Conferences and Reviews

Lumbar Spinal Stenosis

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Lumbar spinal stenosis, the result of congenital and degenerative constriction of the neural canal and foramina leading to lumbosacral nerve root or cauda equina compression, is a common cause of disability in middle-aged and elderly patients. Advanced neuroradiologic imaging techniques have improved our ability to localize the site of nerve root entrapment in patients presenting with neurogenic claudication or painful radiculopathy. Although conservative medical management may be successful initially, surgical decompression by wide laminectomy or an intralaminar approach should be done in patients with serious or progressive pain or neurologic dysfunction. Because the early diagnosis and treatment of lumbar spinal stenosis may prevent intractable pain and the permanent neurologic sequelae of chronic nerve root entrapment, all physicians should be aware of the different neurologic presentations and the treatment options for patients with spinal stenosis.

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Lumbar spinal stenosis is defined as narrowing of the neural canal and foramina to an extent that results in compression of the lumbosacral nerve roots or cauda equina. The embryologic and developmental factors that determine the anatomic configuration and dimensions of the spinal canal are poorly understood. As the articulating facets and supportive ligaments undergo degenerative hypertrophy and osteophytic ridges form, they encroach on neural structures passing through or exiting the spinal canal. Although the prevalence of spinal stenosis is difficult to determine because no population-based studies have been done, the disorder is relatively common and has been recognized more frequently since the introduction of advanced radiographic imaging techniques. A thorough understanding of the anatomic relationship of structures within the spinal canal is necessary to facilitate accurate diagnosis and appropriate surgical intervention.

Etiology and Pathogenesis

The amount of space available for nerve roots in the lumbar spinal canal and foramina is determined both by spinal developmental variations and by articular degenerative responses. The morphogenesis of the lumbar vertebrae begins after the seventh week of gestation, when a pair of chondrification centers forms in each vertebral arch (Figure 1).[1,2] Ossification and bony union of the centrum with its neural arch define the dimensions of the neural canal and foramina and are not completed until several years after birth.[3] If the paired dorsolateral ossification centers of one or more lumbar vertebrae stop growing prematurely, the lumbar spinal canal may become stenotic even in an otherwise normally developed person.[4]

The vertebrae of patients with congenital lumbar stenosis are characterized by short and thick pedicles. The transverse interpediculate diameter may also be smaller than the normal distance of 25 mm.[5] The vertically oriented laminae are short and thick, bringing the facets almost to the midline.[6] The facets are enlarged and bulbous and may nearly touch the spinous processes. Although primary lumbar stenosis is the sole cause of entrapment radiculopathy in only 2% of patients with sciatica at presentation, it is a contributing factor associated with superimposed disc herniation or spondylosis in 30% of these patients.[7]

Stenotic lesions can occur in any of three anatomic sites: the central canal, bordered by the vertebral bodies, discs, and articular processes; the subarticular canal or lateral recess, which extends from the thecal sac to the pedicle; and the intervertebral foramen or nerve root canal lying below the pedicle.[8,9] Stenosis in the central canal usually results from concomitant ligamentous hypertrophy and disc protrusion, especially in patients with a congenitally small spinal canal. Stenosis in the lateral recess is caused by degenerative ligamentous and superior facet hypertrophy. In the foramen, stenosis may result from osteophyte formation under the pars interarticularis where the ligamentum flavum is attached, or from bursal tissue hypertrophy at a spondylolytic defect. The clinical presentation of stenosis is similar at each of these anatomic sites, and the site can be determined only with radiographic imaging.[9-12]

Several important variations of lumbar stenosis are unilateral stenosis caused by a short pedicle, asymmetric stenosis caused by unequal posterior hypertrophy, discontinuous or skipped levels of stenosis, a normal canal in the midline with constricted lateral recesses, a narrow canal in the midline with normal lateral recesses, and sacral stenosis.[13-15] These variations are difficult to diagnose with plain films or standard myelography, but computed tomography (CT) with intrathecal contrast and magnetic resonance imaging (MRI) allow them to be more easily recognized.[16]

Lumbar spinal stenosis can be either congenital or acquired. Congenital or developmental stenosis was described originally in children by Sarpyener and later in adults by Verbiest and is most frequently caused by an idiopathic reduction in the normal spinal canal dimensions or by achondroplastic dwarfism.[17,18] Verbiest performed decompressive laminectomy in middle-aged men who had radicular symptoms in the lower extremities that were aggravated by walking or standing. In all of these patients, the anteroposterior

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ABBREVIATIONS USED IN TEXT

CT = computed tomography
MRI = magnetic resonance imaging

diameter of the lumbar spinal canal was 12 mm or less, much smaller than the 15- to 23-mm diameter in normal cadaver skeletons.[19] Reports of familial developmental lumbar stenosis suggest that the dimensions of the spinal canal, at least in some patients, are regulated by genetic factors.[20,21]

Developmental stenosis of the entire spinal canal is a well-known feature of achondroplastic dwarfism.[22] The vertebrae of patients with this disorder have short pedicles and decreased interpediculate distances, resulting in both anteroposterior and lateral stenosis of the canal. As adults, these patients often have syndromes involving progressive compression of the spinal cord or cauda equina.[4] Minor trauma and disc extrusion in the thoracic or lumbar spine may cause the sudden onset of flaccid or spastic paraplegia. The vertebral deformity has been explained by premature cessation of growth of the paired vertebral body and dorsolateral neural arch ossification centers.[23]

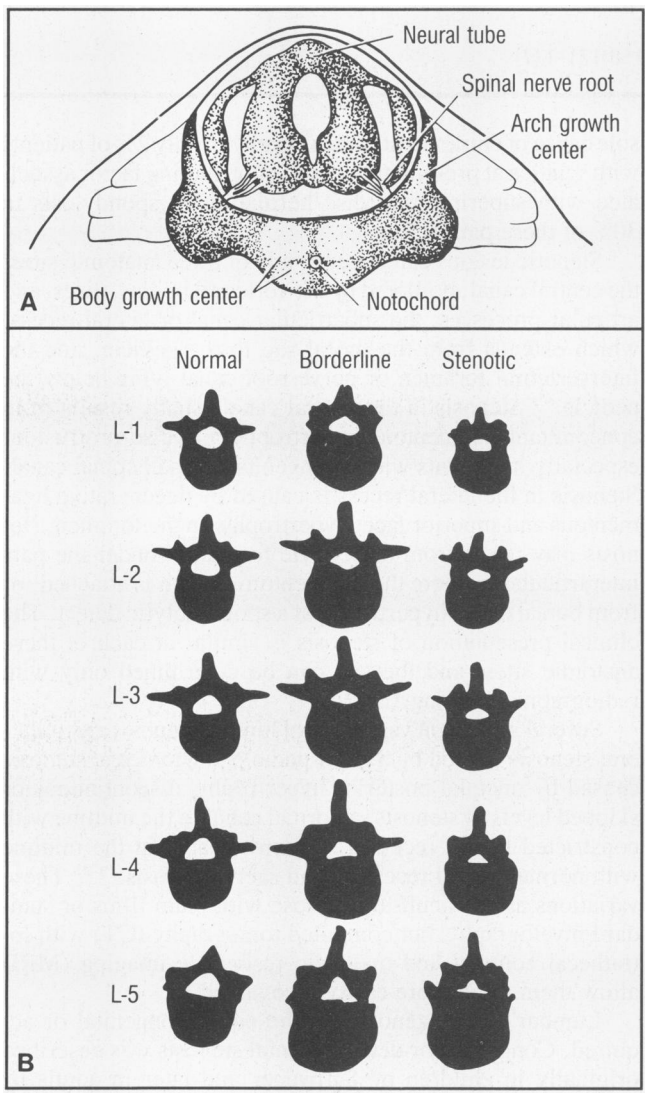

Figure 1.—A, The diagram of lumbar vertebral embryogenesis illustrates paired vertebral body chondrification sites, which unite at 9 weeks' gestation to form a single growth center. Premature cessation of vertical growth in the paired pedicle growth centers could explain developmental stenosis caused by a failure of the neural arch to achieve adequate dimensions (from Weinstein[1]). **B,** Photo tracings of three sets of cadaver lumbar vertebrae show developmental variations in the size and shape of the neural canal, with **A** being normal, **B** borderline, and **C** stenotic. In the stenotic examples, the typical trefoil shape with severe narrowing of the lateral recesses is seen at L-5 (adapted from Epstein et al[2]).

Acquired lumbar stenosis is caused primarily by degenerative disease of the spine. Spontaneous or posttraumatic tears, degeneration, fibrosis, and collapse of the disc lead to the failure of mechanical function and then to subperiosteal osteogenesis at sites where the herniating annulus is attached to the junction of cartilaginous plates with the epiphyseal ring of the vertebral body lip.[24,25] As patients grow older, biomechanical failure of the lumbar discs increases the stress on facet joints and ligamentous attachments, resulting in hypertrophy of the facets and ligaments.[26] Bone also thickens at the pedicles, laminae, and facets as a result of chronic stress or repeated minor trauma.[27-31] A loss of disc height further narrows the neural foramina as the superior facets migrate rostrally. Collapse of the disc reduces the interlaminar space, narrowing the spinal canal as the dorsal edge of the upper lamina overrides the rostral edge of the lower lamina.[27] Posterior protrusion of the annulus and disc contributes to stenosis and nerve root entrapment in the midline, lateral recesses, and foramina.[32]

Degenerative spondylosis is usually associated with hyperplasia, fibrosis, and cartilaginous metaplasia of the annulus, posterior longitudinal ligament, and ligamentum flavum. This disease may increase the thickness of the ligamentum flavum from its normal of 2 to 5 mm[33] to as much as 5 to 10 mm in patients with spondylosis.[1] In our experience, hypertrophied ligamentum flavum causes the most notable compression of the neural elements, and it may be the major cause of lumbar stenosis in some patients.[13,25,34,35] Histologic examination of surgical specimens of ligamentum flavum reveals fragmentation, degeneration, and disappearance of elastic fibers that do not regenerate after injury.[36] These specimens also show increased vascularity, resulting in excessive collagen deposition and fibrosis[1]; evidence of old hemorrhage and residual granulation tissue are occasionally found.[37]

Both spondylolysis (a defect in the pars interarticularis) and spondylolisthesis (the relative anterior or posterior displacement of one vertebral body on an adjacent vertebra) can contribute to spinal stenosis. In degenerative spondylolisthesis, a frequent complication of advanced lumbar spondylosis,[13] complete myelographic block can occur as a result of associated posterior ligamentous and facet hypertrophy in patients with only 3 to 4 mm of subluxation. Because the neural arch is intact, the term "pseudospondylolisthesis" has been used to describe this condition.[27] Disc protrusion and anterior and superior migration of the superior facet may completely obliterate the foramina.

Although acquired spinal stenosis is usually caused by spondylosis and spondylolisthesis, a number of rarer causes exist. Vertebral deformities, osteophyte formation, and soft tissue calcification are frequent sequelae of disc-space infection, osteomyelitis, and Pott's disease (tuberculosis of the spine).[38] Moreover, calcification or ossification of the posterior longitudinal ligament or ligamentum flavum and the development of intraspinal synovial cysts may contribute to spinal stenosis.

Other causes of acquired stenosis are surgical procedures

(laminectomy or spinal fusion),[39] trauma,[40] and bony overgrowth. This overgrowth may be associated with Paget's disease (osteitis deformans),[41] ankylosing spondylitis,[42,43] diffuse idiopathic skeletal hyperostosis,[44] or rheumatoid arthritis.[45] Finally, various metabolic and endocrine abnormalities, such as acromegaly,[46] pseudogout, hypoparathyroidism, or renal osteodystrophy,[47] have been associated with lumbar stenosis (Table 1). Radiculopathy may also be caused by epidural lipomatosis, which is seen predominantly in patients with endogenous obesity who are taking steroids or who have Cushing's syndrome.[48]

Clinical Features

Patients with lumbar spinal stenosis may present with a variety of signs and symptoms. One such symptom is neurogenic claudication, characterized by poorly localized leg pain, sometimes associated with numbness and weakness. This pain is exacerbated by walking or standing and is relieved by postures that reduce the degree of lumbar lordosis.[38,49,50] Patients also usually report a progressive reduction in the distance they can walk before symptoms are noticed. Pain is usually felt before numbness and weakness and has a radicular pattern, unlike the "cramping calf" pain described by patients with vascular claudication. The symptoms typically resolve after two to five minutes of rest in a leaning or sitting position. The pain may be bilateral and poorly localized in stenosis of the central canal, or it may be well localized to one or more nerve roots when lateral recess stenosis predominates.[38,50-52] Claudication is usually bilateral, but asymmetric canal stenosis sometimes causes unilateral symptoms. All of these symptoms may be preceded by chronic low back pain, and they often follow a gradually progressive and prolonged course. The disorder is far more common in men than in women and requires surgical treatment more often in men.[5,50,53]

TABLE 1.—*Major Causes of Spinal Stenosis*

Congenital-developmental stenosis
Idiopathic
Achondroplasia or hypochondroplasia
Hypophosphatemic vitamin D-resistant rickets
Morquio's mucopolysaccharidosis
Spinal dysraphism
Acquired stenosis
Degenerative
Spondylosis
Spondylolisthesis
Scoliosis
Ossification of the posterior longitudinal ligament
Ossification of the ligamentum flavum
Intraspinal synovial cysts
Postoperative
Laminectomy
Fusion
Posttraumatic
Metabolic or endocrine
Epidural lipomatosis—Cushing's disease
Osteoporosis
Acromegaly
Pseudogout—calcium pyrophosphate dihydrate deposition disease
Renal osteodystrophy
Hypoparathyroidism
Other
Paget's disease of bone
Rheumatoid arthritis
Ankylosing spondylitis
Diffuse idiopathic skeletal hyperostosis

The pathogenesis of nerve root dysfunction in patients with neurogenic claudication, characterized by poorly localized leg pain exacerbated with walking and relieved by rest or back flexion, is unclear. Pain and weakness may result from intermittent ischemia caused by compression of the radicular microcirculation during periods of increased axonal activity.[54-58] During tourniquet constriction, nerve fibers first become hyperexcitable and then fire volleys of spontaneous afferent impulses.[56] Later, reversible blockade of conduction by sensory or motor fibers may occur, possibly accounting for the sequence of pain and paresthesias followed by weakness seen in patients with neurogenic claudication.

Positional radiculopathy is a more common initial symptom of lumbar stenosis than is neurogenic claudication and is manifest as radiating leg pain, paresthesias, numbness, or weakness occurring when the patient stands erect or bends backward. Lumbar spine extension induces radiculopathy by relaxing and buckling the ligamentum flavum; it also increases disc protrusion.[59] Extension also moves the laminae closer together and projects the superior facets farther upward, further narrowing the spinal canal and foramina by as much as 60% compared with their diameter during lumbar flexion.

The cauda equina syndrome, characterized by intermittent or progressive symptoms of urinary or fecal incontinence, impotence, and sensory loss in a saddle distribution, is an infrequent presentation of lumbar spinal stenosis. In patients with stenosis, symptoms may be related to standing or walking, whereas in patients with acute disc herniation, symptoms occur abruptly. The symptoms may not be recognized by the patients, making rectal examination an invaluable part of the neurologic examination.

Demonstrable sensory or motor deficits are often absent in patients with lumbar stenosis. Because spinal stenosis has a predilection for the midlumbar spine,[38,60] quadriceps weakness or atrophy and a depressed or absent knee jerk may be the only physical findings. Some patients describe the onset of major weakness such as a footdrop only after prolonged ambulation. The results of the straight-leg–raising test are usually normal, perhaps because this sciatic stretch maneuver also flexes the lumbar spine, thus relieving compression of the nerve roots caused by stenosis.[38,59]

Diagnosis

When a patient's history and the results of the physical examination suggest lumbar stenosis, the diagnosis may be confirmed by other tests. Because of the expense and invasiveness of radiographic imaging techniques, however, further diagnostic workup of patients with suspected lumbar spinal stenosis should be reserved for those with progressive neurologic deterioration, intractable pain, or cauda equina syndrome, or for those in whom surgical intervention is anticipated.

Myelography followed by thin-section CT has been the radiographic imaging modality most often used to demonstrate neural encroachment.[61-64] Defects apparent on myelography range from an "hourglass" configuration to complete obstruction of the subarachnoid space.[65] Computed

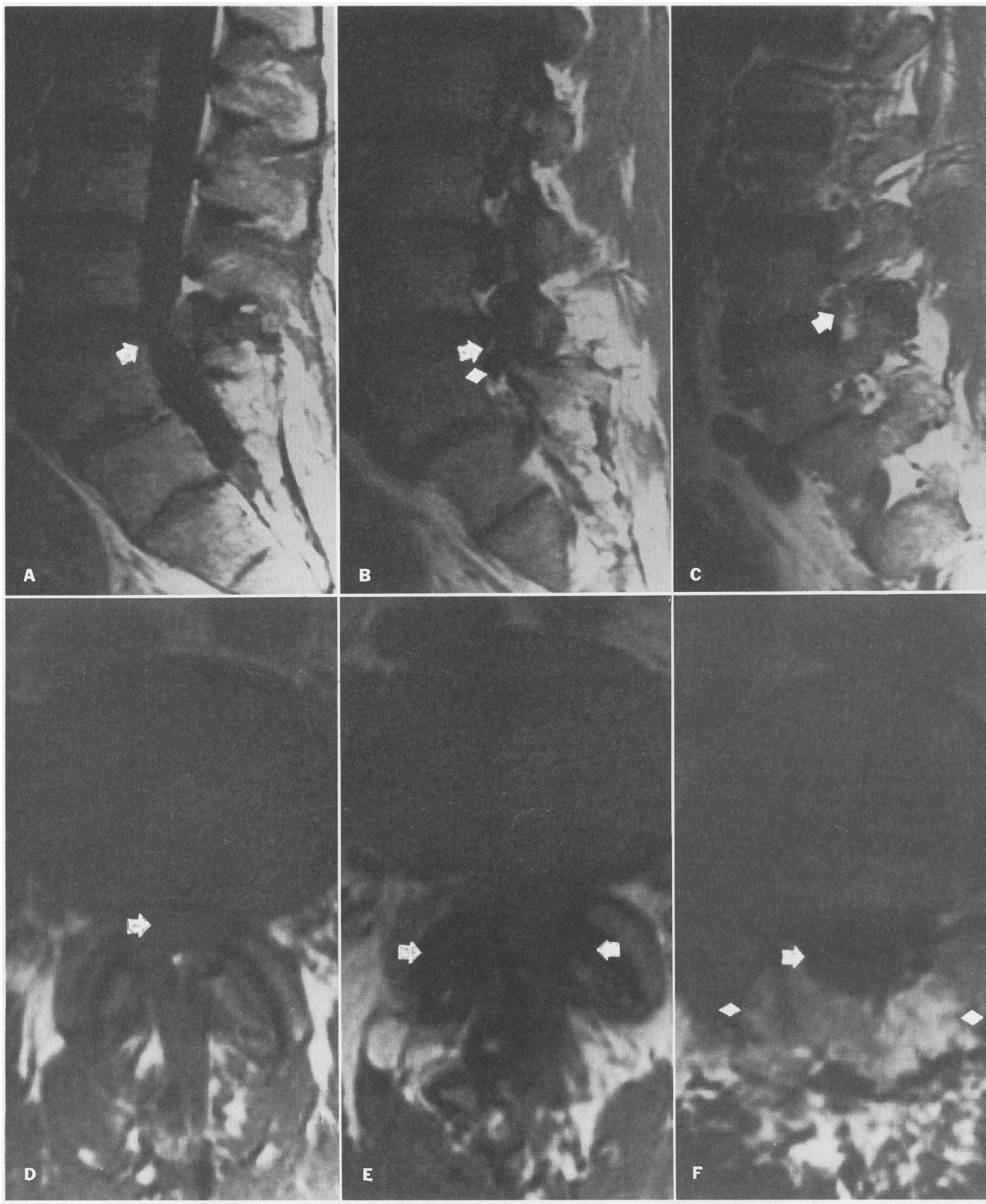

Figure 2.—Magnetic resonance images are shown of a 72-year-old man with intractable bilateral leg pain in the L-5 distribution, initiated and aggravated by standing erect and walking. He had undergone an L5-S1 discectomy and fusion 5 years previously. The results of the neurologic examination and the straight-leg-raising test were normal. The midline image in the sagittal plane, **A,** shows dorsolateral encroachment with stenosis of the neural canal (**arrow**) that is most severe at L3-4. The left lateral sagittal image, **B,** shows L3-4 lateral recess stenosis (**arrow**) caused by disc bulge anteriorly and by hypertrophy of the superior facet posteriorly. The nerve root (**diamond**) is compressed. **C,** Only minimal foraminal narrowing is seen (**arrow**) in the far lateral image. **D,** Axial images confirm almost complete obliteration of the subarachnoid space and constriction of the thecal sac (**arrow**) at L3-4 by dorsal facet, ligamentous hypertrophy, and a ventral discogenic osteophyte. **E,** Moderately severe stenosis seen at L4-5 is primarily a result of hypertrophy of the facets (**arrows**). **F,** The rounded triangular thecal sac configuration (**arrow**) of a relatively normal canal is seen beneath the fusion mass (**diamonds**) at L-5. This patient returned to unrestricted work and recreational activities including playing golf and tennis within 2 months after an L-4 laminectomy with bilateral foraminotomies at L3-4 and L4-5.

tomography allows the configuration and dimensions of the canal to be determined and the contribution of disc displacement, facet and soft tissue hypertrophy, and spondylolysis or occult fractures to be recognized.[63,66,67] Sagittal reconstructions can provide a clear picture of the neural foramina and the relationships of the exiting nerve roots to the pedicles and facets. Computed tomography using a water-soluble intrathecal contrast agent is more sensitive than myelography, which often fails to distinguish posterior from anterior sources of nerve root compression and does not demonstrate the configuration of the lateral recesses and foramina.[16,68-70] Contrast-enhanced CT is especially helpful in patients with persistent postoperative symptoms.[67,71,72]

The features of spinal stenosis apparent on CT are broad-based disc bulging with or without a vacuum disc phenomenon, a congenitally small neural canal, hypertrophic facet disease or facet cyst formation, and ligament hypertrophy.[60] On images taken through the level of the disc space, decreased sublaminar epidural fat is a good indication of spinal stenosis; on images taken through the level of the pedicle, however, this may be a normal finding.[73-75] Some fat may be seen in the midline beneath the spinous process even in patients with severe sublaminar and subarticular stenosis.

Magnetic resonance imaging, when available, is an alternative to CT in the radiographic diagnosis of spinal stenosis. Its advantages include the lack of radiation, the avoidance of invasive intrathecal contrast administration, and the capability of direct multiplanar image construction. In addition to its greater sensitivity in detecting disc disease, MRI also allows visualization of the entire lumbosacral and lower thoracic spine, as well as the conus medullaris and cauda equina (Figure 2).[76] Although MRI provides better soft tissue detail than CT,[77,78] particularly in the lateral recess, it does not provide the detail of bony structures available with CT.[75] Nevertheless, we agree that high-quality MRI should now be considered the most effective and least invasive modality for spinal imaging.[76-78]

The results of electrodiagnostic studies are more often abnormal than are those of the neurologic examination.[51,79] In nearly 80% of patients with proven stenosis, electromyography demonstrates radiculopathy.[61] Abnormalities are often bilateral and involve several root segments. Electromyography is particularly useful in stenosis of the lateral recesses, which may be overlooked if only routine or poor-quality spinal radiographic studies are available. Normal neurophysiologic studies, however, do not rule out spinal stenosis because radiculopathy may be intermittent and evident only after standing and walking.

Plain radiographs may show degenerative changes that contribute to canal stenosis, such as spondylosis and spondylolysis, or underlying bone diseases, such as Paget's disease or dwarfism.[38,80-82] The canal diameter can be measured from anteroposterior and lateral lumbar spinal radiographs, but these measurements are less important than the configuration of the spinal canal as visualized with axial scanning at various levels.[61,83] Although plain radiography is an inexpensive initial diagnostic study, the diagnosis of lumbar spinal stenosis cannot be made on the basis of plain films alone.

Treatment

Conservative, nonoperative therapy includes nonsteroidal anti-inflammatory medications and exercise programs to strengthen abdominal musculature and reduce lumbar lordosis.[38,84] Bed rest, opiate analgesics, muscle relaxants, and lumbosacral bracing may help during periods of increased pain.[47] In our experience, some patients may benefit from a brief course of systemic steroid therapy or from a series of epidural steroid injections, administered under fluoroscopic control, below the level of stenosis. No large controlled studies have been published, however, that address the efficacy of steroid therapy in the treatment of lumbar stenosis. The use of an exercise bicycle, which allows aerobic activity and strengthens the back and leg musculature while the lumbar spine is flexed forward, may be better tolerated than walking. These treatments have been shown to diminish symptoms in some patients with lateral recess stenosis[70] and should be tried in all patients who do not have significant neurologic deficits such as weakness or sphincter disturbances. Medical management may be the only alternative in elderly patients with substantial systemic illness in whom surgical treatment is contraindicated.[85] Although these nonoperative measures may provide temporary relief, they usually do not prevent the recurrence of painful radiculopathy when normal activity is resumed.

Surgical intervention should be considered when medical management fails to relieve intolerable pain during activities of daily living. It should also be considered in patients with initial signs and symptoms of cauda equina dysfunction or progressive neurologic deterioration. Many surgical approaches have been described.[6,13,84,86-92] The goal of each of these approaches is to decompress the thecal sac and exiting nerve roots while minimizing the possibility of subsequent spinal instability.

The degree of surgical decompression must be individually tailored to the patient on the basis of clinical, radiologic, and surgical observations. Wide laminectomy with bilateral foraminotomies is the standard decompressive operation (Figure 3, left).[38,91] When this technique is used, postoperative spinal instability occurs in about 2% of patients older than 35 years,[93] although rates as high as 15% have been reported.[94] Fusion of the spine is therefore generally not necessary except in patients with degenerative spondylolisthesis in whom the likelihood of instability after decompression is high.[84,86,95] Internal fixation with instrumentation and fusion may also be necessary in patients with scoliosis.[96] Arthrodesis may be done at a later date if necessary to relieve intractable back pain or to correct postoperative instability.

Patients with focal midline or lateral recess stenosis may be treated with a limited interlaminar exposure and bilateral laminotomies to remove hypertrophic bone, ligamentous tissue, and osteophytes (Figure 3, right).[91] Decompression of the lateral recesses requires resection of the medial third of both inferior and superior facets and additional undercutting of hypertrophic facet bone and ligament or joint capsule. Complete facetectomy is rarely required, but it may be done if necessary for complete decompression. In elderly patients with spondylosis, complete facetectomy is usually well tolerated if it is unilateral. Discectomy and the removal of ventral marginal osteophytes are rarely desirable in patients with lumbar spondylosis. Unless discs are extruded, they should not be removed because discectomy may increase the possibility of spinal instability after wide decompressive laminectomy and medial facetectomy.

Among patients treated surgically, 80% to 85% obtain good or excellent results, defined as the relief of symptoms and a return to the premorbid level of activity.[5,6,38,51,97-100]

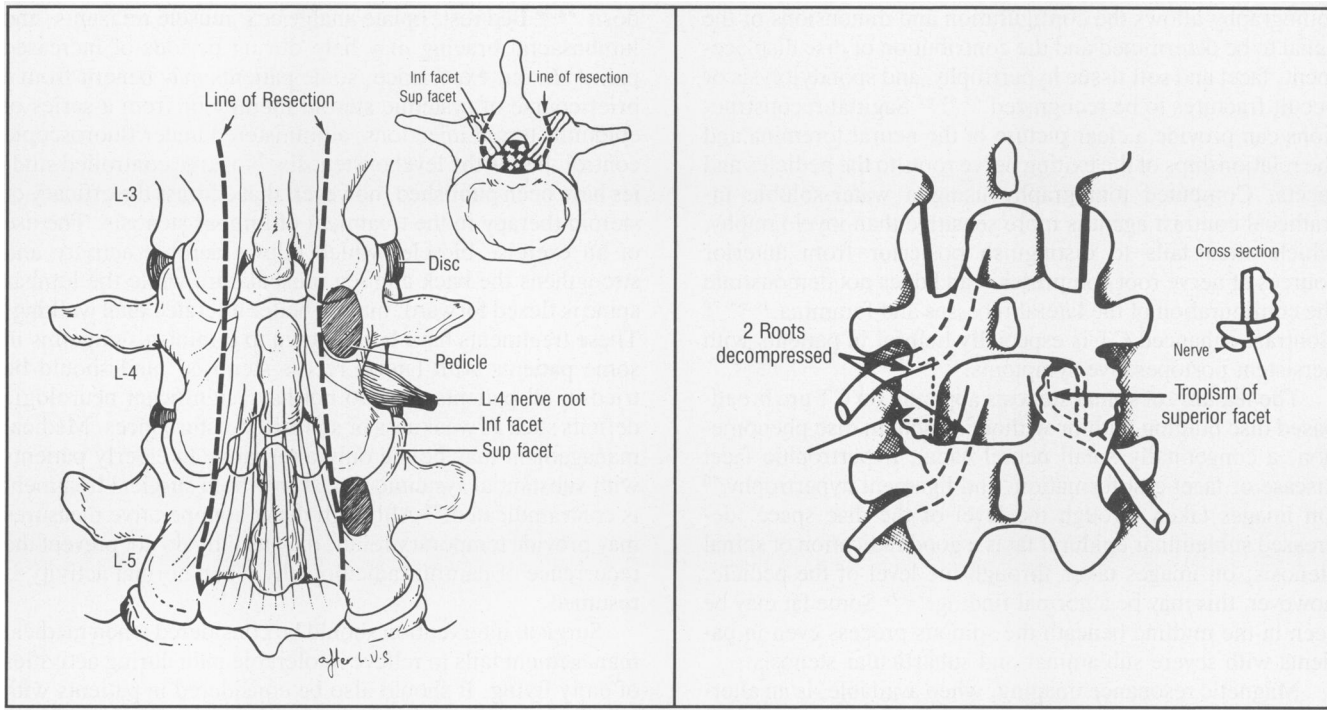

Figure 3.—Left, The diagram of decompressive laminectomy for lumbar spinal stenosis illustrates the extent of lamina and medial facet resection **(broken line)** required for adequate decompression of the neural canal and its lateral recesses. The axial view **(inset)** shows that the line of resection is placed parallel to the medial surface of the pedicle while sparing the lateral half of the facet. Hypertrophic laminae and facets are debrided and thinned with the power drill before completing removal with fine curettes and rongeurs. Inf = inferior, Sup = superior **Right,** The diagram shows the interlaminar approach for decompression of focal lumbar stenosis with bilateral laminotomies rather than a complete laminectomy. Using a microsurgical technique, the entire ligamentum flavum is exposed and removed along with the overlying facet **(arrows)** and laminar bone. Both the lateral recesses and foramina can be satisfactorily decompressed, leaving the lamina, spinous processes, and midline ligaments intact (from Lin[91]).

Many patients, however, are unable to return to work that requires strenuous lifting or prolonged walking or sitting. Because of the likelihood that irreversible nerve root damage has already occurred, full recovery should not be anticipated in patients with muscle atrophy and sphincter dysfunction. Mechanical low back pain is the symptom least often relieved after surgical decompression because this procedure alleviates nerve root entrapment but does not alter the underlying degenerative osteoarthritis that causes it.[39] The role of surgical fusion for internal fixation, with or without instrumentation and as either a primary or second-stage procedure, remains controversial.

Summary

Lumbar spinal stenosis, a common cause of painful and disabling radiculopathy initiated or aggravated by standing and walking, may not be diagnosed until irreversible neurologic damage has occurred. In elderly patients with spondylosis and symptoms of stenosis, MRI or CT with intrathecal contrast should be used to localize the site of nerve root entrapment. Although nonoperative treatment may be successful initially, surgical decompression should be done in patients with serious or progressive pain or neurologic dysfunction. Wide laminectomy or interlaminar decompression alleviates symptoms and improves physical capacity in most patients without interfering with spinal stability. Early diagnosis and treatment of spinal stenosis may prevent intractable pain and the permanent neurologic sequelae of chronic nerve root entrapment.

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