Spinal Stenosis

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Key Points

- Spinal stenosis is defined as a narrowing of the spinal canal. It can be classified in terms of location (central, foraminal, or lateral recess stenosis) or cause (congenital, acquired, or degenerative).
- Degenerative spinal stenosis most commonly affects individuals in their 60s and is the most common diagnosis for spinal surgery for individuals over 65 years old.
- The diagnosis of spinal stenosis is from a combination of symptoms and their correlation with pathology found on radiology imaging.
- Radiographs provide limited information but are wildly available and low cost and have low radiation exposure. Magnetic resonance imaging is the most commonly utilized imaging modality with high sensitivity for detecting spinal stenosis and soft tissue pathology.
- There are no generally accepted radiologic criteria for diagnosing spinal stenosis. In the anteroposterior (AP) diameter, central canal stenosis is compatible with a bony canal diameter of less than 10 mm in the cervical spine and 12 mm in the lumbar spine.
- Central canal stenosis most commonly presents with neurogenic claudication. Neuroforaminal and lateral recess stenosis most commonly presents with radiculopathy.
- Symptoms of cervical spinal stenosis include impaired gait, numbness of the hands, hyperreflexia, atrophy of the intrinsic hand muscles, positive Hoffmann's test, and positive Babinski reflex. In the thoracic region, fatigue, leg heaviness, loss of proprioception, and pseudoclaudication are more common. Lumbar spinal stenosis stereotypically presents with neurogenic claudication and radiculopathy.
- Degenerative changes contribute to spinal stenosis. These include discal degeneration, disc herniation, facet hypertrophy, hypertrophy of the ligamentum flavum, bone remodeling, and osteophyte formation. Degeneration can furthermore lead to instability, scoliosis, and spondylolisthesis.
- Commonly employed conservative treatment includes physical therapy, exercise, patient education, and medication. Epidural steroid injections may offer some benefit as well. In cases of severe symptoms, surgical decompression may improve symptoms and functional capacity.

Introduction

The terminology of spinal stenosis is derived from the Greek word *stenos*, which is translated as narrow. Spinal stenosis refers to the abnormal anatomic narrowing of the spinal canal and can be classified in terms of location (central, foraminal, or lateral recess stenosis) or cause (congenital, acquired, or degenerative). Degenerative spinal stenosis most commonly begins in the sixth decade of life [1, 2]. Age-related changes result in diminished space for the neural and vascular structures. There are significant variations in the description and reporting of spinal stenosis; however, it has been cited as the most common diagnosis for spinal surgery in patients over 65 years old [2].

Congenital Stenosis is a normal variant in the population as well as a feature of achondroplasia. In congenital stenosis, defects in cellular metabolism lead to retardation of skeletal

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growth and hypoplasia, premature fusion of endplates, and irregular intracartilaginous bone formation. Major contributors to congenital stenosis are shortened pedicles, thickened lamina, and hypertrophied facet joints [2, 3]. These changes result in reduction of the AP (anteroposterior) diameter of the spinal canal, and spinal stenosis commonly occurs earlier in life [4–6]. Stenosis can also be acquired due to trauma, neoplasms, and infection or through disorders such as acromegaly, Paget's disease, fluorosis, or ankylosing spondylitis.

Central Stenosis is defined as a narrowing of the central canal, between the medial edges of the two zygapophysial joints (facet joints), resulting in reduced available space for nerve rootlets within the cerebrospinal fluid (CSF) in the dural sac [7, 8] (Fig. 11.1). In the axial plane, the central canal can be compromised anteriorly by disc protrusions or osteophytes, posterolaterally by facet hypertrophy, posteriorly by ligamentum flavum (LF) hypertrophy and buckling, and, in the cervical spine, anterolaterally by uncovertebral hypertrophy [9].

Foraminal Stenosis Foraminal Stenosis causes encroachment of the exiting neural structures at the intervertebral foramen, defined by the medial and lateral pedicle borders (see Fig. 11.1). Lateral recess stenosis is the impingement of the exiting nerve in the lateral recess or proximal foramen at the level of the facet joints, between the medial edge of the facet joint and the medial pedicle border [4, 7, 10, 11]. In the anteroposterior or craniocaudal orientation, foraminal stenosis can occur as a result of disc herniation, facet hypertrophy, or subluxation [9]. The definition of severity of foraminal stenosis, as well as the differentiation between foraminal and lateral recess stenosis, varies and is often inconsistent. Some definitions focus on deformation or obliteration of the perineural fat portraying an increase in the degree of foraminal stenosis [12], while others focus on direction of perineural fat obliteration or nerve root collapse in the foramen [13, 14].

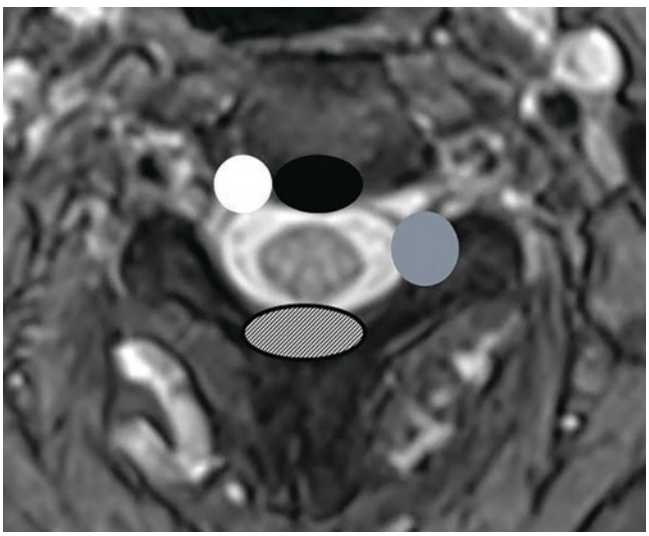

Fig. 11.1 Axial gradient recalled echo image of the cervical spine at the level of the disc space. The canal can be compromised in the following ways: anteriorly by a disc protrusion, osteophytes, or OPLL (black oval); posterolaterally by facet hypertrophy (gray circle); and posteriorly by ligamentum flavum infolding (white and gray oval). Anterolaterally, uncovertebral hypertrophy can narrow the spinal canal in the cervical spine (white circle). (Reprinted with permission of Anderson Publishing LTD. from Talekar et al. [9]. ©Anderson Publishing Ltd.)

Basic Anatomy

A further understanding of spinal stenosis relies on an understanding of the underlying bony and soft tissue anatomy, the physiology of aging, degeneration, and other acquired causes. The spinal column consists of 33 vertebrae, divided into 24 presacral vertebrae (7 cervical, 12 thoracic, and 5 lumbar vertebrae), 5 fused sacral vertebrae which make up the sacrum, and 4 frequently fused coccygeal vertebrae which make up the coccyx. The lumbar vertebrae articulate with the sacrum, which articulates with the five ossicles of the coccyx [15–17]. Each vertebra consists of a vertebral body anteriorly, joining pedicles bilaterally which connect the body to the transverse process, and lamina, which connects the transverse processes to the spinous process bilaterally. Superior articular processes from the vertebrae below articulate with the inferior articular processes from the vertebrae above to form the zygapophyseal, or facet joint [15–17].

The spinal canal is bordered anteriorly by the vertebral body, disc, and posterior longitudinal ligament (PLL), laterally by the pedicles, ligamentum flavum, and neural foramen and posteriorly by the facet joints, lamina, and ligamentum flavum. Anatomic variants exist in the shape of the canal. These include a circular, ovoid, and trefoil shape, the circular and ovoid shapes presenting the largest cross-sectional area [17]. The intervertebral foramen is bounded anteriorly by the posterior wall of the vertebral body and disc, posteriorly by the lateral aspect of the facet joint and ligamentum flavum, and superiorly and inferiorly by the pedicles and vertebral bodies [15–17]. Spinal stenosis can occur due to changes in any of these bordering structures. Descending from the cervical to lumbar vertebrae, characteristic changes occur at the various anatomical bony segments.

Imaging

The diagnosis of spinal stenosis is commonly a combination of symptomatology, imaging, and evidence of neurovascular compromise [4, 9, 18, 19]. In general, radiographs provide limited information. They are insensitive to soft tissue hyper-

trophy and non-osseous causes of spinal stenosis. However, they are rapidly available and low cost and produce low radiation exposure to the patient. Radiographs are able to provide some information on alignment and deformity, degenerative changes, and loss of disc height and aid in ruling out other pathological causes of pain such as fractures [4, 9].

Electromyography is not commonly utilized, and its utility may be limited to differentiating spinal stenosis from peripheral neuropathies, particularly in circumstances where a clear spinal etiology is not found to explain the patient's symptoms of pain or radiculopathy [4]. Computed tomography (CT) is helpful in diagnosing metastasis and infection and visualizing bony anatomy. In a patient with prior back surgery, CT can reduce artifact from metallic hardware. Disadvantages of CT examination include the reduced ability to detect nerve root impingement and the amount of radiation exposure [9, 20–22].

Traditionally, myelography has been used to provide dynamic information about spinal pathology, narrowing of the spinal canal with axial loading and extension. However, this test is invasive, requiring intrathecal injection of contrast and, when CT myelography is utilized, subjects the patient to ionizing radiation. Contrast in the subarachnoid space outlines neural structures for detecting stenosis and impingement and also is useful in diagnosing CSF leak and nerve root avulsion [4, 23].

MRI

Magnetic resonance imaging (MRI) is the most commonly utilized imaging modality for diagnosing spinal stenosis. MRI is noninvasive, has no ionizing radiation, and has high sensitivity in diagnosing spinal stenosis and identifying soft tissue pathology. Contrast may be added to further detect infection, tumor, and postsurgical changes [9, 22, 23]. There is no generally accepted radiologic criteria for diagnosing spinal stenosis [4, 9, 24]; thus, MRIs are usually described qualitatively as mild, moderate, or severe [7, 11]. In the anteroposterior diameter, a bony canal of less than 10 mm in the cervical spine and 12 mm in the lumbar spine is compatible with central canal stenosis. On MRI, a midsagittal diameter of the dural sac less than 10 mm is also compatible with central canal stenosis. Additionally, the cross-sectional area and transverse diameter have also been described [24].

Neuroforaminal stenosis diagnosis requires an AP diameter less than 3 mm on sagittal images and, for lateral recess stenosis, a lateral recess height less than 3 mm or lateral recess angle less than 30°. In the sagittal plane, the combined task force of the North American Spine Society, American Society for Spine Radiology, and the American Society of Neuroradiology recommends defining stenosis as demarcated by the pedicle, defining it, in terms of levels, as supra-pedicle, pedicle, infra-pedicle, and disc level. In the axial plane, they define spinal stenosis with zones of central, subarticular, foraminal, and extraforaminal [9].

Many have proposed alternative grading systems to better describe spinal stenosis. On sagittal images, compromise of neural structures can be inferred by noting degree of CSF obliteration and structural impingement of the spinal cord. Abnormal signal of the spinal cord, crowding of nerve fibers, and redundancy may also provide useful information [9]. Pfirrmann and colleagues created a grading system for disc herniation-related nerve compromise, correlating with intraoperative findings. This scheme uses four grades based on displacement, contact, and compression of neural structures [25]. Schizas and colleagues have described a grading system of CSF-to-rootlet ratio [26], whereas Barz and colleagues have described sedimentation of the nerve roots in the dural sac [27]. However, neither of these two grading systems correlates with functional status, symptomology, or outcomes.

Symptomatology

Lumbar central canal stenosis most commonly results in neurogenic claudication described as pain, dysthesias, paresthesias, or weakness in the back or buttocks radiating to the lower extremities. The abnormal sensations are most commonly bilateral and do not follow a dermatomal distribution. They are worse with extension of the lumbar spine and often relieved by flexion. Patients characteristically can state a duration of walking that brings on the symptoms, and this distance and duration shorten as the stenosis worsens. In comparison to the neurogenic claudication of central canal stenosis, foraminal and lateral recess stenosis typically results in radiculopathy. Radiculopathy is described as pain and paresthesias in a dermatomal distribution of the corresponding nerve root [2, 11, 14, 23].

Many patients have asymptomatic narrowing, with degenerative changes commonly occurring with increasing age, and thus imaging often correlates poorly with symptomatology [28, 29]. This may be, at least partially, due to an individual's ability to compensate for accumulating pathologic changes, which, in turn, is affected by the rate of changes that lead to stenosis. Many studies have looked at compression of the thecal sac leading to increased pressure at the nerve roots [30, 31]. This compression can lead to neural dysfunction, capillary constriction, and venous congestion, which can lead to altered local nutrition, as well as inflammatory chemical mediator accumulation, and electrophysiologic alteration. Mechanical compression and chemical mediators may both affect the experience of symptoms, and, since only the structural anatomy is visible on imaging, this may contribute to the poor correlation [2, 32, 33].

Characteristics of Cervical Spinal Stenosis

Cervical spinal stenosis can have both congenital and acquired causes. Acquired degenerative changes at the disc level are the most common cause of stenosis. These degenerative disc changes such as disc herniations and ossification can cause altered mechanical function and hypertrophy of the posterior elements, such as the facet joints and ligamentum flavum, ultimately leading to cervical stenosis and cord compression. Spondylotic changes of the cervical spine are more prevalent at the C5–C6 segment, followed by the C6–C7 and C4–C5 segments [34]. Symptoms of cervical stenosis can include impaired gait and numbness of the hands, while clinical signs can include hyperreflexia, atrophy of the intrinsic hand muscles, positive Hoffmann's test, and positive Babinski's reflex [35].

The first (atlas) and second (axis) cervical vertebrae are perhaps the most unique spinal segments with a significant portion of forward flexion and rotation occurring at these segments. Characteristics unique to the third to seventh cervical vertebrae are the uncinate processes, transverse foramen, and orientation in the sagittal and transverse planes [15, 16, 36]. An uncinate process is located at the superior lateral edge of the vertebral body where it connects to the transverse process, and the articulation is the joint of Luschka. The transverse processes project laterally with an anterior and caudal tilt. Within the transverse process, the transverse foramen houses the vertebral artery ascending from the sixth to the first cervical vertebrae. The transverse foramen is lateral to the pedicles and medial to the sulcus for the spinal nerves and, descending caudally, becomes more lateral. Anterior disc height is greater than dorsal height, and a slight lordotic curve exists in the cervical spine [15, 16, 36].

The normal AP diameter of the entire cervical spinal canal in adults is 17–18 mm with the spinal cord itself having a diameter of 5–6 mm. Other soft tissue components of the spinal canal such as the posterior longitudinal ligament, dura, and ligamentum flavum occupy another 2 mm of the canal diameter. As such, the common threshold for absolute cervical spinal canal stenosis is 10 mm given that any AP diameter less than this value will cause compression of the spinal cord. There is also an AP diameter threshold for relative cervical stenosis of 12–13 mm given that the AP diameter of the cervical spine reduces by 2–3 mm on neck extension [4, 37]. Furthermore, the intervertebral foramen with the nerve roots becomes smaller in extension and larger in flexion. Defining the parameters of neuroforaminal stenosis in the cervical region is further confounded by the fact that the neuroforamen are at a nearly 45-degree oblique orientation [14].

Cervical canal stenosis has also been defined by the Torg-Pavlov ratio (TPR), which is the ratio of the spinal canal to the vertebral body on conventional radiographs. The TPR is defined by dividing the distance from the midpoint of the posterior aspect of the vertebral body to the nearest point on the corresponding spinolaminar line by the AP width of the same vertebral body [37, 38]. The TPR was originally studied as a parameter to correlate with transient neuropraxia in football players, identifying normal as 1 and congenital cervical stenosis as 0.8 or less [4, 38]. The TPR has a low positive predictive value for compressive myelopathy. Moreover, spondylotic changes commonly occur at the level of the intervertebral disc as opposed to the vertebral body, and conventional radiographs cannot assess canal narrowing by soft tissue. As such, MRI has been deemed the best imaging modality for cervical spinal stenosis as it can provide information on disc and other soft tissue pathology, as well as any changes within the spinal cord itself [4, 37, 39].

Characteristics of Thoracic Spinal Stenosis

The prevalence of thoracic spinal stenosis is much less than that of the cervical and lumbar regions. Thoracic stenosis is radiographically defined as a spinal canal narrowed <10 mm in the AP diameter best measured on MRI or CT imaging. Unlike lumbar stenosis, pain is not the most common symptom of thoracic stenosis. Clinical presentation is mainly one of fatigue, leg heaviness, loss of proprioception, and pseudoclaudication in which symptoms are exacerbated with standing and walking and relieved by rest or forward flexion [40, 41].

The unique anatomy of the thoracic spine leads to its limited range of motion, which protects it from stenosis. Unlike the cervical and lumbar regions, the thoracic spine consists of 12 rib-bearing segments. The first seven ribs are directly connected to the sternum through the costal cartilage; the 8th, 9th, and 10th ribs connect to the sternum through an elongated costal cartilage and are known as false ribs, while the 11th and 12th ribs do not directly connect to the sternum at all and are known as floating ribs. The ribs and sternum provide stability to the thoracic spine along with a decreased range of motion. This stability decreases from higher to lower levels, which in turn affects the level of degeneration seen at various levels. Interestingly, the spinal canal is narrowest at the thoracic spine [41].

A variety of local and systemic metabolic diseases can cause thoracic stenosis. The most common cause is due to degenerative disc disease causing hypertrophy of the posterior elements including the facet joints and the ligamentum

flavum [42]. Posterior compression can also be caused by congenitally short pedicles. Ventral spurs of the uncinate processes, discal intrusions, limbus fractures, and ossification of the posterior longitudinal ligament can also impinge on the canal ventrally [40]. Indeed, these degenerative changes are seen more commonly in the lower thoracic levels where there are greater flexion, extension, and rotation movements and less stability as well as more of a lumbar-like configuration to the vertebrae [40, 43]. Thoracic disc herniations themselves can cause stenosis although this is rare given they account for less than 1% of all disc protrusions. The most common level for disc protrusions is T11/T12 with 75% of all thoracic disc protrusions occurring, again, at the lower levels and below T8 [43]. Systemic metabolic diseases that affect the thoracic spine tend to not only involve longer segments of the spine but also create circumferential narrowing lesions. Examples of these disease processes include acromegaly, achondroplasia, osteochondrodystrophy, Scheuermann's disease, fluorosis, ankylosing spondylitis, and Paget's disease [40, 42]. Space-occupying lesions such as epidural lipomatosis, hematomas, abscesses, and tumors can also cause thoracic stenosis [41].

Characteristics of Lumbar Spinal Stenosis

Due to the increased mechanical load carried by the lumbar vertebrae, lumbar spinal stenosis is more prevalent than cervical or thoracic stenosis. The increased weight supported by the intervertebral discs also renders them more prone to degeneration, resulting in further disc bulging, facet joint hypertrophy, and buckling of the ligamentum flavum (Fig. 11.2). The incidence of lumbar spinal stenosis in asymptomatic individuals aged 60–69 years is estimated to be 47% for relative stenosis and 19% for absolute stenosis [7, 22, 44].

The spinal cord in adults ends at the upper border of the first lumbar vertebral body and continues as nerve roots, the cauda equina. The central canal anteroposterior diameter ranges from 15 to 23 mm. Commonly, the threshold for radiographic lumbar spinal stenosis is an anteroposterior diameter of less than 12 mm, with relative spinal canal stenosis, and less than 10 mm, with absolute spinal stenosis [7, 44]. Alternatively, some clinicians define spinal stenosis not on a specifically quantified diameter but as relative reduction in cross-sectional area with mild as narrowing of the anteropos-

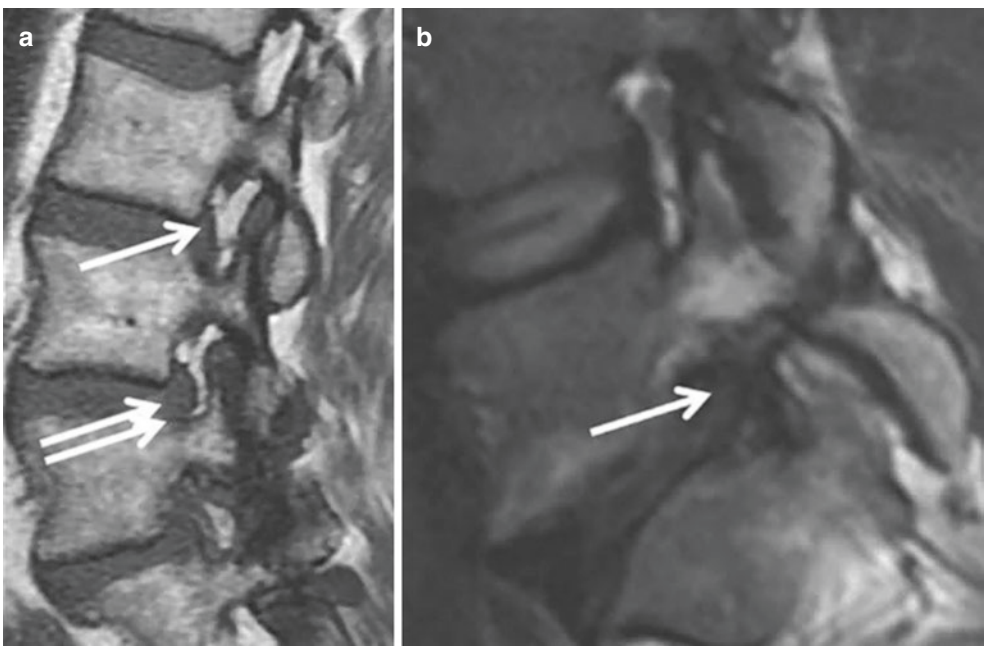

Fig. 11.2 Sagittal, T1-weighted image of the lumbar spine demonstrates intervertebral disc material protruding into the neural foramen, narrowing its inferior portion (single arrow, **a**). There is progressive narrowing extending more superiorly in the neural foramen at the two lower levels due to disc bulge and facet hypertrophy (double arrows, **a**). Foraminal fat is preserved at all these levels. Image (**b**) is a sagittal T2-weighted image demonstrating craniocaudal subluxation as well as disc bulge severely narrowing the neural foramen (arrow). Note the obliteration of the perineural fat. (Reprinted with permission of Anderson Publishing LTD. from Talekar et al. [9]. ©Anderson Publishing Ltd.)

terior canal by one-third or less, moderate by narrowing by one- to two-thirds, and severe as more than two-thirds [7, 11].

The symptoms common to lumbar spinal stenosis, such as neurogenic claudication, can be explained by transient encroachment of structures on the cauda equina with sensory and motor nerve dysfunction. In addition, the symptoms of spinal stenosis may be caused, or exacerbated by, disrupted blood flow and venous congestion [2, 7, 33]. This presents as intermittent low back pain with radiation into the buttock and bilateral legs [2]. A defining characteristic of the pain associated with lumbar spinal stenosis, with high specificity, is that it is triggered by ambulation and relieved by rest or forward flexion [7]. When a patient exhibits forward flexion, the diameter of the spinal canal is increased and the compression of nerve axons is reduced [2, 7].

Weakness is not a prominent symptom but may be present, especially after prolonged walking. If stenosis occurs in the lumbar neuroforamina or lateral recess, symptoms are more commonly radicular. No clear relationship between the severity of symptoms and degree of stenosis exists [2, 14, 23]. The third and fourth lumbar vertebrae exhibit a higher degree of rotational movement, and degeneration at these levels is more common. Anterolisthesis of the L4 on L5 is also more common. Thus, central canal stenosis is more prevalent at L4/L5, followed by L3/L4. Facet joint arthritis is also more prevalent in these locations with the more sagittal orientations of the facets between L4 and L5 exhibiting a predisposition to instability. The iliolumbar ligaments attach the fifth lumbar vertebrae to the iliac crest, creating increased stability in this area, and L5/S1 anterolisthesis is less common than L4/L5 or L3/L4 [10, 45, 46]. In neuroforaminal or lateral recess stenosis, L4/L5 and L5/S1 are the most common locations of narrowing [47].

The Anatomy of Aging (Degeneration)

The spinal degenerative process starts in the first decade of life and progresses throughout an individual's lifetime [48, 49]. In this section, the anatomy of aging and degeneration is briefly discussed. Changes associated with the aging spine can narrow the diameter of the spinal canal, causing spinal stenosis. These changes include disc degeneration, disc herniation, facet hypertrophy and laxity, hypertrophy, and buckling of the ligamentum flavum and can lead to spondylolisthesis or scoliosis [9, 48, 50].

Disc degeneration is considered one of the earliest changes and can predispose to changes of the disc itself as well as deterioration of the bony elements and ligaments [4, 9, 48]. The intervertebral disc is composed of nucleus pulposus confined within the annulus fibrosis. The nucleus pulposus is composed of collagen fibers in a random orientation and radially arranged elastin fibers embedded in an aggrecan rich gel-like matrix. The annulus is composed of concentric collagen fibers with elastin fibers between them. The extracellular matrix and composition of the disc is normally balanced by modeling and enzymatic degradation. The hydrophilicity and gel-like nature of the disc allow it to increase the hydrostatic pressure and handle the axial compressive load. Loss of proteoglycans and water content, alterations in the collagen network, and increase in metalloproteinases result in decreased osmotic pressure in the disc and decreased ability to accommodate compressive forces. The demarcation between the annulus fibrosis and nucleus pulposus also becomes less distinct, predisposing to concentric fissuring and radial tear and disc herniation [48, 51].

The facet joints are the major posterior load-bearing unit of the spine, stabilizing the motion of flexion and extension. As the disc degenerates, it is no longer able to appropriately stabilize the spine anteriorly leading to increased stress on the facet joints. This furthermore leads to subluxation and cartilage degradation, which in turn leads to facet malalignment and hypertrophy, erosions, sclerosis, and osteophyte formation [17, 48]. Healthy ligaments of the spine are highly flexible and restrain motion in multiple dimensions. As ligaments degenerate, elastin increases in concentration, reducing the tensile properties and weakening the ability to stabilize structures. Degeneration of the ligamentum flavum also occurs, leading to increased thickness and buckling [52, 53].

With continued mechanical compression over time, changes in the bony structures can occur. This includes sclerosis and remodeling, formation of osteophytes, and decreased stability. Osteoporosis furthermore weakens the bony elements, predisposing to bone remodeling, rotational deformities, or subluxation. Discal degeneration increases with a reduced blood supply from the surrounding vertebral endplate. This results in tissue breakdown, progressing the degenerative cycle [48].

Ligamentum Flavum Hypertrophy

The ligamentum flavum (LF) is a posterior structure formed during the 10th to 12th week of gestation [54]. It connects the laminae of adjacent vertebrae from C2 to S1 [55]. At each level, the LF inserts on the inferior and anteroinferior aspects of the cranial vertebral arch and on the superior and posterosuperior aspect of the caudal lamina [56]. Thinnest at the cervical and high thoracic levels [57], the histologic characteristics of the LF separate it from other ligaments of the spine in that it consists of 80% elastin and 20% collagen [58]. Indeed, the LF is also called the yellow ligament because of the color given to it by this higher concentration of elastin fibers [59].

It is postulated that this histologic difference assists with the unique function of the LF compared to other ligaments of the spine. One theory is that its elastic nature may help with restoring a flexed lumbar spine back to the extended position. Other theories have focused on the location of the LF rather than its possible biomechanical composition – given its immediate position posterior to the vertebral canal, should the ligament be more collagenous, it would buckle with approximation of the laminae causing encroachment on the spinal cord or nerve roots. An elastic ligament, however, would stretch thin minimizing any buckling during spine flexion and therefore prevent nerve root compromise [60]. Nonetheless, under pathologic conditions, the LF does in fact contribute to spinal stenosis.

Elsberg first reported the hypertrophy of the LF as a possible cause of spinal stenosis in 1913 [61]. Since then, multiple studies have confirmed that thickening of the LF can reduce the diameter of the spinal canal resulting in spinal stenosis [46, 53, 62]. The exact etiology of LF hypertrophy remains poorly understood and is most likely multifactorial. One possible etiology is a disturbance in the ratio of elastin to collagen alluded to before through fibrosis. Fibroblast growth factors play a crucial role in cell proliferation and tissue repair. Several cytokines and growth factors have been reported to play a role in LF hypertrophy including TFG-β, platelet-derived growth factor-BB, and basic fibroblast growth factor [59, 63, 64]. Fibrotic changes can lead to increased levels of collagen and reduced levels of elastin with elastin degeneration [65, 66]. These inflammatory mechanisms may be the result of degenerative processes such as facet arthropathy [67] or from scarring prompted by the accumulation of mechanical stress with the normal aging process [68].

Other studies have proposed the etiology of LF hypertrophy to be secondary to infolding and buckling into the spinal canal as a result of degenerative disc disease as opposed to actual LF thickening. Decreased disc height causes a laxity of spinal column ligamentous tissue leading to LF buckling [66, 69]. As such, factors such as disc bulging, collapsed disc height, mechanical stress, and body mass index may also play a role in the LF's contribution to spinal stenosis. While most studies do show a correlation between increasing age and increasing LF thickening at the L4–L5 level, others have questioned any such association [67].

Disc Herniation

Disc herniation and radiculopathy is covered in detail in another chapter. Here we will briefly review its role in spinal stenosis. The intervertebral discs are the major axial load-bearing structures that absorb compressive forces. The annulus fibrosis consists of concentric fibers that resist tensile forces and confines the gel-like substance of the nucleus pulposus. In healthy discs, axial loads increase hydrostatic pressure within the nucleus pulposus which is resisted by tensile stresses of the annulus fibrosis. Bending and torsion are furthermore resisted by the tensile forces of the annulus [70].

Breakdown of the extracellular matrix and desiccation of the disc result in less distinct demarcation of the annulus and the nucleus, decreased ability to handle a mechanical load, loss of disc height, annular fissure, and eventually herniation [48]. Historically, disc degeneration is thought to be the cornerstone of other degenerative changes with more stress placed on the facet joint leading to degeneration, hypertrophy, and osteophyte formation. Genetic factors may play a role in disc herniation. Genetic mutations in collagen type IX alpha 2 and alpha 3 chains as well as genes involved with cytokines interleukin-1beta and interleukin-6 have been suggested to predispose to herniation [70, 71].

The vertebral endplate, the tissue interface between the vertebral body and the intervertebral disc, is essential in maintaining the integrity of the disc [72]. It balances load distribution, manages metabolite transport, and encases the nucleus within the annulus. Endplate lesions, along with degeneration and desiccation of the disc itself, predispose to herniation of the nucleus pulposus through the annulus fibrosis. Herniated disc materials then result in mechanical narrowing of the space available for the nerve root, causing impingement. Furthermore, chemical mediators and inflammation may play a role in pain symptoms produced [73]. In response to herniated material, increased angiogenesis and microglia and astrocyte can infiltrate the area. An inflammatory milieu is also created, consisting of mediators and cytokines such as IL-1alpha, IL-6, and TNF-alpha which furthermore activate the immune system and upregulate the expression of proteinases [33, 74–76]. The inflammatory mediators themselves can be a chemical irritant to the nerve as well as cause disorganization of the myelin sheath and Wallerian degeneration of the peripheral axons.

MRI allows clinicians to evaluate the relationship between protruding disc material and the nerve roots. Disc herniation is commonly quantified by the Combined Task Force, Jensen, and van Rijn classification systems. The Combined Task Force definition classifies disc bulges as broad based or focal, based on the circumference involved, and as protrusion or extrusion, depending on the shape of material displaced [9, 50]. The classification system by Jensen and colleagues is also commonly used, separating lumbar disc herniation into four grades [77]. Van Rijn further classifies disc bulges by nerve root compression [78].

When the disc herniates, it can lead to a functional narrowing of the spinal canal. In an individual with narrowing of the bony structures of the spinal canal, disc material can further reduce the spinal canal diameter. Posterolateral her-

niations can compress individual nerve roots and lead to radiculopathy, while central herniations can compress the cord or cauda equina and lead to symptoms more consistent with neurogenic claudication [79]. It can also present as muscle weakness or asymmetric reflexes [73, 80]. In absence of underlying stenosis of the bony vertebral canal, disc herniation resulting in symptoms most commonly presents in the fourth or fifth decade of life. The most commonly affected segments are the lower lumbar segments below the third lumbar vertebrae [81].

Spondylolisthesis

Spondylolisthesis is defined as the translational movement of one vertebra on another. The movement can be anterograde or retrograde and most commonly occurs in the middle lumbar spine, rarely in the cervical or thoracic spine. Anatomic and environmental factors can lead to spondylolisthesis, and these are commonly caused by congenital abnormalities, degeneration, trauma, and fracture of the pars interarticularis [82, 83]. Activities that result in repetitive hyperextension of the lumbar spine can also predispose to spondylolisthesis [84].

Commonly utilized classification systems to describe the grade of spondylolisthesis are the Meyerding, the Wiltse, or the Marchetti and Barolozzi classification systems. Meyerding and colleagues grade spondylolisthesis based on percent of slippage [83–85]. Grade 1 is 0–25%, Grade 2 is 25–50%, Grade 3 is 50–75%, and Grade 4 is 75–100% [85]. Wiltse uses the etiologies of dysplastic, isthmic, degenerative, traumatic, and pathological to categorize spondylolisthesis [82]. Marchetti et al. also use an etiology-based system with the categories of iatrogenic, traumatic, and pathologic [83].

Spondylolisthesis of any orientation can cause narrowing of the spinal canal and encroachment of the neural structures. Lower-grade spondylolisthesis more commonly affects the nerve at the subarticular zone and results in radiculopathy, whereas higher-grade spondylolisthesis can reduce the central canal diameter and presents with either radiculopathy or neurogenic claudication [86–88]. Because of weight-bearing mechanics, degenerative spondylolisthesis most commonly occurs between the L4 and L5 or L5 and S1 vertebrae, resulting in an L4 or L5 radiculopathy. In the lumbar spine, facet joints are oriented in a sagittal plane, allowing them to resist rotation but less able to resist flexion and extension. When in extension, they support an axial load. Hyperextension stress as well as hyperflexion and compression can cause excessive force and deformation of the area [89, 90]. At the L5 and S1 junction, a greater lumbosacral joint angle is associated with a greater translational force, and traumatic spondylolisthesis is more common in this location [90].

When spondylolisthesis occurs in the cervical spine, the most common symptom is radiculopathy. However cervical spondylolisthesis can also present with static or dynamic myelopathy [91, 92]. Spondylolisthesis in these areas is rare, with the upper cervical segments more commonly affected. The more coronal nature of the facet joints can predispose to facet dislocations [90, 93]. Traumatic injuries can also cause subluxation when associated with hyperextension injuries on an axial load [91, 92].

Treatment Options-Interventions

Since treatment of spine pain is covered extensively in other chapters, we will briefly touch on intervention options targeting spinal stenosis specifically. Once it is decided that the etiology of back pain is from spinal stenosis, the question of treatment arises. Conservative management, minimally invasive procedures and injections, or surgery may be employed. The most commonly employed conservative treatments for spinal stenosis are physical therapy, exercise, patient education, and nonsteroidal anti-inflammatories, muscle relaxants, or TENS for pain control. Exercise has been shown to improve walking distance, but no specific type of exercise has been shown to be superior [94].

Epidural steroid injections provide analgesia by inhibiting phospholipase A2 as well suppress conduction in C fibers and ectopic discharges of injured fibers. Phospholipase A2 is an inflammatory mediator itself, and its inhibition furthermore reduces the hydrolysis of phospholipids into arachidonic acid and lysophospholipids [95]. Epidural steroid injections may decrease pain and improve walking distance; however, this may be temporary [94, 96]. Evidence for improved efficacy in the long term, beyond 3–6 months, is conflicting [95, 97]. Epidural steroid injections also do not appear to reduce the need for surgery [95].

When symptoms are severe, surgical options are frequently sought, and lumbar spinal stenosis is the most common reason for spinal surgery in patients over 65 years of age [2]. Since the symptoms and functional impairment associated with lumbar spinal stenosis occur secondary to compression of neural structures, surgery aims at decompression techniques [98, 99]. Traditionally a wide laminectomy and open decompressive techniques can create more space to the neural structures. With improved imaging and surgical advances, more directed laminotomy and segmental interlaminar decompression with preservation of the paraspinal musculature and posterior stabilizing structures may be employed [97, 98, 100]. Surgery has been shown to improve symptoms and disability, at least temporarily, with decreased pain and increased function most evident in the first year [99, 101–103].

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