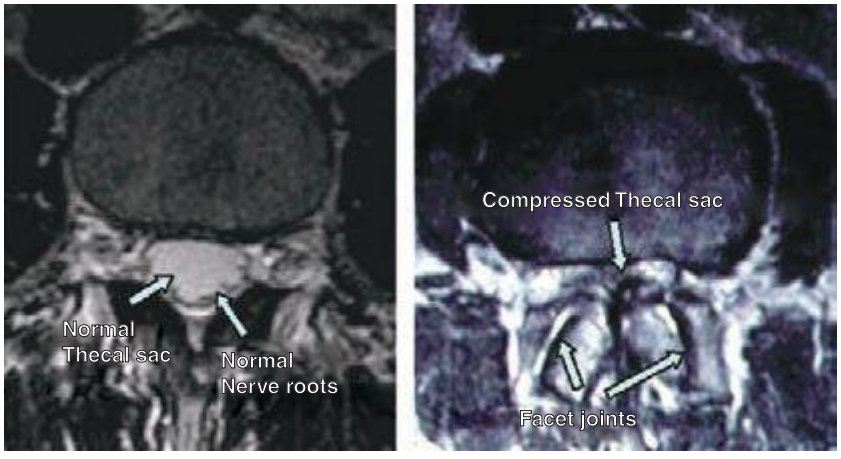

FIGURE 22-5 **Axial T2-weighted images of the lumbar spine. *A.*** The image shows a normal thecal sac within the lumbar spinal canal. The thecal sac is bright. The lumbar roots are dark punctuate dots in the posterior thecal sac with the patient supine. ***B.*** The thecal sac is not well visualized due to severe lumbar spinal canal stenosis, partially the result of hypertrophic facet joints.

LSS by itself is frequently asymptomatic, and the correlation between the severity of symptoms and degree of stenosis of the spinal canal is variable. LSS can be acquired (75%), congenital, or both. Congenital forms (achondroplasia, idiopathic) are characterized by short, thick pedicles that produce both spinal canal and lateral recess stenosis. Acquired factors that contribute to spinal stenosis include degenerative diseases (spondylosis, spondylolisthesis, scoliosis), trauma, spine surgery, metabolic or endocrine disorders (epidural lipomatosis, osteoporosis, acromegaly, renal osteodystrophy, hypoparathyroidism), and Paget's disease. MRI provides the best definition of the abnormal anatomy (Fig. 22-5).

Conservative treatment of symptomatic LSS includes nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, exercise programs, and symptomatic treatment of acute pain episodes. There is insufficient evidence to support the routine use of epidural glucocorticoid injections. Surgical therapy is considered when medical therapy does not relieve symptoms sufficiently to allow for resumption of activities of daily living or when focal neurologic signs are present. Most patients with neurogenic claudication who are treated medically do not improve over time. Surgical management can produce significant relief of back and leg pain within 6 weeks, and pain relief persists for at least 2 years. However, up to one-quarter develop recurrent stenosis at the same spinal level or an adjacent level 7–10 years after the initial surgery; recurrent symptoms usually respond to a second surgical decompression.

Neural foraminal narrowing with radiculopathy is a common consequence of osteoarthritic processes that cause lumbar spinal stenosis (Figs. 22-1 and 22-6), including osteophytes, lateral disk protrusion, calcified disk-osteophytes, facet joint hypertrophy, uncovertebral joint hypertrophy (cervical spine), congenitally shortened pedicles, or, frequently, a combination of these processes. Neoplasms (primary or metastatic), fractures, infections (epidural abscess), or hematomas are other considerations. These conditions can produce unilateral nerve root symptoms or signs due to compression at the intervertebral foramen or in the lateral recess; symptoms are indistinguishable from disk-related radiculopathy, but treatment may differ depending on the specific etiology. The history and neurologic examination alone cannot distinguish between these possibilities. A spine neuroimaging (CT or MRI) procedure is required to identify the anatomic cause. Neurologic findings from the examination and EMG can help direct the attention of the radiologist to specific nerve roots, especially on axial images. For *facet joint hypertrophy,* surgical foraminotomy produces long-term relief of leg and back pain in 80–90% of patients. The usefulness of therapeutic facet joint blocks for pain is controversial. Medical causes of lumbar or cervical radiculopathy unrelated to anatomic spine disease include infections

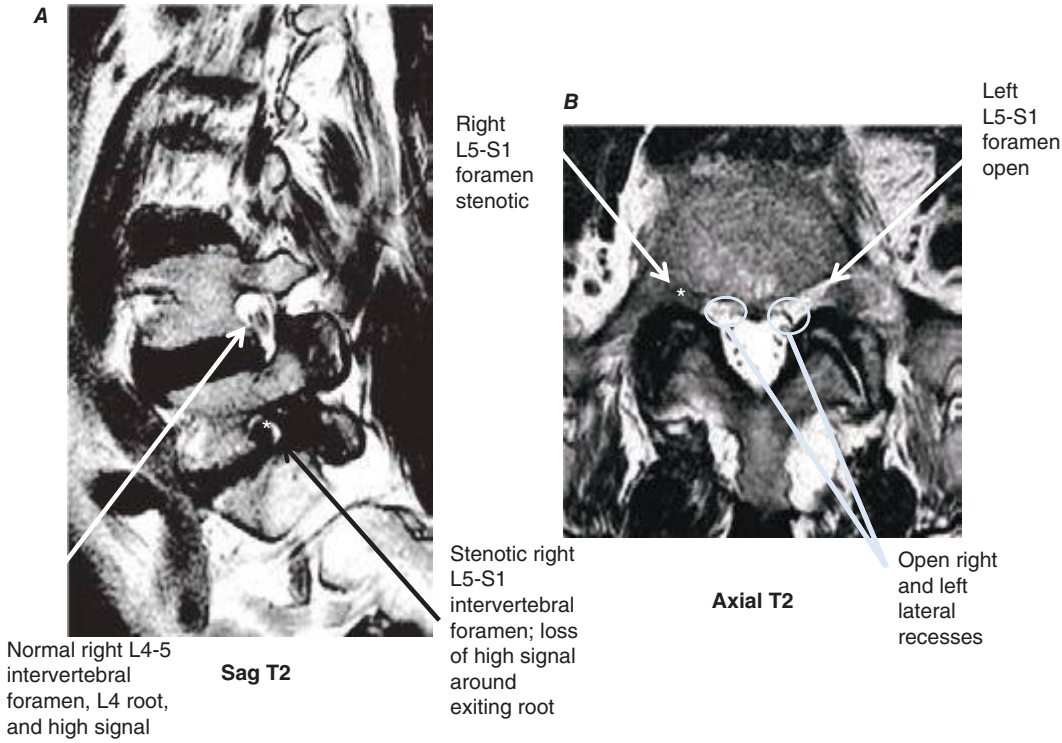

FIGURE 22-6 **Right L5 radiculopathy. *A.*** Sagittal T2-weighted image. There is normal high signal around the exiting right L4 nerve root in the right neural foramen at L4-L5; effacement of the high signal in the right L5-S1 foramen is present one level caudal on the right at L5-S1. ***B.*** Axial T2-weighted image. The lateral recesses are normal bilaterally; the intervertebral foramen is normal on the left, but severely stenotic on the right. *Severe right L5-S1 foraminal stenosis.