# Acquired degenerative changes of the intervertebral segments at and suprajacent to the lumbosacral junction
## A radioanatomic analysis of the nondiscal structures of the spinal column and perispinal soft tissues

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## Abstract

A review of the imaging features of normal and degenerative anatomy of the spine on medical imaging studies shows features that have been largely overlooked or poorly understood by the imaging community in recent years. The imaging methods reviewed included computed tomography (CT) with multiplanar reconstructions and magnetic resonance imaging (MRI). A routine part of the MRI examination included fat-suppressed T2 weighted fast-spin- or turbo-spin-echo acquisitions. As compared to the normal features in asymptomatic volunteers, alterations in the observed CT/MRI morphology and MR signal characteristics were sought in symptomatic individuals.

Findings in symptomatic subjects which departed from the normal anatomic features of the posterior spinal elements in asymptomatic volunteers included: rupture of the interspinous ligament(s), neoarthrosis of the interspinous space with perispinous cyst formation, posterior spinal facet (zygapophyseal joint) arthrosis, related central spinal canal, lateral recess (subarticular zone) and neural foramen stenosis, posterior element alterations associated with various forms of spondylolisthesis, and perispinal muscle rupture/degeneration.

These findings indicate that the posterior elements are major locations of degenerative spinal and perispinal disease that may accompany or even precede degenerative disc disease. Although not as yet proven as a reliable source of patient signs and symptoms in all individuals, because these observations may be seen in patients with radicular, referred and/or local low back pain, they should be considered in the evaluation of the symptomatic patient presenting with a clinical lumbosacral syndrome. Imaging recommendations, in addition to the usual close scrutiny of these posterior spinal elements and perispinal soft tissues on CT and MRI, include the acquisition of high-resolution multiplanar CT reconstructions, and fat-suppressed T2 weighted fast-spin- or turbo-spin-echo sequence MRI in at least one plane in every examination of the lumbar spine.
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## 1. Introduction

Spine related pain and disability are some of the greatest preoccupations of clinicians and patients. Beyond 'normal' aging of the elements of the spine, absolute degeneration of these spinal substructures eventually occurs. This at some point entails a superoinferior narrowing and eventual collapse of the intervertebral disc. Preceding or accompanying these discal alterations, significant degenerative changes also occur in the nondiscal structures of the spinal column and related tissues, including the posterior spinal facet joints, the spinal ligaments, the underlying bone of the posterior bony elements of the spine and the perispinal muscles. This article outlines the potentially clinically relevant spinal and perispinal consequences of, and phenomena contributing to, acquired degenerative changes of the discal and nondiscal structures of the intervertebral segments at and immediately suprajacent to the lumbosacral junction (i.e., L5-S1, L4-L5, L3-L4 levels), and illustrates how these pathoanatomic findings relate to the normal and variant anatomy as well as dysfunction of this region of the spine [24,27,44,51,66,75,79,95, 127,135].

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## 2. Normal and variant anatomy of the lumbosacral spine

The discal and nondiscal structures of the spine that may undergo degenerative changes include the intervertebral disc itself, the posterior spinal facet (e.g., zygapophyseal) joints; the spinous processes; the intraspinal ligaments; the spinal nerves and spinal innervation; and the perispinal (intraspinal) muscles (Figs. 1–4) [39]. Normal gross anatomic variations in these structures include those of lumbosacral spinal curvature (e.g., straight spine: hypolordosis, exaggerated spinal curvature: hyperlodorsosis (Fig. 5); lateral and rotational scoliosis); central spinal canal diameter (e.g., developmental spinal stenosis); vertebral morphology (e.g., normal anterior wedge shape of L2-L5 vertebral bodies); diskal morphology



(A)

(B)

(C)

Fig. 1. Normal osseous and ligamentous anatomy of the lumbosacral spine. (A) Midline sagittal section diagram of the lumbosacral spinal ligaments and related spaces. (B) Coronal section diagram through the mid-portions of the lower lumbar vertebrae and intervertebral disks with the anterior tissues removed. Note also the transverse processes and inter-transverse ligaments. (C) Coronal section diagram through anterior aspect of interspinous ligament and adjacent spinous processes. (D) Coronal section diagram through mid-aspect of interspinous ligament and adjacent spinous processes. 1, Spinal subarachnoid space; 2, thecal sac; 3, anterior epidural space; 4, posterior longitudinal ligament; 5, retrovertebral subligamentous space; 6, median retrothecal fat pad (posterior epidural space); 7, junction of halves of interspinous ligament with the ligamenta flava; 8, normal spinous processes of L3 and L4; 9, normally mildly hypoplastic spinous process of L5; 10, midline sacral spinous tubercles; 11, supraspinous ligament; 12, erector spinae muscle tendons and deep fibers of the lumbosacral fascia; 13, variation in termination of supraspinous ligament (L3–L5); 14, filum terminale; 15, sacral spinal segments; 16, lumbar vertebral bodies; 17, nucleus pulposus of the lumbar intervertebral disk(s); 18, anterior longitudinal ligament; 19, prevertebral subligamentous space; 20, coccyx; 21, right half of interspinous ligament anteriorly; 22, left half of interspinous ligament anteriorly; 23, combined right and left halves of the interspinous ligament in its middle and posterior extent; 24, fibers of interspinous ligament shown crossing the midline; 25, anterior (ventral) segment of interspinous ligament originating in ligamenta flava on each side and terminating in inferior margin of the suprajacent spinous process; 26, middle segment of interspinous ligament originating in superior margin of subjacent spinous process and terminating in suprajacent spinal process; 27, posterior (dorsal) segment of interspinous segment originating in the superior margin of the subjacent spinous process and terminating in the supraspinous ligament (at levels where it exists) or the erector spine muscle tendons (below the level of the supraspinous ligament); 28, transverse recesses; 29, intertransverse ligaments and intertransversarii muscles; 30, annulus fibrosus of the intervertebral disk(s). Note the *posterocranial* orientation of the fibers of the interspinous ligament (B–D). (E) Medical imaging studies: (i) midline sagittal CT reconstruction; normal posterior wedge configuration of the L5 vertebral body (white asterisk) and the L5-S1 intervertebral disk (white dot); normal hypoplasia of the L5 spinous process (black asterisk: compare with (A)). (ii) Midline T1-weighted MRI showing again the posterior wedge configuration of the L4 and L5 (white asterisk) vertebral bodies and the lower lumbar intervertebral disk(s) (white dot), and the hypoplastic L5 spinous process (black asterisk: compare with (A)). Note the shape and intensity of the retrothecal fat pad(s) (black dot) and the interspinous ligament(s) (white circle). Also note the supraspinous ligament (white arrowhead), the midline inferior extension of the lumbosacral fascia (black arrowhead), and the junction between the two (black arrow).
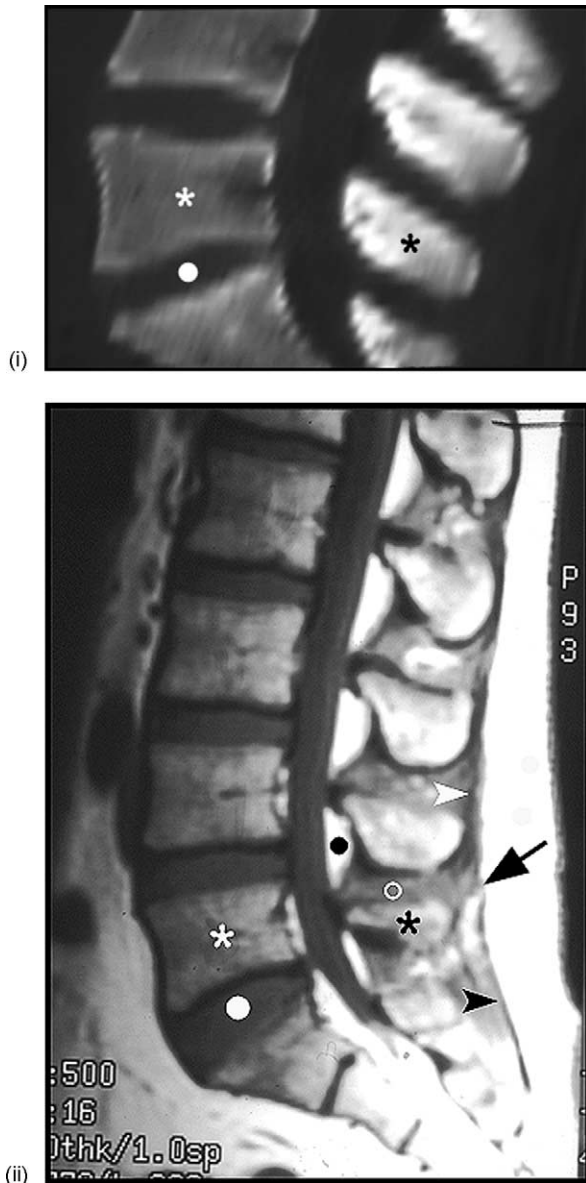
(i)

(ii)
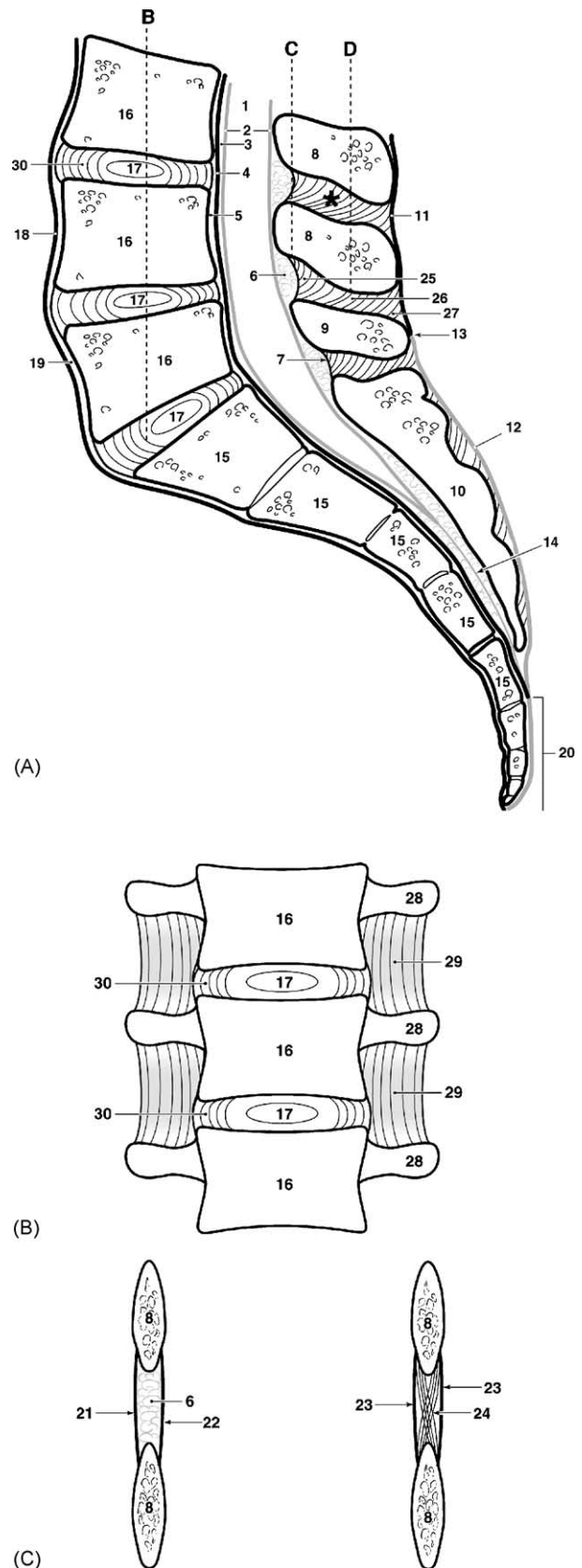
Fig. 1. (*Continued*).

(e.g., normal anterior wedge shape of L2-L3 through L5-S1 intervertebral discs); spinous process morphology (e.g., normal hypoplasia of L5 spinous process); and posterior spinal facet joint angulation in the axial plane (e.g., sagittal or coronal orientation; facet joint tropism or lateral asymmetry of angulation) [2–5,21,28,46,73,98,99,101,105,107,108, 121,122,130,132,133]. These variations may predispose or accelerate degenerative changes in predictable ways. In turn, these degenerative alterations may in some cases result in signs and symptoms including low back pain and lower-extremity referred pain, both of which may respond to therapies specific to the underlying problem. The anatomic foundation for these signs and symptoms is clear and is found within the innervation of these spinal and perispinal structures and the central nervous system pathways serving the peripheral nervous system [19,20,67].

# 3. Pathologic anatomy of the lumbosacral spine related to or accompanying collapse of the intervertebral disc

## 3.1. Vertebral end plate approximation with degenerative disc space narrowing

*Posterior bulging of redundant posterior disc surface with narrowing of the central spinal canal and inferior recesses*
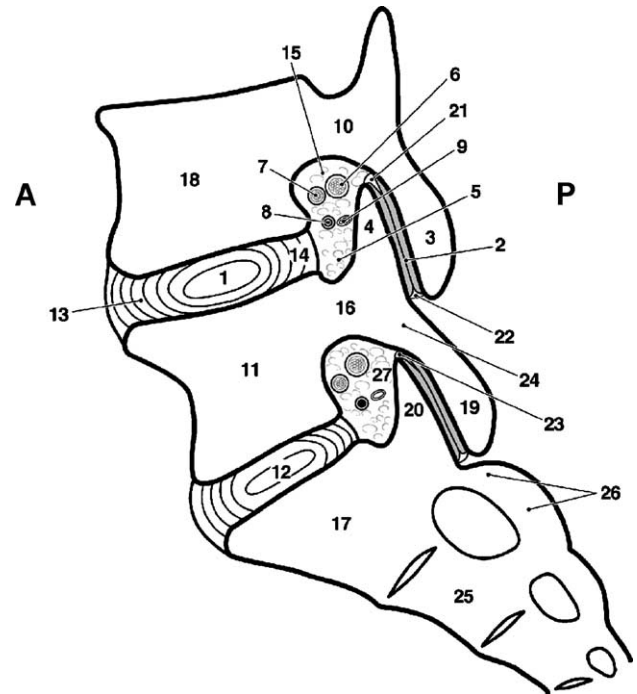


Fig. 2. Normal sagittal appearance of the L4-L5 and L5-S1 neural foramen. (A) Parasagittal section diagram of the spinal neural foramen. 1, L4-L5 intervertebral disk; 2, L4-L5 posterior facet (i.e., zygapophyseal) joint space and articular cartilage; 3, inferior articular facet process of L4; 4, superior articular facet process of L5; 5, inferior recess of the L4-L5 spinal neural foramen; 6, dorsal root ganglion of the L4 spinal nerve dorsal root; 7, ventral root; 8, radiculomedullary artery at L4-L5; 9, radiculomedullary vein at L4-L5; 10, L4 pedicle; 11, L5 vertebral body; 12, L5-S1 intervertebral disk; 13, anterior anular fibers of the L4-L5 intervertebral disk; 14, posterior anular fibers of the L4-L5 intervertebral disk; 15, superior recess of the L4-L5 spinal neural foramen; 16, L5 pedicle; 17, S1 vertebral segment; 18, L4 vertebral body; 19, inferior articular process of L5; 20, superior articular process of S1; 21, superior articular recess of the L4-L5 posterior spinal facet joint; 22, inferior articular recess of the L4-L5 posterior spinal facet joint; 23, superior articular recess and meniscoid of the L5-S1 posterior spinal facet; 24, pars interarticularis of L5; 25, S2 vertebral segment; 26, intermediate sacral crest at S1-S2; 27, intraforaminal fat. A: anterior, P: posterior. (B) Medical imaging studies: (i) lateral parasagittal CT reconstruction showing the bony structure defining the spinal neural foramen (compare with (A)). (ii) Lateral parasagittal T1-weighted MRI showing the pedicle (white circle) of L5, the superior articular process (white asterisk: zygapophysis) of L5, the inferior articular process (black asterisk: zygapophysis) of L5, the pars interarticularis (white dot) of L5; and a portion of the neurovascular bundle (arrow) exiting-entering the L4-L5 neural foramen. (iii) Lateral parasagittal T2-weighted fat-suppressed MRI showing normal fluid within the superior (arrow) and inferior (arrowhead) recesses of the L5-S1 posterior spinal facet (zygapophyseal) joint on one side, and the normal high intensity of the dorsal root ganglia (black asterisks) within the neural foramina (compare with (A)).
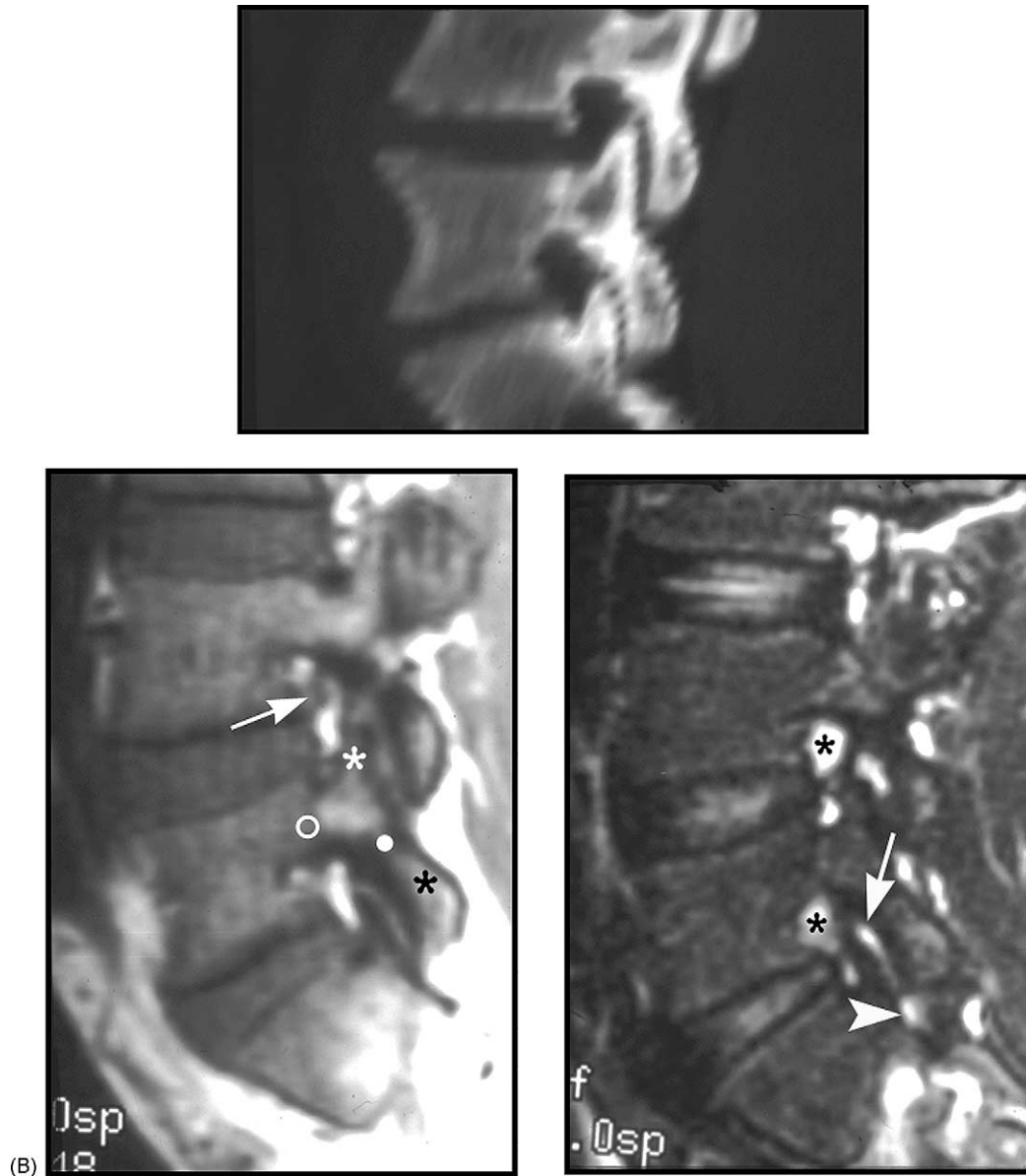
Fig. 2. (*Continued*).

*of the neural foramina*. With superoinferior degenerative collapse of the intervertebral disc, the peripheral annulus fibrosus becomes redundant and bulges outward. Accompanying posterior bulging of the redundant posterior aspect of the disc surface of the annulus fibrosus is regional narrowing of the inferior recesses of the neural foramina (Fig. 6A) [111].

*Anterior bulging of redundant ligamenta flava and posterior spinal facet (i.e., zygapophyseal) joint capsule, with narrowing of the central spinal canal and the lateral recesses of the central spinal canal*. When the intervertebral disc undergoes a degenerative reduction in height, there is a consonant redundancy in the ligamenta flava and posterior spinal facet joint capsule that protrudes anteriorly into the central spinal and lateral recesses of the central spinal canal
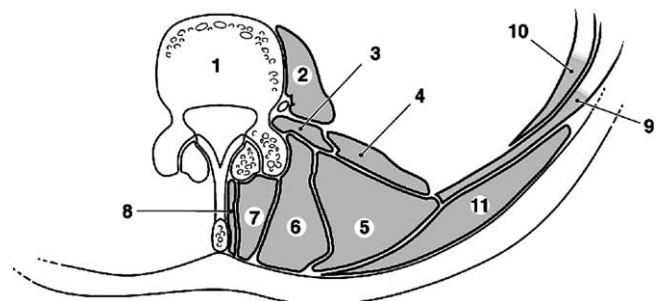


Fig. 3. Axial view of the perispinal muscles in the lumbar region on the left. 1, Lumbar vertebra; 2, psoas muscle; 3, intertransversarius muscle; 4, quadratus lumborum muscle; 5, thoracocostalis muscle; 6, longissimus muscle; 7, multifidus muscle; 8, interspinalis muscle; 9, external oblique muscle; 10, internal oblique muscle; 11, latissimus dorsi muscle.
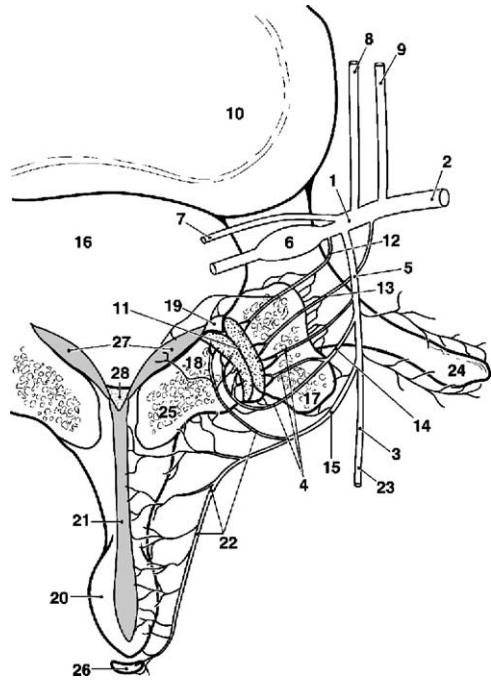
Fig. 4. Innervation of structures of dorsal (posterior) aspect of spinal column. 1, Main trunk of spinal nerve; 2, ventral ramus of spinal nerve; 3, lateral branch of dorsal ramus of spinal nerve; 4, neural fibers to posterior dorsal ramus of spinal nerve; 5, dorsal ramus of spinal nerve; 6, dorsal nerve root and ganglion; 7, ventral nerve root; 8, gray ramus communicans; 9, white ramus communicans; 10, intervertebral disk; 11, articular cartilage of posterior spinal facet (i.e., zygapophyseal) joint; 12, neural fibers from main trunk of spinal nerve; 13, neural fibers to posterior facet joint from ventral ramus of spinal nerve; 14, neural fibers to posterior facet joint from dorsal ramus; 15, medial branch of dorsal ramus of spinal nerve; 16, central spinal canal; 17, superior articular facet process; 18, inferior articular facet process; 19, zygapophyseal joint space and capsule; 20, spinous process; 21, interspinous ligament; 22, medial neural branches ramifying within posterior facet joint, the lamina, spinous process, interspinous ligament, and supraspinous ligament; 23, branch of dorsal ramus ramifying within posterior perispinous tissues; 24, transverse process; 25, lamina; 26, supraspinous ligament; 27, ligamenta flava; 28, median retrothecal fat pad.

and spinal neural foramen, resulting in further narrowing of these regions (Fig. 6B) [13].

*Posterior bulging of a redundant posterior longitudinal ligament with narrowing of the central spinal canal.* With degenerative collapse of the intervertebral disc, there is a consonant focal redundancy of the posterior longitudinal ligament that protrudes posteriorly into the central spinal canal, resulting in further anteroposterior narrowing of this region.

*Posterior paradiscal vertebral arthrosis and osteophytosis with anteroposterior narrowing of the central spinal canal, lateral recesses of the central spinal canal and neural foramina.* With degenerative narrowing of the intervertebral disc, the periphery of the adjacent vertebral bodies typically develop rim osteophytes that extend into the central spinal canal itself, the lateral recesses of the central spinal canal, and the neural foramina. This results in further anteroposterior narrowing of these regions (Fig. 6C) [47,109,110].

*Radial expansion vertebral remodeling with narrowing of the central spinal canal, the lateral recesses of the central spinal canal and the spinal neural foramina.* Accompanying degenerative narrowing of suprajacent and subjacent intervertebral discs, the intervening vertebral body may undergo stress-related remodeling. This remodeling consists of a radial enlargement of the vertebral body in the horizontal plane and a height reduction, causing a type of "pancaking" of the corpus. This results in anteroposterior narrowing of the central spinal canal, the lateral recesses of the central spinal canal and the spinal neural foramina (Fig. 7) [6,7,34,40–42].

## 3.2. Pediclo-pedicular approximation with superoinferior narrowing of the spinal neural foramina

With a loss of height in the intervertebral disc, there is a consonant narrowing of the superoinferior dimension of the narrowing of the spinal neural foramina (see Fig. 6A) [31,58,87].

## 3.3. Posterior spinal facet (zygapophyseal) joint degenerative craniocaudal partial subluxation

With collapse of the intervertebral disc, there is a consonant craniocaudal partial subluxation of the posterior spinal facet (i.e., zygapophyseal) joints [36,56,57,78,80]. This facet subluxation and the subsequent alterations may be asymmetric from side to side. Posterior spinal facet joint effusions accompany these subluxations (see Figs. J-i and -ii).

*Posterior spinal facet joint arthrosis and osteophytosis with narrowing of the lateral recesses of the central spinal canal and the spinal neural foramina.* When the posterior spinal facet joint undergoes subluxation secondary to degenerative intervertebral disc narrowing, new stresses on the facet joint result in arthrosis and osteophytosis. This causes further anteroposterior narrowing of the lateral recesses of the central spinal canal and the spinal neural foramina. Posterior spinal facet joint effusions may accompany these alterations at some point during these degenerative disease processes (see Fig. 6A and C) [38,127].

*Superior articular facet process and pars interarticularis collision.* When the intervertebral disk collapses, the resulting posterior facet joint craniocaudal partial subluxation causes a collision of the apex of the superior articular facet process and the undersurface of the pars interarticularis (see Fig. 6B) [56,57,78,80,81].

- *Superoinferior articular facet process and pars interarticularis collisional osteophytosis with further narrowing of the superior recess of the spinal neural foramen*: Collision of the superior articular facet process with the overlying pars interarticularis results in collisional osteophytosis with further narrowing of the superior recess of the spinal neural foramen (see Fig. 6B, J-iii).
- *Collisional blunt erosion of the apex of the superior articular facet process with superoinferior narrowing of the*
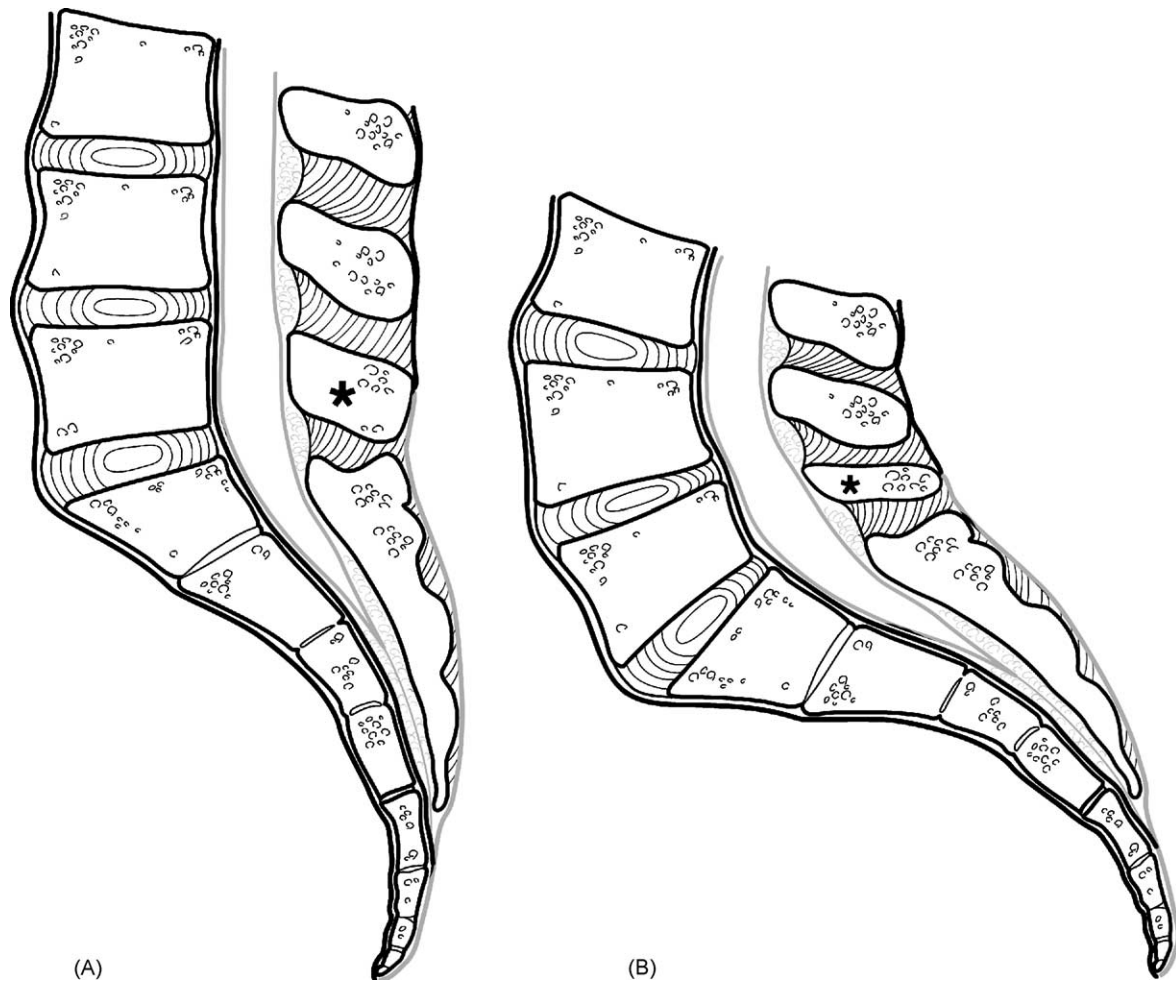
Fig. 5. Variant anatomy of the lumbosacral spine. (A) Straight or hypolordotic spine. The spinal lordotic curvature in this example overall is minimal, or absent in extreme cases, in part because the lower lumbar vertebrae and intervertebral disks are less than normally wedge-shaped (i.e., rectangular shape in this example). Note the mildly hyperplastic L5 spinous process (asterisk). (B) Hyperlordotic (i.e., swayback) spine. The spinal lordotic curvature in this example is exaggerated and the sacrum tends to be more horizontal than normal. Note the hypoplastic L5 spinous process (asterisk) and the exaggerated wedge shape of the vertebral bodies and intervertebral disks. Compare with Fig. 1A. Also note the somewhat horizontally angled sacrum.

*spinal neural foramen: collisional excavative erosion of the undersurface of the pars interarticularis with further craniocaudal narrowing of the spinal neural foramen.* At the same time erosion of the apex of the colliding superior articular facet process is taking place, there is a corresponding excavative erosion of the undersurface of the suprajacent pars interarticularis. This results in yet further superoinferior narrowing of the spinal neural foramen (see Fig. 6B, J-iv–vi) [56,57,78,80].

- *Collisional anterior curved remodeling of the superior articular facet process with further narrowing of the superior recess of the spinal neural foramen:* With progression, the superior articular facet process degenerative collision with the overlying pars interarticularis and pedicle may result in an anterior curved osteophytic remodeling of the superior articular facet process (see Fig. 6C, J-vii–viii). This results in further encroachment on the superior recess of the spinal neural foramen on one or both sides [55].

- *Superior or inferior articular facet process of collisional fracture with fracture fragment displacement and further encroachment on the spinal neural foramen:* Osteophytic insufficiency fracture-dislocation of the superior and/or inferior articular process(es) may occur, alterations which may allow further narrowing of the intervertebral disc space and spinal neural foramina (Fig. 6H, I, J-xiv).

- *Superior articular facet process and pedicular neoarthrosis with osteophytosis with further encroachment on the central spinal canal and spinal neural foramen:* As the superior articular facet process continues to erode the overlying pedicular bone, a neoarthrosis may develop between the two. This neoarthrosis may communicate with the adjacent articular space of the posterior spinal facet joint (Fig. 6D).

- *Superior articular facet process and pedicular communicating neocyst formation with encroachment on the spinal neural foramen and central spinal canal:*

Subsequently, a neocyst (i.e., not synovium lined) may form and similarly communicate with the posterior spinal facet joint (see Fig. 6D, J-ix). If the neocyst extends into the central spinal canal and neural foramen, there is consonant encroachment on these areas. In the past, reported cases of periarticular ganglion and ligamentum flavum cysts were probably representative of this entity [1,8,12,53,64,72,83,84,128,134].

*Posterior spinal facet joint true synovial cyst formation (intraspinal or perispinal) with encroachment on the central spinal canal, the lateral recesses of the central spinal canal, and the spinal neural foramen.* True synovium-lined cysts may also develop, extending from the posterior spinal facet joints. These synovial cysts encroach on the central spinal canal, the lateral recesses of the central spinal canal, and the spinal neural foramen in cases

Fig. 6. Alterations in the posterior spinal facet (i.e., zygapophyseal) joints related to intervertebral disk collapse. (A) Posterior or spinal facet joint subluxation associated with early intervertebral disk collapse. Note the mild L4-L5 intervertebral disk space narrowing (solid arrow), the superoinferior narrowing of the spinal neural foramen (open arrows), the superoinferior subluxation of the posterior spinal facet joint (dashed arrows), and the early collision and sclerosis of the apex of the superior articular process of L5 and the overlying pars interarticularis of L4 (circled area). This will result in many or all cases in posterior spinal facet joint(s) gapping (asterisk) and effusion (coarse stippling) at some time during this pathologic process. (B) Collision and blunt erosion of the apices of the superior and inferior articular processes of the posterior spinal facet (i.e., zygapophyseal) joints and related bony spinal structures. With further narrowing of the intervertebral disk (asterisk), there may be blunting of the apex of the superior articular process and associated erosion of the anteroinferior aspect of the anteroinferior aspect of the overlying upper pars interarticularis of the suprajacent vertebra (solid arrow with shading). Note the early narrowing/erosion of the posterior spinal facet joint articular cartilage. (C) Curved remodeling of superior articular facet process and neoarthrosis formation between the apex of the superior articular facet process and the suprajacent pars interarticularis. The superior articular process may eventually undergo a curved remodeling (asterisk), partially associated with osteophytic overgrowth. At the same time, a neoarthrosis (curved arrow) may form between the remodeled superior facet process and the overlaying bone of the undersurface of the suprajacent pedicle and pars interarticularis. Concomitantly a neoarthrosis (open arrow) may form between the apex of the inferior facet process (dot) and the posterior surface of the subjacent pars interarticularis. Anterior and posterior spinal facet joint articular cartilage has been eroded (solid arrowhead). (D) Neocyst formation off of the neoarthrosis between the superior articular facet process and the superjacent pedicle-pars interarticularis. A neocyst (arrow: coarse stippling) that may communicate with the adjacent posterior spinal facet joint may form off of the neoarthrosis between the superior articular facet process and the inferior surface of the suprajacent pedicle-pars interarticularis. This neocyst may extend into the spinal neural foramen and central spinal canal, thereby narrowing these areas, and into the lateral/posterior perispinal soft tissues. Pathologically the cyst typically has a thick wall and a small central cavity. Histologically this cyst may not be lined by synovial tissue in which case it is a neocyst or pseudocyst, or may be lined by synovial tissue, thereby representing a true synovial cyst. (E) Neocyst formation off of the apex of the inferior articular facet process and the subjacent pars interarticularis. A neocyst (arrow: coarse stippling) that may communicate with the adjacent posterior spinal facet joint may form off of the neoarthrosis between the apex of the inferior articular facet process and the posterior surface of the subjacent pars interarticularis. This neocyst will extend into the perispinous soft tissues. The neocyst characteristically has a thick wall and small cavity. As noted above, this cyst may be a neocyst of a true synovial cyst, a histologic distinction. (F) Erosive pars interarticularis thinning: With collapse of the adjacent intervertebral disks at and suprajacent to the lumbosacral junction (open arrows), erosion of the intervening pars interarticularis (solid arrow) may occur anteriorly and posteriorly: the suprajacent inferior articular facet process (dot) erodes posteriorly and the subjacent superior articular facet process (asterisk) erodes anteriorly. This thins and structurally weakens the pars interarticularis. (G) Degenerative insufficiency fracture of the pars interarticularis. With further erosion and continued stresses, an insufficiency fracture of the pars interarticularis may occur (dashed circle). This may then allow unrestricted degrees of acquired anterolisthesis to result (dashed arrow). (H) Collisional articular facet process fracture. With continued stresses placed upon the remodeled and osteophytically overgrown posterior spinal articular facet processes, the superior (asterisk) or inferior (dot) articular facet process or attached brittle osteophytes may fracture (solid arrows: 1, 2). This may yet further narrow the spinal neural foramen at this level (open arrows). (I) Articular process fracture fragment distraction/displacement. With continued somatic movements, the superior (asterisk) and inferior (dot) articular fracture fragments may go to nonunion and become distracted or displaced (curved arrows). The former further narrow the involved spinal neural foramen. The loss of this buttressing effect then allow further degenerative narrowing or absolute collapse of the intervertebral disk (open arrows), and consequently further narrowing (i.e., stenosis) of the spinal neural foramen at this level. (J) Medical imaging studies: (i) parasagittal T1-weighted MRI showing collapse of the L5-S1 disk (asterisk) and superoinferior narrowing of the spinal neural foramen (arrow: compare with (A)). (ii) Parasagittal T2-weighted fat-suppressed MRI showing the posterior spinal facet (zygapophyseal) joint effusion (arrows: compare with (A)). (iii) Parasagittal CT reconstruction showing the collision of the superior facet process (asterisk) with the overlying pars interarticularis-pedicle junction (dot), and the minor associated osteophytosis (arrow). (iv) Parasagittal CT reconstruction showing the bony excavation/erosion of the undersurface of the pars interarticularis at the L4 and L5 levels (arrows). (v) Parasagittal CT reconstruction showing truncation of the tip of the superior articular facet process of S1 (arrow: compare with (B) and the sharp, pointed termination of the superior articular process at the L4-L5 level above: arrowhead). (vi) Parasagittal T1-weighted MRI showing again the truncation of the tip of the articular facet process (asterisk) of S1 (compare with (B)). (vii) Parasagittal CT reconstruction showing curved remodeling of the superior articular facet process (arrow) of L4-L5 (compare with (C) and the straight, pointed configuration of the level below: arrowhead). (viii) Parasagittal T1-weighted MRI showing another case of curved remodeling of the superior articular facet process (arrows; compare with (C)). (ix) Parasagittal T2-weighted, fat-suppressed MRI showing an anteriorly directed periarticular neocyst (arrow: i.e., synovial cyst) extending into the spinal neural foramen (compare with (D)). (x) Parasagittal CT reconstruction showing the collision of the tip of the inferior articular facet process of L4 (arrow) with the posterior–superior aspect of the subjacent pars articularis of L5 (asterisk) (compare with (B)). (xi) Parasagittal T1-weighted MRI showing a posterior neocyst (arrow: i.e., synovial cyst) extending posteriorly from the inferior aspect of the zygapophyseal joint at L4-L5 (compare with (E)). (xii) Parasagittal T1-weighted fat-suppressed, IV gadolinium-enhanced MRI showing enhancement of the thickened rim of the posterior neocyst (arrow); note the small non-enhancing central area of the cyst (arrowhead; compare with (E)). (xiii) Parasagittal T1-weighted MRI showing minor spondylolisthesis of L4 on L5 (arrow) secondary to acquired lysis of the pars interarticularis (asterisk) of L5; note the separation of the superior (circle) and inferior (dot) articular processes, the gap between the two (asterisk), and the complete obliteration (i.e., stenosis: arrowhead) of the superior recess of the L4-L5 neural foramen (compare with (G)). (xiv) Parasagittal CT reconstruction showing fatigue fractures of the inferior (arrow to reader's right) and superior (arrow to reader's left) articular facet processes of L3 (dot) and L4 (circle), respectively (compare with (I)).
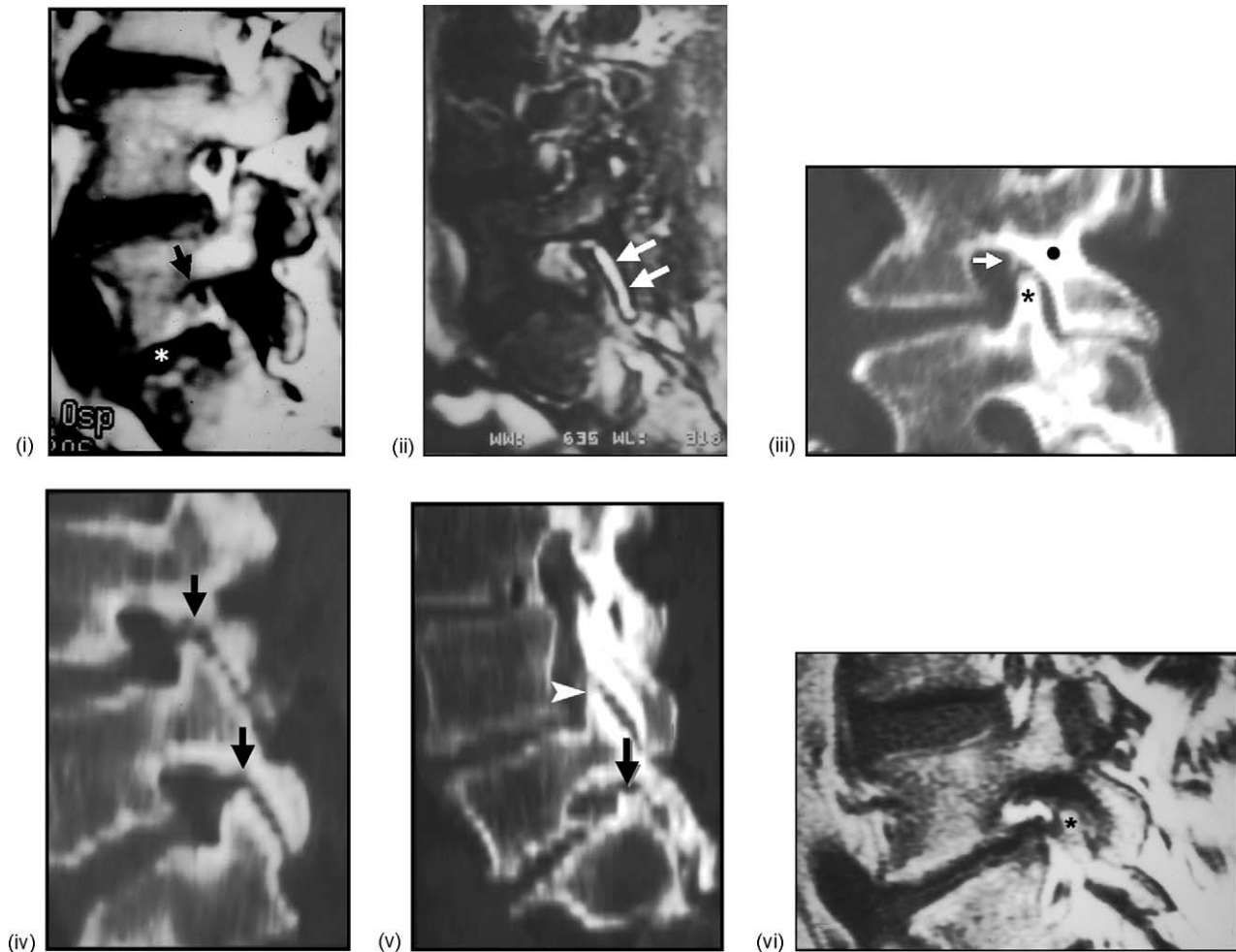
(A)

(B)

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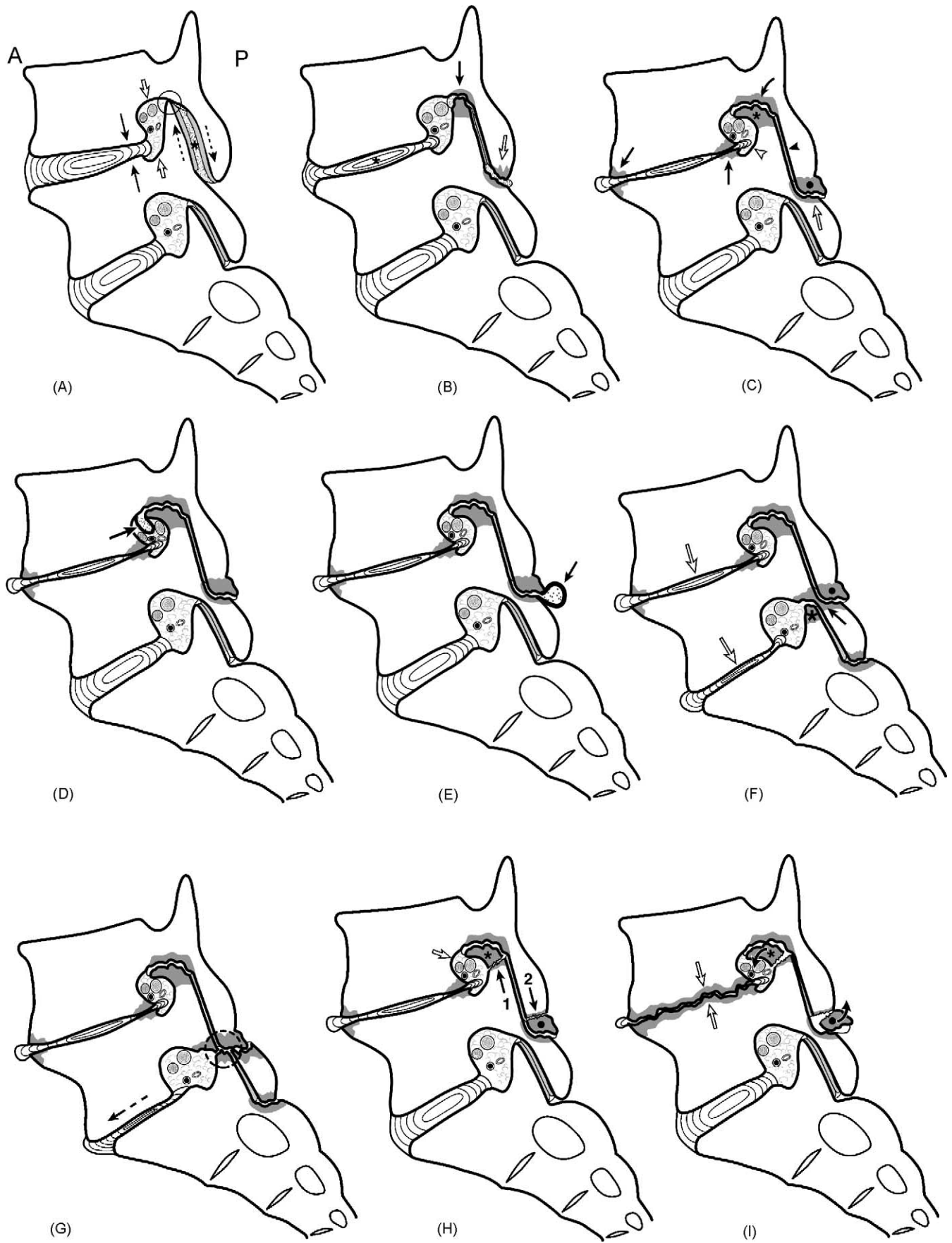
Fig. 6. (*Continued*).

of cyst extension into these areas [30,59,65,76,77,90,112,117, 123].

*Inferior articular facet process and pars interarticularis collision posteriorly at the lumbosacral lordosis.* When the intervertebral disc narrows, the apex of the inferior articular facet process collides with the posterior aspect of the pars interarticularis of the subjacent vertebra [56,57,78,80,106]. This type of collision only occurs at the lumbosacral lordosis (see Fig. 6B, J-x).

- *Posteroinferior collisional osteophytosis:* With collision of the apex of the inferior articular facet process and the underlying pars interarticularis, a collisional osteophytosis results (see Fig. 6B).
- *Posteroinferior collisional blunt erosion of the apex of the inferior articular facet process:* With progressive degenerative intervertebral disc narrowing, collisional blunt erosion of the apex of the inferior articular facet process occurs. This may allow further superoinferior narrowing of the neural foramen (see Fig. 6C).
- *Collisional excavative erosion of the posterior surface of the pars interarticularis:* At the same time that erosion of the apex of the colliding inferior articular facet process is

taking place, there is a consonant excavative erosion of the posterior surface of the pars interarticularis (see Fig. 6C) [56,57,78,80].

*Inferior articular facet process and pars interarticularis communicating neocyst formation.* Subsequently a neocyst (i.e., not synovium lined) may develop as an extension of the neoarthrosis between the apex of the inferior articular process and the posterior aspect of the pars interarticularis. This neocyst may communicate with the articular space of the posterior spinal facet joint (Fig. 6E, J-xi–xii). If the cyst is lined with synovium, this could constitute a true synovial cyst at this location. The differentiation between communicating neocyst and true synovial cyst in all cases is a histologic one.

### 3.4. Interspinous ligament sprain with or without ligamentous rupture, interspinous neoarthrosis and neocyst formation and secondary paraspinal muscle degeneration

Increased intervertebral stresses may induce an interspinous ligament sprain [97,100]. This may include tears

Fig. 6. (*Continued*)

(ruptures) of the fibers of the interspinous ligament. With a progressive loss of intervertebral disc height, there is a consonant loss of the interspinous space and further increased axial stresses on the interspinous and supraspinous ligaments.

*Interspinous ligament redundancy and sprain with hyperplasia, and eventual collisional osteophytosis and neoarthrosis.* With a near or true collision of the vertebral spinous processes (i.e., Baastrup's phenomenon) there is an interspinous ligament redundancy of the opposing spinous process, osteophytosis, and eventual neoarthrosis formation. The redundancy and hyperplasia of the interspinous ligament may extend into the posterior aspect of the central spinal canal in the midline resulting in replacement
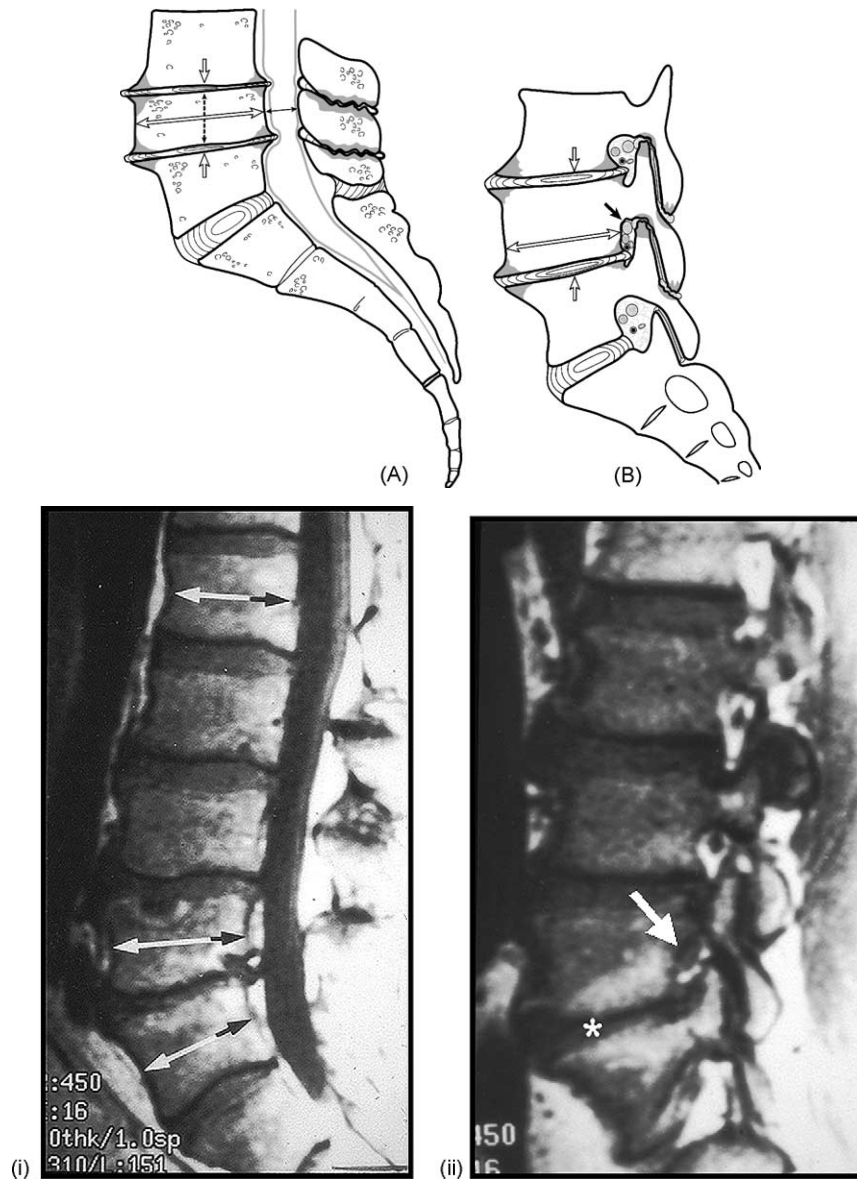
Fig. 7. Radial expansion remodeling of vertebral body. (A) Radial expansion remodeling of the vertebral body between suprajacent and subjacent intervertebral disk collapse associated with central spine canal stenosis. The vertebral body between two adjacent collapsed intervertebral disks (open, single-headed arrows) may undergo radial expansion remodeling circumferentially in the horizontal plane (open, double-headed arrow). At the same time, there will be a superoinferior narrowing of the vertebral body (open, dashed double-headed arrow) producing a bony flat remodeling, or *pancaking* of the vertebra. This results in anteroposterior narrowing of the central spinal canal (solid double-headed arrow) and its lateral recesses. (B) Radial expansion remodeling of the vertebral body associated with spinal neural foramen stenosis. The radially expanded vertebral body (open, double-headed arrow) between two collapsed intervertebral disks (open, single-headed arrows) results in anteroposterior narrowing of the spinal neural foramen (solid arrow) at this level. This stenosis alteration may be asymmetric, side-to-side. (C) Medical imaging studies: (i) midline sagittal T1-weighted MRI showing degeneration of the L3-L4 and L4-L5 intervertebral disks and associated flattening in the superoinferior dimension, and elongation in the anteroposterior dimension of the intervening L4 vertebral body (middle double-headed arrow); note the relative normal dimensions of the L1 and L5 vertebrae (upper and lower double-headed arrows, respectively) by comparison (compare with (A)). (ii) Parasagittal T1-weighted MRI showing anteroposterior narrowing of the L4-L5 spinal neural foramen (arrow) as a consequence of the vertebral body elongation at the L4 level (compare with (B)); some superoinferior narrowing of the L4-L5 neural foramen is also present as a result of L4-L5 degenerative intervertebral disk collapse (asterisk).

of the retrothecal fat pad and narrowing of the central spinal canal. Acute, subacute and chronic autotrauma to the interspinous ligament may result in minor intrinsic sprain or frank rupture-avulsion of the interspinous ligament (Fig. 8A). These alterations accompany consonant intervertebral disk disease in most cases (75%); however, in the remainder interspinous ligament disease may occur before and be more severe than some isosegment disk disease [9–11,25,26,49,50,62,80,86,113–115,118].

*Interspinous neoarthrosis and neocyst formation with anteroposterior narrowing of the central spinal canal in cases of neocyst extension into the central spinal canal.* When a
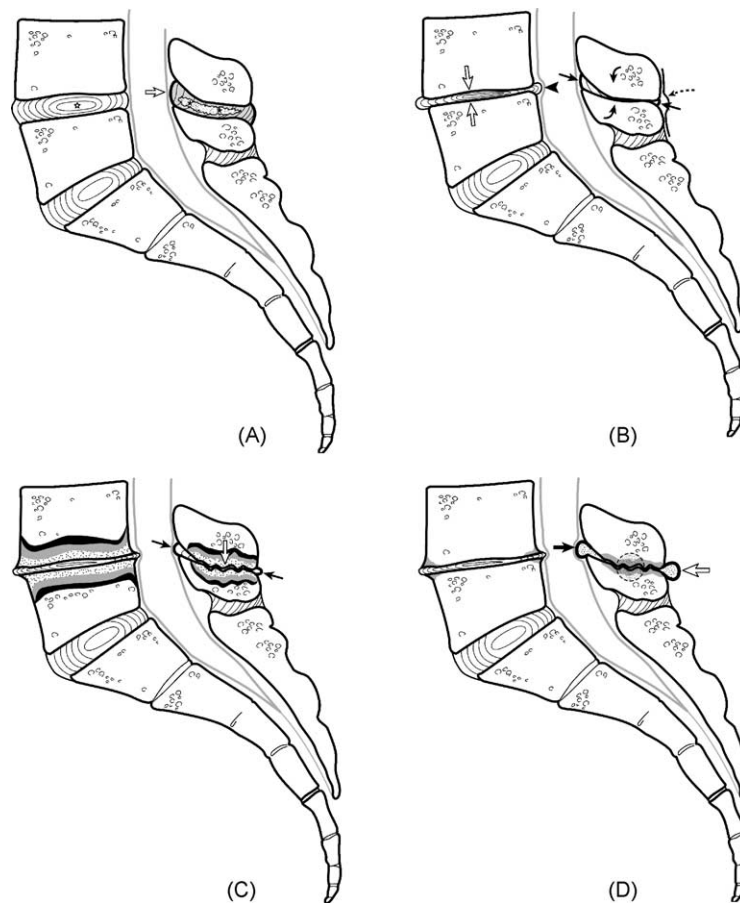
Fig. 8. Degenerative alterations in the interspinous ligaments and interspinous space. *A*, Interspinous ligament sprain with or without intervertebral disk degeneration and associated spinal instability with segmental motion-related stresses. Acute, subacute and chronic motion-related stresses may lead to a type of degenerative ligamentous sprain (i.e., edema, ligamentous fiber tears, frank rupture/avulsion) of the interspinous ligament (*asterisk/shading*). Interspinous ligament redundancy will bulge posteriorly and anteriorly; the latter will replace/displace varying degrees of the segmental retrothecal fat pad(s) (open arrow). These interspinous ligament sprains may be hyperintense on T1- and T2-weighted imaging sequences, presumably as a result of high protein content. Ligamentous degenerative change may occur before, simultaneously with, or following intervertebral disk degeneration (asterisk). (B) Spinous process collision associated with progressive interspinous degenerative alteration (i.e., Baastrup's phenomenon). With progressive intervertebral disk collapse (open arrows), there may be a bony collision of the spinous processes of the adjacent vertebrae (solid curved arrows) at and suprajacent to the lumbosacral junction. Interspinous ligament redundancy (solid straight arrow*s*) together with bulging of the posterior aspect of the intervertebral disk (arrowhead) into the central spinal canal will produce some degree of central spinal canal stenosis. Note that the redundant supraspinous ligament (dashed arrow) will bulge into the perispinous soft tissues (dashed curved arrow). (C) Interspinous neoarthrosis associated with intervertebral disk collapse; associated stress-related marrow alterations within the spinous process marrow and vertebral bodies. With further collapse of the intervertebral disk and increased segmental instability/motion, a neoarthroses (i.e., pseudoarthrosis) may develop between the spinous processes of adjacent vertebral levels (open arrow). The thickened interspinous ligament will protrude peripherally/radially in the axial plane (solid arrows). These phenomena will be predisposed to in individuals with spinous processes that are larger in the superoinferior dimension and in individuals with marked lumbosacral lordosis (i.e., hyperlordosis: "sway back"). Spinous process and vertebral body marrow edema (coarse stippling: type 1 marrow alteration), fatty marrow infiltration (gray shading: type II marrow alteration) and/or bony sclerosis (black shading: type III marrow alteration) may result from these ongoing intervertebral interspinous stresses. (D) Neocyst (i.e., pseudocyst) formation extending from an interspinous neoarthrosis. Continued stresses exerted upon the interspinous ligament and adjacent spinous processes may eventually result in neocyst formation extending off of the interspinous neoarthrosis. These neocysts may be multiple and may extend posteriorly (open arrow), laterally (dashed circle), or anteriorly (solid arrow). The latter may significantly contribute to stenosis of the central spinal canal. (E) Medical imaging studies: (i) midline sagittal T1-weighted MRI showing hyperintensity of the interspinous ligaments at multiple levels (asterisks). (ii) Midline sagittal T2-weighted, fat-suppressed MRI showing isolated hyperintensity of the L5-S1 interspinous space (arrow) indicating degeneration and possible cystic alteration of the interspinous ligament (compare with (A)). (iii) Coronal T1-weighted MRI showing the rounded appearance of the interspinous ligament (arrow), between the spinous processes of L5-S1 (dots). (iv) Coronal T2-weighted, fat-suppressed MRI showing again the apparent cystic degeneration of the interspinous ligament situated between the spinous processes of L4 and L5 (dots; compare with iv). (v) Axial T1-weighted MRI showing multiple rounded paraspinous soft tissue structures (arrows); also note the spinous process of L5 (black dot) and the redundant-hypertrophic inter-supraspinous ligament(s) (white dot) (compare with (D)). (vi) Axial T2-weighted, fat-suppressed MRI showing the hyperintense nature of the paraspinous cysts (arrows; same patient in (E) v–vi). (vii) Sagittal T2-weighted, fat-suppressed MRI showing two interspinous cysts extending into the posterior aspect of the central spinal canal at L3-L4 and L4-L5 (arrows; compare with (D)). (viii) Midline sagittal T1-weighted MRI showing multilevel posterior redundancy-hypertrophy of the supraspinous ligament (arrows; compare with (B)).
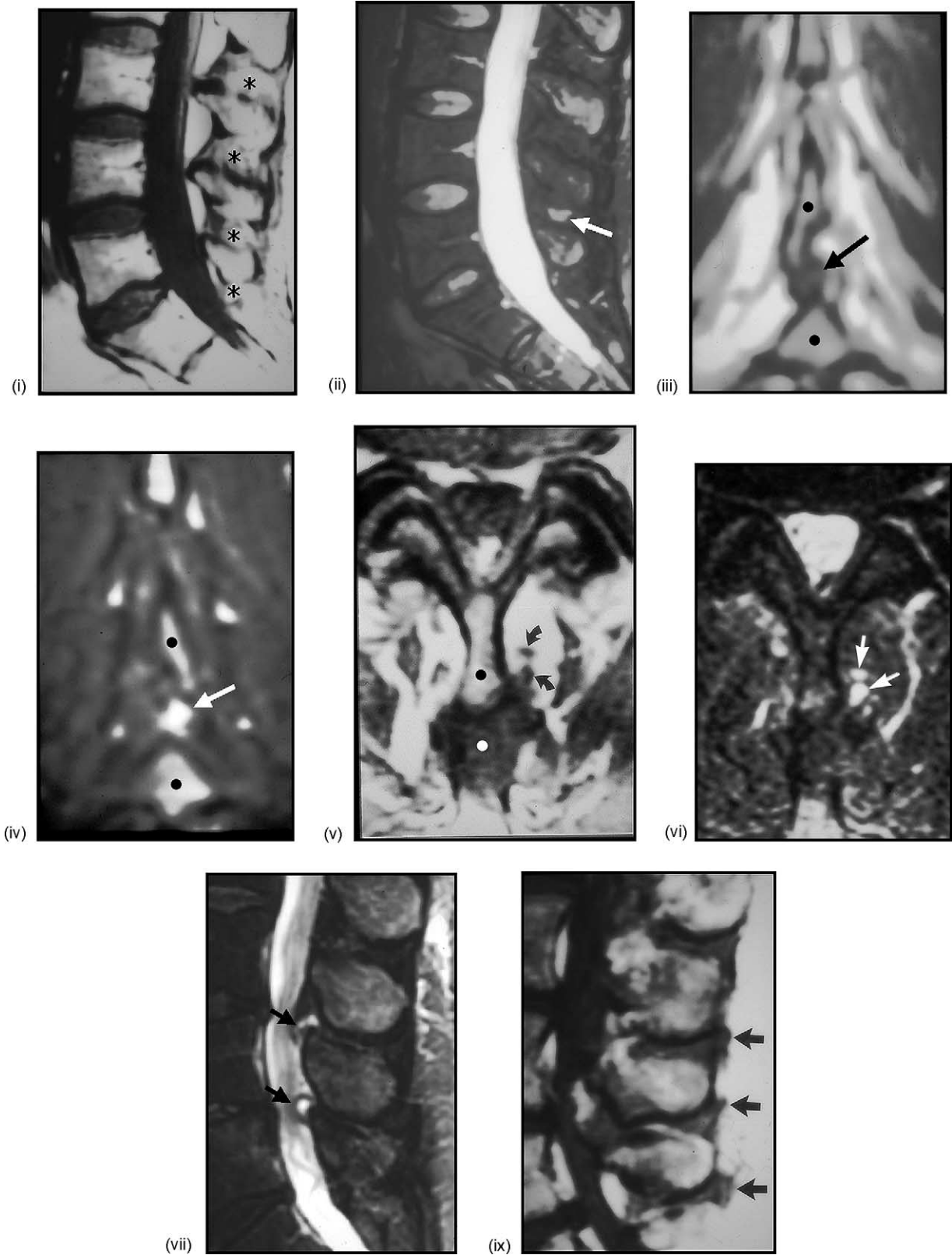
Fig. 8. (*Continued*).

neoarthrosis develops between two colliding spinous processes, a communicating neocyst (i.e., pseudocyst) may evolve (Fig. 8B–D, E-i–iv). This neocyst formation may extend in any radial direction in the axial plane. Extension of the neocyst into the central spinal canal results in additional





Fig. 9. (*Continued*).

replacement of the retrothecal fat pad and further narrowing (i.e., stenosis) of the central spinal canal (Fig. 8E-v–vii).

## 3.5. Supraspinous ligament redundancy

With degenerative approximation of the spinous processes, the intervening supraspinous ligament becomes redundant and bulges into the posterior perispinous soft tissues (see Fig. 8B, E-viii).

## 3.6. Segmental degenerative intervertebral instability

Concomitant with collapse of the intervertebral disc, the spine may undergo segmental intervertebral degenerative instability [82]. Depending upon the individual case, the suprajacent vertebral body may slip backward (i.e., retrolisthesis) forward (i.e., anterolisthesis), lateral (i.e., laterolisthesis) or rotationally (i.e., rotolisthesis) with relation to the subjacent one.

### 3.6.1. Degenerative spinal retrolisthesis

- *Anterior and superior displacement of the superior articular facet process with narrowing of the anteroposterior and superoinferior dimensions of the spinal neural foramen*: In degenerative retrolisthesis (Fig. 9A, D-i), the superior articular facet process is displaced anteriorly. With associated narrowing of the intervertebral disc, the superior articular facet process is displaced superiorly. This results in anteroposterior and superoinferior narrowing of the spinal neural foramen. The apex of the superior articular facet process in some instances may be displaced
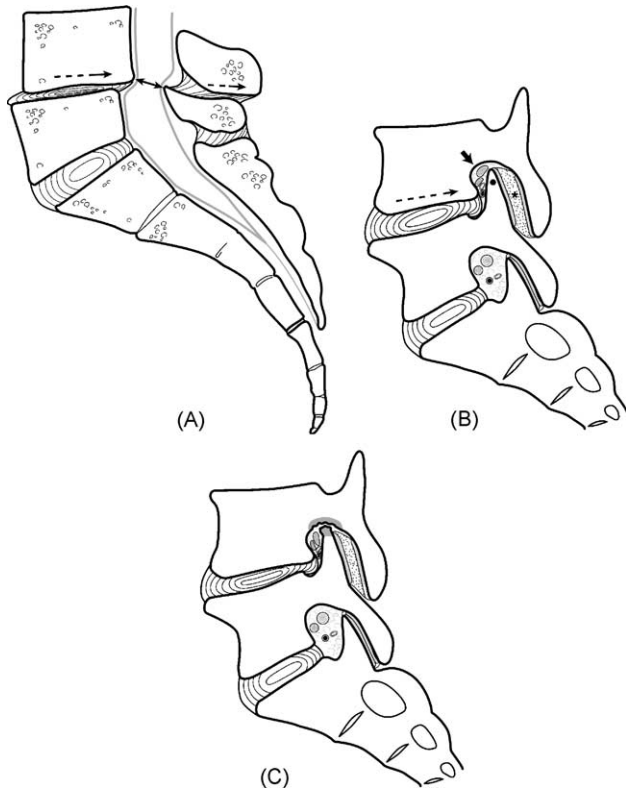


Fig. 9. Degenerative retrolisthesis associated with intervertebral disk collapse and degeneration of related spinal structures. (A) Degenerative retrolisthesis with central spinal canal stenosis. With intervertebral disk collapse and degeneration of related spinal structures (e.g., intraspinal ligaments, degenerative retrolisthesis may occur (dashed arrows). This results in stenosis of the central spinal canal (double-headed arrow). (B) Anteroposterior posterior spinal facet (i.e., zygapophyseal) joint dislocation. With the retrolisthesis of the suprajacent vertebral body (dashed arrow), there will be an anteroposterior posterior spinal facet joint dislocation (asterisk) associated with a joint effusion (coarse stippling). This will narrow the anteroposterior diameter of the spinal neural foramen (solid arrow). In addition, the apex of the superior articular facet process (dot) may protrude directly into the superior recess of the spinal neural foramen. (C) Erosion of the apex of the superior articular facet process and pedicle. With further disk collapse there maybe an excavative erosion of the apex of the superior articular facet process and suprajacent pedicle (shading). (D) Medical imaging studies: (i) midline sagittal T1-weighted MRI showing partial disk collapse at L5-S1 (dot) associated with very minor degenerative retrolisthesis of L5 (arrow) on S1 (asterisk; compare with (A)). (ii) Parasagittal CT reconstruction showing anteroposterior posterior spinal facet joint space "gapping" (arrow) resulting from relative anterior displacement of the superior facet process of S1 (dot) into the spinal neural foramen causing anteroposterior narrowing of the foramen (asterisk; compare with (B)). (iii) Parasagittal T1-weighted MRI showing minor retrolisthesis of L5 (arrowhead) on S1, with anterior displacement of the superior articular facet process (white asterisk) of S1 into the superior recess of the spinal neural foramen resulting in foraminal stenosis (arrow); there is relative consonant posterior displacement of the inferior articular process of L5 (black asterisk) compare with (B)).
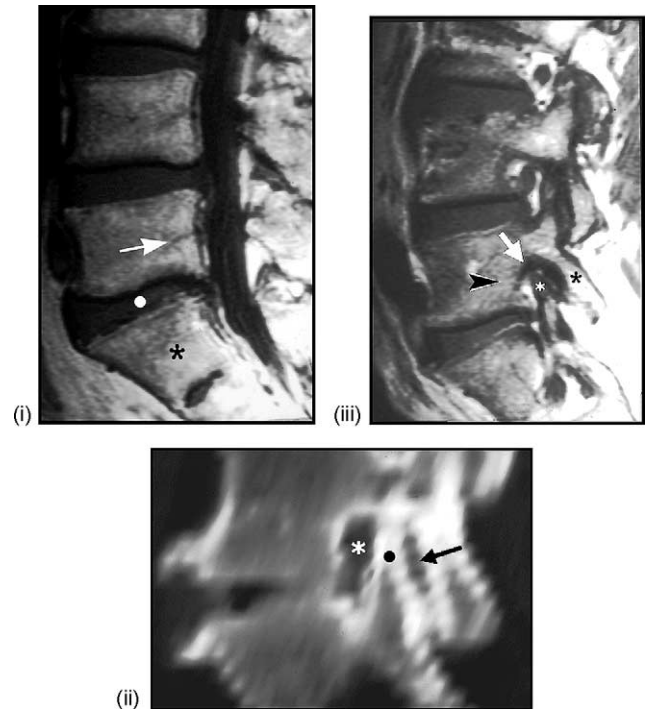
Fig. 10. Degenerative anterolisthesis related to intervertebral disk collapse and degeneration of related spinal structure. (A) Degenerative anterolisthesis. With collapse of the intervertebral disk and degeneration of related spinal structure (e.g., intraspinal ligaments), degenerative anterolisthesis (dashed arrows) of the suprajacent vertebral body may occur. This results in stenosis of the central spinal canal (double-headed arrow) (dashed lines: levels of sections in iii and F). (B) Angular remodeling of the articular facet processes and Type I stress-related marrow alteration. In order for degenerative anterolisthesis (dashed arrow) to occur, segmented anterior angular remodeling (i.e., bending) of the superior articular facet process (open arrows) of the subjacent vertebra and posterior angular remodeling of the inferior articular facet process (solid arrows) of the suprajacent vertebra must occur. Degenerative narrowing of the posterior articular facet joint space also takes place, initially occurring as a result of articular cartilage loss. During the active-progressive phase of this process, marrow edema (coarse stippling: type I marrow alteration) may be present within the affected segmental posterior articular facet processes, pars interarticularis (i) and connected pedicle(s). Radial anterior; posterior and lateral (not shown) paradiskal osteophytes (shaded areas) may be present. Note the narrowing of the inferior recess (asterisk) of the spinal neural foramen that results from anterior angular remodeling of the superior articular process. (C) Type II stress-related posterior articular process/pars interarticularis pedicle marrow alterations. With continued anterior translational (i.e., shear: dashed arrow) stresses on the spine, fatty marrow infiltration (gray shading: type II marrow alteration) may result. Note the erosive changes of the respective posterior spinal fact joint articular processes. (D) Type III stress-related posterior articular process/pars interarticularis/pedicle marrow alteration. Bony sclerosis (black shading: type III marrow alteration) of the pars interarticularis (i) articular facet process(es) and pedicle(s) of the involved vertebrae may eventually result. (E) Sagittal with primary orientation (i.e., developmental), or secondary stress-related degenerative remodeling reorientation (i.e., acquired), of the posterior spinal facet (i.e., zygapophyseal) joint in the axial plane, consequent degenerative anterolisthesis. (i) Normal posterior spinal facet joint angulation in the axial (transverse) plane. (ii) Increased sagittal plane orientation of the posterior spinal facet joint angulation in the axial plane. This sagittal plane orientation may be primary (i.e., developmental sagittal orientation) or acquired (i.e., degenerative remodeling). (iii) Axial plane relationship of anterolisthetic suprajacent vertebra and its superior articular facet process (solid arrows) compared with the subjacent vertebral body (star) and its inferior articular facet processes (asterisks) in cases of sagittal facet joint angle orientation. Sagittal primary orientation of acquired reorientation of the posterior spinal facet joints in the axial plane results in anterolisthesis. This in turn causes stenosis of the central spinal canal (double-headed arrow), and simultaneous narrowing of the inferior recesses of the spinal neural foramina and lateral recesses of the central spinal canal (curved arrows). 1, Coronal plane of vertebral body; 2, posterior spinal facet (i.e., zygapophyseal) joint angle on the right side in the axial plane; 3, posterior spinal facet joint angle on the left side in the axial plane; 4, posterior spinal facet joint angulation on the left side with reference to the coronal plane of the spine; 5, posterior spinal facet joint angulation on the right side with reference to the coronal plane of the spine; 6, superior articular process of subjacent vertebra; 7, inferior articular process and contiguous pars interarticularis of suprajacent vertebra; 8, spinous process; 9, lamina; 10, vertebral body; 11, bilateral sagittal orientation of the posterior spinal facet joint angles; 12, relative enlargement of posterior spinal facet joint angles. Compare with E-i; 13, bilateral coronal orientation of the posterior spinal facet joint angles; 14, relative narrowing of posterior spinal facet joint angles. Compare with E-i and E-ii. (F) Axial plane relationship of suprajacent anterolisthesis vertebral body and its inferior articular facet processes (open arrows) to subjacent superior articular processes in example of coronally oriented facet joint angulation in the axial plane. In the absence of a primary or acquired sagittal orientation of the posterior spinal facet joint angulations, the shear forces obtained at this level of the spine may still result in anterior angular remodeling of the superior articular processes (solid arrows) of the subjacent vertebra (not shown) and consequent stenosis of the inferior recess of the spinal neural foramina (see Fig. B) and the lateral recesses of the central spinal canal (curved arrows). (G) Segmental sagittal plane tropism (i.e., asymmetry of the posterior spinal facet (i.e., zygapophyseal) joints with consequent degenerative rotolisthesis (i.e., segmental rotoscoliosis). (i) *Tropism* or posterior spinal facet joint asymmetry not infrequently is present before the onset of degenerative disease. Note the more sagittal orientation of the facet joint in this example on the right side, with reference to the axial plane. (Curved, dotted arrow: rotational stresses obtaining in right-foot dominant individual. Note the forces terminate at right angles to the posterior spinal facet joint angulation in the axial plane. Prior to segmental spinal degeneration alterations, this may be a biomechanically advantageous spatial orientation). (ii) Rotational segmental rotolisthesis (i.e., segmental rotoscoliosis) may accompany segmental spinal degenerative changes in cases of posterior spinal facet joint tropism. Note the segmental rotolisthesis that may occur in cases of pre-existing (i.e., developmental) posterior facet joint tropism. The suprajacent vertebral (dotted outline) rotates with reference to the subjacent vertebra (solid outline). This has the effect of unilateral anterior displacement of the posterior vertebral elements (straight arrow) and ipsilateral stenosis of the lateral recess of the central spinal canal and the inferior recess of the spinal neural foramen (curved arrow), on this side (dashed line: rotationally shifted median sagittal plane of suprajacent vertebra). 1, Coronal plane of vertebral body; 2, posterior spinal facet joint relative sagittal angulation on the right side in the axial plane; (3) posterior spinal facet joint on the left side in the axial plane; (4) posterior spinal facet joint angulation with reference to the coronal plane of the spine; (5) posterior spinal facet joint angulation on the right side with reference to the coronal plane of the spine; (6) median sagittal plane of subjacent vertebra. (H) Medical imaging studies: (i) midline sagittal T1-weighted MRI showing a grade I anterolisthesis of L4 on L5 (arrow: compare with (A)). (ii) Parasagittal T1-weighted MRI showing the anterolisthesis of L4 (arrow) on L5, the anterior angular bending/remodeling of the superior articular process of L4 (asterisk), and the stenosis of the inferior recess (black arrowhead) and relative patency of the superior recess (white arrowhead) of the L4-L5 spinal neural foramen; same case as in (i) (compare with (B)). (iii) Midline sagittal T1-weighted MRI showing grade I anterolisthesis of L5 (arrow) on S1. (iv) Axial T1-weighted MRI at L5-S1 showing the near sagittal orientation of the angulation of the posterior spinal facet joints (arrows) in the axial plane on both sides; note the stenosis of the central spinal canal (dot) and the spinal neural foramina bilaterally (asterisks) (same case as in H,iii; compare with (E)). (v) Midline sagittal T1-weighted MRI in a different patient showing the very minor anterolisthesis of L4 on L5 (arrow). (vi) Axial T1-weighted MRI showing a clockwise rotoscoliosis of L4 on L5, oblique orientation of the facet joint angulation in the axial plane on the left side (arrowheads), and partial sagittal orientation of the posterior facet joint angulation on the right side (asterisks); note the associated anterior displacement of the lamina and inferior articular facet process (arrow) on the right as compared to the patient's left side, and the displacement of the spinous process (dot) toward the right side as a result of the rotoscoliosis (compare with (G)). (vii) Parasagittal T2-weighted, fat-suppressed MRI showing Type I hyperintense edematous vertebral marrow alterations involving the pedicles, pars interarticulari, and articular facet processes at the L4 and L5 levels (arrows; compare with (B)). (viii) Parasagittal T1-weighted MRI showing Type II hyperintense fatty marrow alteration within the pedicle (arrows) and inferior articular facet process (arrowhead) of L5; note the complete obliteration (i.e., stenosis) of the neural foramen at L4-L5 (dot) (compare with (C)). (ix) Parasagittal T1-weighted MRI showing the Type III hypointense sclerotic alteration of the vertebral marrow within the posterior bony structures at multiple spinal levels inferiorly (asterisks; compare with (D)).

directly into the superior recess of the spinal neural foramen, a process which may result in direct impingement upon the exiting neurovascular bundle (Fig. 9B, D-ii–iii) [69]. Eventually, erosive alterations occur within the colliding bony elements (Fig. 9C).

### 3.6.2. Degenerative spinal anterolisthesis

- *Anteroposterior narrowing of the central spinal canal*: With anterolisthesis of the suprajacent vertebral body on the subjacent one, anteroposterior narrowing of the central spinal canal occurs (Fig. 10A, H-i).
- *Anterior angular remodeling (bending) of the superior articular facet process*: In order for segmental anterolisthesis to occur, the posterior facet joint articular cartilage, the spinal ligaments, and the relevant bone itself must give way. Following arthrosis of the posterior facet joint with a loss of articular cartilage, anterior angular remodeling (i.e., bending) of the superior articular facet process occurs (Fig. 10B, H-ii) [43,96,120].
- *Posterior angular remodeling (bending) of the inferior articular facet process*: With anterior angular remodeling of the superior articular facet process, there may be a similar posterior angular bending of the inferior articular process (see Fig. 10B) [43,96,120].
- *Developmental sagittal plane orientation of the angle of the posterior spinal facet joints in the axial plane*: The angulation of the posterior spinal facet joints in the axial plane may be oriented in the sagittal plane developmentally; this may predispose to eventual premature degenerative anterolisthesis (see Fig. 10E, and H-iii–iv). Alternatively, the axial plane angulation of the posterior

spinal facet joints may remodel over time into the sagittal plane on one or both sides. This stress-related remodeling may ultimately lead to accelerated anterolisthesis.

- *Facet arthrosis and osteophytosis with narrowing of the central spinal canal and lateral recesses of the central spinal canal*: Accompanying the previously mentioned facet changes, facet arthrosis and osteophytosis occurs. This may result in central spinal canal and lateral recess stenosis. Because of the anterolisthesis, elongation of the spinal neural foramen initially takes place. With further progression of these alterations, spinal neural foramen stenosis may occur [60,61,85,104].
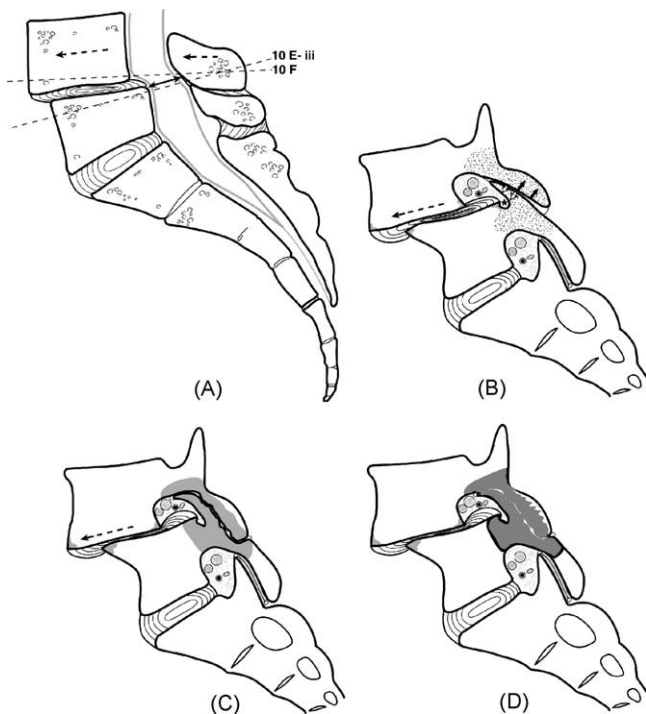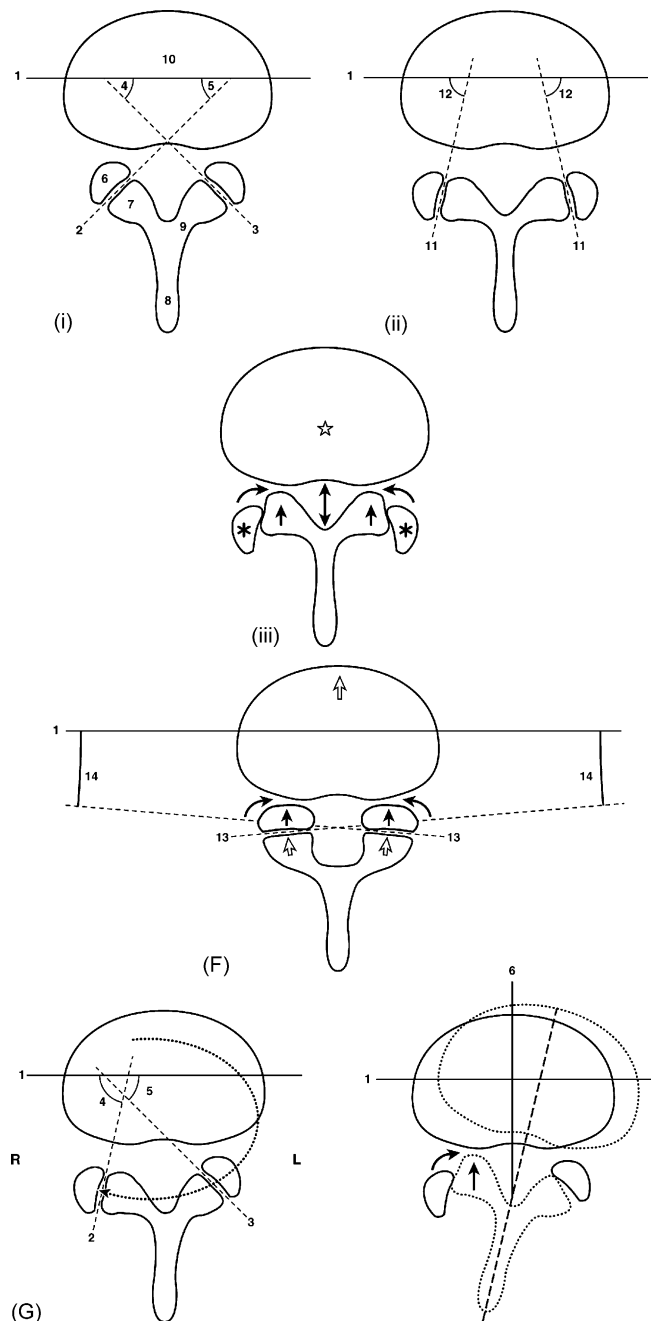


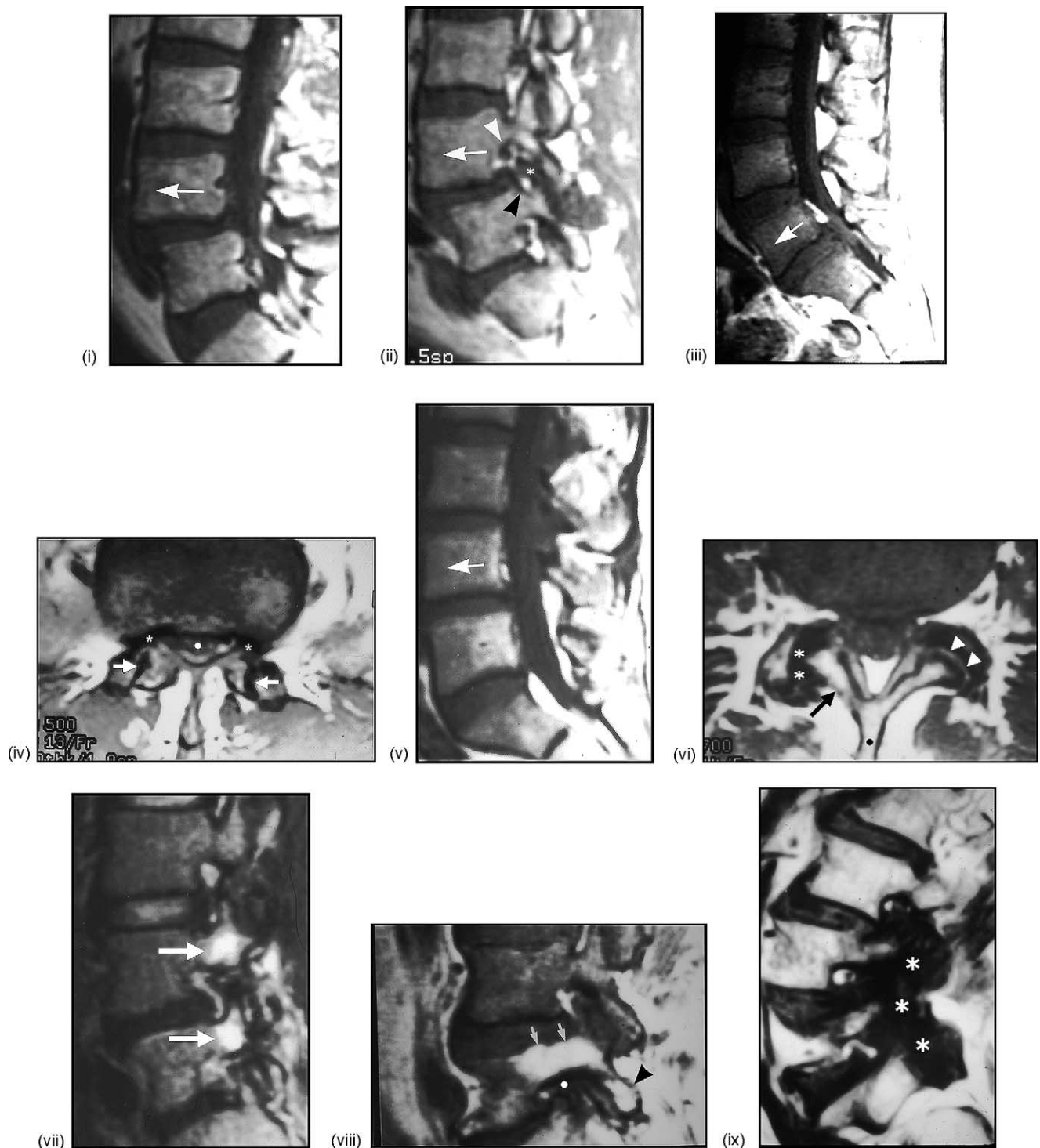Fig. 10. (*Continued*)



Fig. 10. (*Continued*)

Fig. 10. (*Continued*).

- *Erosion of the facet joint components*: Erosion of the involved facet joint components typically occurs resulting in further anterolisthesis (see Fig. 10C).
- *Erosion of the anterior surface of the pars interarticularis*: Erosion of the anterior surface of the pars interarticularis of the suprajacent vertebra resulting from collision with the superior facet process of the subjacent

vertebra may take place allowing yet further anterolisthesis (see Fig. 10C).

### 3.6.3. Degenerative spinal laterolisthesis

Underlying predisposing factors, such as lateral scoliosis or perispinal muscle asymmetry may cause a suprajacent vertebrae to slip laterally on a subjacent one, resulting in

*laterolisthesis*. This leads to an alteration in stresses and acceleration of asymmetric discal and posterior spinal facet joint degenerative changes (see Fig. 11) [48].

### 3.6.4. Degenerative spinal rotolisthesis

In part, underlying predisposing anatomic factors, such as posterior spinal facet joint tropism (i.e., lateral asymmetry in joint surface angulation in the axial plane), may lead to a rotational slip or *rotolisthesis* of a suprajacent vertebra on a subjacent one (see Fig. 10G, H-v–vi) [5,94]. This results in accelerated stresses and asymmetric discal and posterior spinal facet joint degenerative changes. These changes to-

gether may cause ipsilateral stenosis of the lateral recess of the spinal canal and ipsilateral spinal neural foramen [14,15,17,29,32,52,74,119,131].

### 3.7. Degenerative segmental widening of the anteroposterior diameter of the central spinal canal

Paradoxically, degenerative segmental widening of the anteroposterior dimension of the central spinal canal may occur in unusual cases (Fig. 12B, C-i). The mechanism behind this widening is not certain but must centered around one or both of the following phenomena.
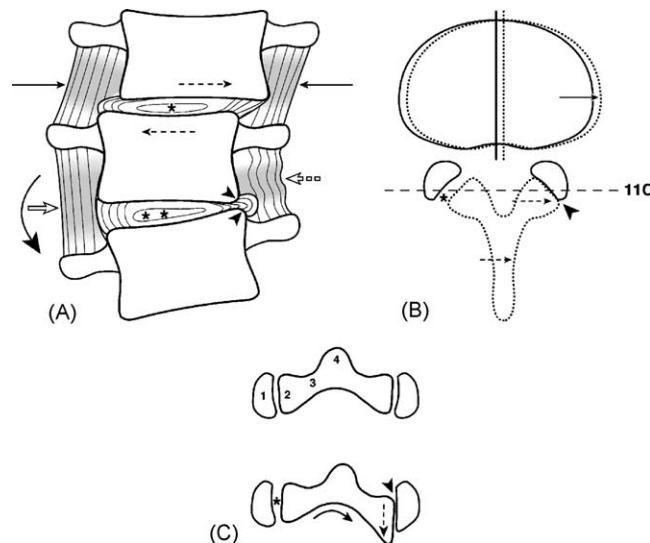


Fig. 11. Degenerative laterolisthesis, alterations related to segmental scoliosis and degeneration of related spinal structures (e.g., intraspinal ligaments and muscles, intervertebral disk). (A) Degenerative laterolisthesis scoliosis and lateral intervertebral disk herniation (coronal plane). With degeneration of the intervertebral disk (single asterisk) and related spinal structures, degenerative laterolisthesis (dashed arrows) may occur (upper segmental levels shown). This will cause the intertransverse ligaments and intertransversarii muscles (straight solid arrows) to become stretched and taught. At the lower level shown, a segmental scoliosis with the convex curve on the reader's left (curved arrow) is present. The intervening intervertebral disk becomes laterally wedge-shaped (double asterisks), the intertransverse musculoligamentous structures on the convex side of the curve become overstretched (open arrows), while the same tissues contralaterally on the concave side of the scoliotic curve become redundant (open dashed arrow). In some cases focal direct lateral intervertebral disk extensions (arrowheads) occur that may engender perispinal sterile inflammation and involve the medial surface of the psoas muscle on that side. (B) Degenerative laterolisthesis (axial plane). In laterolisthesis the suprajacent vertebral body (dotted curved outline), will be shifted to one side (solid arrow) together with the posterior bony elements (dashed arrows), as compared with the subjacent vertebral body (solid curved outline). This will result in narrowing of the posterior spinal facet joint on the side toward the laterolisthesis (arrowhead) and consonant gapping and joint effusion of the contralateral joint (asterisk). With direct laterolisthesis, the median sagittal plan of the suprajacent vertebral body (dotted straight line) will be shifted laterally with reference to that of the subjacent vertebral body (solid straight line). This laterolisthesis may have a rotational component, especially if posterior spinal facet joint tropism exists before segmental degeneration (see Fig. 10 G-ii). (C) Level of section in (C)). (C) Vertical vertebral cant (i.e., lateral tilt) in the coronal plane. Especially in cases of scoliosis, or in instances of asymmetric lateral (i.e., lateral wedge-shaped) disk narrowing, there will be, in addition to the laterolisthesis outlined above, a vertical vertebral cant or tilt (curved arrow) in the coronal plane. This will result in a caudal subluxation of the superior articular process (straight dashed arrow) of the posterior spinal facet joint of the subjacent vertebra in relationship to the inferior articular process of the posterior facet joint of the suprajacent vertebra, on the concave side of the scoliotic curve. The posterior spinal facet joint (arrowhead) at the same level on the contralateral convex side of the scoliotic curve may not undergo significant craniocaudal subluxation, but it may sublux laterally, leaving a wider articular gap than normal. Hypertrophic, stress-related spondylosis (shading), and joint space narrowing (arrowhead) will usually accompany these distortional alterations (compare with more normal configuration at the segmental level above). 1, Inferior articular process of suprajacent vertebra; 2, superior articular process of subjacent vertebra; 3, lamina; 4, base of spinous process. (D) Medical imaging studies: (i) axial T1-weighted MRI showing laterolisthesis of L3 on L4 (arrows), and relative gapping of the posterior facet joint (arrowheads) on the patient's right side (compare with (B)). (ii) Coronal T1-weighted MRI showing laterolisthesis of L3 on L4 (dashed arrow), angulation with asymmetric (left greater that right) frontal plane intervertebral disk collapse at L4-L5 (asterisk), and lateral disk herniation (dot) with paradiscal osteophyte formation (solid arrow) at L4-L5 (compare with (A)). (iii) Coronal T1-weighted MRI showing inferior subluxation of the superior articular facet process of L3 (arrow) on the inferior articular facet process of L4 (asterisk); note the even height of the lower aspects of the articular processes at the level above (arrowheads) and on the opposite side (same case as D-I and ii; compare with (C)).
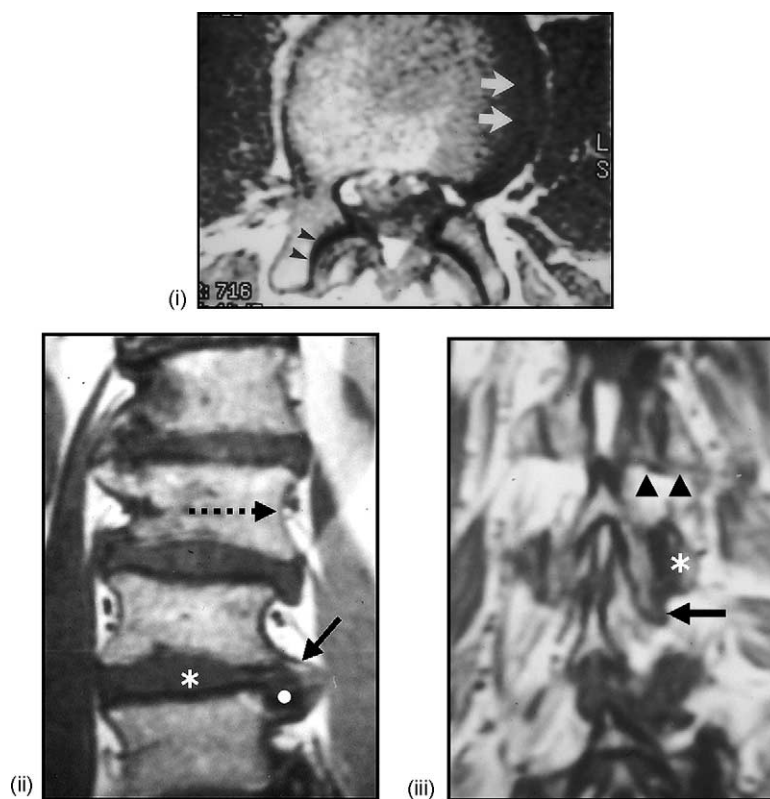
Fig. 11. (*Continued*).

*Stretch longitudinal remodeling of the segmental pedicle resulting in a true elongation of the pedicle.* Long-term anteroposterior vectorial (i.e., shear) stresses on the vertebral pedicles might conceivably result in a stretch longitudinal remodeling of the pedicles themselves. This constitutes a true physical elongation of the pedicle, and an accompanying increase in the segmental anteroposterior dimension of the central spinal canal (see Fig. 12A, C-ii).

*Erosive longitudinal remodeling of the segmental pedicle resulting in an effective elongation of the pedicle.* Long-term stress and erosive changes of the junction of the pedicle and pars interarticularis and the facet joint processes may result in erosive remodeling. This accounts for an effective elongation of the pedicle on one or both sides (see Fig. 12A), and a consonant increase in the segmental anteroposterior dimension of the central spinal canal.

### 3.8. Segmental hypermobile instability with paraspinous muscle degeneration

*Segmental hypermobile segmental instability.* Because of a loss of segmental support following degenerative changes of the spinal ligaments and related spinal structures (e.g., posterior spinal facet joints, anterior and posterior spinal ligaments, and intervertebral disk), hypermobile segmen-

tal instability takes place in flexion-extension and rotation (Fig. 13B). This allows the spine to move through a range of motion that is greater than normal (Fig. 13B) [37,63,85,116].

*Acute–subacute intrinsic spinal muscle degeneration.* Because of this segmental hypermobility, the muscles that originate and insert into the spine (e.g., multifidus and interspinalis muscles) may undergo acute–subacute degeneration. Hypothetically this degeneration is caused by either one or both of two mechanisms related to neuromuscular autotrauma: (1) rupture or avulsion of the insertion intrinsic spinal muscles (e.g., rupture-avulsion of the multifidus and interspinalis muscles) (Fig. 13D, E-i–iv); or (2) traumatic denervation of the interspinal muscles (e.g., rupture-avulsion of the medial branch of the dorsal ramus of the spinal nerve) (Fig. 13C) [35,45,70,93,102,136,137].

### 3.9. Stress-related marrow edema, fatty marrow infiltration, and bony sclerosis of the bony posterior spinal elements

Increased stresses placed upon the posterior elements engendered by intervertebral disk collapse and related instability results in alterations of marrow edema (type 1), fatty marrow infiltration (type 2), or bony sclerosis (type 3) of the superior and inferior articular facet processes, the pars interarticularis, the pedicles, and the spinous processes of the involved vertebrae (see Fig. 10B–D, and H-vii–ix) [22,33,89,92,129].
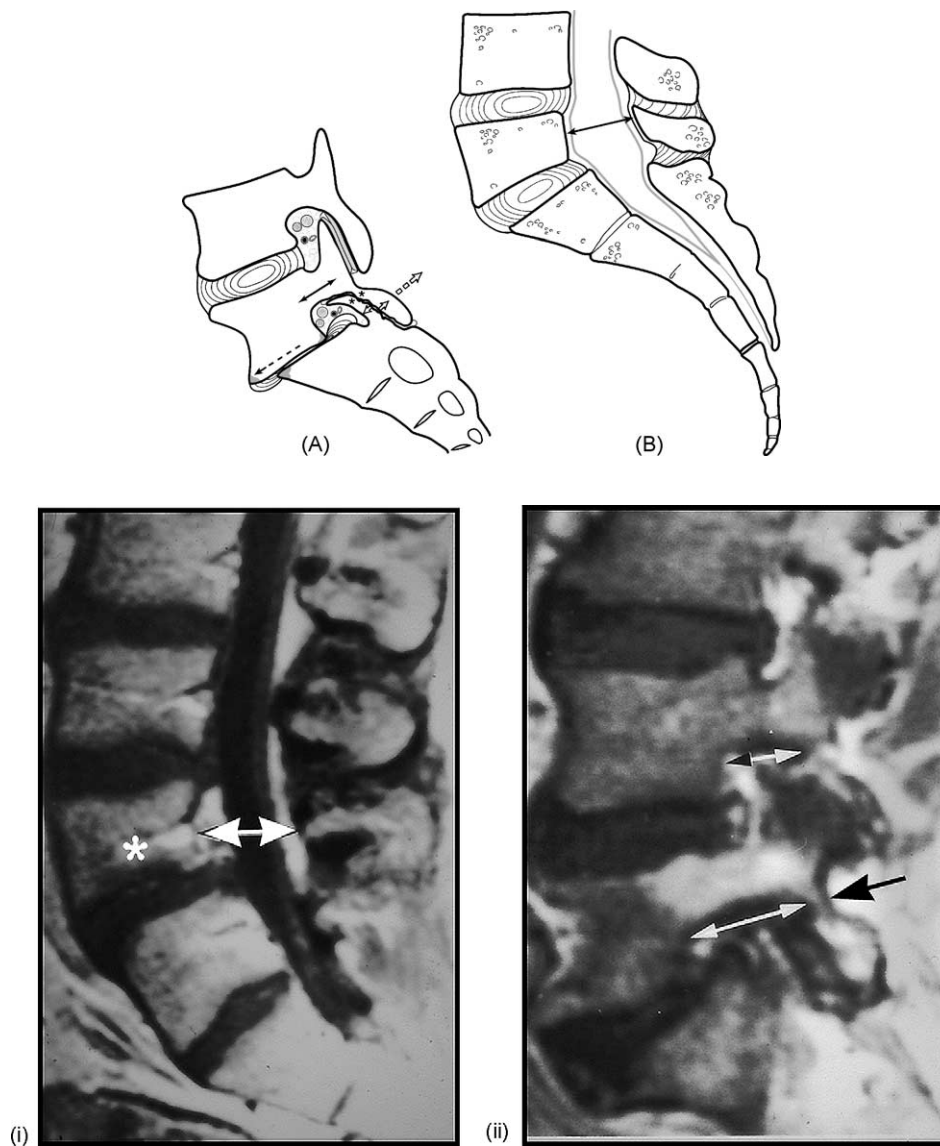
Fig. 12. Segmental degenerative elongation of the anteroposterior dimension of the central spinal canal. (A) True or effective pedicle elongation. Prolonged anteroposterior stresses may induce a true stretch remodeling of the involved pedicle(s) (double-headed arrow). Alternatively or in combination, superior and inferior angular remodeling (open arrows) and degenerative erosive changes (asterisks) of the segmental facet processes and pars interarticularis may result in an effective elongation of the pedicle length. This will result in anterior displacement of the involved suprajacent vertebral body (dashed arrow) and relative minor posterior displacement of the inferior articular facet process(es), lamina(e), and spinous process of the same vertebra. (B) Segmental enlargement of anteroposterior dimension of central spinal canal. With true or effective elongation of the pedicles at one level, there may be minor segmental enlargement of the anteroposterior dimension of the central spinal canal (double-headed arrow). (C) Medical imaging studies: (i) midline T1-weighted MRI showing monosegmental elongation of the anteroposterior dimension of the central spinal canal (double-headed arrow; compare with (B)), note the minor degenerative anterolisthesis of L4 (asterisk) on L5 (the pars interarticularis on both sides was intact [not shown]). (ii) Parasagittal T1-weighted MRI in a different patient showing monosegmental elongation of the anteroposterior dimension of the L5 pedicle (lower double-headed arrow) as compared to the suprajacent L4 vertebrae (upper double-headed arrow); note the intact pars interarticularis (black arrow).

### 3.10. Combined effects of adjacent (tandem) intervertebral disk narrowing at and suprajacent to the lumbosacral junction

*Erosion of the anteroinferior surface of the pars interarticularis of L5.* As noted in the foregoing, collision of the apex of the superior articular facet process of S1 erodes the L5 pars interarticularis from below (Fig. 6F).

*Erosion of the posterosuperior surface of the pars interarticularis of L5.* Concomitantly with this, the apex of the inferior articular process of L4 may erode the pars interarticularis from above in cases of collapse of the L4-L5 intervertebral disk (see Fig. 6F).

*Erosional insufficiency fracture of the L5 pars interarticularis.* With simultaneous eroding influences from above and below, continuing stress may eventually result in an in-

sufficiency fracture of the L5 pars interarticularis (Fig. 6G, J-xiii). When bilateral, these insufficiency fractures may allow relatively unrestricted L5-S1 anterolisthesis or fracture related-acquired degenerative spondylolyses. With a distraction of the fracture fragments, enlargement of the anteroposterior dimension of the central spinal canal may occur.

### 3.11. Clinical implications of these alterations

*Stenosis of the central spinal canal, the lateral recesses, and the spinal neural foramina resulting in neural compression and radiculopathy.* The sum total of these alterations results in spinal stenosis in its various forms: the central spinal canal, the lateral recess of the central spinal canal, and the neural foramina. At and suprajacent to the lumbosacral junction this may in turn cause compressive radiculopathy either in one (monoradiculopathy) or more (polyradiculopathy) of the spinal nerve roots [18,23,126].

*Bony collision of the spinal bony elements resulting in low back pain.* At the same time as frank neural compression takes place, a bony collision of the various spinal elements may cause somatically and autonomically mediated low back pain and referred (i.e., pseudoradicular) low back, pelvic and lower-extremity pain and paresthesias [18,23,54,88,97,103,125,126].

*Musculoligamentous injury resulting in low back pain.* Rupture avulsions of the musculoligamentous structures in and surrounding the spine may, at some point, cause low back pain.

### 3.12. Medical imaging recommendations

*Acquire thin-section, stacked axial ct images for high-resolution sagittal reconstructions.* By stacking thin-section axial CT images, high-resolution sagittal reconstructions acquired from side-to-side yield relevant information concerning the consequences of acquired collapse of the intervertebral disks at and suprajacent to the lumbosacral junction.

*Acquire high-resolution far-lateral MR images through the spinal neural foramina.* By acquiring high-resolution far-lateral MR imaging acquisitions through the neural

Fig. 13. Paraspinal intrinsic spinal muscle degeneration secondary to hypermobile spinal instability. *A*, Multifidus and interspinalis muscle origins and insertions an innervation(s) from posterolateral aspect on the left side. The interspinalis muscle typically originates in the craniad aspect of a subjacent spinous process (or sacral equivalent), and inserts into the caudal aspect of the immediately craniad suprajacent spinous process. The multifidus muscle originates from the mammillary process of a superior articular process and joint capsule of a subjacent lumbar vertebra (or the sacral equivalent), and inserts into one or more spinous processes of cranial vertebra. Note the multisegmental nature of the innervation of the medial-posterior spinal and perispinal tissues from the medial branch of the medial branch of the dorsal ramus of the spinal nerve. 1, Interspinalis muscle; 2, multifidus muscle; 3, spinous process; 4, mammillary process; 5, combined spinal nerve; 6, medial branch of dorsal ramus of spinal nerve; 7, lateral branch of dorsal ramus of spinal nerve; 8, mammilloaccessory ligament; 9, interspinal ligament; 10, vertebral body; 11, transverse process; 12, accessory process. (B) Segmental hypermobile instability associated with degenerative osseous and ligamentous spinal changes. The spine may be hypermobile in marked cases of segmental degenerative spinal alteration causing the spinous processes to traverse through a range of motion (dashed arrows) that is greater than its capacity would otherwise normally allow. The interspinous space (star) may thereby expand to a larger than normal degree. (C) Autotraumatic denervation of paraspinal muscles secondary to hypermobile instability. Because the medial branch of the dorsal ramus of the spinal nerve is fixed, hypermobile instability and associated intrinsic spinal ligament degeneration (double-headed arrows), may result in rupture/avulsion of this nerve (open arrows: or nerves if multisegmental spinal hypermobility exists) and lead to paraspinal muscle deinnervative degeneration. (D) Autotraumatic rupture of the specific paraspinal intrinsic spinal muscles. Alternatively, in cases of degenerative segments or multisegmental spinal hypermobility, the abnormal range of spinal motion (asterisks: ruptured interspinal ligaments) may lead to a rupture or avulsion of specific paraspinal intrinsic spinal muscles that originate and terminate in the spine (double-headed arrows: multifidus, interspinalis muscles). (E) Medical imaging studies: (i) axial T1-weighted MRI showing lateral asymmetry of the multifidus muscles (arrows; compare with (C and D)). (ii) Axial T2-weighted, fat-suppressed MRI showing edema within the multifidus muscles bilaterally and within the perimuscular fat (arrows). (iii) Coronal T1-weighted MRI showing lateral asymmetry of the multifidus and interspinalis muscles with a serpentine configuration of these muscles on the right side (arrowheads) indicating muscular rupture and retraction; note the spinous processes (black dots) and the intervening soft tissue intensity interspinalis ligament remnants (white dots). (iv) Coronal T2-weighted, fat-suppressed MRI showing bilateral muscular and perimuscular edema (solid arrows), hyperintensity within the interspinous ligaments at L3-L4 and L4-L5 (asterisks: interspinous neocyst formation), and a left sided perispinal fluid accumulation (arrowhead), emanating from a narrow connection with the interspinous cyst at L4-L5 (i.e., interspinous neocyst rupture).
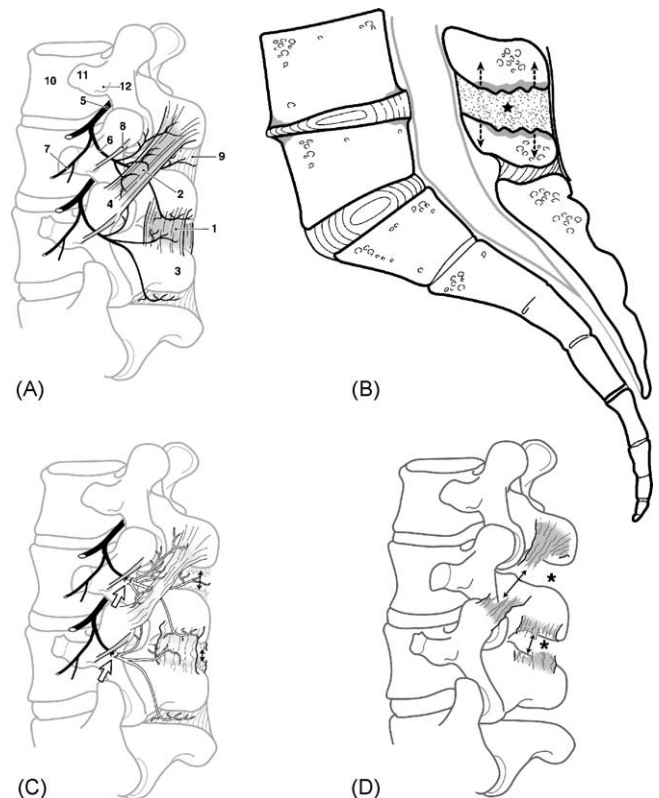


(A)    (B)
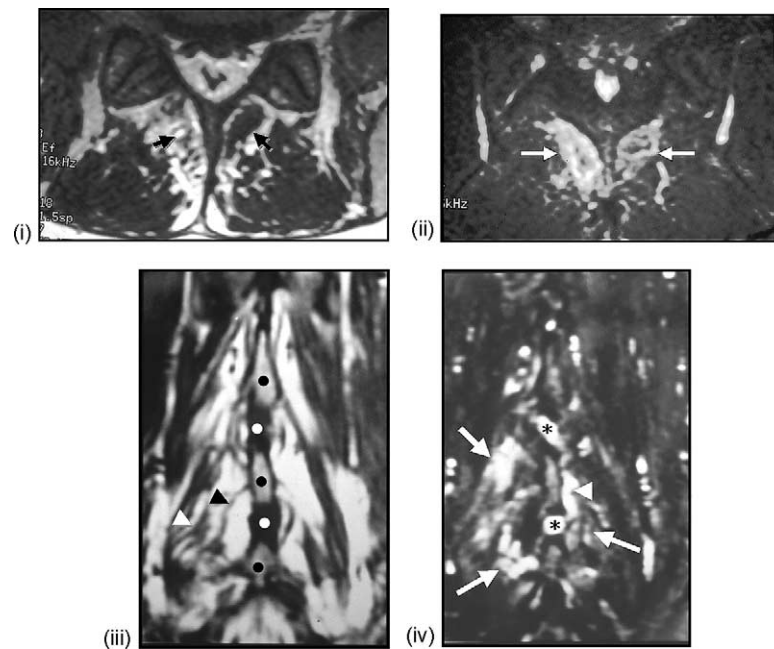
(C)    (D)

Fig. 13. (*Continued*)

Fig. 13. (*Continued*).

foramina, the images yield relevant information concerning the alterations in the posterior spinal facet joints, pedicles, intervertebral disks and their margins, and neural foramina themselves resulting from or accompanying collapse of the intervertebral disks at and suprajacent to the lumbosacral junction. These MR imaging changes complement the information gained from the far-lateral CT reconstructions.

*Analyze the midline sagittal magnetic resonance images and computed tomography reconstructions through the intervertebral disks, the adjacent vertebrae, the spinous processes and the interspinous spaces.* By carefully evaluating the midline sagittal MR images and the sagittal CT reconstructions, the pathologic alterations of the intervertebral disks, the spinal alignment, the retrothecal fat pads and the interspinous spaces will become evident.

*Acquire T2-weighted fast spin echo, fat-suppressed images in the sagittal, axial, and coronal planes.* These T2-weighted, fat-suppressed images allow the visualization of the interspinous ligament degeneration, the neocysts and synovial cysts forming off of the posterior spinal facet (zygapophyseal) joints, and the neocysts emanating from the interspinous neoarthrosis. Also noted is the related perispinous muscle degeneration that sometimes accompanies interspinous ligament degeneration or rupture, and the degenerative marrow alterations [16,91,124].

## 4. Summary

In earlier evolutionary times, mammals were primarily quadrupeds. However, other bipeds have also been represented during the course of the Earth's several billion-year history. In many cases, either the bipedal stance yielded a large tail and hypoplastic upper extremities (e.g., *Tyrannosaurus rex*, the kangaroo), or it culminated in hypoplasia of the tail and further development and specialization of the upper extremities (e.g., nonhuman primates and human beings). In the human species this relatively recently acquired posture resulted in a more or less pronounced lumbosacral kyphosis. In turn, certain compensatory anatomic features have since occurred. These include the normal characteristic posteriorly directed wedge-shape of the L5 vertebral body and the L5-S1 intervertebral disk; the L4 vertebral body and the L4-L5 disk may be similarly visibly affected.

These compensatory mechanisms, however, have proved to be functionally inadequate over the long term of the human life span. Upright posture also leads to increased weight bearing in humans that progressively causes excess stresses at and suprajacent to the lumbosacral junction. These combined factors result in accelerated aging and degenerative changes and a predisposition to frank biomechanical failure of the subcomponents of the spinal column in these spinal segments.

One other specific problem that occurs at the lumbosacral junction that predisposes toward premature degeneration is the singular relationship that exists between a normally mobile segment of spine (i.e., the lumbar spine), and a normally immobile one (i.e., the sacrum). It is well known that mobile spinal segments adjacent to congenitally or acquired fused segments have a predilection toward accelerated degenerative changes. The only segment of the spine in which this is invariably normally true is at the lumbosacral junction (i.e., the unfused lumbar spine adjoining the fused sacrum).

Nevertheless, biomechanical failures of the human spine are not lethal traits; in most cases today, mankind reaches sexual maturity before spinal biomechanical failure pre-

cludes sexual reproduction. For this gene-preserving reason, degenerative spinal disorders will likely be a part of modern societies for the foreseeable eternity of the race.

The detailed alterations accruing from the interrelated consequences of and phenomena contributing to acquired degenerative changes of the lumbosacral intervertebral segments as detailed in this discussion highlight the extraordinary problems that are associated with degenerative disease in this region of the spine. Further clinicoradiologic research in this area will progressively determine the clinical applications and clinical efficacy of the various traditional and newer methods of therapy in patients presenting with symptomatic acquired collapse of the intervertebral disks at and suprajacent to the lumbosacral junction and the interrelated degenerative alterations of the nondiskal structures of the spine [68,71].

## Acknowledgements

## References

[1] Abdullah AF, Chambers RW, Daut DP. Lumbar nerve root compression by synovial cysts of the ligamentum flavum. J Neurosurg 1984;60:617–20.

[2] Adams MA, Hutton WC. The mechanical function of the lumbar apophyseal joints. Spine 1983;8:327–30.

[3] Adams MA, Hutton WC, Stoff JRR. The resistance to flexion of lumbar intervertebral joint. Spine 1980;5:245–53.

[4] Aharinejad S, Bertagnoli R, Wicke K, et al. Morphometric analysis of vertebrae and intervertebral disks as a basis of disk replacement. Am J Anat 1990;189:69–76.

[5] Ahmed AM, Duncan NA, Burke DL. The effect of facet geometry on the axial torque-rotation response of lumbar motion segments. Spine 1990;15:391–401.

[6] Amonoo-Kuofi HS. Age-related variations in the horizontal and vertical diameters of the pedicles of the lumbar spine. J Anat 1995;186:321–8.

[7] Amonoo-Kuofi HS. Morphometric changes in the heights and anteroposterior diameters of the lumbar intervertebral discs with age. J Anat 1991;175:159–68.

[8] Awwad EE, Martin DS, Smith KR. MR imaging of lumbar justaarticular cysts. J Comput Assist Tomogr 1990;14:415–7.

[9] Baastrup CI. Le 'lumbago' et les affections radiologiques des apophyses épineuses des vertèbres lombaires, de la 1st vertèbre sacrèe et des parties interepincuses. J Radiol 1936;20:78–93.

[10] Baastrup CI. On the spinous processes of the lumbar vertebrae and the soft tissue between them and on pathological changes in that region. Acta Radiol 1933;14:52–4.

[11] Baastrup CI. Proc. Spin. Vert. lumb. Und einige zwischen diesen liegende gelenkbildungen mit pathologischen prozessen in dieser region. RöFo 1933;48:430–5.

[12] Baker JK, Hanson GW. Cyst of the ligamentum flavum. Spine 1994;19:1092–4.

[13] Beamer YB, Garner JT, Shelden CH. Hypertrophied ligamentum flavum. Arch Surg 1973;106:289–92.

[14] Berlemann U, Jeszenszky DJ, Buhler DW, et al. Facet joint remodeling in degenerative spondylolisthesis: an investigation of joint orientation and tropism. Eur Spine J 1998;7:376–80.

[15] Berlemann U, Jeszenszky DJ, Buhler DW, et al. The role of lumbar lordosis, vertebral end-plate inclination, disc height, and facet orientation in degenerative spondylolisthesis. J Spinal Disord 1999;12:68–73.

[16] Bobman SA, Atlas SW, Listerud J, et al. Postoperative lumbar spine: contrast-enhanced chemical shift MR Imaging. Radiology 1991;179:557–62.

[17] Boden SD, Riew KD, Yamaguchi K, et al. Orientation of the lumbar facet joints: association with degenerative disc disease. J Bone Joint Surg Am 1996;78:403–11.

[18] Bogduk N. The anatomy of the lumbar intervertebral disc syndrome. Med J Aust 1976;1:878–81.

[19] Bogduk N. Lumbar dorsal ramus syndrome. Med J Aust 1980;2:537–41.

[20] Bogduk N, Wilson AS, Tynan W. The human lumbar dorsal rami. J Anat 1982;134:383–97.

[21] Boos N, Wallin A, Schmucker T, et al. Quantitative MR imaging of lumbar intervertebral discs and vertebral bodies: methodology, reproducibility, and preliminary results. Magn Reson Imag 1994;12:577–87.

[22] Bram J, Zanetti M, Min K, et al. MR abnormalities of the intervertebral discs and adjacent bone marrow as predictors of segmental instability of the lumbar spine. Acta Radiol 1998;39:18–23.

[23] Brown R, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984;9:7–15.

[24] Butler D, Trafimow JH, Anderson GBJ, et al. Discs degenerate before facets. Spine 1990;15:111–3.

[25] Bywaters EGL. Lesions of bursae, tendons and tendon sheaths. Clin Rheum Dis 1979;5:883–925.

[26] Bywaters EGL, Evans S. The lumbar interspinous bursae and Baastrup's syndrome. Rheumatol Int 1982;2:87–96.

[27] Cauchoix J, Benoist M, Chassaing V. Degenerative spondylolisthesis. Clin Orthop Rel Res 1976;115:122–9.

[28] Cheng XG, Sun Y, Boonen S, et al. Measurements of vertebral shape by radiographic morphometry: sex differences and relationships with vertebral level and lumbar lordosis. Skeletal Radiol 1998;27:380–4.

[29] Cinotti G, Postacchini F, Fassari F, et al. Predisposing factors in degenerative spondylolisthesis: a radiographic and CT study. Int Orthop 1997;21:337–42.

[30] Conrad MR, Pitkethly DT. Bilateral synovial cysts creating spinal stenosis: CT diagnosis. J Comput Assist Tomogr 1987;11:196–7.

[31] Crock HV. Isolated lumbar disk resorption as a cause of nerve root canal stenosis. Clin Orthop Rel Res 1976;115:109–15.

[32] Cyron BM, Hutton WC. Articular tropism and stability of the lumbar spine. Spine 1980;5:168–72.

[33] DeRoos A, Kressel H, Spritzer C, et al. MR imaging of marrow changes adjacent to end plates in degenerative lumbar disk disease. AJR Am J Roentgenol 1987;149:531–4.

[34] Diacinti D, Acca M, D'Erasmo E, et al. Aging changes in vertebral morphometry. Calcif Tissue Int 1995;57:426–9.

[35] Dofferhof ASM, Vink P. The stabilizing function of the mm. iliocostales and the mm. multifidi during walking. J Anat 1985;10:329–36.

[36] Dunlop RB, Adams MA, Hutton WC. Disc space narrowing and the lumbar facet joints. J Bone Joint Surg Br 1984;66:706–10.

[37] Dupuis PR, Yong-Hing K, Cassidy JD, et al. Radiologic diagnosis of degenerative lumbar spinal instability. Spine 1985;10:262–76.

[38] Eisenstein SM, Parry CR. The lumbar facet arthrosis syndrome: clinical presentation and articular surface changes. J Bone Joint Surg Br 1987;69:3–7.

[39] El Mahdi MA, Farouk Y, Latif A, et al. The spinal nerve root "innervation. Neurochirurgia 1981;24:17–141.

[40] Ericksen MF. Aging in the lumbar spine: II. L1 and L2. Am J Phys Anthropol 1978;45:241–5.

[41] Ericksen MF. Aging in the lumbar spine. III. L5. Am J Phys Anthropol 1978;48:247–50.

[42] Ericksen MF. Some aspects of aging in the lumbar spine. Am J Phys Anthropol 1976;45:575–80.

[43] Farfan HF. The pathological anatomy of degenerative spondylolisthesis: a cadaver study. Spine 1980;5:412–8.

[44] Fitzgerald JAW, Newman PH. Degenerative spondylolisthesis. J Bone Joint Surg Br 1976;58:184–92.

[45] Fleckenstein JL, Chason DP. Skeletal muscles. In: Stark DD, Bradley WG, editors. Magnetic resonance imaging. St. Louis: Mosby; 1999. p. 1057–77.

[46] Gallagher JC, Hedlund LR, Stoner S, et al. Vertebral morphometry: normative data. Bone Miner 1988;4:189–96.

[47] Giles LGF, Kaveri MJP. Some osseous and soft tissue causes of human intervertebral canal (foramen) stenosis. J Rheumatol 1990;17:1474–81.

[48] Giles LGF, Taylor JR. The effect of postural scoliosis on lumbar apophyseal joints. Scand J Rheumatol 1984;13:209–20.

[49] Goobar JE, Clark GM. Sclerosis of the spinous processes and low back pain (cock spur disease). Arch Interam Rheum 1962;5:587–98.

[50] Goobar JE, Sartoris DJ, Hajek PC, et al. Magnetic resonance imaging of the lumbar spinous processes and adjacent soft tissues: normal and pathologic appearances. J Rheumatol 1987;14:788–97.

[51] Grenier N, Kressel HY, Schiebler ML, et al. Normal and degenerative posterior spinal structures: MR imaging. Radiology 1987;165:517–25.

[52] Grobler LJ, Robertson PA, Novotny JE, et al. Etiology of spondylolisthesis: assessment of the role played by lumbar facet joint morphology. Spine 1993;18:80–91.

[53] Haase J. Extradural cyst of ligamentum flavum L4: a case. Acta Orthop Scand 1972;43:32–8.

[54] Hackett GS. Referred pain from low back ligament disability. Arch Surg 1956;73:878–83.

[55] Hadley LA. Anatomico-roentgenographic studies of the posterior spinal articulations. AJR Am J Roentgenol 1961;86:270–6.

[56] Hadley LH. Apophyseal sublaxation. J Bone Joint Surg 1936;18:428–33.

[57] Hadley LH. Intervertebral joint subluxation: bony impingement and foramen encroachment with nerve root changes. AJR Am J Roentgenol 1951;65:377–402.

[58] Hasegawa T, An HS, Haughton VM, et al. Lumbar foraminal stenosis: critical heights of the intervertebral discs and foramina. J Bone Joint Surg Am 1995;77:32–8.

[59] Hemminghytt S, Daniels DL, Williams AL, et al. Intraspinal synovial cysts: natural history and diagnosis by CT. Radiology 1982;145:375–6.

[60] Herkowitz HN. Spine update: degenerative lumbar spondylolisthesis. Spine 1995;20:1084–90.

[61] Herkowitz HN, Kurz LT. Degenerative lumbar spondylolisthesis with spinal stenosis. J Bone Joint Surg Am 1991;73:802–8.

[62] Heylings DJA. Supraspinous and interspinous ligaments of the human lumbar spine. J Anat 1978;125:127–31.

[63] Howes RG, Isdele IC. The loose back: an unrecognized syndrome. Rheum Phys Med 1971;11:72–7.

[64] Hsu KY, Zucherman JF, Shea WJ, et al. Lumbar intraspinal synovial and ganglion cysts (facet cysts). Spine 1995;20:80–9.

[65] Jackson DE, Atlas SW, Mani JR, et al. Intraspinal synovial cysts: MR imaging. Radiology 1989;170:527–30.

[66] Jinkins JR. Acquired degenerative changes of the intervertebral segments at and suprajacent to the lumbosacral junction: a radioanatomic analysis of the discal and nondiscal structures of the spinal column and perispinal soft tissues. In: Kaech DL, Jinkins JR, editors. Spinal Restabilization Procedures: Diagnostic and Therapeutic Aspects of Intervertebral Fusion Cages, Arificial Discs and Mobile Implants. Amsterdam: Elsevier, 2002. p. 3–36.

[67] Jinkins JR. The pathoanatomic basis of somatic and autonomic syndromes originating in the lumbosacral spine. Neuroimaging Clin North Am 1993;3:443–63.

[68] Jinkins JR. Acquired degenerative changes of the intervertebral segments at and suprajacent to the lumbosacral junction: a radioanatomic analysis of the discal and nondiscal structures of the spinal column and perispinal soft tissues. Rivista Neuroradiol 2002;15:359–92.

[69] Johnson RW. Posterior luxations of the lumbosacral joint. J Bone Joint Surg 1934;16:867–76.

[70] Jonsson B. The functions of individual muscles in the lumbar part of the spinal muscle. Electromyography 1970;1:5–21.

[71] Kaech DL, Jinkins JR. Clinicoradiologic assessment after disc cage implantation. Rivista Neuroradiol 1999;12:129–40.

[72] Kao CC, Uihlein A, Bickel WH, et al. Lumbar intraspinal extradural ganglion cyst. J Neurosurg 1968;29:168–72.

[73] Kénési C, Lesur E. Orientation of the articular processes at L4, L5, and S1: possible role in pathology of the intervertebral disc. Anat Clin 1985;7:43–7.

[74] Kim NH, Lee JW. The relationship between isthmic and degenerative spondylolisthesis and the configuration of the lamina and facet joints. Eur Spine J 1995;4:139–44.

[75] Kirkaldy-Willis WH, Weddge JH, Yong-Hing K, et al. Pathology and pathogenesis of lumbar spondylosis and stenosis. Spine 1978;3:319–28.

[76] Kjerulf TD, Terry DW, Boubelik RJ. Lumbar synovial or ganglion cysts. Neurosurgery 1986;19:415–20.

[77] Knox AM, Fon GT. The appearances of lumbar intraspinal synovial cysts. Clin Radiol 1991;44:397–401.

[78] Lewin T. Anatomical variations in lumbosacral synovial joints with particular reference to sublaxation. Acta Anat 1968;71:229–48.

[79] Lewin T. Osteoarthritis in lumbar synovial joints: a morphologic study. Acta Orthop Scand 1964;73:1–12.

[80] Lewin T, Reichman S. Anatomical variations in the S-shaped contour of the lumbar aricular processes with special reference to subluxation. Acta Morphologica Scandinavica 1968;7:179–84.

[81] Lewin T, Reichman S. Bony contacts between the tips of the articular processes and adjacent parts of the cerebral arch in the lumbar spine in young individuals. Acta Morphologica Neerlando-Scandinavica 1968;7:185–93.

[82] Louis R. Spinal stability as defined by the three-column spine concept. Anat Clin 1985;7:33–42.

[83] Mahallati H, Wallace CJ, Hunter KM, et al. MR imaging of a hemorrhagic and granulomatous cyst of the ligamentum flavum with pathologic correlation. AJNR Am J Neuroradiol 1999;20:1166–8.

[84] Maresca L, Meland NB, Maresca C, et al. Ganglion cyst of the spinal canal: case report. J Neurosurg 1982;57:140–2.

[85] Matsunaga S, Sakou T, Morizono Y, et al. Natural history of degenerative spondylolisthesis: pathogenesis and natural course of the slippage. Spine 1990;15:1204–10.

[86] Mayer: Ueber zwei neu entdeckte gelenke an der wirbelsäule des menschlichen korpers. Z Physiol 1825;2:29–35.

[87] Mayoux-Benhamou MA, Revel M, Aaron C, et al. A morphometric study of the lumbar foramen: influence of flexion-extension movements and of isolated disc collapse. Surg Radiol Anat 1989;11:97–102.

[88] McCall IW, Park WM, O'Brien JP. Induced pain referral from posterior lumbar elements in normal subjects. Spine 1979;4:441–6.

[89] McCarthy EF, Dorfman HD. Idiopathic segmental sclerosis of vertebral bodies. Skeletal Radiol 1982;9:88–91.

[90] Mercader J, Gomez JM, Cardenal C. Intraspinal synovial cyst: diagnosis by CT. Follow-up and spontaneous remission. Neuroradiology 1985;27:346–8.

[91] Mirowitz SA, Shady KL. Gadopentetate dimeglumine-enhanced MR imaging of the postoperative lumbar spine: comparison of fat-suppressed and conventional T1-weighted images. AJR Am J Roentgenol 1992;159:385–9.

[92] Modic MT, Steinberg PM, Ross JS, et al. Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. Radiology 1988;166:193–9.

[93] Morris JM, Benner G, Lucas DB, et al. An eletromyographic study of the intrinsic muscles of the back in man. J Anat 1962;96:509–20.

[94] Murtagh FR, Paulsen RD, Rechtine GR. The role and incidence of facet tropism in lumbar spine degenerative disc disease. J Spinal Disord 1991;4:86–9.

[95] Nachemson AL, Schultz AB, Berkson MH. Mechanical properties of human lumbar spine motion segments: influences of age, sex, disc level, and degeneration. Spine 1979;4:1–8.

[96] Nagaosa Y, Kikuchi S, Hasue M, et al. Pathoanatomic mechanisms of degenerative spondylolisthesis: a radiographic study. Spine 1998;13:1447–51.

[97] Newman PH. Sprung back. J Bone Joint Surg Br 1952;34:30–7.

[98] Nissan M, Gilad I. Dimensions of human lumbar vertebrae in the sagittal plane. J Biomech 1986;19:753–8.

[99] Panjabi MM, Goel V, Oxland T, et al. Human lumbar vertebrae: quantitative three dimensional anatomy. Spine 1992;17:299–306.

[100] Panjabi MM, Goel VK, Takata K. Physiologic strains in the lumbar spinal ligaments: an in vitro biomechanical study. Spine 1983;7:192–203.

[101] Panjabi MM, Oxland T, Takata K, et al. Articular facets of the human spine: quantitative three-dimensional anatomy. Spine 1993;18:1298–310.

[102] Peck D, Nicholis PJ, Beard C, et al. Are there compartment syndromes in some patients with idiopathic back pain? Spine 1986;11:468–75.

[103] Pfaff R. Treatment of acute lumbosacral strain. J Fla Med Assoc 1967;54:968–71.

[104] Postacchini F, Penigia D. Degenerative lumbar spondylolisthesis. I. Etiology, pathogenesis, pathomorphology, and clinical features. Ital Orthop Traumatol 1991;17:165–73.

[105] Postacchini F, Rauschning W. Anatomy of the vertebral canal and the neural structures in the lumbar spine. In: Postacchini F, editor. Lumbar spinal stenosis. Vienna: Springer-Verlag; 1989. p. 87–99.

[106] Postacchini F, Rauschning W. Pathomorphology. In: Postacchini F, editor. Lumbar spinal stenosis. Vienna: Spring-Verlag; 1989. p. 87–99.

[107] Putz R. The functional morphology of the superior articular processes of the lumbar vertebrae. J Anat 1985;143:181–7.

[108] Rauschning W. Detailed sectional anatomy of the spine. In: Rothman SLG, Glenn WV, editors. Multiplanar CT of the spine. Baltimore: University Park Press; 1985. p. 33–85.

[109] Rauschning W. Normal and pathologic anatomy of the lumbar root canals. Spine 1987;12:1008–19.

[110] Rauschning W. Pathoanatomy of lumbar disc degeneration and stenosis. Acta Orthop Scand Suppl 1993;251:3–12.

[111] Resnick D. Degenerative diseases of the vertebral column. Radiology 1985;156:3–14.

[112] Reust P, Wendling D, Lagier R, et al. Degenerative spondylolisthesis, synovial cyst of the zygapophyseal joints, and sciatic syndrome: report of two cases and review of the literature. Arthritis Rheum 1988;31:288–94.

[113] Rissanen PM. Comparison of pathologic changes in intervertebral discs and interspinous ligaments of the lower part of the lumbar spine in the light of autopsy findings. Acta Orthop Scand 1964;1:54–65.

[114] Rissanen PM. "Kissing-spine" syndrome in the light of autopsy findings. Acta Orthop Scand 1962;32:132–9.

[115] Rissanen PM. The surgical anatomy and pathology of the supraspinous and interspinous ligaments of the lumbar spine with special reference to ligament ruptures. Acta Orthop Scand 1960;46:1–100.

[116] Rosenberg NJ. Degenerative spondylolisthesis: predisposing factors. J Bone Joint Surg Am 1975;57:467–74.

[117] Rosenblum J, Mojtahedi S, Foust RJ. Synovial cysts in the lumbar spine: MR characteristics. AJNR Am J Neuroradiol 1989;10:94.

[118] Sartoris DJ, Resnick D, Tyson R, et al. Age-related alterations in the vertebral spinous processes and intervening soft tissues: radiologic-pathologic correlation. AJR Am J Roentgenol 1985;145:1025–30.

[119] Sato K, Wakamatsu E, Yoshizumi A, et al. The configuration of the laminas and facet joints in degenerative spondylolisthesis: a clinicoradiologic study. Spine 1989;14:1265–71.

[120] Satomi K, Hirabayashi K, Toyama Y, et al. A clinical study of degenerative spondylolisthesis: radiographic analysis and choice of treatment. Spine 1992;14:1329–36.

[121] Scoles PV, Linton AE, Latimer B, et al. Vertebral body and posterior element morphology: the normal spine in middle life. Spine 1988;13:1082–6.

[122] Sharma M, Langrana NA, Rodriguez J. Role of ligaments and facets in lumbar spinal stability. Spine 1995;20:887–900.

[123] Silbergleit R, Gebarski SS, Brunberg JA, et al. Lumbar synovial cysts: correlation of myelographic, CT, MR and pathologic findings. AJNR Am J Neuroradiol 1990;11:777–9.

[124] Simon JH, Szumowski J. Chemical shift imaging with paramagnetic contrast material enhancement for improved lesion depiction. Radiology 1989;171:539–43.

[125] Sinclair DC, Feindel WH, Weddell G, et al. The intervertebral ligaments as a source of segmental pain. J Bone Joint Surg Br 1948;30:515–21.

[126] Spencer DL, Irwin GS, Miller JAA. Anatomy and significance of fixation of the lumbarsacral nerve roots in sciatica. Spine 1983;8:672–9.

[127] Taylor JR, Twomey LT. Age changes in lumbar zygapophyseal joints: observations on structure and function. Spine 1986;11:739–45.

[128] Tournade A, Patay Z, Krupa P, et al. A comparative study of the anatomical, radiological and therapeutic features of the lumbar facet joints. Neuroradiology 1992;39:257–61.

[129] Toyone T, Takahashi K, Kitahara H, et al. Vertebral bone-marrow changes in degenerative lumbar disc disease. J Bone Joint Surg Br 1994;76:757–64.

[130] Tulsi RS, Hermanis GM. A study of the angle of inclination and facet curvature of superior lumbar zygapophyseal facets. Spine 1993;18:1311–7.

[131] Vanharanta H, Floyd T, Ohnmeiss DD, et al. The relationship of facet tropism to degenerative disc disease. Spine 1993;18:1000–5.

[132] van Schaik JPJ, van Pinxteren B. Curvature of the lower lumbar facet joints: variations at different levels and relationship with orientation. J Spinal Disord 1999;1:341–7.

[133] van Schaik JPJ, Verbiest H, Frans DJ, et al. The orientation of laminae and facet joints in the lower lumbar spine. Spin 1985;10:59–63.

[134] Vernet O, Fankhauser H, Schnyder P, et al. Cyst of the ligamentum flavum. Report of six cases. Neurosurgery 1991;29:277–83.

[135] Vernon-Roberts B, Pirie CJ. Degenerative changes in the intervertebral discs of the lumbar spine and their sequelae. Rheumatol Rehabil 1977;16:13–21.

[136] Warfel JH. Multifidus, interspinales. In the Head, Neck, and Trunk. Philadelphia: Lea and Febiger; 1985. p. 57–9.

[137] Waters RL, Morris JM. Electrical activity of muscles of the trunk during walking. J Anat 1972;111:191–9.