Imaging of Spinal Stenosis
Neurogenic Intermittent Claudication and
Cervical Spondylotic Myelopathy

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KEYWORDS
- Spinal stenosis • Claudication • Myelopathy cervical spondylotic myelopathy (CSM)
- Neurogenic intermittent claudication (NIC) • Diffusion tensor imaging (DTI) • Foraminal stenosis

KEY POINTS
- Lumbar spinal stenosis is an anatomic observation; it may cause the clinical syndrome of neurogenic intermittent claudication (NIC).
- The pathophysiology of NIC remains controversial; the best evidence suggests venous congestion from multiple sites of compression initiates an inflammatory reaction causing neural dysfunction and irritability.
- The imager must identify all sites of neural compression in the central canal, subarticular recess, and foramen.
- The clinical syndrome of cervical spondylotic myelopathy (CSM) occurs in the setting of cervical central canal compromise, frequently with underlying congenital narrowing.
- Intramedullary signal change, enhancement, and diffusion characteristics can aid in selecting CSM patients for decompressive surgery.

SPINAL STENOSIS

Although spinal stenosis is one of the most common reasons for spinal imaging, and most radiologists likely perceive they “know it when they see it,” it remains a more elusive phenomenon when one carefully examines the literature. It is first important to distinguish between the anatomic observation of central canal narrowing and the clinical syndromes that it may provoke: neurogenic intermittent claudication (NIC) and/or radiculopathy in the lumbar spine, or myelopathy in the cervical or thoracic spine. Anatomic central canal narrowing is a frequent observation in an asymptomatic population, and increases in prevalence as an asymptomatic finding with age.¹ Compromise of the central canal, with or without associated subarticular recess (lateral recess) or foraminal compromise, provides the necessary but insufficient anatomic substrate for the clinical syndromes of NIC, radiculopathy, or myelopathy.

This article initially examines the literature describing the pathophysiology and imaging of NIC caused by lumbar central canal, lateral, and foraminal stenosis, followed by consideration of thoracic central canal compromise, and finally the pathophysiology and imaging literature of cervical spondylotic myelopathy (CSM). It is assumed that the reader can make the observations regarding compromise of the various spinal compartments; the emphasis is on the clinical significance of these observations as they affect the patient’s clinical care and ultimate outcome.

NEUROGENIC INTERMITTENT CLAUDICATION

NIC caused by central and/or lateral and foraminal stenosis in the lumbar spine is a clinical syndrome of significant frequency and debility in the elderly population. The North American Spine Society (NASS) 2011 evidence-based guidelines on the diagnosis and treatment of spinal stenosis define...
it in this fashion: “degenerative lumbar spinal stenosis describes a condition in which there is diminished space available for the neural and vascular elements in the lumbar spine secondary to degenerative changes in the spinal canal. When symptomatic, this causes a variable clinical syndrome of gluteal and lower extremity pain and/or fatigue, which may occur with or without back pain. Symptomatic lumbar spinal stenosis has certain characteristic provocative and palliative features. Provocative features include upright exercise such as walking or positionally induced neurogenic claudication. Palliative features commonly include symptomatic relief with forward flexion, sitting and /or recumbency.” The most common symptoms in patients with lumbar spinal stenosis are back pain (prevalence of 95%), claudication (91%), leg pain (71%), weakness (33%), and voiding disturbances (12%). There may be a paucity of physical findings, even in the presence of symptoms. NIC as a result of lumbar spinal stenosis is the most common cause of spine surgery in patients older than 65 years. Critical points regarding NIC are summarized in Box 1.

Prevalence, Natural History

Despite the significance of lumbar spinal stenosis and NIC, there is relatively little epidemiologic literature. A study from Denmark suggested an annual incidence of symptomatic disease of 272 per million inhabitants. This is a fourfold higher incidence than symptomatic cervical central canal compromise. In the United States, Kalichman and colleagues used data from the Framingham study to establish the prevalence of congenital and acquired lumbar central canal stenosis in a community population. Using the anterior-posterior dimension of the central canal derived from computed tomography (CT) studies (12 mm = relative stenosis, 10 mm = absolute stenosis), they noted congenital central canal narrowing of relative degree in 4.7% of the population and absolute stenosis in 2.6%. Acquired stenosis was identified in 22.5% (relative) and 7.3% (absolute) of individuals. Their review of the literature noted a range of prevalence of acquired lumbar stenosis from 1.7% to 13.1%. As would be expected, the congenital central canal stenosis did not change with age, but the prevalence of acquired stenosis (absolute) increased from 4% in patients younger than 40 years to 14.3% in patients older than 60. The presence of absolute central canal stenosis was significantly associated with low back pain in this study population; it was not significantly associated with leg pain. The correlation between imaging findings and symptomatology is discussed in detail below.

Pathophysiology

The pathogenesis of NIC in lumbar spinal stenosis has been a subject of investigation for half
a century. Verbiest initially described mechanical compression of the nerve roots of the cauda equi- 
a as a cause of NIC in 1954. Subsequent investigators have postulated that arterial or venous ischemia, perhaps exacerbated by restriction of cerebrospinal fluid (CSF) flow (which participates in nerve root nutrition), are major contributors to the clinical syndrome. The current preponderance of evidence would favor venous congestion secondary to mechanical compression. This hypothesis emphasizes the importance of multiple levels of compression, and the physiologic effects of lumbar extension. Both of these observations have significant relevance to imaging.

Takahashi and colleagues demonstrated in a porcine model that blood flow within the cauda equina was reduced by 64% in the segment between 2 zones of modest (10 mm Hg) compression, providing early evidence that there are profound microcirculatory changes even in an un-compressed segment of nerve between 2 zones of compression. Olmarker and Rydevik postulated that venous stasis between 2 zones of modest compression may cause proinflammatory compounds to leak from capillaries, stimulating a local inflammatory response. Also in a porcine model, they demonstrated reduced amplitude of action potentials in nerves subjected to 2 zones of modest compression. Furthermore, when 2 vertebral segments rather than 1 separated the zones of compression, there was a significant further reduction in the amplitude of action potentials. Thus, venous congestion can precipitate neural dysfunction.

Kobayashi and colleagues, in a canine model, examined cauda equina histology after the application of a modest stenosis (30% of cross-sectional area) to the dural tube. The cauda equina demonstrated congestion and dilatation of intraradicular veins and an inflammatory cellular infiltrate. There was disruption of the blood–nerve barrier, both at the site of the compression and also in more distant sites of Wallerian degeneration. The necrotic debris created by Wallerian degeneration stimulates macrophage activity; macrophages are known to generate inflammatory molecules, such as interleukin-1 and tumor necrosis factor α. Macrophages may also stimulate cytotoxic activity by the release of nitric oxide and proteases. They are considered the chief effector cells causing an inflammatory neuritis that results in aberrant ectopic neural discharge and conduction disturbance leading to the pain and neural dysfunction of NIC. In this canine model, the histologically demonstrated disruption of the blood–nerve barrier correlated with strong gadolinium enhancement on magnetic resonance imaging (MRI). It should be emphasized, paralleling the evidence presented earlier on the genesis of radicular pain caused by disc herniations, that an inflammatory pain reaction is intimately involved in the generation of pain.

There are thus experimental data to suggest that multilevel central canal compromise may provoke clinical symptoms even at modest levels of compression owing to venous congestion and a secondary inflammatory response. There are clinical studies supporting this contention. Sato and Kikuchi stratified 81 patients with lumbar central canal stenosis caused by spondylosis and degenerative spondylolisthesis into those with a single-level stenosis at the L4-L5 level and a second group with 2-level stenosis at L3-L4 and L4-L5. The patients with 2-level stenosis were significantly more likely to have neurogenic claudication symptoms than those with single-level stenosis. It was also observed that in 2-level stenosis, the symptomatic expression most closely matched the radicular distribution of the more caudal of the 2 stenotic levels; in those patients with compromise at the L3 and L4 disc levels, the pain pattern matched that of the traversing L5 roots.

The study of Porter and Ward noted that the sites of compression may be either in the central canal or the neural foramina. In their cohort of 49 patients with symptomatic NIC, 94% had either a multilevel central canal stenosis or central canal plus neural foraminal stenosis. They defined neural foraminal stenosis as a foraminal diameter smaller than 4 mm (the plane of the dimension was not specified). Symptomatic single-level stenosis was rare. Drawing on the earlier work of Takahashi and colleagues and Olmarker and Rydevik, they postulated that modest compression at 2 distant sites will cause venous congestion over a long segment and this will be exacerbated by the dynamic effects of walking (ie, vasodilatation of the arterioles and capillaries will worsen the venous hypertension and capillary stasis rendering a long segment of nerve ischemic and irritable, ultimately producing pain). This hypothesis can explain bilateral and unilateral claudication with a varied combination of central and lateral lesions, which may be near normal venous pressure in the static state but then exceed venous pressure under the stress of walking. It also emphasizes the importance of imaging detection of all stenotic zones, both within the central canal, the subarticular recess, and the neural foramina. Failure to address all zones of stenosis during a decompression procedure may result in an undesirable clinical result.

The work of Morishita and colleagues emphasized the importance of the neural foramen as a potential zone of compression, particularly with dynamic changes in posture. In 2006, they studied 41 patients with central canal stenosis or disk...
herniations; intraoperatively, micro catheters with pressure transducers were placed in the neural foramina of L5. Action potentials were measured in the anterior tibial musculature. Intraforaminal pressures significantly increased when the patient posture was passively moved from lumbar flexion to extension. The amplitude of the action potentials diminished in concert with the rise in intraforaminal pressure. These patients did not have foraminal stenosis by imaging. The findings suggest that the neural foramen may be a site of venous constriction with the spine in extension even in the absence of stenosis detectable by imaging. This stenosis may act in concert with central or subarticular recess compromise to produce symptoms in positions of lumbar extension, such as walking. In a second study, Morishita and colleagues again measured intraoperative L5 intraforaminal pressures during passive positional changes of the lumbar spine in a cohort of patients with spinal stenosis. These patients were stratified into single or 2 levels of central canal compromise, as well as into greater or lesser disability. The group with greater disability had significantly greater rises in intraforaminal pressures in the movement from flexion to extension. The patients who had 2-level central canal compromise had greater disability even though the rises in intraforaminal pressure were more modest. The study suggests that there is a cumulative effect of multiple sites of central canal compromise that conspire with the rise in intraforaminal pressures during lumbar extension to produce neural ischemia, presumably via venous congestion. One can postulate that static foraminal stenosis would further exacerbate this process. All levels of neural compromise, central, subarticular, and foraminal, are potentially significant and must be detected by imaging.

**Imaging**

**Congenital spinal stenosis**

It is widely accepted, although poorly documented, that in a portion (10%–15%) of patients who present with NIC, the spinal canal is congenitally or developmentally narrow, and only modest spondylotic changes are necessary to produce clinical symptoms (Figs. 1 and 2). Szpalski and Gunzburg, in a recent review article on lumbar spinal stenosis, contended that there is a Gaussian distribution of both canal size and dural sac size; when a canal size is too small for the dural sac it contains, stenosis occurs. In this construct, disregarding the true achondroplasia syndromes, so-called congenitally narrow canals are merely the extreme of Gaussian distribution of healthy subjects. In contrast, Singh and colleagues studied the morphologic characteristics of a cohort of surgically treated patients carrying the clinical diagnosis of congenital lumbar stenosis. They noted that these patients had a significantly shorter pedicle length and, as a result, smaller cross-sectional spinal canal area when compared with controls matched for age and sex. The patients with a congenitally narrowed lumbar central canal typically exhibit these morphologic characteristics over several vertebral segments, maximal at the L3 level. This contrasts with purely “degenerative” stenosis, which is often more focal, particularly at the L4 disc level. Patients with congenital central canal stenosis tend to present at a younger age (40–50) and with less spondylotic change than typical. In this cohort of patients, congenital narrowing could be recognized on lateral radiographs by identifying a pedicle length to vertebral body ratio of less than 0.43 at the L3 vertebral level.

The common terminology of “congenital” spinal stenosis may in fact be flawed; “developmental” may be a more accurate designation. These morphologic changes may not simply represent genetic variation, but rather reflect a developmental insult. Papp and colleagues studied patients with “congenital” spinal canal stenosis and noted significant correlations with prenatal factors. The spinal canal at the L3 level (>L4>L5) was found to be the most sensitive to the influence of prenatal events. In a multiple regression analysis, external factors accounted for 43% of the variance of the spinal canal cross-sectional area at L3; gestational age was by far the most significant factor. Shorter gestational age resulted in a smaller adult spinal canal. Significant factors of less importance included low placental weight, greater maternal age, primiparity, low socioeconomic class, and low birth weight. The L3 and L4 vertebral bodies are fully developed by age 1; L5, in contrast, is not fully mature until age 6. There is little opportunity for catch-up growth at L3 or L4 following a prenatal insult. The trunk and limbs can recover from a prenatal disturbance, as they continue to grow until adulthood, resulting in normal external patient morphology concealing a mid-lumbar developmental stenosis.

**Acquired spinal stenosis**

The great majority of patients presenting with NIC have acquired “degenerative” spondylotic change as their primary cause of central canal, subarticular recess, and/or foraminal compromise. As noted by Dr Bogduk earlier in this issue, the term “degenerative” is unduly pejorative and may contribute to negative patient perceptions regarding their prognosis. Maturational or age change is preferred.
The changes that result in compromise of the central canal are rooted in the 3-joint structure of the spine motion segment: the disc and the paired facet joints. In the anterior column, degradation of the nuclear compartment of the disc places excessive load on the posterior annulus, resulting in end-plate hypertrophs, or with annular failure, disc herniation. These changes encroach on the ventral aspect of the central canal or LRs. Loss of disc space height obligates narrowing of the neural foramina, and contributes to increased facet load and ultimately arthrosis; facet capsular hypertrophy and superior articular process (SAP) osteophytes compromise the subarticular recesses. Synovial cysts, particularly at the L4 level, may contribute to central canal, subarticular recess, or foraminal compromise. The reduced height of the segment, and loss of elasticity of the ligamentum flavum, result in its buckling centrally as a dominant cause of loss of cross-sectional area of the central canal. The ligamentum flavum may also thicken, although it is unclear if this represents true hypertrophy. It is known to undergo fibrotic chondrometaplastic change, diminishing its elasticity, and to calcify more commonly in patients presenting with NIC. Thickening of the ligamentum flavum has also been associated with arthropathy in the adjacent facet joints, suggesting there may be an inflammatory component to this process, independent of simple mechanical buckling of ligament into the central canal as a result of loss of disc height. These several anterior and posterior column phenomena conspire to narrow the central canal most commonly at the L4-5 disc level, followed by L3-L4, L5-S1, and L1-L2. This cascade of age changes compromise the central canal, subarticular recesses, and/or neural foramina; there are a number of measurable parameters that could quantify the degree of stenosis depicted by radiography, myelography, CT, or MRI. Verbiest, in his early descriptions of the entity of spinal stenosis, suggested that a 10-mm to 12-mm anterior-posterior (AP) diameter of the dural sac on conventional myelography constituted relative stenosis, with a measurement of less than 10 mm denoting absolute stenosis. Steurer
and colleagues,\textsuperscript{21} in a 2011 review, surveyed the numerous measurements applied by various investigators in the intervening decades in 25 unique studies and 4 systematic reviews. These parameters are detailed in Tables 1 and 2.

The most common descriptors of central stenosis include the AP dimension of the osseous canal or the dural sac and the cross-sectional area of the dural sac. Dural sac AP dimension of less than 10 mm or dural sac cross-sectional area of less than 100 mm\textsuperscript{2} constitutes stenosis. Descriptors of lateral recess or subarticular stenosis include the height and depth of the recess and the subarticular recess angle. Height is defined as the shortest distance between the most anterior point of the SAP and the posterior vertebral body, depth as the distance from the SAP to the junction of the pedicle and the vertebral body, and recess angle as the angle formed by the posterior vertebral body and the pars interarticularis. Although defined differently by different investigators, there is little real distinction between height and depth. Subarticular recess stenosis is typically defined as height less than 2 mm, depth less than 3 mm, or angle smaller than 30°. Descriptors of foraminal stenosis most commonly used suggest a diameter of 2 to 3 mm or smaller as indicative of stenosis. Steurer and colleagues\textsuperscript{21} correctly observed that the lack of a uniform quantitative description of anatomic central canal, subarticular recess, or foraminal stenosis confounds the evaluation of the role of imaging in the diagnosis of the clinical entity of NIC. This criticism was echoed by Genevay and associates,\textsuperscript{22} who lamented the high degree of variability in the eligibility criteria for studies examining neurogenic claudication caused by lumbar spinal...

Fig. 2. CT of developmental stenosis. This 24-year-old man presented with incapacitating leg pain with walking and voiding disturbance. His standing radiographs (A, B) show only short pedicles at L4 and L5. He was not MRI compatible. Sagittal CT reconstruction (C) demonstrates disc herniations at L4 and L5 that severely constrict the thecal sac. Axial CT images at L3 (D), L4 (E), and L5 (F) confirm severe central canal stenosis at both L4 and L5 owing to the disc herniations and a small canal. This case is unusual in that congenital central canal narrowing is usually maximal at L3. It does demonstrate the ability of CT to provide all the needed information for surgical planning.
<table>
<thead>
<tr>
<th>Imaging Method</th>
<th>Author</th>
<th>Site of Measurement</th>
<th>Level, Where Measured (Measurement Points)</th>
<th>Definition of Stenosis (Cut-Off Values)</th>
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<tbody>
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<td>Magnetic resonance imaging</td>
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<td>Midsagittal diameter of thecal sac</td>
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<td>Herzog</td>
<td>Midbody of each vertebra</td>
<td>Cross-section area of thecal sac area in % of normal midsagittal diameter: Grade 1: anterior &lt;15%, posterior &lt;10%; Grade 2: anterior 15%–30%, posterior 10%–20%; Grade 3: anterior &gt;30%, posterior &gt;20%</td>
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<td>Hamanishi</td>
<td>Intervertebral levels: L2/3, L3/4, L4/5</td>
<td>&lt;100 mm², at more than 2 of 3 intervertebral levels</td>
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<td>Laurencin</td>
<td>Motion segment: intervertebral disc level coincident with flexible joint; stable segment: level coincident with the midpedicle unaffected by stenosis</td>
<td>Stenosis ratio: Cross-sectional area of dural sac of motion segment divided by stable segment cross-sectional dural sac area: Level: L3–L4 &lt;0.66 L4–L5 &lt;0.62 L5–S1 &lt;0.73</td>
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<td>Ligamentous interfacet distance</td>
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<td>Herzog</td>
<td>Distance between the inner surface of flaval ligaments on a line connecting the joint space of facet joints at the level of the intervertebral disc</td>
<td>&lt;10 mm (L2–L3) &lt;10 mm (L3–L4) &lt;12 mm (L4–L5) &lt;13 mm (L5–S1)</td>
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<td>Koc</td>
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<td>Ullrich&lt;sup&gt;107&lt;/sup&gt;</td>
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<td>4 zones of measurement: upper, middle, lower zone of vertebral body and disc space</td>
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<td>Bolender&lt;sup&gt;111&lt;/sup&gt;</td>
<td>5-mm intervals from L2 to L5</td>
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<td>Haig&lt;sup&gt;23&lt;/sup&gt;</td>
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<td>&lt;15 mm (suggesting narrowing)</td>
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<td>&lt;10 mm (usually diagnostic)</td>
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<td>Midvertebral body level</td>
<td>10–12 mm (relative)</td>
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<td>Herzog&lt;sup&gt;104&lt;/sup&gt;</td>
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<td>Compression of thecal sac area in % of normal midsagittal diameter: Grade 1: anterior &lt; 15%, posterior &lt; 10%; Grade 2: anterior 15%–30%, posterior 10%–20%; Grade 3: anterior &gt;30%, posterior &gt;20%</td>
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<td>5-mm intervals from L2 to L5</td>
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<td>&lt;100 mm&lt;sup&gt;2&lt;/sup&gt; (present stenosis)</td>
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<td>&lt;10.5 mm (lower limit)</td>
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<td>&lt;5.5–7.0 mm (considerable)</td>
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<td>10–12 mm (relative)</td>
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<td>&lt;130 mm&lt;sup&gt;2&lt;/sup&gt;</td>
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Available at: [http://www.biomedcentral.com/1471-2474/12/175](http://www.biomedcentral.com/1471-2474/12/175). Accessed November 19, 2011.

stenosis, both radiological and clinical. The lack of a validated classification criterion impairs the quality of knowledge of this condition.

The multiplicity of quantitative parameters suggests that no single measurement has proven satisfactory. Indeed, the very notion of a readily quantifiable measure of stenosis may be flawed. There is wide individual variation in the size of the central canal and dural tube; the relative mismatch of these structures, exacerbated by spondylotic change, produces the increased pressure that results in venous congestion and Wallerian degeneration. Thus, any imaging criteria must address not just size of the dural tube or central canal, but imaging manifestations of crowding or compression of the neural elements. This was well described by Speciale and colleagues; in examining the reliability of rating of central canal stenosis they noted a very poor correlation between the cross-sectional area of the spinal canal and the degree of crowding of neural elements. Some patients in whom the central canal was stenotic by cross-sectional area criteria showed little crowding of neural elements, whereas other patients exhibited adequate cross-sectional area but significant crowding of the cauda equina. A qualitative assessment of crowding is indeed what most radiologists examine, and is the basis of the simplistic criteria put forth by the joint study commission of radiological and surgical societies.

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<th>Imaging Method</th>
<th>Author</th>
<th>Site of Measurement</th>
<th>Level, Where Measured</th>
<th>Definition Of Stenosis (Cut-Off Values)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Computed tomography</td>
<td>Ciric</td>
<td>Lateral recess height</td>
<td>Not reported</td>
<td>5 mm (normal)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>≤3 mm (highly indicative)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>≤2 mm (diagnostic)</td>
</tr>
<tr>
<td></td>
<td>Strojnik</td>
<td>Between the most medial point of the superior articular facet and the posterior point of the vertebral body</td>
<td></td>
<td>≤3.6 mm</td>
</tr>
</tbody>
</table>

### Depth of lateral recess

<table>
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<tr>
<th>Imaging Method</th>
<th>Author</th>
<th>Site of Measurement</th>
<th>Level, Where Measured</th>
<th>Definition Of Stenosis (Cut-Off Values)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dincer</td>
<td>Between superior articular facet and the top part of the pedicle.</td>
<td>&gt;5 mm (normal)</td>
<td>4–5 mm (Group 3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3–4 mm (Group 2)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>2–3 mm (Group 1)</td>
<td></td>
</tr>
<tr>
<td>Mikhael</td>
<td>Between the posterior surface of the vertebral body and the anteromedial portion of the superior articular facet at the level of the superior border of the corresponding level</td>
<td>&gt;5 mm (normal)</td>
<td>3–5 mm (suggestive)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>≤3 mm (definitive)</td>
<td></td>
</tr>
</tbody>
</table>

### Lateral recess angle

<table>
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<tr>
<th>Imaging Method</th>
<th>Author</th>
<th>Site of Measurement</th>
<th>Level, Where Measured</th>
<th>Definition Of Stenosis (Cut-Off Values)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strojnik</td>
<td>Between the bottom and the roof of the triangular space (= lateral recess)</td>
<td>&lt;30°</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
in 2001. This lexicon of spine “degenerative” conditions defines central canal stenosis as mild if there is compromise of one-third or less of the expected canal area, moderate if the compromise is one-third to two-thirds, and severe if the compromise exceeds two-thirds. This more subjective standard better addresses the crowding of neural elements, but has reliability challenges, described later.

The observation of nerve root redundancy as a qualitative marker of central canal compromise dates from the original description of the entity of spinal stenosis by Verbiest. This is presumed to originate from mechanical entrapment of the root at the site of compression, with subsequent elongation of the nerve above this site under the tensile stress of physiologic flexion and extension motion. Although frequently observed, this sign has been subjected to little study. Redundant nerve roots are present in 34% to 42% of surgical candidates with clinical NIC. In a 2007 study by Min and colleagues, redundant nerve roots were more commonly seen in older patients, but there was no significant association with duration of symptoms, diameter of the spinal canal, preoperative symptom intensity, or surgical outcomes. There was a nonsignificant trend toward poorer surgical outcomes in patients with redundant roots.

**Degenerative spondylolisthesis**

Degenerative spondylolisthesis was initially described by McNab as “spondylolisthesis with an intact neural arch.” It is highly associated with disk failure at this level, as well as significant facet arthrosis; it frequently results in single-level central canal compromise. Degenerative spondylolisthesis is present in 4% to 14% of elderly patients. It is most frequent at the L4 level, followed by the L5 and L3 levels. It is significantly more common in women than men.

As degenerative spondylolisthesis is usually present at a single segmental level, it most commonly causes radiculopathy or radicular pain rather than NIC. Associated foraminal stenosis or modest central canal or LR compromise at other levels can provoke NIC (Fig. 3).

**Reliability of imaging parameters**

Speciale and associates studied the reliability of a normal, mild, moderate, severe classification of central canal stenosis. They observed only fair interobserver reliability (κ = 0.26). Stratified by specialties, reliability was higher among radiologists (κ = 0.40), followed by neurosurgeons (κ = 0.21) and orthopedic surgeons (κ = 0.15). Intraobserver reliability was poor at κ = 0.11. Linear regression models showed that the classification was highly correlated with central canal area; that is, although the grade of stenosis at a given spinal level showed wide variability among observers, smaller canals were invariably diagnosed as more severely stenotic than larger canals. In this study, the observers were not given criteria or examples defining the mild, moderate, or severe classification.

In contrast, Lurie and associates studied the reliability of subjective grading of stenosis of the central canal, subarticular recesses, and neural foramina and measurement of central canal and dural sac area aided by specific definitions and imaging examples of the criteria. Stenosis was subjectively rated as none, mild, moderate, or severe using the Fardon and Millette definitions; nerve root compromise in the foramen was categorized as none, touching, displacing, or compressing. Inter-reader reliability in assessing the central canal was substantial, with κ = 0.73. There was moderate to substantial reliability for foraminal stenosis and nerve root impingement (κ = 0.58 and 0.51, respectively). Reliability for subarticular stenosis was only moderate at κ = 0.49. Intrareader reliability was greater than inter-reader reliability for all features. These results emphasize the importance of clear definition of criteria for reliable grading of stenosis by subjective scales.

**Specificity: correlation of imaging findings with clinical state**

The ultimate challenge in establishing the utility of diagnostic imaging in the diagnosis of NIC is the lack of a gold standard against which to measure imaging parameters. Surgical findings may be subjective. Clinical outcomes are highly dependent on the technical success of the instituted surgical therapy, and the outcome instruments used in any such measurement. Comparison against the best available cross-sectional imaging results in a circular argument.

The specificity fault in imaging of central canal stenosis can be seen in studies of asymptomatic volunteers. Boden and associates noted significant central canal stenosis on MRI in 21% of asymptomatic subjects older than 60. Jarvik and colleagues demonstrated that asymptomatic stenosis on MRI increases in prevalence with age: moderate to severe central canal stenosis was seen in 7% of subjects younger than 45, 6% of subjects age 45 to 55, 11% of subjects age 55 to 65, and in 21% of subjects older than 65.

Several studies involving patients with NIC suggested quantitative imaging correlates, which may aid in the diagnosis. The study of Hamanishi and colleagues showed that a decrease in the dural...
cross-sectional area (DCSA) to less than 100 mm$^2$ at more than 2 of 3 lumbar levels (L2–3, L3–4, L4–5) was highly associated with the presence of clinical NIC. Similarly, Bolender and colleagues$^{31}$ suggested that a DCSA of 100 mm$^2$ or less on CT was unequivocal evidence of central canal compromise. Schöneckstroem and Hansson,$^{32}$ in a cadaver study, demonstrated that a DCSA of 75 mm$^2$ or less corresponded to the initial increase in pressure in the cauda equina and represented a more physiologic marker of central canal stenosis.

On the contrary, numerous studies examining patients with NIC have shown a poor correlation between imaging parameters of stenosis and clinical state. Haig and colleagues$^{33}$ studied 126 patients stratified into 3 groups: those with no back pain, those with mechanical back pain, and those with NIC by clinical diagnosis. MRI measurements did not differentiate between patients with clinical NIC and controls by better than chance. Sirvanci and colleagues$^{34}$ examined 63 patients undergoing decompressive surgery for NIC. Morphologic stenosis was assessed by dural cross-sectional area (normal was greater than 100 mm$^2$; 76 to 100 mm$^2$ was moderately stenotic; less than 76 mm$^2$ was severely stenotic) and a 4-point grading of subarticular and foraminal stenosis. There was no correlation between any of the measured parameters in any spine compartment and patient disability as measured by the Oswestry Disability Index (ODI). This applied to both patients with multilevel central stenosis and a subset with degenerative spondylolisthesis.

Yasar and colleagues$^{35}$ performed a prospective analysis of 125 patients with a clinical diagnosis of NIC and anatomic central canal stenosis (DCSA <100 mm$^2$) who underwent surgical decompression. Preoperative evaluation included time to first claudicatory symptoms (FST) on treadmill testing, maximum walking distance (MWD), and ODI. There was no correlation between DCSA and the FST, MWD, or ODI preoperatively or at a 3-month postoperative evaluation. Results emphasize that the decision to operate and assessment at

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**Fig. 3.** Degenerative spondylolisthesis. Sagittal T1-weighted (A) and T2-weighted (B) images demonstrate grade 1 anterolisthesis of L4 on L5 with central canal compromise. Axial T2-weighted images through the L4 disc space (C, D) confirm severe central canal stenosis. Paramedian T2 sagittal images through the left and right foramina (E, F) show only modest foraminal narrowing on the right. This patient presented with a radicular pain syndrome (bilateral L5) rather than NIC, likely owing to the single-level compressive disease.
follow-up are better done with functional than anatomic parameters.

Geisser and colleagues, in a 2007 study of 50 patients with clinical NIC, showed no correlation between the AP dimension of the spinal canal (using 10-mm or 13-mm criteria for stenosis) and functional assessments of patient disability. Zeifang and colleagues, in a 2008 evaluation of 63 patients, found no correlation between MWD and DCSA. Although the NASS guidelines conclude that there is insufficient evidence to recommend for or against a correlation between clinical symptoms or function and the presence of anatomic narrowing of the spinal canal on cross-sectional imaging, the preponderance of evidence argues against such a correlation in this author’s judgment.

**Sensitivity: dynamic lesions**

There is also a basic sensitivity flaw in advanced imaging. NIC is by definition intermittent; most patients with NIC report exacerbation of symptoms with extension and weight bearing. The cross-sectional area of the central spinal canal, subarticular zone or lateral recess, and neural foramina are maximized with flexion positioning; the dimensions of these structures diminish with extension and axial load. Intradiscal pressures are significantly lower in a recumbent position than when sitting or standing. Schmid and associates noted a 40 mm² reduction in cross-sectional area of the dural sac at the L3-4 level in moving from flexion to extension. Danielson and Willen studied asymptomatic volunteers and noted a significant decrease in the DCSA with axial loading in 56% of the subjects, most commonly at L4-5. This finding was more common with increasing age. Decrease in DCSA with loading was less frequent in healthy volunteers than in a population of patients with NIC. In Willen and Danielson’s study, 80% of symptomatic patients crossed a threshold of relative (100 mm² or absolute (75 mm²) in DCSA when axial load was applied using a loading device on a standard MRI scanner. Willen and colleagues reported on the follow-up of 25 patients who underwent lumbar decompression based on central canal compromise detected solely on images obtained under axial load; 96% of the patients were improved or much improved regarding their clinical NIC after decompression. The proportion of patients with a walking capacity of 500 m increased from 4% preoperatively to 87% postoperatively. The NASS guidelines suggest that MRI or CT with axial loading is a useful adjunct to standard imaging in patients who have clinical signs or symptoms of NIC, a DCSA of less than 110 mm² at 1 or more levels, and suspected but not verified central or lateral stenosis on unloaded MRI or CT.

A 2009 study by Hansson and associates identified the ligamentum flavum as the greatest dynamic contributor to central canal compromise with axial load and extension. The average cross-sectional area of the central canal diminished by 23 mm² at the L3 disk level and 14 mm² at the L4 level under load. The ligamentum flavum was responsible for 50% of the reduction at the L3 level and 85% of the reduction at the L4 level. Madsen and associates attempted to distinguish between the effects of axial load and extension; their work suggested that lumbar spine extension is the dominant cause of reduction in DCSA in the standing patient. Axial load was less significant. The study of Feng and colleagues similarly identified a direct correlation between the degree of lumbar angular motion and the observed reduction in central canal diameter in the movement from flexion to extension. These studies would suggest that recumbent imaging in lumbar extension, with standard MRI scanners, may improve the sensitivity to the detection of dynamic lesions without the cost and diminished signal-to-noise ratio of dedicated upright scanners. This remains to be demonstrated in direct comparative studies.

**Imaging modalities: CT, CT/myelography, MRI**

There are no comparative effectiveness studies addressing the use of CT versus CT/myelography versus MRI in the documentation of central canal, subarticular recess, or foraminal stenosis using current technologies. The NASS systematic review of this question revealed that there is little strong evidence preferring MRI over CT or CT/myelography. The studies of Kent and colleagues, Bischoff and colleagues, Modic and colleagues, and Schnebel and colleagues show good agreement between the modalities and no substantive evidence for the superiority of any modality. Based primarily on its noninvasive character, and lack of radiation exposure, NASS suggests MRI as the most appropriate initial study in patients with clinically suspected NIC; CT/myelography or CT alone are appropriate examinations in the non–MRI-compatible patient (Figs. 4 and 5).

**Gadolinium**

MRI studies in patients with clinical evidence of NIC are typically performed without the administration of intravenous gadolinium, with the exception of the postoperative setting. Given the specificity challenges of purely anatomic imaging, the more physiologic parameter of gadolinium enhancement may have a role. This is not a new observation; Jinkins in 1993 observed abnormal
intrathecal nerve root enhancement at the site of stenosis on enhanced MRI in patients with NIC. He postulated that this represented breakdown of the blood–nerve barrier at sites of nerve root injury with subsequent Wallerian degeneration. This has been elegantly confirmed in a canine model by the recent work of Kobayashi and colleagues. Histologic examination demonstrated congestion and dilatation of intraradicular veins and an inflammatory cellular infiltrate at sites of gadolinium enhancement. Gadolinium enhancement may provide added specificity in the correlation of imaging and the clinical symptomatology of NIC; this remains to be proven in clinical studies.

Recent Observations

A 2010 study by Barz and colleagues described the “nerve root sedimentation sign” as a marker of symptomatic NIC. In patients without central canal compromise, the roots of cauda equina lie in the dorsal aspect of the dural sac on supine MRI. A positive sedimentation sign was defined as the absence of nerve root sedimentation to the dorsal dural sac on at least 1 image of axial MRI at a level above or below the zone of compression; the 2 nerve roots leaving the dural sac at the next most caudal segment are exceptions. This retrospective study used a total of 200 patients: 100 patients with low back pain but without clinical NIC, and a DCSA greater than 120 mm², and a cohort of 100 patients with clinical NIC, a maximum walking distance of less than 200 m, and a DCSA of less than 80 mm² on at least 1 level. There was no correlation between the smallest DCSA and patient disability as measured by the ODI. The sedimentation sign, however, was identified in 94 of the patients in the NIC cohort but in none of the low back pain group. It remains to be demonstrated that this sign provides additional specificity over quantitative measurement of the DCSA.

THORACIC SPINAL STENOSIS

Symptomatic central canal stenosis in the thoracic segment of the spine owing to age-related change is far less common than in the cervical and lumbar regions, likely because of the added mechanical stability imparted by the rib cage. Systemic disease...
accounts for a correspondingly greater proportion of cases. Systemic processes leading to thoracic central canal compromise include achondroplasia, osteochondrodystrophy, Scheuermann disease, diffuse idiopathic skeletal hyperostosis (DISH), and Paget disease. Age-related causes and select unique entities are addressed in the following paragraphs.

Compromise of the thoracic spinal canal may be manifest clinically as myelopathy, radiculopathy, or a mixed presentation. In a surgical series reported by Palumbo and colleagues, all patients reported pain at presentation; half exhibited a clinical myelopathy. In another surgical series of Chang and associates, a myelopathy picture dominated in 86% (spastic paraparesis, hyperreflexia, sensory level), whereas 14% exhibited a mixed pattern (paraparesis, radicular pain, and normal deep tendon reflexes). In this series, back pain was less frequent.

The segmental level of canal compromise is most commonly reported to be in the lower thoracic region. In the Palumbo and colleagues’ series, the stenotic zone was always in the lower half of the thoracic spine; two-thirds bridged the thoracolumbar junction, and most were multilevel. The larger series by Chang and colleagues had 54% of cases with lesions in the T9 to T12 region, with 25% from T5 to T8, and the remainder in the upper thoracic region. Earlier surgical series also demonstrated stenotic segments exclusively in the lower thoracic spine. Thoracic spine mobility, particularly flexion-extension motion, is greatest near the thoracolumbar junction, likely the biomechanical underpinning to this distribution of age-related pathology.

Posterior element age-related changes play a greater role in the genesis of thoracic central canal compromise. This takes the form primarily of unilateral or bilateral facet joint hypertrophy. Thoracic disc herniations or disc-osteophyte complexes may also contribute. Both the ventral and dorsal contributions to thoracic central canal compromise are well seen on CT, CT/myelography, and MRI.

Ossification of the thoracic ligamentum flavum (OTLF), although rare in a white population, is a well-recognized cause of myelopathy or mixed myelo-radiculopathy in an Asian population. The prevalence of OTLF in an Asian population is
estimated as 6.2% of men and 4.8% of women.\textsuperscript{53} The ossified, thickened ligamentum flavum is readily demonstrated by either CT or MRI, and is typically seen in the lower third of the thoracic region. Its pathogenesis is poorly understood. It may be associated with ossification of the posterior longitudinal ligament (OPLL), which is discussed later in this article.

Epidural lipomatosis is a rare cause of central canal compromise in the thoracic or lumbar spine; it may be idiopathic or secondary to endogenous or exogenous steroid excess. Obesity is a common factor in both groups. Excess epidural fat acts as a mass compressing the dural sac, most commonly from a dorsal vector in the thoracic region; it is more likely to be circumferential in the lumbar region. A recent literature review by Al-Khawaja and associates\textsuperscript{54} noted that thoracic involvement was more frequent in secondary versus idiopathic cases; lumbar involvement dominated in idiopathic cases. Conservative therapy with weight reduction (and removal of any endocrine stimulus) was beneficial when no neurologic compromise was evident; in the face of neurologic deficit, surgery was indicated. Surgical intervention was moderately successful (50%–60% full recovery) with the exception of secondary thoracic involvement, where surgical outcomes were much poorer (Fig. 7).

**CERVICAL STENOSIS**

Cervical spondylotic myelopathy (CSM) is the most common cause of spinal cord dysfunction...
This section examines the pathophysiology of CSM, the role of imaging in the detection and characterization of the underlying anatomic abnormality, and the use of imaging in the selection of patients for therapeutic interventions, particularly surgical decompression. It is insufficient to simply describe morphologic alterations in cervical spondylosis; the imager must be familiar with the literature identifying the clinical significance of imaging findings, guide the use of advanced imaging, and inform its use in patient selection for decompression. The prognostic significance of MRI findings in CSM are discussed in depth. The critical points are summarized in Box 2.

Stookey originally described CSM in 1928. Although its pathophysiology remains incompletely understood, it is widely acknowledged to involve static factors causing stenosis of the cervical canal and dynamic factors causing repetitive cord injury. These mechanical factors both directly injure neural tissue and initiate secondary ischemia, inflammation, and apoptosis. The histologic characteristics of CSM include cystic cavitation and gliosis of the central gray matter and demyelination of the medial portions of the white matter long tracks. There is Wallerian degeneration in the posterior columns and posterolateral tracts cephalad to the site of compression. Loss of anterior horn cells and corticospinal tract degeneration are seen at and caudal to the site of compression. Imaging correlates are discussed later in this section.

**Pathogenesis: Static Factors**

The developmentally narrow spinal canal is a more universal substrate for CSM than is the case with NIC in the lumbar region. The sagittal diameter of the adult spinal cord is nearly constant, measuring about 8 mm from C3 to C7; the cervical cord enlargement occurs primarily in the transverse plane. The normal cervical spinal canal sagittal diameter (posterior vertebral body to spinolaminar...
(C3–C7) in a white population; such subjects will rarely develop sufficient age-related change to provoke CSM. Edwards and LaRocca observed that patients with developmentally narrowed midcervical sagittal diameters smaller than 10 mm were often myelopathic, patients with canals of 10 to 13 mm were at risk for CSM, canals of 13 to 17 mm were seen in patients with symptomatic spondylosis but rarely myelopathy, and subjects with canals larger than 17 mm were not prone to develop spondylosis.

Morishita and colleagues recently examined the kinematics of subjects with congenitally narrow cervical canals, and noted significant differences when compared with normal canals. In congenitally narrowed canals, there is more segmental mobility in the lower cervical segments, C4-5 to C6-7, and significantly greater disc age-related change in the lower cervical spine. Hence, the individual with a congenitally narrowed canal is at risk both because of the limited space available for the cord, and a greater propensity to age-related spondylotic change (Fig. 8).

Acquired cervical central canal stenosis encompasses age-related spondylotic change (most common), OPLL, and ossification of the ligamentum flavum (OLF). As in the lumbar region, age-related degradation of the matrix of the disc nucleus transfers load to the annulus, resulting in disc-osteophyte complexes or protrusions encroaching on the ventral canal. Increased loading of the uncovertebral joints provokes hypertrophic change, compromising the lateral recesses and proximal neural foramina. These anterior column changes excessively load the facet joints, with resultant hypertrophy. The ligamentum flavum loses elasticity and buckles centrally. This cascade of events circumferentially narrows the canal and may directly compress the cord, exiting spinal nerves, and the anterior spinal artery. In Morio and colleagues’ surgical series, the most common levels of compromise were C3-4 (27%), C4-5 (37%), and C5-6 (29%). This series included patients with spondylosis only and patients with OPLL.

OLF is a multifactorial disease, whose genetic basis is a defect in the nucleotide pyrophosphatase (NPPS) gene. The prevalence is 1.9% to 4.3% of the Japanese population, and approximately 3.0% in Korea and Taiwan. It is implicated in up to 25% of the North American and Japanese cases of CSM. It has a significant association (up to 50%) with DISH, and is considered by some a subtype of DISH. It is progressive with age. OPLL imaging characteristics are discussed later in this section.

OLF is more common in the thoracic spine, where it was previously described. It may also extend into the cervical spine; its prevalence in the Japanese population older than 65 years is up to 20%.

Pathogenesis: Dynamic Factors

In addition to static compression, dynamic forces contribute to repetitive cord injury. Chen and colleagues measured dynamic flexion-extension changes in cadavers; the flexion to extension motion increased disc bulging by 10.8% of the canal diameter, and ligamentum flavum bucking by 24.3% of the canal diameter. Zhang and colleagues performed flexion and extension MRI studies on patients with CSM. The cross-sectional area of the central canal was greater in the neutral position than in either flexion or extension; it was greater in flexion than extension. There was functional cord impingement in 12% of patients in flexion, 34% in the neutral position, and 74% in extension. Muhle and colleagues noted an increase in central canal stenosis in 48% of patients moving from the neutral to an extension position, with 20% exhibiting cord

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**Box 2**

**Cervical spinal stenosis**

The clinical syndrome of cervical spondylotic myelopathy (CSM) very frequently occurs in the setting of a congenitally narrowed spinal canal.

Fixed stenosis, dynamic injury with motion, and a secondary cascade of ischemia, inflammation, and cell death cause the clinical syndrome of CSM.

The specificity fault common to all spine imaging persists in the cervical region: patients meeting objective criteria of central canal stenosis may be asymptomatic.

Intramedullary T2 hyperintensity may reflect a spectrum of pathology, from reversible edema to demyelination and cystic necrosis; more intense and well-defined T2 hyperintensity suggests irreversible injury.

Intramedullary T1 hypointensity implies irreversible necrotic change.

Intramedullary gadolinium enhancement is a negative prognostic finding in patients with CSM.

Diffusion tensor imaging is more sensitive than T2 hyperintensity in the detection of early cord injury; decreased fractional anisotropy values at the site of compression are the most reliable parameter.

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line) is 17 to 18 mm (C3–C7) in a white population; such subjects will rarely develop sufficient age-related change to provoke CSM. Edwards and LaRocca observed that patients with developmentally narrowed midcervical sagittal diameters smaller than 10 mm were often myelopathic, patients with canals of 10 to 13 mm were at risk for CSM, canals of 13 to 17 mm were seen in patients with symptomatic spondylosis but rarely myelopathy, and subjects with canals larger than 17 mm were not prone to develop spondylosis.

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compromise; the movement from neutral into flexion increased central canal stenosis in 24% and cord compromise in 11%. Cervical neural foramina diminish in cross-sectional area in extension, and increase slightly in flexion. In addition to the mechanical trauma to the cord imparted by static compression or dynamic compression and tension, secondary biochemical processes, especially ischemia, contribute to the development of CSM. Ventral compression of the cord compromises flow through the arterioles arising from the anterior spinal artery in the ventral sulcus; dorsal compression reduces perfusion to the central gray matter. Oligodendrocytes are extremely sensitive to ischemic injury; resultant apoptotic cell death may cause the demyelination characterized seen in CSM. Recent animal evidence further supports the role of an inflammatory cascade and Fas-mediated apoptosis in CSM. In an animal model, an antibody directed against the Fas-ligand diminished the inflammatory response and increased neurologic recovery from compressive injury. Such medical management may become a useful adjuvant to surgical decompression.

**Imaging**

Imaging of patients with CSM may include radiographs, CT, CT/myelography, and MRI. Imaging parameters, reliability, specificity challenges, correlation with clinical CSM, and imaging predictors of response to surgical decompression are discussed.

Historically, radiographic assessment of cervical anatomic stenosis relied on the AP dimension of the central canal as measured from the...
posterior vertebral body to the spinolaminar line. As noted previously, the normal sagittal diameter from C3 through C7 is considered to be 17 to 18 mm. Edwards and LaRocca\textsuperscript{56} noted that a cervical canal with a sagittal diameter smaller than 13 mm was at risk for myelopathy; absolute stenosis was defined as being smaller than 10 mm. To compensate for magnification variables, Pavlov and Torg (Pavlov and colleagues\textsuperscript{64}) promulgated the ratio of the sagittal diameter of the spinal canal to the sagittal diameter of the mid-vertebral body; a value greater than 1 was regarded as normal, and a value less than 0.82 was considered absolute stenosis. More recent studies have shown a poor correlation between the Pavlov/Torg ratio and the space available for the cord, suggesting its usefulness is limited.\textsuperscript{65}

The advent of cross-sectional imaging has allowed us to directly measure the diameters and cross-sectional areas of the cervical spinal canal and the cervical cord. MRI has also given us the ability to evaluate physiologic parameters: T2 hyperintensity, T1 hypointensity, gadolinium enhancement, and, with diffusion tensor imaging (DTI), fractional anisotropy (FA) and apparent diffusion coefficient (ADC).

Reliability
A 2010 study by Naganawa and associates\textsuperscript{66} demonstrated good intraobserver and interobserver reliability in evaluation of the cross sections of the cervical canal and spinal cord with both CT/myelography and MRI. They noted that dural sac diameter and cross-sectional area measurements were slightly but significantly larger with CT/myelography than fast spin-echo T2-weighted MRI; conversely, the diameters and cross-sectional areas of the spinal cord were slightly but significantly larger with MRI. These findings may reflect halation by the myelographic contrast material, overestimating the CSF space. They also evaluated a semiquantitative 4-point rating scale of the degree of stenosis; the interobserver kappa values were 0.69 for CT/myelography and 0.68 for MRI. MRI graded the stenosis as slightly, but significantly, more severe than CT/myelography, likely because of the halation effect. This study examined fast spin-echo T2-weighted images; it could be anticipated that T2$^*$ (gradient echo) imaging would have further accentuated this effect. Note that the very good reliability figures in this study may reflect the use of defined criteria and representative images in training observers. A similar 2009 study by Song and associates\textsuperscript{67} found no significant difference in interobserver or intraobserver reliability between CT/myelography and MRI. With its superior spatial resolution, CT/myelography was somewhat better in assessment of foraminal stenosis, and much better in discriminating bony versus soft tissue lesions. MRI was more reliable in identifying direct nerve root compression. An earlier (1999) study by Shafai and colleagues\textsuperscript{58} showed a poorer agreement between MRI and CT/myelography; given the interval evolution in technology, this is of questionable relevance.

Imaging specificity: correlation with pathophysiology, clinical state
As in the lumbar region, there is a basic specificity fault in all cervical imaging: subjective and quantitative evidence of central canal compromise may be seen in asymptomatic subjects. The prevalence of asymptomatic findings increases with age. Teresi and colleagues\textsuperscript{69} studied asymptomatic volunteers 65 and older with MRI; 57% exhibited cervical disc protrusions and 7% had frank cord compression. Hayashi and colleagues\textsuperscript{70} noted 10% of subjects older than 60 to demonstrate significant stenosis in canal diameter (<13 mm) without signs or symptoms. Matsumoto and colleagues,\textsuperscript{71} in a large study of 500 asymptomatic subjects, observed direct cervical cord compression in 8%.

When a population of patients with a clinical diagnosis of CSM is studied, the correlations appear more favorable. The transverse area of the spinal cord as measured by MRI correlates well with the severity of myelopathy and the pathologic changes seen in the cord in CSM.\textsuperscript{72,73}

The physiologic parameters of T2 hyperintensity or T1 hypointensity have provided further insight into the evolution of CSM. Takahashi and colleagues\textsuperscript{74} initially suggested that T2 hyperintensity within the cord represents the myelomalacia or gliosis seen pathologically in patients with CSM. Ramaanuas and colleagues\textsuperscript{75} considered that early in the process of myelomalacia evolution, T2 hyperintensity may reflect edema; this may progress to cystic necrosis of the central gray matter, which will ultimately manifest itself as T1 hypointensity and T2 hyperintensity. Al-Mefty and associates\textsuperscript{76} considered T2 hyperintensity to represent myelomalacia, with T2 hyperintensity accompanied by T1 hypointensity to reflect cystic necrosis or syrinx formation. Direct correlation with histology in a canine model by Al-Mefty and associates\textsuperscript{76} showed motor neuron loss, necrosis, and cavitation in areas of cord signal abnormality. Correlation with human autopsy findings led Ohshiro and colleagues\textsuperscript{77} to conclude that T2 hyperintensity alone represents edema, gliosis, and minimal gray matter cell loss, whereas T1 hypointensity heralds necrosis.
myelomalacia, and spongiform change. The observation that T2 hyperintensity may resolve led Taneichi and colleagues\textsuperscript{78} to ascribe it to reversible edema, whereas stable T2 hyperintensity reflects gliosis (Fig. 9).

From these studies, and additional outcomes studies detailed in the following paragraphs, we can conclude that

1. Intramedullary T2 hyperintensity represents a range of reversible (edema) and irreversible (demyelination, gliosis, cystic necrosis) pathology
2. Faint and indistinct T2 hyperintensity is more likely to reflect reversible edema
3. Very intense and well-defined T2 hyperintensity more likely represents fixed gliosis or cystic necrotic change
4. Intramedullary T1 hypointensity represents irreversible necrosis and myelomalacia.

A 2010 study by Ozawa and colleagues\textsuperscript{79} and a 2011 study by Cho and colleagues\textsuperscript{80} compared patients with CSM who exhibited gadolinium enhancement with a nonenhancing control group. The zone of enhancement was always within and smaller than a zone of T2 hyperintensity at the site of maximal compression, with extension caudally; it was typically seen in the posterior or posterolateral cord. There was no correlation of enhancement with preoperative clinical symptoms. Enhancement disappeared in most patients within 1 year of surgical decompression; patients who exhibited preoperative enhancement had a poorer postoperative prognosis than those who did not (Fig. 10).

Floeth and colleagues\textsuperscript{81} studied 20 patients with CSM using 18F-fluorodeoxyglucose (18F-FDG) positron emission tomography (PET) in the setting of a single-level stenosis at C3-4 or C4-5. All the patients with CSM showed a significant decrease in 18F-FDG uptake in the lower cord below the stenosis, relative to healthy controls. A cohort of these patients also exhibited increased uptake at the level of the stenosis. The patients with increased 18F-FDG uptake at the stenosis had a significantly shorter duration of symptoms, a more precipitous decline in function.

Fig. 9. Cervical spondylotic myelopathy. This patient had a moderate myelopathy preoperatively. Sagittal T2 (A, B) and T1 (C) images show severe central canal stenosis at the C4 level with impingement from both ventral and dorsal vectors. Discrete, well-marginated T2 hyperintensity is seen off the midline (B). Axial gradient echo T2-weighted image (D) confirms the cord deformity, central disc protrusion, and left >right T2 hyperintensity. Post decompression sagittal T1 and T2 images (E, F respectively) show that the central canal is well decompressed, with some reexpansion of the cord. The zone of T2 hyperintensity, however, has expanded, also demonstrated on axial FSE (G) and gradient echo (H) images. The patient had a fixed deficit without clinical improvement. The well-marginated intramedullary T2 hyperintensity preoperatively was a negative prognostic sign, as was the postoperative expansion of the T2 hyperintensity.
in the 3 months before decompression, and ultimately exhibited significant improvement after decompression. Patients without increased uptake at the stenotic zone did not recover neurologic function after decompression. Animal experiments have shown increased immunoreactivity (postulated to cause increased metabolic activity) in neurons and glial cells in the early stages of cord compression; chronic compression leads to cell loss, atrophy, and diminished metabolic activity.

**Imaging correlates: diffusion tensor imaging**

Early reports suggest DTI may offer greater accuracy in identification of symptomatic cord compromise than T2 hyperintensity or T1 hypointensity. The measured parameters are FA, mean diffusivity (MD), or ADC. The ADC or MD values reflect overall diffusivity in the tissue irrespective of directional dependence. Anisotropy (directional dependence) of diffusion in white matter tracts results from linearly oriented membrane structures (ie, axons and myelin). Diminished FA values may reflect loss of directionally oriented membrane structures, increased extracellular edema, or both. Animal studies have demonstrated that diminished FA values are seen in mechanical disruption, tearing of fibers and myelin sheaths, Wallerian degeneration, and demyelination. Kara and colleagues speculate that decreased FA values in the spinal cord devoid of T2 signal abnormality represent early demyelination owing to oligodendrocyte ischemia and subsequent apoptosis. Direct histologic confirmation of this attractive hypothesis is not yet available.

Several recent studies have addressed DTI in patients with CSM. Kara and colleagues examined 16 patients with a clinical diagnosis of CSM but no T2 hyperintensity within the cord. All of the patients showed a statistically significant reduction in FA values and an increase in ADC values at the site of maximum stenosis, prompting the hypothesis that low FA reflects early demyelination. Facon and colleagues studied DTI parameters in 15 patients with cervical or thoracic cord compressive lesions, both spondylotic and metastatic. FA values in the compressed cord were reduced compared with healthy controls in 10 patients; ADC values were significantly increased in only 2 patients. Seven patients showed T2 hyperintensity in the cord. One patient with an acute clinical onset of myelopathy had an increased FA value. Facon and colleagues suggest this may be because of acutely restricted diffusivity; all other compressive processes have shown decreased FA.

Budzik and colleagues studied 20 patients with symptomatic CSM and 15 volunteers. FA values were significantly lower at the compressed levels in patients than at comparable uncompressed levels in volunteers. There was a significant correlation between clinical state as measured by validated functional scores and the FA values. There was no such correlation between clinical state and the T2 hyperintensity; T2 hyperintensity did not correlate with FA or MD values. Patients who exhibited T1 hypointensity in the cord showed very low FA values.

Finally, Kerkovsky and colleagues studied 52 patients with CSM and 13 volunteers using DTI. They demonstrated good interobserver reproducibility in DTI values. The patients all exhibited cord compression, but were stratified into those...
exhibiting signs and symptoms of CSM and those with nonspecific neck pain. The morphologic parameters of AP dimension of the central canal or cross-sectional area of the spinal canal did not discriminate between symptomatic and asymptomatic patients. The FA values were significantly reduced and ADC values elevated at the site of maximal compression in the symptomatic patients with CSM compared with the patients with cord compression but no symptoms. The FA and ADC values also discriminated between the 2 patient subgroups versus healthy controls.

DTI holds significant promise in identifying patients early in the course of CSM who are at risk for progression. Currently available evidence suggests diminished FA is more sensitive in the detection of cord injury than T2 hyperintensity and is better correlated with symptoms. This may assist in selection of patients for surgical decompression, although additional work remains to be done.

**Imaging: prognosis, selection of patients for decompression**

The ultimate goal of imaging must be to improve the clinical outcomes of patients. In the CSM population, this currently implies timely and appropriate selection of patients for therapeutic interventions, primarily surgical decompression. There is a large body of literature that has examined the role of imaging in predicting clinical response to surgical decompression; this literature is reviewed, hopefully adding clarity to a sometimes confusing mass of work. The imaging parameters under consideration include the cross-sectional area of the cord, intramedullary T2 hyperintensity, including its degree of intensity and multifocality, intramedullary T1 hypointensity, change or stability of intramedullary signal after decompression, recovery of cord cross-sectional area after decompression, and intramedullary gadolinium enhancement. The correlates are summarized in Box 3.

There is a consensus that there is a negative correlation in surgical prognosis with cross-sectional area of the cord at the site of compression; patients with greater degrees of cord compression do less well.\(^{72,86,87}\)

There is reasonable consensus that symptomatic patients with CSM who have no intramedullary signal change have a better prognosis for surgical decompression than those who exhibit T2 hyperintensity.\(^{74,75,80,86,88,89}\) There are contradictory studies in which focal T2 hyperintensity did not greatly affect prognosis.\(^{87,90,91}\) The studies of Shin and colleagues,\(^{92}\) Zhang and colleagues,\(^{61}\) Yasutsugu and colleagues,\(^{93}\) and Mastronardi and colleagues\(^{86}\) all semiquantitatively scored the intensity of T2 signal elevation on 3-point or 4-point scales; patients with intense T2 signal abnormality had the longest duration of disease and the poorest objective recovery rates. More intense and well-defined intramedullary T2 signal likely reflects fixed cavitative disease as opposed to reversible edema. The study of Avadhani and colleagues\(^{94}\) also used a 3-point evaluation of T2 intensity, but did not see a difference in recovery rates. The systematic review of patient selection by Munmaneni and colleagues\(^{95}\) noted there is also evidence that multilevel intramedullary T2 hyperintensity predicts a poorer outcome from surgical decompression than single-level T2 hyperintensity.

There is a strong consensus that intramedullary T1 hypointensity is a very poor prognostic sign for recovery following cervical decompression.\(^{59,76,86,90,91,94–96}\) The diminished T1 signal represents cavitative change or necrotic tissue. Patients exhibiting intramedullary T1 hypointensity recovered less well than patients with T2 hyperintensity alone, and much less well than patients without intramedullary signal abnormality. As noted previously, intramedullary gadolinium enhancement is a negative prognostic sign for postoperative recovery.\(^{75,80}\)

### Box 3

**CSM prognostic factors for surgical decompression**

1. Intramedullary T2 hyperintensity diminishes prognosis relative to normal signal.
   - a. Intense, focal T2 hyperintensity is a more negative prognostic sign than ill-defined hypointensity.
   - b. Multilevel T2 hyperintensity is a more negative prognostic sign than single-level change.
   - c. Resolution of T2 hyperintensity postoperatively improves prognosis.
   - d. Expansion of T2 hyperintensity postoperatively diminishes prognosis.
2. Intramedullary T1 hypointensity greatly diminishes prognosis.
   - a. Evolution of T1 hypointensity postoperatively diminishes prognosis.
3. Intramedullary gadolinium enhancement greatly diminishes prognosis.
4. Increased metabolic activity at the site of compression on 18F-FDG PET improves prognosis over no increased activity.
5. Postoperative residual compression and failure of reexpansion of the cord cross section are negative prognostic signs.
The regression of intramedullary T2 hyperintensity following surgical decompression has also been studied. The consensus of multiple studies is that patients who undergo regression of T2 hyperintensity on postoperative MRI examinations recover better than those in whom T2 hyperintensity is stable.\textsuperscript{59,74,86,89,90} Mastronardi and colleagues\textsuperscript{86} reported several patients who exhibited regression of T2 hyperintensity on intraoperative imaging obtained immediately after decompression, emphasizing that T2 hyperintensity may reflect rapidly reversible edema. Patients with preoperative T1 hypointensity never exhibited improvement in this signal abnormality postoperatively in any study.

Avadhani and colleagues\textsuperscript{94} described patients who developed T1 hypointensity postoperatively; as would be anticipated, they did very poorly.\textsuperscript{94} Yagi and colleagues\textsuperscript{97} described a cohort of patients who developed expansion of a zone of T2 hyperintensity postoperatively. The recovery rates were significantly reduced in these patients; the development of increasing T2 hyperintensity was significantly related to development of instability (defined as >3 mm subluxation) postoperatively, as well as residual cord compression.

Postoperative cord morphology may also affect prognosis; it is not surprising that patients with no residual cord compression did better.\textsuperscript{59,90} Evidence of cord reexpansion in the postoperative period is also a positive prognostic sign.\textsuperscript{59,86}

**Ossification of the Posterior Longitudinal Ligament**

As is noted previously, OPLL is implicated in up to 25% of patients with cervical myelopathy, and it is

![Fig. 11. OPLL. This 77-year-old white man had slowly progressive myelopathy on clinical examination. Lateral radiograph (A) demonstrates OPLL in the upper cervical spine; note the coexistent manifestations of DISH. CT sagittal reconstruction (B) and sagittal T2-weighted (C) and T1-weighted (D) images better depict the severe cord deformity at C2. The patient underwent C2 to T2 instrumented fusion, and C1 to C5 decompression (E). He is neurologically intact. Another patient (F) demonstrates coexistence of DISH and OPLL.](image)
typically grouped with age-related changes into the CSM population. Most of the previously referenced studies describing the pathophysiologic correlates of imaging and prognostic signs for decompression include large proportions of patients with OPLL. Like age-related causes of structural central canal narrowing, it is often asymptomatic. The ossification is most common in the cervical region, where it causes static narrowing of the canal; repeated impact of the ventral cord on the bony mass also contributes to myelopathic injury. Patients may present in their 40s and 50s with pain, chronic myelopathy, or acute neurologic injury after modest trauma. The natural history of the ossification is progression. From this flows the recommendation for surveillance imaging and close clinical follow-up (Fig. 11).

The ligamentous ossification may be identified on radiographs, CT, and MRI. On lateral radiographs, reduction of the sagittal canal diameter available for the cord by more than 60% correlates strongly with myelopathy. The ossification is located from C2 to C4 in 70% of cases, T1 to T4 in 15%, and L1 to L3 in 15%. On CT, the ossification may be classified as segmental (posterior to individual vertebrae: 39%), continuous (bridging across vertebrae: 27%), mixed type (29%), and other (ossification posterior to discs, variable sagittal extension: 5%). It may also be classified as central or laterally deviated. Matsunaga and colleagues noted that clinical myelopathy was more frequent in the laterally deviated type. They also observed that myelopathy was more frequent when the range of flexion-extension motion was greater, emphasizing the importance of dynamic trauma to the cord.

CT imaging is also critical to identify characteristics that suggest penetration of the dura. CSF leaks are a significant risk in anterior decompression surgery, particularly when the dura is ossified/inseparable from the bony ligamentous mass. Hida and colleagues described the single-layer and double-layer signs. A double-layer sign describes ossification continuous with the dorsal vertebral body, an intervening layer of hypodense hypertrophied PLL, and another central ossified layer. The single-layer sign implies a single homogenous ossified mass. The double-layer sign is strongly associated with no identifiable dural plane at surgery and significant risk of postoperative CSF leak. Epstein noted that a large laterally deviated single-layer mass that hooked in a “C” configuration was also seen in association with local dural penetration.

On MRI, mature ligamentous ossification is of diminished signal intensity on all sequences; early OPLL may have inhomogeneous signal and exhibit slight enhancement. When mature, there is no enhancement, allowing differentiation from epidural fibrosis. The secondary signal alterations in the cord are described previously.

SUMMARY

This article has reviewed pertinent pathophysiology, imaging findings, and the predictive value of imaging when evaluating patients with the clinical syndromes of NIC and CSM. Key points are summarized in the boxed comments. It should be apparent that several of the themes of this volume are carried through: (1) imaging has significant specificity and sensitivity faults; (2) the imager must understand the clinical state to provide useful information to the referring physician; and (3) imaging parameters that are based in physiology will be more valuable than a purely morphologic evaluation of spinal structures.

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