

# Lumbar Spinal Stenosis

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## ABSTRACT

Lumbar spinal stenosis refers to a diversity of conditions that decrease the total area of the spinal canal, lateral recesses, or neural foramina. Lumbar stenosis is a common disorder that may be present in isolation, with or without associated disk bulge or herniation, or can be associated with degenerative spondylolisthesis or scoliosis. Symptomatic lumbar spinal stenosis is characterized by neurogenic claudication and/or lumbar or sacral radiculopathy. Sixty percent to 85% of properly selected patients have a satisfactory symptomatic improvement with surgical treatment.

**KEYWORDS:** Lumbar stenosis, neurogenic claudication, spinal canal, spondylosis, radiculopathy

**Objectives:** On completion of this article the reader will understand the pathogenesis, clinical presentation, diagnosis, and management of lumbar spinal stenosis.

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Lumbar spinal stenosis is a developmental and/or acquired condition that results in the formation of a neural arch smaller than normal in diameter and triangular rather than round.<sup>1</sup> The normal dimensions of the neural canal and its pathologic variations have been established from anatomic, radiologic, and surgical observations (Table 1).<sup>2-4</sup> In patients with lumbar stenosis, the pedicles and laminae are short and thick; therefore, the facets extend downward almost to the floor of the canal and inward almost to the midline. The anteroposterior diameter of the canal may be less than 12 mm centrally and less than 4 mm in the lateral recesses and

foramina (Table 1). The transverse interpedicular diameter may be less than 25 mm. Disk protrusion anteriorly and massive hypertrophy of the ligamentum flavum posteriorly may encroach further upon the space available for nerve roots,<sup>5</sup> substantially reducing the circumferential area of the thecal sac and eliminating the cushion of cerebrospinal fluid (CSF) surrounding the cauda equina.<sup>6</sup>

Lumbar stenosis can be classified by its cause and by the location of the stenosis. It is generally divided into developmental or congenital and acquired types, although both mechanisms may be present in a given pa-

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**Table 1 Normal and Stenotic Lumbar Spinal Canal Dimensions**

Dimension	Neural Canal (mm)	Lateral Recess (mm)
Normal anteroposterior	15–25	3–5
Stenotic anteroposterior	5–10	1–2

tient. Anatomically, stenosis may be classified as central, lateral, or combined, based on radiographic measurements. Precise anatomic classification of the site of stenosis (central canal, lateral recess, and/or neural foramen) is perhaps the most practical approach and helps determine the nature and extent of surgical treatment.

### CONGENITAL AND DEVELOPMENTAL LUMBAR STENOSIS

Lumbar stenosis was first described by Sarpyener<sup>7</sup> in children born with dysraphic abnormalities, and the associated radicular syndrome and its treatment were described by Verbiest.<sup>8</sup> Embryogenesis of the lumbar vertebra begins after the seventh week of gestation. Two sets of paired chondrification centers form in the vertebral body and neural arch,<sup>9</sup> and fusion and ossification of growth centers in the vertebral body and pedicles are completed several years after birth.

Congenital stenosis is most often due to diffuse skeletal dysplasias, such as achondroplastic dwarfism<sup>10</sup> or spondyloepiphyseal dysplasia (Table 2). This congenital or developmental stenosis results from inadequate growth of the pedicles and lamina, resulting in formation of a disproportionately smaller neural canal. The vertebral neural canal reaches its adult level of cross-sectional area by 4 years of age.<sup>11</sup> Antenatal risk factors, such as prematurity and low birth weight, retard the growth of the lumbar spinal canal.<sup>12</sup>

Developmental lumbar stenosis requiring surgical treatment is three times more prevalent in females.<sup>13</sup> Although most cases are sporadic, familial cases of stenosis not associated with dwarfism have been reported,<sup>14</sup> which suggests that genetic factors may be involved. Relatively low prevalence rates were reported for lumbar stenosis before the era of computed tomography (CT) and magnetic resonance imaging (MRI).<sup>15</sup> Now that more accurate methods of diagnosis are available, prevalence rates need to be updated.

### ACQUIRED LUMBAR STENOSIS

Most commonly, lumbar spinal stenosis arises from degenerative changes in an aging spine, which may already possess an element of congenital or developmental stenosis. Thus, patients with congenitally narrow spinal canals are more likely to develop symptomatic spinal stenosis at an earlier age. The stenosis develops focally at the intervertebral junctions from a complex process of disk

degeneration, facet arthropathy, ligamentum flavum hypertrophy, spondylosis, and sometimes spondylolisthesis. The disorder most frequently affects L4–5, with L3–4, L5–S1, and L1–2 following in descending order.

The pathogenesis of spondylosis, which originates with aging and degeneration of the intervertebral disk, has been described in detail.<sup>5</sup> In the absence of a frank disk herniation, the aging lumbar disk undergoes a progressive collapse that involves fissuring and desiccation of the nucleus with a consequent buckling of the

**Table 2 Causes of Lumbar Spinal Stenosis**

- |  |
|--|
| A. Congenital/developmental  |
| 1. Idiopathic  |
| 2. Achondroplasia  |
| 3. Hypophosphatemic vitamin D-resistant rickets (spondyloepiphyseal dysplasia) |
| 4. Morquio's syndrome  |
| 5. Spinal dysraphism (lipoma, myelomeningocele)                                |
| B. Acquired  |
| 1. Degenerative  |
| a. Spondylosis   |
| b. Spondylolisthesis   |
| c. Scoliosis   |
| d. Ossification of the posterior longitudinal ligament                         |
| e. Ossification of the ligamentum flavum                                       |
| f. Intraspinous synovial cysts   |
| 2. Postoperative   |
| a. Laminectomy   |
| b. Fusion  |
| c. Fibrosis  |
| 3. Traumatic   |
| a. Laminectomy   |
| b. Kyphosis/scoliosis  |
| c. Burst fracture  |
| 4. Metabolic/endocrine   |
| a. Epidural lipomatosis (Cushing's disease)                                    |
| b. Osteoporosis  |
| c. Acromegaly  |
| d. Pseudogout (calcium pyrophosphate dihydrate deposition)                     |
| e. Renal osteodystrophy  |
| f. Hypoparathyroidism  |
| 5. Skeletal  |
| a. Paget's disease   |
| b. Ankylosing spondylitis  |
| c. Rheumatoid arthritis  |
| d. Diffuse idiopathic skeletal hyperostosis (DISH)                             |

dorsal annulus. This buckling, in turn, results in a broad-based bulge and the slow formation of diskogenic osteophytes. Hasegawa and colleagues<sup>16</sup> inversely correlated disk height with histologic evidence of nerve root compression in pathologic specimens. In addition, arthritic changes in intervertebral facet joints, perhaps exacerbated by increased stresses from the collapsing disk, are manifest by bone hypertrophy and synovial tissue overgrowth, which together encroach on the lateral central canal, lateral recesses, and dorsal neural foramina. Extreme degrees of facet arthropathy may be appreciated at surgery, with overgrown facets that obscure the lamina and abut the spinous processes.

Hypertrophy and buckling of the ligamentum flavum are another major cause of central canal stenosis that must be relieved at surgery. The ligamentum flavum tends to become hypertrophic at the enthesis<sup>17</sup> (the insertion point of the ligament to the lamina and facet), where degeneration of elastic fibers and proliferation of type II collagen occur. Subperiosteal osteoneogenesis is stimulated at these sites, which is responsible for osteophyte formation. The major component of neural canal and lateral recess stenosis is situated in the interlaminar space at the level of the hypertrophic ligamentum flavum.

A multitude of disease processes may contribute to the development of acquired lumbar stenosis (Table 2). In addition to the common degenerative changes already described, trauma (e.g., lumbar burst fracture); infections (e.g., diskitis, osteomyelitis, Pott's disease); miscellaneous skeletal conditions, such as Paget's disease, diffuse idiopathic skeletal hyperostosis, ankylosing spondylitis, and rheumatoid arthritis; bone or soft tissue infiltration by tumors such as prostate carcinoma; and metabolic and endocrine abnormalities such as acromegaly, pseudogout, hypoparathyroidism, renal osteodystrophy, and Cushing's disease with epidural lipomatosis may be associated with lumbar stenosis.<sup>18-20</sup> Postsurgical stenosis may occur after bone graft application to the lamina and facets during arthrodesis.

Degenerative spondylolisthesis (pseudospondylolisthesis) is a relatively common complication of lumbar spondylosis that results in anterior subluxation of the upper vertebra not associated with spondylolysis.<sup>21</sup> Although the malaligned intervertebral joint is usually stable, severe nerve root entrapment may develop, especially beneath the hypertrophic ligamentum flavum and anteriorly displaced inferior facet in the lateral recess at the level of or just below the affected disk.

## CLINICAL PRESENTATION

Patients suffering from lumbar spinal stenosis develop pain, paresthesias, numbness, and weakness in the back and legs due to entrapment of the lumbosacral nerve roots in the constricted neural canal and foramina.<sup>22</sup> This back and leg pain usually appears on standing, is

exacerbated by walking, and is relieved by rest in a flexed or seated position.<sup>13</sup> Symptom patterns and neurologic signs vary among patients and in the same patient. For example, the pain may be sharp and lancinating in a lower extremity or it may be dull, aching, and of gradual onset in the sacroiliac and posterolateral thigh areas.<sup>23</sup> In cases of central canal stenosis, the pain may be bilateral, although not necessarily symmetric; in cases in which the lateral recess is the most prominent site of nerve root entrapment, symptoms may resemble a unilateral monoradiculopathy.<sup>13,24-26</sup> Sensory and motor dysfunction, often in the same anatomic distribution as the radicular pain, may also be present.

Lumbar stenosis is usually a chronic condition, often beginning with many years of low back pain punctuated with acute but temporary episodes of incapacitating lumbago with or without sciatica. Initial or recurrent symptoms are associated in some cases with trauma or unusual physical stress. Often the history includes a satisfactory response to medical therapy after a previous diagnosis of acute sprain, disk herniation, or even fracture. Many patients with recurrent symptoms have already undergone discectomy or laminectomy and sometimes fusion as well. In fact, residual or recurrent stenosis is a frequent cause of relapse or persistence of radiculopathy after failed lumbar spinal decompression.<sup>27</sup>

The age at presentation varies inversely with the severity of the developmental component of the stenosis. Thus, in cases of dwarfism or severe primary stenosis, the acute onset of excruciating sciatica, sometimes associated with paralytic lumbosacral radiculopathy, may occur at 20 to 30 years of age. Acute paraplegia may be due to a relatively mild disk protrusion that obliterates an already constricted subarachnoid space. More common is presentation in the seventh, eighth, or even ninth decade with progressive loss of the capacity to stand and walk because of leg pain or paresthesias with or without mild motor and reflex deficit. In very elderly patients, the medical risks of surgical decompression for spondylotic lumbar radiculopathy may be justified to avoid lost independence and restriction to bed or wheelchair. The symptoms that help to distinguish lumbar stenosis from other causes of caudal radiculopathy are the neurogenic claudication, postural aggravation, and cauda equina syndromes.<sup>28</sup>

## Neurogenic Claudication

Neurogenic claudication consists of the progressive onset of radicular pain, paresthesias, numbness, and eventually in some cases weakness, initiated or worsened by walking. Numbness, fatigue, or weakness to the extent of footdrop or knee buckling may occur without or before the appearance of pain. Incapacitating sensory dysesthesias may also occur without pain or weakness. These symptoms are characteristically relieved within

**Table 3 Vascular versus Neurogenic Claudication**

Characteristic	Vascular (Iliofemoral Arterial Insufficiency)	Neurogenic (Lumbosacral Nerve Root Entrapment)
Mechanism	Ischemic	Ischemic and/or mechanical
Pain	Present (cramping)	Present or absent (radicular)
Pain location	Exercised muscles	Lumbosacral (sciatic)
Pain relieved by	Rest	Flexion posture or sitting
Motor deficit	Rare	Variable, exacerbated by walking
Pulses	Decreased	Normal
Arterial bruit	May be present	Absent
Aortography	Diagnostic	Normal
Lumbar MRI, CT, myelogram	Normal	Diagnostic

CT, computed tomography; MRI, magnetic resonance imaging.

minutes by sitting, but not by merely standing, as is often the case with claudication due to iliofemoral arterial stenosis (Table 3).<sup>29</sup>

In cases of neurogenic claudication, patients can often walk farther without rest during shopping while leaning forward than they can while standing erect to cross the parking lot. Bicycling, especially with the handlebars lowered to allow lumbar flexion, is often tolerated for much longer periods than walking.<sup>29</sup> Thus, for elderly patients with lumbar stenosis, an exercise bicycle may be the only way to engage in aerobic activity required for weight reduction or cardiac rehabilitation. The "bicycle test" of van Gelderen can be used to distinguish neurogenic from vascular claudication syndromes.<sup>30</sup> In this test, the patient is asked to exercise on a bicycle in various postures; with vascular claudication the pain will increase in relation to muscle ischemia, whereas with neurogenic claudication the pain will be dependent on posture (relieved by bending forward and worsened by lumbar extension despite equivalent exercise intensity).

#### PATHOPHYSIOLOGY OF NEUROGENIC CLAUDICATION

The pathogenesis of radiculopathy during neurogenic claudication remains speculative.<sup>29</sup> The nerve roots of the cauda equina derive their metabolic energy requirements from the CSF via diffusion and from the arterial circulation located on the surface of the nerve roots. These anatomic features have been postulated to place the roots at risk for ischemia in the setting of extrinsic compression as seen in lumbar stenosis. Experimental studies have shown that the compression of the cauda equina at pressures similar to those seen in lumbar stenosis can reduce blood flow and impulse conduction in the compressed roots.<sup>31</sup> During experimental limb tourniquet compression, the nerve fibers first become hyperexcitable and spontaneous volleys of afferent impulses are recorded; later, a reversible conduction block is observed.<sup>32</sup> Paresthesias, pain, and weakness are thought to result from relative nerve root ischemia in the presence of compression of the radicular microcirculation by

stenosis.<sup>33,34</sup> When an ambulation-induced increase in the metabolic rate of the nerve root cannot induce a compensatory increase in blood flow and substrate availability, the resulting microvascular insufficiency is thought to result in the ischemic radiculopathy of neurogenic claudication.<sup>33</sup>

Venous congestion is another proposed mechanism of intermittent postural radiculopathy in lumbar stenosis.<sup>11</sup> Myelography reveals congestion rather than ischemia in patients reporting neurogenic claudication.<sup>35</sup> Experiments indicate that compression of 10 mm Hg (a subarterial pressure) at a single level in a porcine cauda equina model has no significant effect, whereas the same compression applied at two levels decreases blood flow by 64%.<sup>36</sup> This decrease is thought to be caused by reduced venous return in the two-level compression model. Although the exact mechanism for the neurologic symptoms in patients with lumbar stenosis remains unresolved, it seems likely that mechanical compression of nerve roots results in arterial, venous, and CSF circulatory disturbances that impair neural transmission.

#### Postural Aggravation of Radiculopathy

The syndrome of postural aggravation of radiculopathy, or spinal claudication, is also characteristic of lumbar stenosis and consists of the immediate onset of back and leg pain upon standing in an erect or hyperextended position. The pain is rapidly relieved by sitting or bending forward. Thus, patients may have greater difficulty walking uphill or upstairs than downhill or downstairs.

Postural aggravation of radiculopathy is more readily understood than neurogenic claudication, as lumbar lordosis and extension are known to exacerbate stenosis of the neural canal and foramina.<sup>22</sup> In the extended position, disk protrusion may increase if spondylotic ossification has not already stabilized the annulus or fused the joint. The interlaminar space is reduced, and "shingling," or overlapping, of the rostral edge of the lower lamina under the caudal edge of the upper lamina oc-

curs. The ligamentum flavum relaxes and buckles inward, increasing its dorsolateral mass effect. Rostral and anterior migration of the superior facets further constricts the foramina, while caudal movement of the inferior facets may further narrow the lateral recesses. Thus, the postural aggravation of radiculopathy may be explained by intermittent direct neural compression, inhibiting nerve root function, as the patient moves into the erect or extended position.

Takahashi and colleagues<sup>37</sup> demonstrated a relationship between posture and epidural pressures in patients with spinal stenosis. In a study of 12 patients with lumbar stenosis compared with 7 age-matched controls, epidural pressures were higher in the stenosis patients and there was significant postural aggravation of epidural pressure on ambulation.

### Cauda Equina Syndrome

The cauda equina syndrome includes intermittent or progressive symptoms of urinary or fecal incontinence, impotence, and sensory loss in the “saddle” distribution. In lumbar stenosis, in contrast to acute disk extrusion or chronic tumor progression, the onset of sacral paresthesias, incontinence, and paraparesis may be related to standing and walking. Bladder control may be lost only if ambulation continues after the onset of pain, paresthesias, and weakness because rest is impossible. Severe compromise of the neural canal that causes sacral nerve root compression, usually at L3-4 or L4-5 in the midline, is responsible for the impairment of sphincter control and perineal sensation. Patients with cauda equina syndrome often have a complete myelographic block; however, the prognosis for recovery is good.<sup>13</sup>

### Other Signs and Symptoms

Neurologic deficits may be absent or intermittent in patients with lumbar stenosis, being temporarily detectable only after ambulation and not at rest. Involvement of the midlumbar segments is most common, as such sensory and motor deficits and reflex changes suggest L4 and L5 rather than S1 root involvement. However, correlation of observed deficits with the anatomic sites of root entrapment demonstrated radiographically does not always occur. For example, absence of the patellar reflex may result from L4 root compression due to canal stenosis at L2-3 or L3-4, disk protrusion and inferior facet or ligamentous osteophyte at L3-4, lateral recess stenosis at L3-4, or far lateral disk and superior facet encroachment in the foramen at L4-5. Often, bilateral stenosis is associated with only unilateral symptoms. Pain and deficit patterns that implicate only one root pair may be associated with radiographic abnormalities at more than one level. Finally, the stenosis may be unilateral and asymmetric or discontinuous with a skipped

level in between. A narrow central canal may be associated with a normal lateral recess and foramina, or the midline anteroposterior canal diameter may be normal and the lateral recess severely stenotic.

Surprisingly, the straight leg-raising test (Lasègue's sign) is most often negative in patients with lumbar stenosis.<sup>13</sup> This finding may help to differentiate spondylotic radiculopathy from that due to disk herniation, where nerve root displacement and inflammation may lower the threshold to stretch-induced, mechanical stimulation of the nerve root. In fact, the straight leg-raising maneuver may relieve the root compression by reducing the baseline lumbar lordosis that is present in the supine position or even by flexing the lower spine as the hip is flexed and the pelvis tilts backward.<sup>28</sup>

### DIAGNOSIS

The clinical history often distinguishes lumbar stenosis from other causes of radiculopathy. The level, side, and severity of involvement may be identified by the neurologic examination, including tests of anal sphincter tone and strength and sacral sensation. When a history of dysuria or incontinence is obtained, urodynamic studies of bladder function should be obtained. Electromyography and sensory conduction studies may also help to localize the site(s) of involvement.<sup>23</sup> Electrophysiologic studies may be normal at rest but abnormal after ambulation induces symptoms of neurogenic claudication.

Spinal radiographs are needed to demonstrate degenerative changes in the disks and facet joints as well as evidence of instability, traumatic deformity, spondylolisthesis, or scoliosis. Skeletal disorders such as osteoporosis, Paget's disease, and dwarfism may be identified. Lumbar stenosis may be diagnosed in some cases by measuring the anteroposterior diameter of the neural canal and foramina on lateral radiographs. Accurate localization of lumbar lesions also requires radiographic identification of anatomic variations in the number of lumbar vertebrae because of such anomalies as lumbarization of S1 or sacralization of L5.

Definitive diagnostic information is most readily obtained from lumbar spinal MR images and/or CT scans with sagittal reconstructions. These studies clearly show the size, shape, and anatomic relationships of spinal and neural elements and can demonstrate the relative contribution of developmental stenosis as well as disk, facet, and ligamentous elements of nerve root compression. MRI, when available, is the preferred initial scanning procedure.<sup>38</sup> The classic findings of a smoothly marginated *waist* or *hourglass* shape on sagittal images and a *trefoil*-shaped neural canal on axial images can be seen. In addition, the conus medullaris and cauda equina, as well as the individual nerve roots, can be seen. Direct multiplanar MR image construction provides more precise anatomic detail on sagittal images than CT.

In addition, MRI usually provides anatomic information sufficient for surgical planning. Axial MRI scan of central stenosis typically demonstrates a circumferentially narrowed canal. Hypertrophic bone appears as a dark region of low signal of T1-weighted and T2-weighted images, hypertrophic ligamentum flavum as an intermediate signal on T1-weighted and T2-weighted images, and loss of fat in the epidural space due to prolonged compression as a loss of high T1 signal. T2-weighted sagittal images are useful for their myelogram-like representation of the thecal sac. Lateral stenosis appears on axial and sagittal views as bone encroachment and loss of fat signal (best appreciated on T1-weighted images) surrounding the exiting nerve root.

CT remains the best choice for detailed imaging and analysis of osseous anatomy and pathology, especially in situations (such as degenerative spondylolisthesis) in which consideration is being given to surgical fusion in addition to decompression. Myelography is now rarely, if ever, required to diagnose lumbar stenosis. Approximately 55% of patients with neurogenic claudication have stenosis severe enough to cause a complete myelographic block of the subarachnoid space.<sup>39</sup> However, myelography does provide a remarkable demonstration of the pathophysiology of the postural effects that produce spinal claudication. A severe hourglass constriction or complete obstruction of the subarachnoid space seen in the prone (extended) position is usually partially or completely relieved in the flexed knee-chest or sitting position.

For postoperative studies, intravenous administration of a paramagnetic contrast agent is an invaluable aid in differentiating peridural scar from recurrent disk herniation or stenosis on MRI. When MRI is not available, a CT myelogram may be useful to demonstrate the subarachnoid space and neural elements.<sup>40</sup>

## TREATMENT

### Conservative Management

Conservative management begins with physical therapy, exercise (e.g., stationary bicycle), and analgesic, antispasmodic, and, when tolerated, nonsteroidal anti-inflammatory medications. Some patients may benefit from a brief (3- to 5-day) course of systemic corticosteroid administration or a series of three epidural steroid injections above the stenotic levels every 2 weeks. The aim is to reduce inflammation, which may contribute to nerve root compression by worsening edema. Use of a lumbar corset or flexion orthosis may relieve pain and facilitate ambulation. A walker may provide support for walking while the lower back remains flexed. Physiotherapy may help to alleviate pain, improve mobility and posture, and strengthen the abdominal and paraverte-

bral musculature. Although these nonsurgical therapies may provide temporary relief, painful radiculopathy may recur upon resumption of normal activity.

Such conservative measures result in overall clinical improvement, defined as decreased pain, increased ambulation tolerance, and reduced neurologic symptoms, in a majority of patients<sup>18,41</sup>; however, symptoms often recur. The disease is generally slowly progressive, but most results suggest that it is safe and often efficacious to attempt a trial of conservative (nonsurgical) management.<sup>42</sup>

### Surgical Therapy

Surgical treatment is indicated in radiographically confirmed cases of lumbar stenosis who have progressive neurologic deficit and/or severe neurogenic claudication that do not respond to conservative therapy. In patients without neurologic deficit, the most common indication for surgery is recurrent intolerable pain that restricts or prevents activities of daily living.

Selecting the most effective surgical procedure requires detailed analysis of the clinical and radiographic data.<sup>24</sup> The goals of surgery are to decompress fully the thecal sac and exiting nerve roots and to minimize the risk of resulting spinal instability. Decompression of the stenotic lumbar spine is a technically difficult operation. The initial muscle dissection for vertebral exposure demands meticulous hemostasis to avoid excessive blood loss. Intraoperative radiographic localization of the involved levels is essential. Removal of hypertrophic bone and osteophytes requires the use of heavy bone-cutting instruments and a power drill. The final ligament removal, dural exposure, and nerve root decompression are delicate dissections that require powerful illumination and microsurgical technique. The normal epidural fat layer may be atrophic; adhesions between ligamentum flavum and dura may increase the risk of dural laceration and nerve root injury. Repair within the narrow spinal canal may be difficult because the surgical exposure is also limited by increased venous bleeding that follows drainage of CSF. To avoid further injuring compressed nerve roots, care must be taken not to place large instruments, such as curettes or rongeurs, within the stenotic neural canal or foramina.

The standard surgical approach for the relief of lumbar stenosis is a wide laminectomy with bilateral foraminotomies, although many variations in technique have been proposed (Table 4). The number of levels to be operated on and the lateral extent of decompression are determined by inspecting and measuring the canal on MRI images or CT scans.

When relative stenosis is primarily due to spondylosis, interlaminar decompression by bilateral laminotomies and foraminotomies may be performed at one or

**Table 4 Operations for Lumbar Spinal Stenosis**

Operation	Reference
Complete laminectomy	J Bone Joint Surg Br 1954;36:320
Interlaminar decompression	Neurosurgery 1982;11:546
Unilateral decompression and contralateral fusion	J Neurosurg 1996;84:166
Trumpet laminectomy	J Spinal Disord 1993;6:232
Transforaminal arthroscopic decompression	J Neurosurg 1996;84:462
Expansive lumbar laminoplasty	Eur Spine J 1997;6:191

more levels to unroof the neural canal and lateral recesses without sacrificing the laminae, spinous processes, and supraspinous ligaments.<sup>43</sup> Although this procedure is more difficult and lengthy than the standard laminectomy approach, especially if performed at multiple levels, it has two major advantages: it preserves the neural arch, which protects the dura and nerve roots from epidural scarring; and it provides a site for paravertebral muscles and ligaments to reattach. This operation is thought to assist in maintaining spinal stability, especially in patients with degenerative spondylolisthesis or scoliosis. However, comparisons of interlaminar decompression and complete laminectomy have failed to demonstrate a difference in either postoperative outcome or spinal stability.<sup>44,45</sup>

In patients with lateral recess syndrome due to focal lumbar spondylosis and stenosis causing unilateral radiculopathy at a single level, simple interlaminar laminotomy and foraminotomy on the affected side should relieve symptoms. However, as in a full laminectomy procedure, substantial surfaces of the superior and inferior facets are required for an adequate decompression that completely unroofs the lateral recess and exposes the medial surface of the pedicle. Care must be exercised to preserve the pars interarticularis to prevent inadvertent inferior facetectomy or resultant spondylolysis. Undercutting techniques are used to remove the ligamentum flavum entirely and to resect the rostral portion of each superior facet that contributes to foraminal stenosis. A malleable probe or angled dissector is used to verify that the foraminotomies are adequate.

Diskectomy or removal of ventral osteophytes is rarely if ever required for adequate decompression of lumbar stenosis. Unless an extruded soft disk is encountered, the disk should be left intact to preserve the stability of the degenerated and partially resected intervertebral joint postoperatively. Because instability occurs after laminectomy in only 2 to 15% of patients over 35 years of age,<sup>46,47</sup> spinal fusion is usually not indicated as a part of the initial decompressive procedure. However, in patients with severe back pain or preoperative instability due to degenerative spondylolisthesis demonstrated on lateral flexion-extension radiographs and in advanced cases of rotoscoliosis requiring complete bilateral face-

tectomy, fusion and internal fixation may be indicated as part of the primary procedure.<sup>48</sup> Secondary fusion is rarely necessary in patients over 50 years of age.

## OUTCOME

After surgical treatment, good or excellent results and return to premorbid activity levels have been reported in 60 to 85% of cases.<sup>23,49-53</sup> The Maine Lumbar Spine Study prospectively compared surgical with medical treatment in 148 patients with lumbar stenosis and found at 1-year follow-up that 55% of the surgical group versus 28% of the medical group reported improvement in their symptoms.<sup>54</sup> In a more recent 4-year follow-up study in 119 of the same patients, 70% of the surgical group versus 52% of the medical group reported that their predominant symptom was improved. In addition, surgical treatment was associated with greater improvement in patient satisfaction (63%) than nonsurgical treatment (42%) at 4-year evaluation. Other studies have shown that surgical outcome correlates with the severity of preoperative stenosis.<sup>55</sup> Meta-analysis of 74 series on lumbar stenosis demonstrated good to excellent results in 64% of cases.<sup>56</sup> Longer-term (up to 8-year) outcome reviews suggest slowly decreasing levels of patient satisfaction,<sup>57</sup> which may be associated with bone regrowth in some cases.<sup>58</sup>

Despite adequate decompression, some patients may not be able to resume work requiring heavy physical labor. Patients with advanced chronic radicular neurologic deficits associated with muscle atrophy are unlikely to recover fully. Low back pain in the paravertebral area, which may be due to underlying degenerative arthritis rather than to an entrapment radiculopathy, is the least likely symptom to be relieved by decompressive surgery<sup>59,60</sup>; however, in many patients surgery eliminates the preoperative claudication-like low back and sacroiliac pain worsened by ambulation.

## CONCLUSION

Lumbar stenosis is an important cause of painful and incapacitating radiculopathy that has been diagnosed more frequently as the population has aged and as improved spinal imaging studies have become available.

Surgical decompression is indicated when back and leg pain initiated and exacerbated by standing and walking becomes disabling or intolerable or when progressive neurologic deficit develops. The results of wide laminectomy or more limited interlaminar lateral recess decompression are gratifying when pain is relieved and normal ambulation and activity are restored.

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