Pathoanatomy of lumbar disc degeneration and stenosis

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This paper details the characteristic pathoanatomical features of the normal lumbar spine and of degenerative changes of the lumbar motion segment that cause both central and lateral canal stenosis. Since the pathoanatomical changes are degenerative derangements of the normal motion segment, the normal anatomy is briefly outlined. The terminology adheres to the Glossary on Spinal Terminology published by the American Academy of Orthopedic Surgeons' Committee on the Spine with the exception of the term "spine" which is replaced by "spinous process" and "vertebral canal" which is used synonymously with the established term "spinal canal" (1).

Conventional dissection, by definition, inevitably destroys and distorts potentially crucial topographic-anatomical relationships. By contrast, sectional images from *in situ* frozen specimens show the unequivocal relationships of the musculoskeletal spinal column to the delicate neurovascular spinal elements. Moreover, freezing specimens in functional postures also allows to study functional topographic relationships and the effect of extension, rotation and bending (and combinations of the above) on the neurovascular spinal elements in both normal and degenerated specimens because these relationships are "frozen" (2, 20). Plain radiography and high resolution CT-scanning of the specimens prior to to sectioning on the cryomicrotome also facilitates accurate and direct point-to-point radio-anatomical correlations.

A distinction should be made between the *vertebral column* and the *spine*. The mobility and motion pattern greatly varies between the different storeys of the spine. Every spinal motion segment has a typical range and pattern of motion that is determined by the shape and three-dimensional arrangement of bony and articular surfaces, intervening discs, ligaments and joint capsules (24). Mobility is also determined and controlled by the insertion and origin of the muscles that attach to the spine, their mass, fiber composition and orientation in space.

Diagnostic imaging correlations

Among the computerized tomographic diagnostic imaging modalities that have evolved within a few years, high resolution computed tomography (CT) depicts the osseous changes in exquisite detail. Magnetic resonance imaging (MR) depicts the spinal soft tissues with increasingly high resolution and in exquisite detail. MR also provides physiological information on blood circulation, inflammatory and neoplastic changes with high sensitivity and rapidly improving specificity. Both CT and MR have significantly contributed to the understanding of a variety of stenosing processes of the spinal spaces by degenerative changes, not only by bony overgrowth, but they by soft tissue compromise (16, 22, 23, 25). Dynamic radiographic studies such as functional myelography have shown that the dimensions of the spinal canal change dramatically in flexion, extension and rotation of the spine (9, 11, 15).

Overall spinal anatomy

Figures 1 to 7 illustrate both normal and degenerative anatomy of the lumbar spine. The vertebral column houses osseoligamentous spaces which accommodate, protect and transmit the delicate neurovascular structures. These spaces are the vertebral canal, and the two intervertebral foramina (root canals) at each spinal level. The root canal morphology significantly varies from one vertebral level to the next and they also display a wide and significant range of interindividual variations with respect to size
and configuration. The most mobile portions of the spine are the cervical and the midlumbar spine (17, 19, 21, 26).

The lumbar spinal canal can be divided into a central portion and two lateral portions. The central portion represents the central spinal canal, whereas each lateral portion constitutes a root canal or radicular canal. The central portion of the spinal canal is the round area occupied by the thecal sac. Stenosis of the

central portion of the vertebral canal has often been called central spinal stenosis in order to distinguish it from lateral spinal stenosis, corresponding to root canal stenosis. This terminology implies that in central spinal stenosis the root canals may not be stenotic. However, this is not the case because stenosis of the central portion is consistently associated with stenosis of the lateral corners of the spinal canal (12, 21).

Intervertebral disc anatomy

At the thoracolumbar junction, the intervertebral discs rapidly increase in height. The anterior wall of the vertebral canal is straight and the intervertebral discs have no tendency to bulge into the canal. The lower lumbar vertebrae are slightly wedge-shaped, causing the lumbar lordosis and lumbosacral angulation. The lumbar pedicles are strong and project posteriorly behind the vertebral bodies. An important exception is the pedicle of L5 which is broader and takes an oblique posterior and lateral course.

The intervertebral discs increase in thickness relative to the height of the vertebrae (Figures 1 and 2). Whereas the discs of L1 and L2 have a straight posterior margin, the discs of L3, L4 and L5 have a distinctly posteriorly convex, bulging configuration. Contrary to the discs of the thoracic spine, where the peripheral layers of the annulus fibrosus insert into the apophyseal ring, the outer annular lamellae of the lower lumbar discs insert beyond the assimilated ring apophysis into the vertebral bodies, blending with their Sharpey's fibers of the periosteum. This normal disc architecture is shown in sagittal CT and MR scans and should not be confused with pathological bulging of the discs due to degeneration or instability.

Only at the L5-S1 disc level a small amount of areolar soft tissue may be interposed between the dura and the annulus. At all other levels the dura is firmly attached to the disc. At the disc level the ventral internal veins have merged into one vertically running venous trunk which connects adjacent retrovertebral veins in a "ropeladder" fashion (14). This vascular architecture is apparent in ascending epidural phlebograms. In the lumbar spine, the thecal sac is normally round.

The *posterior longitudinal ligament* attaches to the endplate portion of the vertebrae and to the posterior circumference of the intervertebral discs where it fans out to a thin fibrous layer of diverging collagenous strands, indistinguishably interwoven with the outermost lamellae of the annulus fibrosus. At the pedicle level this ligament measures barely one millimeter square in axial cross section (Figure 3). Biomechanical studies attest to its low tensile strength. It bridges the concavity behind the vertebral bodies in a bowstring

fashion, creating an osseomembranous compartment for the voluminous ventral internal venous plexus.

The ventral internal veins communicate with a system of venous channels that transgress the midportion of each vertebra through vascular foramina that commonly are referred to as the Batson vein outlets. The posterior longitudinal ligament is a suspensory ligament for the thecal sac and transmits the movements of the spinal column to the position of the thecal sac in the vertebral canal (28). The posterior longitudinal ligament thus appears to be another component of the suspensory Hofmann ligaments (10). It also has a rich nervous and vascular supply. This ligament also has a rich supply of sensory nervous fibers and probably acts more as a strain gauge type of positional transducer than an actual mechanical-ligamentous restraint. Hypermobility and/or reduction of the intervertebral disc height (e.g. following chemonucleolysis) may cause a relatively sudden decrease in foramen dimensions and compression of the root which may be further accentuated in extension and rotation.

Intervertebral foramina or root canals

The root canals—anatomical concepts rather than canals *sensu strictu*—are tubular conduits in which the nerve root bundles take their course from the lateral aspect of the dural sac to their entrance into the intervertebral foramen. The root canals consist of three components: the proximal (subarticular or intervertebral) portion, the distal portion that corresponds to the lateral recess, and the pedicle zones, i.e. the lateral corners of the vertebral foramen at the level of the pedicles (3).

The *proximal portion* of the root canal is bounded anteriorly by the intervertebral disc and posterolaterally by the anterior border of the superior articular process which is covered by the ligamentum flavum, which at these levels merges with the articular capsule of the facet joint. The root canals are defined as the tubular structures in which the "nerve root" takes its course from the lateral aspect of the dural sac to the entrance into the intervertebral foramen.

The *distal portion* of the root canal begins at the level of the superior endplate and ends at the entrance into the intervertebral foramen. The root canal does not have a real medial wall, but is rather bounded by the lateral aspect of the dural sac. The root canal may be stenotic in the absence of a significant narrowing of the central spinal canal or in isolated root canal stenosis. The neuroforamen proper is the irregularly shaped exit zone or outlet of the root canal. Root canal stenosis is a common condition, either isolated or associated with stenosis of the central spinal canal. By contrast, compression of the root bundle at the foraminal outlet

is rare. The division of the root canals into three por-tions (proximal, intermediate and distal), brings confu-sion into the taxonomy and complicates the terminol-ogy of the stenotic conditions.

Dynamic neurovascular compression

The sagittal dimensions of the spinal canal increase in flexion and decrease in extension of the spine. The flexion, the laminae of two adjacent vertebrae move apart and the interlaminar space widens by the length-ening and thinning of the ligamentum flavum. The sagittal dimensions of the spinal canal increase in flex-ion and decrease in extension of the spine. Concur-rently, the vertebral bodies move closer anteriorly and further apart posteriorly, stretching the posterior por-tion of the annulus fibrosus. These changes increase the length and the sagittal dimensions of the spinal canal. The opposite occurs in extension of the spine: the ligamenta flava shorten, become thicker and buckle and the annulus fibrosus bulges posteriorly, causing a decrease of the sagittal dimensions of the canal at intervertebral level.

Changes in size and shape of the vertebral canal that occur in flexion or extension are mainly caused by thickening and retraction of the ligamenta flava and they involve the central area of the spinal canal with the thecal sac, which undergoes constrictions at inter-vertebral level, often associated with a simultaneous dilatation at the midvertebral levels. Dynamic changes in the root canal, while less dramatic than those of the central spinal canal, are caused by bulging or increased bulging of the posterolateral region of the disc into the subarticular portion of the root canal and buckling of the anterior capsule of the facet joint covered by the ligamentum flavum as a result of a downward sliding of the inferior articular process with respect to the superior during extension of the spine (Figure 4).

Rotational deformation of the vertebral canal affect the subarticular portion of the root canal. Torsion of a vertebra with respect to the vertebra below results in a posterior displacement of the lateral portion of the proximal vertebra and increased posterior bulging of the disc on the side towards which torsion occurs. On this side the root canal decreases in width. This may be cause compression of the nerve root or increased com-pression if the canal is narrow or stenotic.

In most instances the vertebral canal has a sufficient reserve space to accommodate the nervous structures, even in its decrease size caused by extension of the spine. If this reserve space is limited by pre-existing stenosis of the vertebral canal, (for example, in spines with short pedicles), even minor additional narrowing causes compression of the nervous and vascular struc-tures. Changes in size and shape of the vertebral canal

occurring in flexion and extension, particularly those caused by the ligamenta flava, essentially involve the central area of the spinal canal and thus the dural sac, which undergoes constrictions at intervertebral level, often associated with a simultaneous dilatation at mid-vertebral level. Dynamic changes in the root canal are less marked than in the central spinal canal. They are caused by bulging or increased bulging of the postero-lateral region of the disc into the subarticular portion of the root canal and buckling of the anterior capsule of the facet joint covered by the ligamentum flavum as a result of a downward sliding of the inferior articular process with respect to the superior during extension of the spine.

Rotational forces affect the subarticular portion of the root canal. Torsion of a vertebra with respect to the vertebra below results in a posterior displacement of the lateral portion of the proximal vertebra and marked posterior protrusion of the disc on the side towards which torsion occurs. On this side the root canal decreases in width and this may be responsible for compression of the nerve root or may increase com-pression if the canal is narrow or stenotic. The dimen-sions and the volume of the lumbar spinal canal vary considerably in both flexion and extension and rotation of the motion segment. Dynamic variations in flexions and extension are related to changes in bulging of the intervertebral disc and in thickness and buckling of the ligamentum flavum. Rotation mainly affects the root canals which narrows due to both changes in the soft tissues and vertebral displacement.

Movement of the vertebral column dramatically affects the size and volume of the vertebral canal and foramina (root canals). These canals contain the spinal cord and the lumbosacral roots of the cauda equina which are contained and ensheathed by the thecal sac, a tubular structure which is continuous at the foramen magnum level with the thecae of the skull. The dura mater spinalis is continuous with the dura mater of the skull. It is completely lined internally by the arachnoid membrane. The thecal sac with its subarachnoid space is filled with the cerebrospinal fluid in which the spi-nal cord and the cauda equina roots and some free sub-arachnoid blood vessels float freely. The neural thecal envelopes attain fixation to the vertebral column at the level of the neuroforamina where a variable number of cauda equina roots converge towards a strut-shaped infundibular expansion of the thecal sac. The free intrathecal roots become ensheathed by the meninges; whereas the arachnoid membrane terminates at the medial border of the ganglion, the dura is contiguous with the capsule of the ganglion and the perineurium of the segmental nerves.

Whereas the intrathecal neural elements are mobile, the thecal sac is segmentally attached to the vertebral

Figure 1. Sagittal section through a normal lower lumbar spine and upper sacrum of a young female adult at the level of the lateral portion of the thecal sac. This level displays the segmental root bundles converging towards each intervertebral level. The L3, L4 and L5 discs all display a slight posterior convexity and attachment of the peripheral layers of the annulus fibrosus into the border of the vertebral bodies some millimeters beyond the apophyseal ring. The vertebral bodies all display concavities posteriorly and at L5 a large venous vascular foramen. These concavities are occupied by epidural fat and the ventral internal venous plexus which communicates with the veins crossing the vertebral body through the vascular foramina. Note that the posterior vertebral body wall is weak at the level of these foramina and that fractures usually run through these channels. The laminae have a characteristic shape in sagittal cross section. Towards the vertebral canal only a narrow vertical band of cortical bone is exposed. Superiorly the laminae have a sharp ridge. The lamina is sloping postero-inferiorly and the ligamentum flavum attaches to a large area of its inferior, posteriorly receding surface. At the infrajacent lamina the ligamentum flavum attaches to the sharp upper ridge and a small area posterior to this ridge. Note that the ligamentum flavum at the lumbosacral level is much thinner than at the levels above. Anomalies and variations are common in the transitional lumbosacral segment. In this specimen a very thin "ligamentum flavum membrane" bridges the posterior arch equivalents of the first and second sacral segment. The cul de sac (here filled with blood-tinted CSF) terminates slightly below the S1-S2 level which is delineated by the vestige of the S1 disc.

Figure 2. Sagittal section through the pedicles of the same specimen. The discs display the texture of the lamellae of the lateral portion of the annulus fibrosus. Some of the endplates show markedly concave configuration. The pedicles ae relatively short, causing an inverted teardrop shape of the foramina. The important neurovascular structures are located in the "subpedicular notch" which corresponds to the deep incisura vertebralis inferior of the vertebral body. The foramen cross-section can be divided into a subpedicular (vertebral) portion and a disc- or retrodiscal portion. The former is bounded by bone anteriorly and superiorly and by ligamentum flavum/joint capsule posteriorly, the latter is frequently obliterated (especially at L4-L5) because the disc inserts into the upper aspect of the pedicle and physiologically "bulges" and because the joint capsule or ligamentum flavum broadly attaches to the anterior surface of the superior articular process. This attachment usually extends to the upper surface of the pedicle. Note the dark dorsal root ganglia and the ventral roots at L4 and L5.
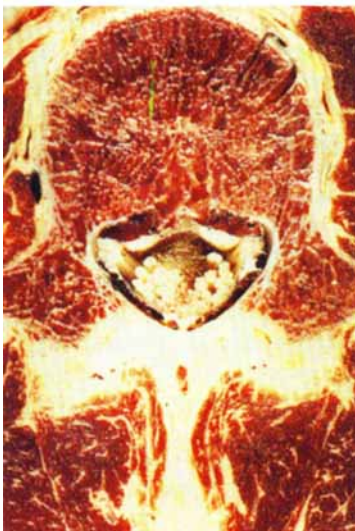

Figure 3. Axial section through a typical vertebra at the level of the mid-pedicle. The cancellous bone is coarse and strong. Venous vascular channels traverse the vertebral body and transgress the cortical bone posteriorly in the midline and anterolaterally on the right side. Anteriorly, the vertebra is braced by the thick and wide anterior longitudinal ligament. The posterior longitudinal ligament is narrow and thin and always attached to the dura. Behind the base of the transverse processes a pointed bony spike, the accessory process, lies exactly in the trajectory along which screws typically are inserted into the pedicle. This explains occasional difficulties encountered during percutaneous insertion of pedicle screws which tend to slide out of the "bullseye" image of the pedicle. Posteriorly, the upper sharp margin of the lamina and of the spinous process are visualized. The thecal sac is displayed at the level of the axillary outpouchings of the dura which constitute the steeply inferiorly directed offset of the root sleeves. Posteriorly the thecal sac is bounded by the sublaminar veins. These dorsal internal veins are far less voluminous than the ventral internal veins.

Figure 4. Sagittal high power photograph of the L4-L5 facet joint and foramen in a spine which was positioned in extension in the intact cadaver and frozen in situ to maintain the undistorted soft tissue-bone relationships. The extension was done without axial loading. The extension induces a sagittal rotation of L4 posteriorly on L5. In addition, the slight obliquity of the facet joint forces a slight posterior translation of L4. Extension causes the tip of the inferior articular process to hit the pars interarticularis of L5 which also is hugged from inferiorly by the superior articular process (SAP) of S1. The joint capsule is elongated and severely compressed against the interarticular pars. The degree of angulation is obvious from the wedge-shaped opening of the superior joint space into which a loose areolar meniscoid synovial tag is projecting. The ligamentum flavum joint capsule attaches broadly to the anterior surface of the SAP. It is pushed into the subpedicular notch and compresses and flattens the dorsal root ganglion posteriorly. Anterior to the ganglion the separate ventral roots are discernible.
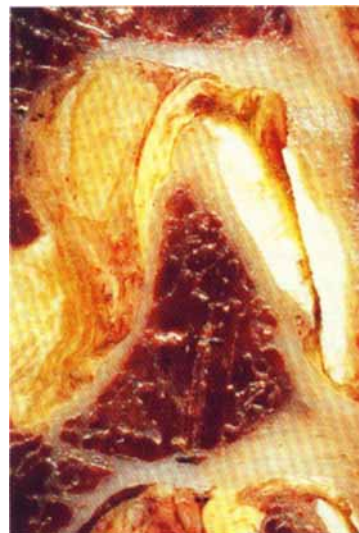

Figure 5. Severe degenerative spinal stenosis at the L4-L5 level in a 70 year-old man who had a history of intermittent claudication. The central and lateral stenosis is most pronounced at this motion segment level. The encroachment is almost exclusively caused by soft tissues. Anteriorly, the circumferentially "balooning" disc narrows the thecal sac anteriorly and also completely obliterates the retrodiscal portion of the root canals. The facet joints, especially the superior articular processes are moderately hypertrophied, rendering a ball-and-socket configuration of the facet joints. Note the effusion posteriorly of the left facet joint and the sclerotic meniscoid tag posteriorly into the right facet joint. The thecal sac is severely compressed posterolaterally by the thick ligamentum flavum and assumes a triangular slit-shaped configuration in which the roots of the cauda equina are tightly packed without any cerebrospinal fluid surrounding them. The two ligamenta flava are continuous posteriorly with the thick and degenerated interspinous ligament.

Figure 6. A few millimeters above the level of Figure 5 the morphology changes dramatically. The posterior contour of the vertebral body appears beveled and slightly V-shaped. From posteriorly large enthesopathic insertion- site osteophytes of the ligamentum flavum into the superior articular processes cause complete obliteration of the subpedicular notch and of the lateral recess. The ganglia are seen laterally, flattened against the inferior surface of the pedicles by the disc underneath (see Figure 5). Anteriorly in the vertebral canal, thick and congested veins are visible (black). The thecal sac is flattened from the sides but much has a much wider diameter in this less mobile portion of the motion segment and the thin cauda equina roots are surrounded by CSF.
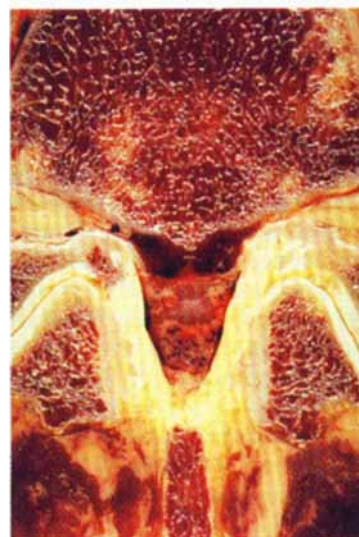
column by several important ligaments and membranes some of which are commonly known as the Hofmann ligaments. The thecal sac does not expand appreciably when intrathecal pressure raises because the dura is not very elastic, but the thecal sac cannot resist compression. The size of the vertebral canal and its volume increases slightly in flexion and decreases in extension. The size and volume of the intervertebral foramina (root canals) increases and decreases dramatically in flexion as well as extension. These volume changes are most pronounced in the cervical spine. The thecal sac is surrounded by epidural fat and epidural veins. The latter are somewhat arbitrarily divided into the anterior and posterior internal veins. The former constitute a plexus of veins behind the vertebral bodies, the latter, fewer and smaller, line the inner aspect of the laminae.

The ligamenta flava may be thick and the articular processes hypertrophic as a result of degenerative changes, which are generally mild since this type of stenosis is usually observed in middle or early senile age due to the shortness of the pedicles, the intervertebral foramina are narrow and slit-shaped in the sagittal plane, whereas the vertical dimensions are generally normal on account of the normal height of the intervertebral discs (19, 31). The middle and outer portions of the neuroforamina may be abnormally narrow due to the posterolateral bulging of the intervertebral discs, but the roots are rarely compressed, since they occupy the cranial portion of the foramina.

Degenerative changes of the motion segment

Degenerative changes of the spine occur at the level of the mobile portion of the segment and only in rare cases at the level of the bony ring (pedicles and lamina). At the disc level the ballooning of the disc anteriorly and the marked thickening of the ligamenta flava in combination with the hypertrophy of the superior articular processes compress the vertebral canal to a small triangular conduit in which the thecal sac is compressed to a small tube in which the roots lie tightly packed. In the same specimen, the subpedicular portion of the foramen is severely encroached on by arthrosis-type of osteophytes at the insertion areas of the ligamentum flavum. The various components of the nerve roots are compressed from inferiorly by the protruding disc and posteriorly by the large facet joint osteophytes and pressed and flattened against the unyielding pedicle (4, 6, 8, 13, 27, 30).

At the disc level the ballooning of the degenerated disc anteriorly and the marked thickening of the ligamenta flava in combination with the hypertrophy of the superior articular processes may compress the vertebral canal to a small triangular conduit in which the thecal sac is compressed to a small tube in which the roots lie tightly packed and the subpedicular portion of the foramen may be severely encroached on by arthrosis-type of osteophytes at the insertion areas of the ligamentum flavum. The various components of the nerve roots are compressed from inferiorly by the protruding disc and posteriorly by the large facet joint osteophytes and pressed and flattened against the unyielding pedicle.

In most instances, stenosis is produced by spondylotic changes in subjects in whom the spinal canals have sagittal or interarticular dimensions at the lower limits of normal. In developmental, degenerative or combined stenoses, compression of the nervous structures occurs exclusively at intervertebral levels, since only this portion of the canal is delimited by anatomic structures which may undergo degenerative changes. At the level of the posterior wall of the vertebral body the spinal canal may be narrow but never stenotic, i.e. responsible for compression of the neural contents.

The spinal soft tissues play a decisive role in the compression of the neurovascular structures. Degenerative spondylolisthesis may be associated with spinal stenosis if the canal dimensions are below normal the normal limits. In the presence of a developmentally large spinal canal significant compression of the neural elements may occur only when vertebral slipping is marked and/or degenerative hypertrophy of the facet joints are severe.

The bulging of the intervertebral discs into the spinal canal is markedly increased. The ligamenta flava may be mildly thickened and the articular processes moderately hypertrophic due to arthrotic changes. Degenerative hypertrophy of the superior articular processes, associated with shortness of the pedicles, is responsible for abnormal narrowing of the nerve root canals. The spinal canal, when seen in a midsagittal section, does not show the markedly sinusoidal morphology, which is typical of the former type of idiopathic developmental stenosis and achondroplastic stenosis. Instead, it appears narrow all or almost all its length and shows area of further narrowing at the intervertebral levels as a result of disc bulging, buckling of the ligamenta flava and/or hypertrophy of the articular processes. Narrowing is cause by the arthrotic changes of the facet joints and vertebral slipping such as in degenerative and isthmic spondylolisthesis.

Degenerative changes of the posterior joints consist in a hypertrophy and reactive remodeling of the articular processes which grow longer anteromedially and posterolaterally and tend to assume a transverse orientation this abnormal orientation is probably related to the abnormal reciprocal relationship of the facets as a result of vertebral slipping. It cannot be excluded that

the process of remodeling represents a compensation mechanism aimed at limiting vertebral slipping. Degenerative changes of the superior articular processes are responsible for narrowing of the root canals. Narrowing may be slight and not cause compression of the spinal nerve roots or may produce stenosis of varying entity of the root canals. When hypertrophy and outgrowth of the medial border of the facets are severe, the latter encroach on the central spinal canal and may compress, even markedly, the dural sac.

The hypertrophied inferior articular processes narrow the posterior portion of the central spinal canal which is further constricted to a significant extent by vertebral slipping. In fact due too the integrity of the posterior vertebral arch, the dural sac follows the slipped vertebra and distally to this undergo a sort of guilloutinement between the intervertebral disc and the posterior superior endplate of the vertebra below, on one side and the superior articular processes of this vertebra and the posterior arch of the slipped vertebra as well as the ligamenta flava on the other side. The pathological effects of degenerative changes of the articular processes and olisthesis are strictly related to the original dimensions of the vertebral canal. If the latter is wide and vertebral slipping of slight entity, compression of the dural tube may not occur. When the spinal canal is of medium size, the dural sac is usually encroached upon at the level of its lateral portions, which are in direct contact with the medial border of the facet joints. I the dimensions of the spinal canal are at the lower limits of normal or below these, marked compression of the dural sac and the emerging nerve roots occurs even in the presence of mild degenerative changes of the articular processes or moderate vertebral slipping.

Isthmic spondylolysis

In spondylolisthesis a highly complex pathoanatomical situation is found. The instability owing to the insufficient restraint of the posterior elements inevitably entails early and severe degeneration of the disc which allows pathological translatory and rotatory movements. The "beak" of the pars interarticularis is typically dislodged anteriorly and inferiorly and plunges into the interlaminar ligamentum flavum, pushing it anteriorly against the upper endplate of the vertebra below and the thin remnants of the posterior annulus fibrosus, both compressing the root components from inferiorly.

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Degenerative spondylolisthesis

Narrowing is caused by the arthrotic changes of the facet joints and vertebral slipping. Degenerative changes of the posterior joints consist in a hypertrophy and a sort of remodeling of the articular processes which grow longer anteromedially and posterolaterally and tend to assume a transverse orientation this abnormal orientation is probably related to the abnormal reciprocal relationship of the facets as a result of vertebral slipping. It cannot be excluded that the process of remodeling represents a sort of compensation mechanism aimed at limiting vertebral slipping. Degenerative changes of the superior articular processes are responsible for narrowing of the root canals. Narrowing may be slight and not cause compression of the spinal nerve roots or may produce stenosis of varying entity of the root canals. When hypertrophy and outgrowth of the medial border of the facets are severe, the latter encroach on the central spinal canal and may compress, even markedly, the dural sac (5, 18, 29).

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The intervertebral disc below the olisthetic vertebra may show a normal or decreased height. In both instances the disc is degenerated and bulges into the

spinal canal to a carrying extent, but it never shows a true herniation. In transverse sections the spinal canal is mildly narrowed at the level of the inferior endplate of the slipped vertebra due to degenerative hypertrophy of the inferior articular processes of the olisthetic vertebra and posterior bulging of the intervertebral disc. Narrowing increases opposite to the intervertebral disc and the superior endplate of the vertebra below the olisthetic one, while at the level of and distal to the pedicle of the latter the spinal canal regains a normal shape and size.

Spinal canal stenosis

Spinal canal stenosis includes primary, secondary, combined and mixed forms. In *primary stenosis,* narrowing of the lumbar spinal canal is caused by congenital malformations- congenital stenosis- or a defective postnatal development of the lumbar vertebrae—developmental stenosis. The latter may have a known etiology as in achondroplasia or be idiopathic. In primary stenoses, compression of the nervous structures is exclusively or essentially due to the congenital malformation or developmental narrowing. An example of developmental lumbar spinal stenosis is found in specimens with constitutionally short pedicles. In such specimens, the dorsal wall of the vertebral bodies display a marked concavity which commonly is further accentuated by prominent vertebral endplates. The intervertebral discs physiologically bulge into the spinal canal.

In *secondary stenosis,* in which the spinal canal is developmentally normal, compression of the dural tube and caudal nerve roots is due exclusively to one or more acquired conditions. These are generally represented by spondylotic changes, associated or not with degenerative spondylolisthesis. Late sequels of fractures or infections and systemic bone diseases, like Paget's disease, are additional, rare cause of secondary stenosis.

Stenosis of the spinal canal includes a large spectrum of pathologic condition characterized by developmental narrowing of the vertebral canal and/or degenerative changes of one or more of the anatomic structures forming the walls of the canal. Idiopathic developmental stenosis is a fairly rare condition. In most cases stenosis is produced by spondylotic changes in subjects in whom the spinal canals have sagittal or interarticular dimensions at the lower limits of normal.

Encroachment on the central spinal canal is a constant feature of developmental as well as degenerative changes in which the soft tissues play a dominant role in the compression of the nervous structures. In most cases degenerative spondylolisthesis is associated with spinal canal stenosis if the dimensions of the spinal

canal are at the lower limits of normal or below these on a developmental basis. In the presence of a developmentally large spinal canal significant compression of the neural elements may occur only when vertebral slipping is marked and/or degenerative hypertrophy of the facet joints are severe.

Spinal canal dimensions vary in both flexion and extension and rotation of the motion segment. Dynamic variations in flexions and extension are related to changes in bulging of the intervertebral disc and in thickness and buckling of the ligamentum flavum. Rotation mainly affects the root canals which narrows due to both changes in the sift tissues and reciprocal vertebral displacement.

The intervertebral discs in the lumbar spine, while usually normal in height, may bulge markedly into the vertebral canal, further worsening the posterior prominence of the vertebral endplates (Figure 5). When examined in transverse sections, the vertebral canal appears abnormally narrow in the interarticular diameter and, occasionally, in the interpedicular diameter, midsagittal diameter may be abnormally narrow, but most often it is within the limits of normal despite the shortness of the pedicles.

A true root canal stenosis is usually only seen at the lowest lumbar levels, sometimes but even at the L3-L4 and at the L2-L3 levels. The subarticular portion of the root canals and the lateral recesses are narrow as the result of the shortness of the pedicles and the sagittal orientation of the superior articular processes (Figure 6). Posterior disc bulging and prominence of the vertebral endplates, associated with a transverse narrowing of the spinal canal, produced a segmental narrowing of the latter, which increases in width at the level of the concavity of the posterior surface of the vertebral bodies above and below.

The pedicles are short and the dorsal aspect of the vertebral bodies show a marked concavity in the sagittal plane associated with a posterior prominence of the vertebral endplates. The intervertebral discs protrude into the spinal canal even in the absence of any degenerative changes. The articular processes and the lamina show a more sagittal orientation than normal. The transaxial morphology of the spinal canal is characterized by an abnormally short (less than 11 mm) interarticular distance, i.e. a trefoil shape in the presence of a normally sized midsagittal diameter.

The ligamenta flava may be thick and the articular processes hypertrophic as a result of degenerative changes, which are generally mild since this type of stenosis is usually observed in middle or early senile age. Due to the shortness of the pedicles, the intervertebral foramina are narrow and slit shaped in the sagittal plane, whereas the vertical dimensions are generally normal on account of the normal height of the

Figure 7. Sagittal section through a degenerated lower lumbar spine of a 68-year-old man with no history of back pain or radiculopathy. At L5-S1 there is complete resorption of the intervertebral disc and stable fusion of the cartilaginous endplates. A 1.5-cm-wide band of subchondral endplate sclerosis borders this fusion area. Posteriorly in the disc, hard and dark outer annular layers are extruded into the midzone or pedicle portion of the radicular canal in which the relatively small dorsal root ganglion snugly follows the pedicle. The radicular artery anterior to the ganglion (arrow) is very small and so are the segmental veins which, however, are not entirely collapsed. The total resorption of the disc also entails a severe shortening in the posterior elements as demonstrated here by the axial shortening subluxation of the facet joint. Its vertical, apparently less loaded facet carries macroscopically normal hyaline cartilage whereas the superior tip of the upper articular process erodes into the inferior aspect of the pars interarticularis of L5. There is osteoarthrosis with osteophyte formation of the tip of the SAP.

intervertebral discs. The middle and outer portions of the neuroforamina may be abnormal
ly narrow due to the posterolateral bulging of the intervertebral discs, but the nerve roots are rarely compressed, since they occupy the cranial portion of the foramina (Figure 7).

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