Lumbar Spinal Stenosis: Historical Perspectives, Classification, and Pathoanatomy

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Spinal stenosis is narrowing of the spinal canal (central stenosis), the lateral recess (lateral recess stenosis), or the foramen (foraminal stenosis) with neural impingement that can produce radicular pain, gait disturbances, bowel and/or bladder dysfunction, motor/sensory changes, and neurogenic claudication.

Historical Perspective of Spinal Stenosis

Spinal stenosis is an ancient disorder. It has been noted in Egyptian mummies, and it is a well-known malady in the veterinary world being observed in certain dogs (dachshunds) and horses. Portal of France in 1803 may have been the first to study spinal stenosis in humans when he related the size of the vertebral canal to compression on the spinal sac.[1] Lane of England in 1893 did a decompressive laminectomy to relieve a woman of a cauda equina syndrome caused by spondylolisthesis.[1] Bailey and Casmajor in 1911 emphasized that facet joint exostoses could cause significant compression of the spinal canal and cauda equina.[1] However, only within the last 50 years has the disorder been understood with respect to its clinical signs and symptoms, pathophysiology, pathoanatomy, and treatment. Sarpyener in 1945 described a "congenital stricture" of the spinal canal associated with spina bifida and also in children without any other developmental anomaly of the spine.[2] Van Gelderen in 1948 described a syndrome in two patients who showed signs of compression of the lumbar nerve roots with walking that disappeared with rest. They were cured with laminectomy. He also stated that a hypertrophied ligamentum flavum was the etiology of the compression in the erect position. Verbiest in 1954 described a clinical condition in seven patients (age 37 to 67) of bilateral radicular pains and sensory/motor disturbances in the legs caused by standing or walking. Myelography displayed a block in the lumbar region in every case. At surgery, he found a shallow canal with a compressed dural sac. Encroachment on the canal by enlarged articular processes was a possible etiology. He did not agree with Van Gelderen's theory that a hypertrophied ligamentum flavum caused the narrowing.[3,4] Since then, many investigators have described the pathogenesis and treatment of spinal stenosis.[5-9] Kirkaldy-Willis studied the "spectrum of pathologic change" in spinal stenosis. The two posterior joints and the disc, "the three-joint complex," are all involved in the pathogenesis. Degenerative changes of the three-joint complex occur secondarily to repeated rotational and compression injuries. The invertebral discs develop circumferential and radial annular tears, internal disruption, loss of disc height, and protrusion. The posterior joints undergo synovitis, cartilage destruction, osteophyte formation, capsular laxity, ligamentum flavum hypertrophy or buckling, and joint instability or subluxation. Changes occurring in the posterior joints affect changes in the disc and vice versa. As a result, instability of the three-joint complex occurs creating degenerative spondylolisthesis, retrolisthesis, degenerative scoliosis, as well as rotational deformities that occur with listhesis and scoliosis.[9]

Classification of Spinal Stenosis

Arnoldi et al developed a useful classification scheme based on the causes of spinal stenosis (Table 1).[5] The great majority of spinal stenosis cases are caused by degenerative changes in the intervertebral disc and facet joints creating constriction of the central spinal canal, lateral spinal canal, or intervertebral foramen. Vertebral translations such as in degenerative spondylolisthesis also narrow the canal and may lead to neural compression. There are many other causes of lumbar spinal stenosis. Iatrogenic causes such as postlaminectomy translation and postfusion stenosis

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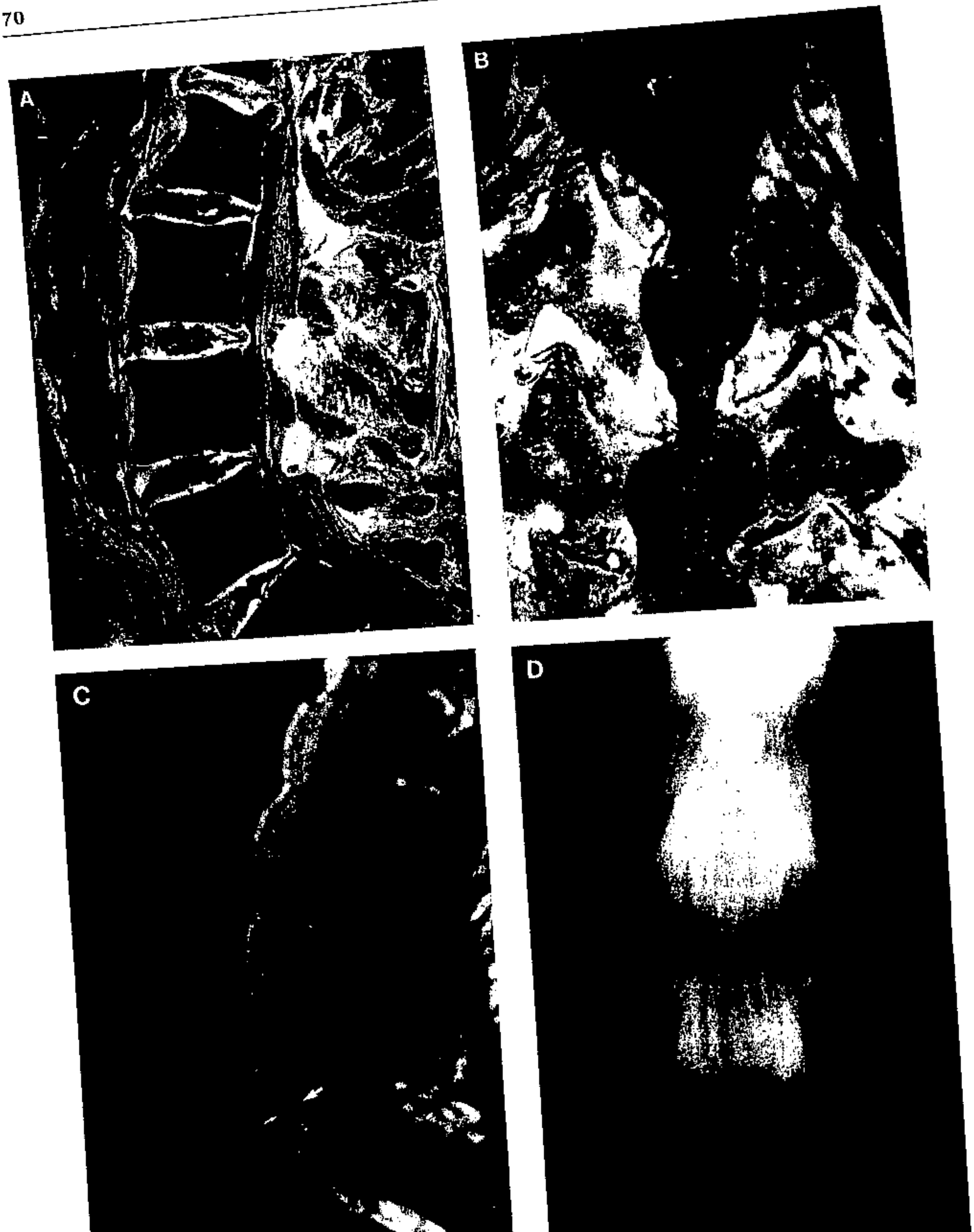

Figure 1

Table 1. Classification of Spinal Stenosis

I. Congenital-developmental stenosis
  A. Idiopathic (hereditary)
  B. Achondroplastic
II. Acquired stenosis
  A. Degenerative
  B. Combined congenital and degenerative stenosis
  C. Spondylolytic/spondylolisthetic
  D. Iatrogenic (ie, postlaminectomy, postfusion)
  E. Posttraumatic
  F. Metabolic (ie, Paget's disease, fluorosis)

should not be overlooked. Other rarer conditions such as achondroplasia and Paget's disease may contribute to spinal stenosis. Any of these various causes of stenosis may lead to symptoms of neural compression with back pain, leg pain, and neurological deficits.[5] Also, variations of the spinal canal may predispose to spinal stenosis. Three types of spinal canals are observed: a round canal, an oval canal, or a trefoil canal. A trefoil canal is seen in 15% of spinal canals and predisposes to lateral recess stenosis.

Symptom onset in patients with spinal stenosis is insidious, and the duration of symptoms is usually longer compared with patients with a disc herniation. Patients are typically over 50 years old. Complaints such as fatigue, weakness, low back pain, and numbness in the lower extremities are frequent. The classic symptom of central spinal stenosis is claudicating leg pain, aggravated by standing or walking and relieved by forward flexion or sitting. Radiculopathy due to lateral canal stenosis consists of pain in a dermatomal distribution and sensory or motor deficits of a particular nerve root. Nerve root compression may occur at more than one site. For example, L5 radiculopathy may be caused by L4-L5 facet hypertrophy with lateral recess stenosis and/or by L5-S1 foraminal stenosis. The diagnosis of symptomatic spinal stenosis can be made with these clinical presentations along with supportive evidence from a myelogram, computed tomography (CT) scan, or magnetic resonance imaging (MRI). One must be cautious interpreting these imaging modalities because false-positive rates are high.[10-12] Clinical correlation with the particular imaging abnormality is critically important.

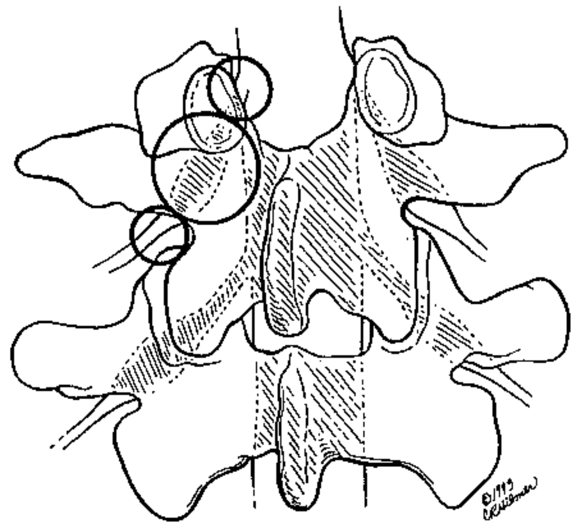

Figure 2. A diagram of three zones of the lateral spinal canal: Entrance zone proximally, midzone under the pars interarticularis, and the exit zone of the intervertebral foramen.

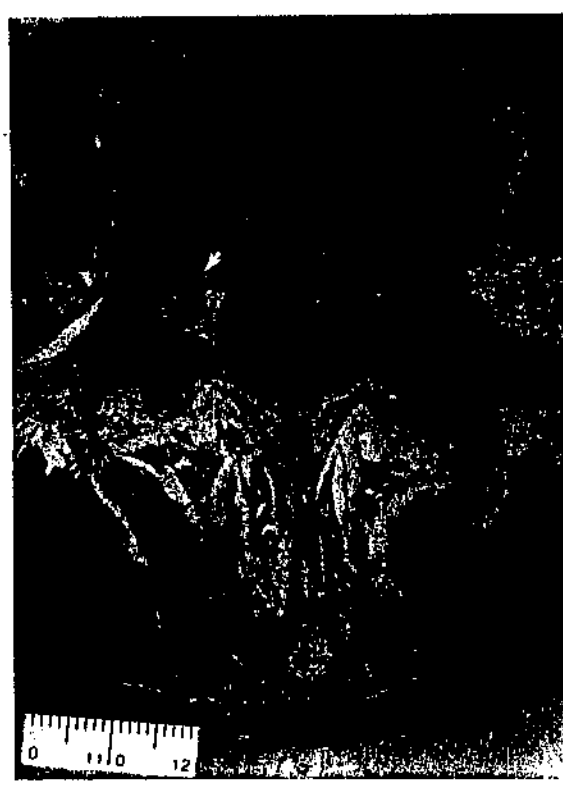

Figure 3. A cryomicrotome that shows normal anatomy within the lateral recess (arrow).

Figure 1. (A) A sagittal cryomicrotome section of the lumbar spine illustrating central lumbar spinal stenosis, bulging discs, and ligamentum flavum hypertrophy (arrow) at L3-4 and L4-5 levels. (B) Coronal cryomicrotome of a severe central lumbar spinal stenosis case, showing hypertrophic facets and ligamentum flavum indenting the thecal sac at multiple levels (arrows). (C) Sagittal T2 MRI showing severe central stenosis at L4-5 with bulging disc (small arrow) and hypertrophied ligamentum flavum (large arrow). (D) Anteroposterior Myelogram demonstrating complete block at L4-5 and attenuation of contrast at L3-4.

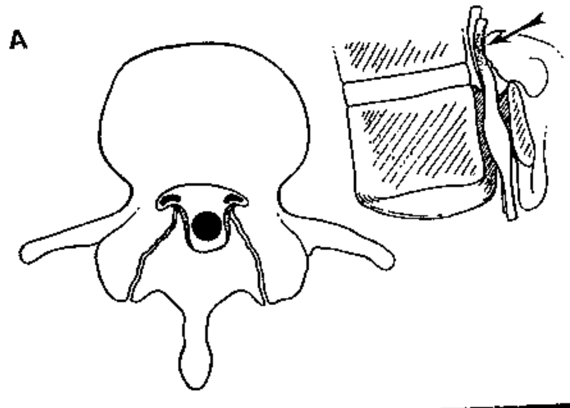

Figure 4. (A) A diagram of the entrance zone stenosis, showing subarticular entrapment of the nerve root. (B) A cryomicrotome demonstrating narrowing within the lateral recess (arrow).

Pathoanatomy of Spinal Stenosis

Understanding the pathological anatomy of spinal stenosis is important when relating the history and physical examination[33] to areas of neural impingement, interpreting imaging studies, and planning surgical approaches. Spinal stenosis can be subdivided into central stenosis and lateral stenosis.

Central stenosis is found at the intervertebral level and is caused by hypertrophic facets, ligamentum flavum buckling or hypertrophy, disc protrusion, and degenerative spondylolisthesis (Figs 1 A and B).[13,14] 40% of central stenosis is secondary to soft-tissue changes.[15] The cauda equina is compressed centrally from the anterior-posterior direction at the intervertebral disc level. The discs bulge posteriorly, and the hypertrophied ligamentum flavum and facet joints intrude posteriorly. Multilevel stenosis is common. Imaging studies such as MRI or myelography can vividly show the pathoanatomy of central stenosis (Figs 1 C and D). Intrathecal contrast from the surrounding bone and soft tissue created by T2-weighted images on MRI or with injectable contrast on myelography show where the cerebrospinal fluid around the roots of the cauda equina is obliterated. With CT, midsagittal lumbar canal diameters less than 10 mm are indicative of absolute stenosis, and less than 13 mm are indicative of relative stenosis.[16,17] Midsagittal lumbar canal diameters are not as reliable as the cross-sectional dimensions at the level of the intervertebral disc because most cases of degenerative spinal stenosis involve the facet joints and disc space. The lumbar epidural fat is usually obliterated with central spinal stenosis, although a small amount of fat may be preserved in the midline posterior to the dural sac even in severe stenosis. With respect to symptomatology, neurogenic claudication is usually the result of central canal stenosis.

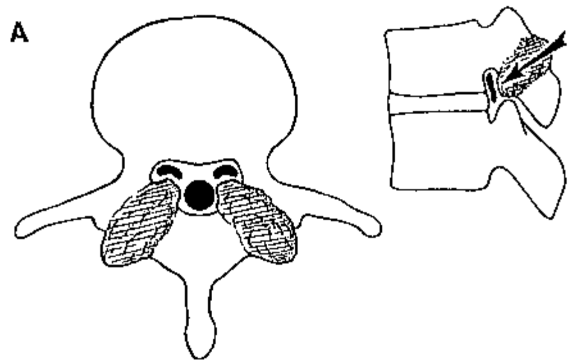

Figure 5. (A) A diagram of the pars interarticularis defect in the sagittal plane and fibrocartilaginous mass compressing the nerve root in the midzone. (B) An axial CT scan demonstrating the pars defect with isthmic spondylolisthesis (arrow).

Lateral stenosis is a common cause of lumbar radicular symptoms. The lateral lumbar spinal canal includes the nerve root canal (lateral recess) and the invertebral foramen. Together they form a tubular canal through which the nerve root exits the spinal canal.[18] The lateral lumbar spinal canal has been subdivided into three anatomic zones by Lee et al[19]: entrance zone, mid zone, and exit zone (Fig 2). The entrance zone is the subarticular area and medial to the pedicle and is synonymous with the lateral recess area. The mid zone is located under the pars interarticularis and the pedicle, and the exit zone is synonymous with the intervertebral foramen.

The entrance zone is located underneath the superior articular process of the facet joint and medial to the pedicle. The entrance zone is the cephalad aspect of the more commonly known lateral recess that begins at the lateral aspect of the thecal sac and runs obliquely downward and laterally toward the invertebral foramen.[20,21] Anatomically, the lateral recess is bordered laterally by the pedicle, posteriorly by the superior articular facet, and anteriorly by the posterolateral surface of the vertebral body and adjacent intervertebral disc.[22] The medial border of the lateral recess is formed by the thecal sac. The narrowest portion of the lateral recess is between the superior border of the pedicle and the broad portion of the superior articular facet.[23] The nerve root in this region is covered by the root sleeve and surrounded by cerebrospinal fluid. The lateral margin of the nerve root sleeve contacts the medial cortical bone of the pedicle, and the medial margin of the nerve root is surrounded by epidural fat tissue (Fig 3). The normal lateral recess measurement have

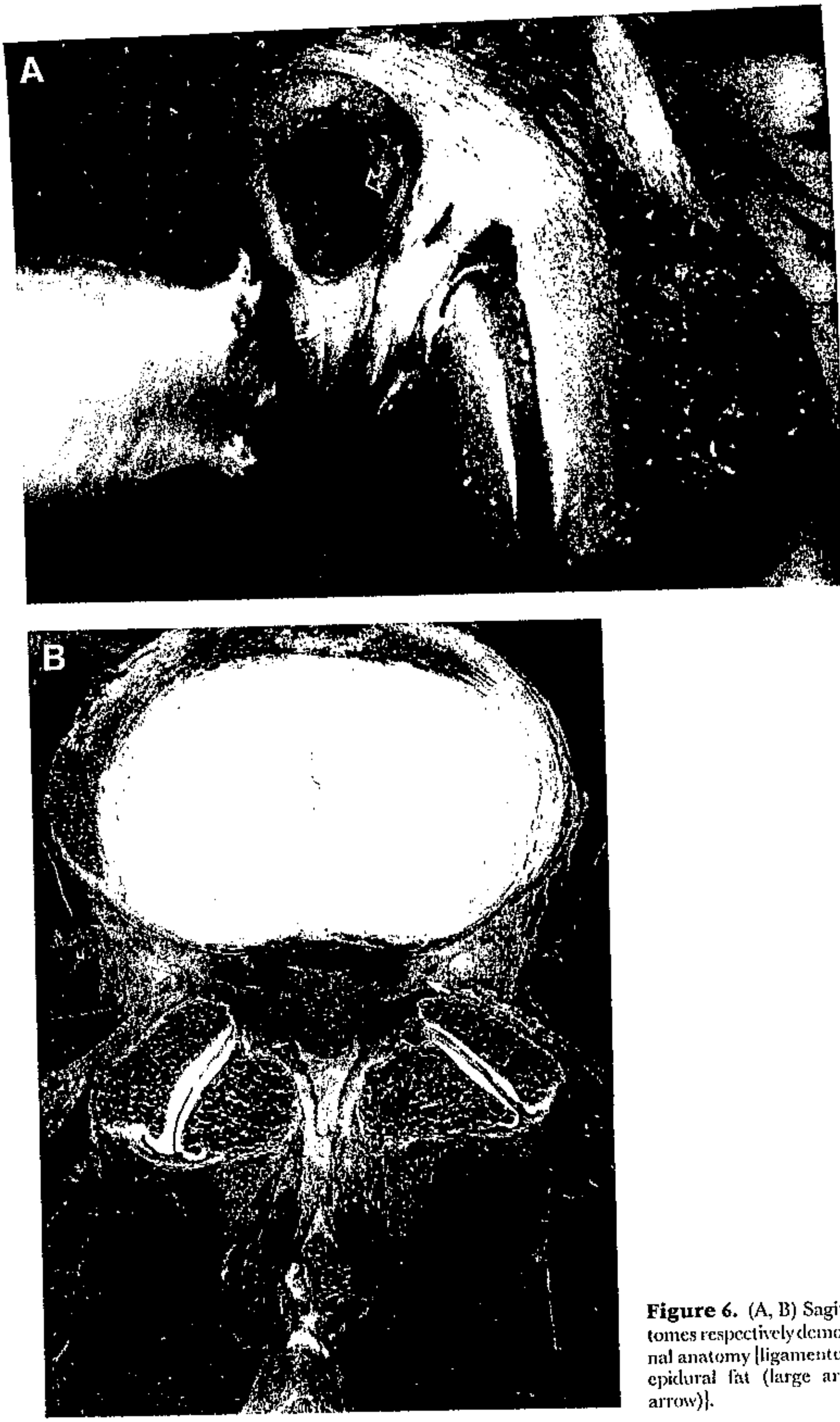

Figure 6. (A, B) Sagittal and axial cryomicrotomes respectively demonstrating normal foraminal anatomy [ligamentum flavum (small arrow), epidural fat (large arrow), nerve root (open arrow)].

been well delineated by CT.[21] A lateral recess height of 5 mm or more is normal. A height of 2 mm or less is pathological, and a height of 3 to 4 mm is suggestive of lateral recess stenosis.[22]

Narrowing of the entrance zone or lateral recess with nerve root compression is most commonly caused by a posterolateral herniated disc, which compresses the nerve root as it emerges from the dural sac. Another common cause of nerve root compression is a hypertrophic superior articular process also known as lateral recess syndrome,[24] superior facet syndrome,[25] or nerve root canal stenosis (Figs 4 A and B).[26] These two causes account for the majority of cases of lateral spinal stenosis.

The mid zone is located under the pars interarticularis and just below the pedicle. It is bounded anteriorly by the posterior aspect of the vertebral body and posteriorly by the pars interarticularis. The medial boundary is open to the central spinal canal.[19] The nerve roots normally run obliquely downward through the lateral recess into the intervertebral foramen. The nerve root travels around the subpedicular notch and contacts posteriorly with the ventral wall of the pars interarticularis where the ligamentum flavum is attached.[18] CT provides the accurate information of the pars interarticularis and shows the adjacent nerve root under the pars.[27] In sagittal MRI, the pathological conditions of the pars interarticularis are well defined.[28] A normal pars on T1-weighted images is shown as high signal intensity bone marrow surrounded by the lower signal intensity of cortex. The bone marrow signals are intact from superior to inferior articular process. Discontinuity in this signal heralds a pars defect.

In midzone stenosis, a defect in the pars interarticularis is most commonly responsible for nerve root compression, For instance, the L5 nerve root may be entrapped by the fibrocartilaginous tissue at the L5 pars defect in isthmic spondylolisthesis (Figs 5A and B). Another common cause of midzone stenosis is pedicular kinking. As the nerve root exits just inferomedial to the pedicle, kinking of the nerve root by the pedicle may be responsible for radiculopathy. This phenomena is more common in patients with scoliosis or spondylolisthesis where one pedicle may be lower than the other owing to rotatory deformity of the vertebral body or asymmetric collapse of the disc space.[29]

The exit zone is formed by the intervertebral foramen. The lumbar intervetebral foramen, which is shaped like an inverted teardrop, forms a tunnel that connects with the spinal canal. It is bounded superiorly and inferiorly by the pedicle of the adjacent vertebrae. The posterior boundary is formed by the pars interarticularis and the ligamentum flavum. The anterior boundary is formed by the posteroinferior margin of the superior vertebral body, the posterior margin of the intervertebral disc, and the posterosuperior margin of the inferior vertebral body (Figs 6A and B).[30,31] The normal foraminal height varies from 20 to 23 mm, and the width at the upper foraminal area varies from 8 to 10 mm. The ventral and dorsal nerve roots occupy 23% to 30% of the area of the foramen and lie anterior to the dorsal root ganglion (DRG).[32] The DRG normally lies within the superior lateral portion of the lumbar intervertebral foramen and directly below the pedicle in 90% of lumbar levels.[33] Foraminal height of less than 15 mm and posterior disc height of less than 4 mm are associated with nerve root compression 80% of the time.[32]

Figure 7. Parasagittal T1-weighted MRI showing normal foraminal anatomy. The small arrows point to the abundance of high-intensity fat signal around the nerve roots.

The shape of the lumbar intervertebral foramen is not well shown by transaxial CT scans. Sagittal reconstruction images may help to identify the bony abnormalities of the intervertebral foramen. MRI displays the soft-tissue structures in the invertebral foramen. Sagittal T1-weighted images are used for evaluating the morphological structures of the intervertebral foramen. On T1-weighted images, the nerve root complex and radicular veins within the intervertebral foramen can be shown as lower signal intensity structures surrounded by the higher signal intensity of fat. The structures that border the intervertebral foramen such as the cortex of the pedicle, vertebral body, the periphery of the disc, and the ligamentum flavum are well outlined (Fig 7).[18]

In exit zone or foraminal stenosis, the nerve root can be impinged in an up/down or front/back fashion (Fig 8A). This can occur secondarily to subluxation of the superior articular facet, a laterally herniated disc, or protruding annulus or an uncinate spur from the posterolateral vertebral body (Figs 8B and C). The nerve root may also be compressed at more than one site. With MRI, the intervertebral forami-

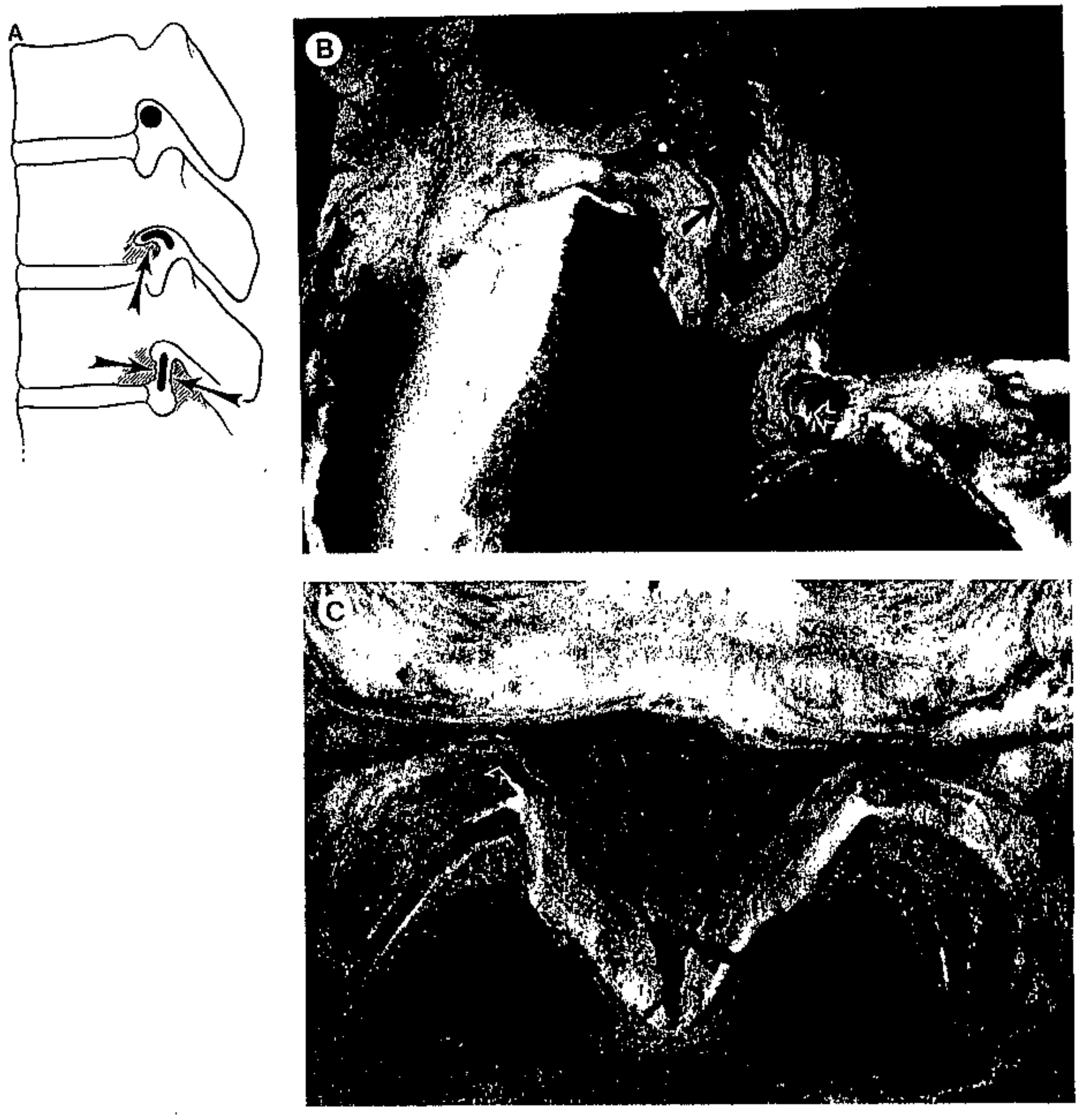

Figure 8. (A) A diagram of the intervertebral foraminal stenosis by osteophytes (midlevel) and subluxating facet joint (lowest level) in the exit zone. (B) A sagittal cryomicrotome section showing degenerative disc disease associated with osteophytes and buckling of the ligamentum flavum adjacent to the facet joint (arrow) in proximity to the nerve root in the exit zone. Note also the bulging disc in the inferior part of the foramina (open arrow). (C) Axial cryomicrotome demonstrating encroachment on the foramen by bulging disc (closed arrow) and ligamentum flavum (open arrow).

nal stenosis is best visualized by the parasagittal scans that show the nerve root compressed or deformed by a laterally herniated disc or facet subluxation with ligamentum flavum impingement on the nerve root. A useful indication of significant foraminal stenosis is absence of the well-defined perineural fat signal on the parasagittal T1 images.

In summary, this review has covered a brief history, classification, and pertinent normal and pathological anatomy of spinal stenosis. A clear understanding of where thecal sac and nerve root impingement occur will aid the spine surgeon in interpreting imaging studies and planning appropriate surgical decompression.

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