically only if accompanied by the clinical syndrome of neurogenic claudication or chronic nerve root compression.

MR imaging is used increasingly for imaging of the lumbar spine. MR imaging is noninvasive, involves no radiation exposure, and provides excellent sagittal and transverse views of osseous and soft tissues. At present, it is about two to three times more expensive than CT. MR image resolution can be further enhanced with intravenous contrast materials such as gadolinium. In the lumbar spine, gadolinium enhancement appears useful in distinguishing epidural fibrosis from recurrent disk herniation in patients with prior back surgery.[12] In most patients evaluated for spinal stenosis with MR imaging, however, contrast enhancement is not necessary.

Modic et al[21] compared the sensitivity of MR imaging, CT, and myelography in the diagnosis of spinal stenosis. The authors studied 62 intervertebral levels in 48 patients who had surgical confirmation of pathology. Disk herniation was suspected in 32 levels, and cauda equina compression, or spinal stenosis, in 30. The surgical findings served as the gold standard. In the diagnosis of spinal stenosis, MR imaging had sensitivity of 0.77, CT 0.79, myelography 0.54, and the combination of CT and myelography 0.84. In the diagnosis of herniated disk, MR imaging had sensitivity of 0.88, CT 0.79, myelography 0.81, and the combination of CT and myelography 0.93. These data suggest that each imaging modality is more sensitive for detection of disk herniation than for cauda equina compression. Also, CT-myelography appears to be the most sensitive approach, followed by MR imaging, then CT alone, and finally myelography alone. The comparatively poor sensitivity of myelography is surprising. A limitation of this study is that MR imaging was performed with a 0.6 Tesla scanner, weak by today's standards. A stronger field strength improves resolution and might have resulted in greater sensitivity. Also, the study was performed in patients with surgically confirmed disease, precluding assessment of specificity. Sensitivity might be lower in a group with less severe symptoms. As with CT scanning, the excellent sensitivity of MR imaging must be balanced against its modest specificity. MR images demonstrate degenerative changes in virtually every older patient[35] and therefore must be interpreted with caution.

NEUROPHYSIOLOGIC TESTING

Electromyography and nerve conduction testing can be extremely useful in differentiating nerve root compression from other causes of neurologic dysfunction such as metabolic polyneuropathy. Electrophysiologic testing can also be used to distinguish patients with ongoing active denervation from patients with well-established, inactive, chronic nerve compression. Classically, spinal stenosis produces polyradicular, frequently bi-

lateral electromyographic abnormalities. Motor conduction velocity may also be diminished in the legs of patients with spinal stenosis, suggesting that nerve conduction at the radicular level may result in axonal changes.[14] In addition, dermatomal somatosensory evoked potentials appear to be sensitive indicators of spinal stenosis.[27] The degree of dural sac stenosis noted at myelography correlates with the severity of electromyographic changes. For example, in one study,[14] electromyographic abnormalities were noted in all patients with complete block on myelogram and neurogenic claudication, in 94% of patients with neurogenic claudication and partial myelographic block, and in 54% with neurogenic claudication and no obstruction in flow of myelographic contrast. Thus, a normal electromyogram does not rule out spinal stenosis, especially in patients with radiographically mild disease. Electrophysiologic tests are particularly useful in the subset of patients with spinal stenosis and clinical evidence of chronic nerve root compression but not neurogenic claudication. Testing generally shows polyradiculopathy. We believe that, with a few exceptions, electrophysiologic testing is not necessary in the routine evaluation of patients with suspected spinal stenosis.

DIFFERENTIAL DIAGNOSIS AND COEXISTING PAIN SYNDROMES

In approaching a patient suspected of having the clinical-anatomic syndrome of spinal stenosis, the clinician should entertain three important considerations. First, other conditions that may be confused with spinal stenosis should be excluded. Second, if the clinical-anatomic diagnosis of spinal stenosis is made, the etiology of stenosis should be identified. Finally, spinal stenosis may be associated with other pain syndromes besides neurogenic claudication and chronic nerve compression. These syndromes should be identified, and their impact upon the patient's disability should be assessed.

Exclude Other Conditions

Neurogenic claudication may produce discomfort over a wide distribution including the back; buttocks; trochanteric region; anterior, lateral, and posterior thighs; knees, calves; and feet. Because of its broad distribution, spinal stenosis may be confused with a variety of musculoskeletal disorders. Piriformis syndrome can cause buttock pain and sciatica but may be distinguished by local tenderness and exacerbation of pain with resisted internal rotation of the hip. Trochanteric bursitis produces pain in the trochanteric region with radiation into the buttock and lateral thigh. Trochanteric bursitis, however, is virtually always accompanied by local tenderness at the trochanteric bursa, allowing its differentiation from neurogenic claudica-

tion. Trochanteric bursitis may represent radiation of spinal pain or may arise secondarily from gait abnormalities in patients with hip arthritis or other problems.

Hip osteoarthritis produces groin and anterior thigh pain that can suggest neurogenic claudication, but pain is reproduced by hip movement. Knee osteoarthritis and pes anserine bursitis are also common in the elderly but have local knee findings. Finally, as mentioned previously, vascular claudication can mimic neurogenic claudication. Lumbar mechanics provide the most useful means of distinguishing spinal stenosis from these other pain syndromes: Pain that is provoked by lumbar extension and relieved by flexion is likely due to symptomatic spinal stenosis. Of course, referred lumbar pain, trochanteric bursitis, and osteoarthritis of the hip and knee are all common in the elderly, and they may coexist with neurogenic claudication.

The chronic lower extremity nerve compressive syndrome that frequently accompanies long-standing spinal stenosis also has a wide differential. Peripheral mononeuropathies resulting from vasculitis or trauma can produce a similar picture but can be distinguished on the basis of history and other clinical features. Generalized neuropathy from diabetes, alcohol use, amyloidosis, and other conditions produces a more uniform stocking pattern of neurologic deficits. In unusual circumstances, electrophysiologic testing is required to differentiate among these possibilities.

Identification of the Etiology of Spinal Stenosis

Once the clinical-anatomic diagnosis of spinal stenosis is established, the precise etiology for stenosis should be defined. Neurogenic claudication and chronic nerve compression may arise from any process causing cauda equina compression. The most common is degenerative spinal stenosis; however, the differential is broad.[22] The onset of neurogenic claudication in the second through fourth decade raises the possibility of congenital spinal stenosis, characterized by reduced bony dimensions of the bony spinal canal. Early onset of symptoms may also arise from spondylolisthesis, frequently associated with spondylolysis. Patients with prior lumbar fusion may develop postfusion spinal stenosis adjacent to the fusion owing to accelerated disk disease and facet joint degeneration. Epidural metastases and epidural abscesses may both present with neurogenic claudication. Systemic features, coexisting tumor, other sites of bacterial infection, and nighttime pain unrelieved by simple changes in position all point to tumor or infection rather than a degenerative etiology. In patients with hypercortisol states, epidural lipomatosis may produce neurogenic claudication and chronic nerve compressive syndromes.[20] Fluorosis and diffuse idiopathic skeletal hyperostosis rarely cause cauda equina compression in advanced stages. Paget's disease may cause expansion of the lumbar vertebrae, result-

ing in neurogenic claudication. Acromegaly and osteoporotic vertebral fracture are also in the differential diagnosis.

Identification of Coexisting Pain Syndromes

Patients with degenerative lumbar spinal stenosis have extensive degenerative disease of the spine, which gives rise to a variety of pain syndromes. Patients may have central lumbar pain due to involvement of pain-sensitive structures including tendon, ligament, muscle, and disk. They may also have myofascial pain syndromes characterized by muscular pain and tight "trigger" points. Involvement of lumbar structures may be referred to the lower extremities, resembling radicular pain. Disk protrusion may also occur in these patients, producing true sciatica. Osteoporotic compression fractures are frequent in this age group. The challenge to the clinician is to identify these distinct entities and determine how much each bothers the patient and compromises functional status. The critical clinical point is that neurogenic claudication is often responsive to epidural corticosteroid injection and surgical decompression, whereas central back pain and myofascial pain are less responsive and should be managed with a comprehensive rehabilitative approach.[17]

CONCLUSIONS

Symptomatic lumbar spinal stenosis is a clinical-anatomic syndrome. Anatomic evidence of cauda equina compression is necessary but by no means sufficient to establish the diagnosis. The critical clinical feature is neurogenic claudication, which if defined broadly as we have, occurs in virtually all patients. Chronic nerve compression occurs in advanced cases. Neurogenic claudication is established by a history of calf or thigh discomfort or both that is exacerbated by lumbar extension and relieved by flexion. The physical examination usually reveals pain with lumbar extension. The neurologic examination in the lower extremities may be entirely normal, but it often reveals evidence of chronic nerve compression. Reduction in vibratory sensibility appears to be the most sensitive physical finding. The clinician evaluating older patients with back and lower extremity pain must first differentiate spinal stenosis from other lower extremity pain syndromes on the basis of the history and physical examination. Second, if a clinical-anatomic diagnosis of spinal stenosis is established, the clinician must determine the cause of the anatomic stenosis, ruling out unusual metabolic, infectious, congenital, and neoplastic processes. Finally, the clinician must estimate the extent to which neurogenic claudication and chronic nerve compression contribute to the patient's disability, and how much is due to coexisting spinal, other musculoskeletal, and medical comorbidities. Therapy should be targeted at the symptoms that bother the patient most, which in many instances will not be neurogenic claudication.

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Address reprint requests to

Jeffrey N. Katz, MD, MS
Department of Rheumatology and Immunology
Brigham and Women's Hospital
75 Francis Street
Boston, MA 02115