Transverse axial tomography of the spine

Part 2: The stenotic spinal canal

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The authors describe the diagnostic value of transverse axial tomography in
developmental lumbar stenosis, spondylosis, facetal hypertrophy, and other abnor-
malities that can constrict the spinal canal.

KEY WORDS • axial tomography • developmental stenosis •
facetal hypertrophy • spondylosis • lumbar disc syndrome
• laminectomy failure • spondylolisthesis • fusion overgrowth

AXIAL radiographs provide a transverse
cross-sectional view of the spinal
canal, and can reveal bone pathology
and constrictions of the canal not seen by
routine radiography or myelography because
of the overlapping of radiological shadows.
Axial tomography can demonstrate
axial distortion of the canal in lumbar
stenotic conditions and can assess the role of
the articular processes and the dorsal arch in
these states. 4, 5

Verbiest 49, 50 first recognized the clinical and
surgical significance of structural stenosis of
the lumbar canal; he found that stenosis was
due to posterolateral encroachment on the
canal by the articular processes rather than a
primary coronal narrowing secondary to
medial displacement of the pedicles. In his
studies of normal disarticulated lumbar
vertebrae, radiographs, and surgical cases, he
found that interpedicular measurements were
usually standard and that the dimensions of
the canal depended on the position, size, and
shape of the articular processes and the dorsal
arch. Operation revealed these structures to
be hypertrophied and distorted, suggesting
that the critical portion of the normal and
pathological canal is dorsal to the pedicles.

Epstein, et al., 9, 11 and Schatzker and Pen-
nal 42 later stressed the importance of patho-
logical changes of the articular processes in
developmental stenosis and spondylosis.
Prominent, bulbous, articular processes
characteristically expand into the dorsal sec-
tion of the canal, constricting the lateral
recesses and the lateral walls medially. 14 The
dorsal arch is distorted by thickened,
vertically-oriented, sclerotic laminae. 18 The
ligamentous interlaminar space is often lost,
and as a consequence the enclosed lumb-
osacral roots and the cauda equina are
compressed.

This report is a study of the lumbar spinal
canal with stenotic pathology. Methodology
and axial anatomy of the normal lumbar
spine have been described previously. 27

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**Fig. 1.** Tomogram (left) and line drawing (right) showing developmental stenosis at the L-4 level. The dorsal arch of L-4 is markedly sclerotic and thickened and the inferior articular processes (if) are bulbous, causing dorsal and dorsolateral constriction of the canal. la = lamina; sf = superior articular process or superior facet.

**Fig. 2.** Tomogram (left) and line drawing (right) showing developmental stenosis at the L-4 level, with associated giant bulbous articular processes leading to circumferential narrowing. A thin column of Pantopaque can be seen. See Fig. 1 for abbreviations.

**Congenital or Developmental Stenosis**

Cross-sectional tomograms in developmental stenosis reveal that the maximum point of stenosis is within the L-4 articular segment of the canal, although the problem may be diffuse and multileveled. A general narrowing of the canal is more marked within the articular segment, regardless of the level of involvement, and the pedicles forming the intrasosseous segment are in a normal position. This encroachment on the canal occurs initially in the dorsal half when the laminar arch becomes sclerotic and thickened, and so constricts the canal medially (Fig. 1). The normal ligamentum flavum is interposed between successive laminae in the interlaminar space; in developmental stenosis, apposition of the laminar arches and the articular processes causes the entire dorsal wall of the canal to become bony.

The articular processes, especially the inferior ones, that form the lateral and dorsolateral walls of the lower lumbar canal, also become sclerotic and bulbous (Fig. 2).26,21,40 The medial aspects of the inferior articular processes bulge convexly into the dorsal half of the canal. The canal loses its normal outwardly convex shape and assumes a scalloped, biconcave configuration.11,12 A deep constricted midline posterior recess usually develops between the medial surfaces of the articular processes. If prominent pathological lateral recesses are associated with a deep dorsal recess, the canal has a trilobed configuration.

With more advanced hypertrophy of the articular process, there is progressive bone encroachment into the ventral portion of the

**Fig. 3.** Tomogram (left) and line drawing (right) showing developmental stenosis at the L-5 level, with asymmetry of the inferior articular processes (if) at L-5. The canal is constricted dorsally by the thickened laminarch and dorsolaterally by the articular processes forming a ventral recess on one side and diffuse flattening of the lateral wall on the opposite side. sp = spinous process; sf = superior articular process or superior facet.
canal. If the inferior articular process is more involved than the superior process, a pathological gutter develops ventral to the overhanging inferior facet. When the subjacent superior articular process is also distorted and hypertrophied, the entire lateral wall is constricted medially, with encroachment on the lateral gutter and deformation of the lateral wall of the canal (Fig. 3). The canal then may appear as a markedly flattened tube. This may be misinterpreted as resulting from medial placement of the pedicles, but is actually caused by a steady overgrowth of facetial bone into the canal from the medial edge of normally placed pedicles.1,2,16,18,22,24,25

Spondylosis

Primary, degenerative spondylosis is characterized by hypertrophy and subluxation of the articular facets, sclerotic, thickened intervertebral disc, hyperplasia or ossification of the ligamentum flavum, and the development of ventral osteophytes and ridging of the canal.8,10,16,21,29,36,46,48 The constriction is multisegmental.8,52 Lumbar spondylosis is pathologically indistinguishable from "developmental" lumbar stenosis when the latter does not have medial displacement of the pedicles.

Cross sections of the canal in lumbar spondylosis reveal primarily posterolateral and lateral encroachment on the dorsal half of the canal by hypertrophied and sclerotic articular processes. The dorsal arch is thickened and sclerotic, although the severe degree of laminar convergence seen in true developmental stenosis is not usually present (Fig. 4). Paradoxically, this allows the canal to widen dorsal to the hypertrophied articular processes. The canal's greatest narrowing is the midfacet point at the middle of the artroposterior dimension of the canal (Fig. 5). The width of the ventral portion of the canal at the level of the pedicles or the intervertebral foramina is normal; the result is an hourglass constriction. When ventral overgrowth of the articular processes also encroaches on the ventral portion of the canal, a concentric constriction of the canal appears that is indistinguishable from developmental stenosis.

Most commonly, the articular facets are asymmetrically involved in degenerative lumbar spondylosis, giving the canal a markedly irregular configuration.21,51 Hypertrophy of the facets is most apparent in the large inferior articular processes.17,19,27,41 This leads to the formation of prominent, asymmetrical, deep lateral recesses, ventral to the medially convex inferior articular process and medial to the subjacent superior articular process (Figs. 6 and 7). In developmental stenosis, similar deep recesses are seen with associated facetial hypertrophy or spondylosis, although the typical finding in developmental stenosis is loss of the lateral recess and constriction of the lateral wall.

The spondylotic recess or gutter must be distinguished from the developmental lateral recess described by Schlesinger,65,64 and Epstein, et al.12 In the latter case, the recess is due to developmental malrotation, frontal orientation and medial overhang of a mildly hypertrophied superior articular process. The recess in these cases is ventral to the superior articular process; in the spondylotic recess, the gutter is ventral to the inferior articular facet. Since the inferior facet is medial to the superior articular process, the spondylotic recess extends much further medially into the canal. Epstein, et al.,12 noted the developmental recess in 3% to 4% of their cases. In the axial studies, there have been only scattered examples, although there have been many instances of the deeper and more medial spondylotic recess.

In addition to primary spondylosis, many cases of focal spondylosis6,16,41,47,52 occur secondary to or associated with discogenic disease at one level.38,41,47,52 In focal spondylosis, the articular process ipsilateral but occasionally contralateral to the clinical symptoms shows evidence of hypertrophy and sclerosis (Fig. 6).40 This leads to the formation of focal, irregular lateral recesses with asymmetrical lateral and dorsolateral constriction of the canal (Fig. 7). This secondary focal spondylosis may be seen concomitantly with acute disc herniation,16,31,40 suggesting the more longstanding degenerative process,36 or as a sequel to the conservative or surgical treatment of herniated discs.14,32,37,40 Follow-up axial tomograms at future intervals may demonstrate the progressive nature of this apophyseal hypertrophy along with the later development of deep spondylotic gutters and distortion of the lumbar canal.
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FIG. 4. Tomogram (left) and line drawing (right) showing focal thickening and sclerosis of the L-4 lamina arch secondary to spondylosis. The thickening is asymmetrical at the level of the L-4 intervertebral disc leading to unilateral foraminal encroachment. See previous figures for abbreviations.

FIG. 5. Tomogram (left) and line drawing (right) showing lumbar spondylosis at the L-4 level. There is marked asymmetry of the hypertrophied inferior articular processes (if), and constriction of the lateral walls with relative preservation of the anteroposterior diameter of the canal in the midsagittal plane. Asymmetric lateral recesses can be seen ventral to the bulbous inferior articular processes. See previous figures for abbreviations.

FIG. 6. Tomogram (left) and line drawings (right) showing focal spondylosis at the L5-S1 level. There is focal sclerosis with mild hypertrophy of the inferior articular process (if) of L-5 unilaterally, and a medial convex bulge of the pathological apophyseal joint constricting the canal. Note the normally medially concave configuration of the articular processes on the opposite side. See previous figures for abbreviations.

FIG. 7. Tomogram (left) and line drawing (right) showing focal hypertrophy of the inferior articular process (if) of L-5 unilaterally. A bulbous L-5 process forms a deep pathological recess (lr) ventral to the inferior articular process and medial to the superior process (sf) of S-1. The canal is distorted dorsolaterally with relative preservation of the ventral aspect of the canal. See previous figures for abbreviations.

Ventral Overgrowth of Spinal Fusions

Ventral overgrowth of posterior lumbar fusions into the lumbar canal leads to bone constriction and encasement of multiple lumbosacral roots and is a recognized iatrogenic cause of lumbar stenosis. McNab and Dall noted that over 20% of their patients subjected to posterior fusions required subsequent decompression for stenosis. Such patients may actually have had undiagnosed bone stenosis and facet hypertrophy prior to the fusion. In these cases, axial tomograms reveal an abnormal canal, typical of developmental stenosis or spondylosis with an associated bone fusion mass presenting exterior to the laminar arch dorsally or dorsolaterally (Fig. 8).

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diverse radicular symptoms and signs. Plain radiographs usually reveal dense sclerotic bone over the dorsal arches with no accurate way of ascertaining if the bone is encroaching on the canal. Axial tomography eliminates the overlap of shadows and provides a cross-sectional view of the canal so that ventral overgrowth is easily detectable.

**Spondylolisthesis**

Spondylolisthesis has long been recognized as a common cause of lumbar stenosis and intermittent claudication of cauda equina. Pathologically and radiologically, spondylolisthesis can be subdivided into spondylolisthesis and pseudospondylolisthesis. Although the latter is less common, it has a greater association with symptomatic lumbar stenosis.

Spondylolytic spondylolisthesis may occur at any level, although the great majority of cases are at L-5. In L-5 spondylolytic spondylolisthesis, marked axial distortion of the canal appears in the anteroposterior plane. The articular facets have been abnormal in the six cases we studied. The inferior articular processes of the rostral subluxed vertebra are densely sclerotic, hypertrophied, and irregular, and display obvious lateral encroachment of the canal.

Pseudospondylolisthesis, with no defect in the pars interarticularis, is due to degenerative molding of the articular processes and progressive slipping of the entire rostral vertebral body and its attached dorsal arch forward onto the more caudal vertebra. This is most commonly seen at L-4. Cross-sectional study of the canal at the involved level in two cases has demonstrated marked constriction of the canal (Fig. 10). The dorsal arch and the attached inferior articular processes slip forward and are ventral to the adjacent superior facets, which have associated sclerosis. The anterior displacement of the inferior processes relative to the subjacent superior facets does not occur in other stenotic conditions, nor in the normal spine.

**Discussion**

In addition to providing a direct radiological cross-sectional view of the canal and aiding in the diagnosis of lumbar...
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stenosis, axial tomography of the lumbar spinal canal clarifies the true pathological anatomy of lumbar stenosis, facetal hypertrophy, degenerative spondylosis, spondylolisthesis, and pseudospondylolisthesis.

Routine radiological studies reveal neither the distortion of the canal nor the hypertrophy of the dorsal arch and articular processes that are characteristic of lumbar stenosis and spondylosis. Plain radiographs do not provide an accurate means of delineating the margins of the canal. The overlapping of radiological shadows prevents accurate delineation of the structures involved, and the dense sclerotic bone commonly seen with lumbar stenosis makes this differentiation even more difficult. The shape of the canal cannot be demonstrated nor extrapolated from routine radiographs.

Myelography gives an indirect gross indication of the shape and size of the canal, but the large epidural space in the lower lumbar spine renders myelography relatively insensitive to early overgrowth and encroachment of bone. Significant bone hypertrophy and constriction of this space may occur without deformity of the dural sac. Again, the overlapping of multiple opacified shadows may lead to a false estimation of the size of the dural sac or may hide pathological recesses.

Surgical and pathological descriptions of the stenotic canal have identified the bone structures constricting and distorting the lumbar canal, but the demonstration of the abnormal configuration of the canal has been limited to the study of disarticulated vertebrae. The relationship of the pathological dorsal arch and articular processes has not been truly appreciated.

From the pathological and surgical descriptions of developmental stenosis and primary degenerative spondylosis, the predominant locations and sources of encroachment are apparently the hypertrophied articular processes and dorsal arch. Axial studies of the lumbar canal in both of these conditions have demonstrated these abnormalities clearly, regardless of the presence or absence of abnormal pedicles, ventral ridging, or disc disease.

The most vulnerable parts of the vertebra are the articular processes and their extension into the dorsal laminar arch. As Verbiest observed, and as axial study of the normal lumbar spine shows, the dorsal half of the canal, especially at L-4 and L-5, is particularly vulnerable to change in the facets or dorsal arch.

It must be stressed that these radiological studies reveal only bone constriction of the canal. When soft tissue structures are involved, the encroachment and constriction of the abnormal canal may be even more marked with or without significant myelographic change.

Although all of these pathological observations regarding the stenotic canal have been described individually, diseases of the dorsal arch and the articular processes have not been presented as a pathological continuum. Pathological descriptions and cross-sectional studies of the canal in stenotic conditions appear to justify an attempt at more precise definition, since the various conditions leading to lumbar stenosis are often indistinguishable clinically, causing a spectrum of signs and symptoms from simple back and radicular pain to intermittent claudication related to the cauda equina. Lumbar stenosis is a frequent cause of the lumbar disc syndrome, and axial tomography allows the preoperative detection of those common bone conditions whose symptoms mimic the lumbar disc syndrome. It also offers a means of evaluating the patient who shows new or recurrent symptoms after laminectomy or fusion.
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