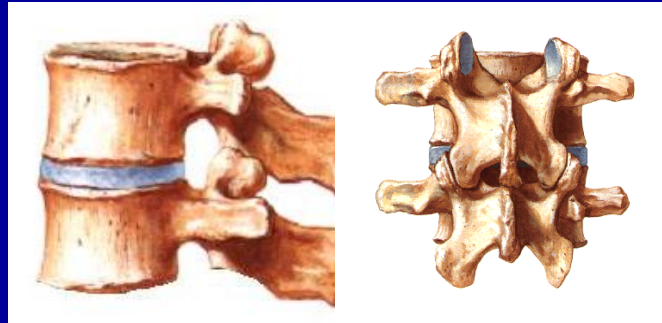


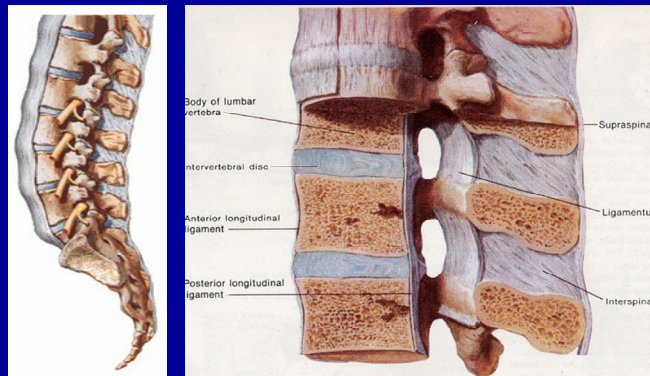
Lumbar Spine Motion Segment

- Three joint complex
- Intervertebral disc + 2 facet joint
- Ligamentous structure, vertebral body



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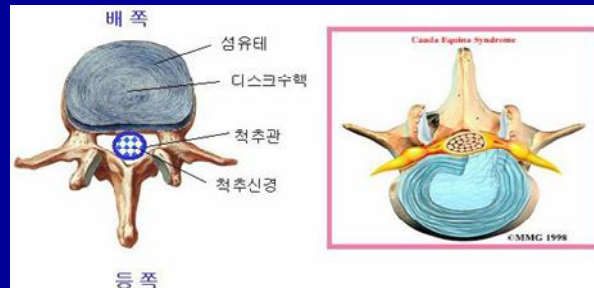


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Intervertebral Disc

- Hydrostatic, load bearing structure between the vertebral bodies
- Nucleus pulposus + annulus fibrosus
- No blood supply
- L4-5, largest avascular structure in the body

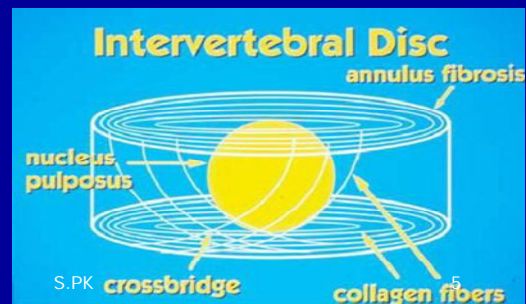


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Nucleus Pulposus

- Type II collagen strand + hydrophilic proteoglycan
- Water content 70 ~ 90%
- Confine fluid within the annulus
- Convert load into tensile strain on the annular fibers and vertebral end-plate
- Chondrocyte manufacture the matrix component

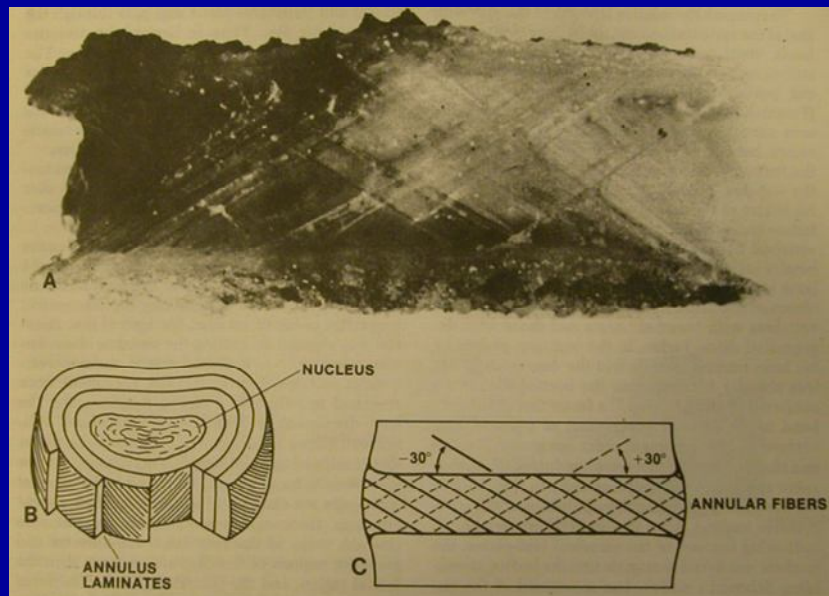


Annulus Fibrosus

- Outer boundary of the disc
- More than 60 distinct, concentric layer of overlapping lamellae of type I collagen
- Fibers are oriented 30-degree angle to the disc space
- Helicoid pattern
- Resist tensile, torsional, and radial stress
- Attached to the cartilaginous and bony end-plate at the periphery of the vertebra

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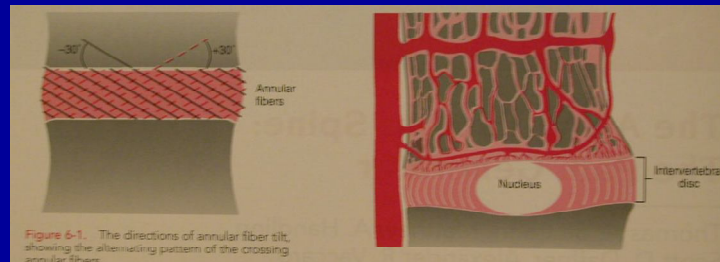


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Vertebral End-Plate

- Cartilaginous and osseous component
- Nutritional support for the nucleus
- Passive diffusion

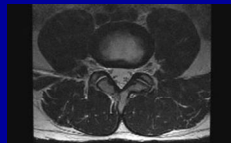


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Facet Joint

- Synovial joint
- Rich innervation with sensory nerve fiber
- Same pathologic process as other large synovial joint
- Load share 18% of the lumbar spine



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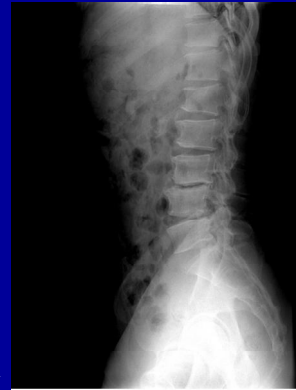
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Spondylosis

- Generalized process of the axial skeleton
- Sequence of degenerative change
- Start biochemical and cellular level
- Manifest biomechanical and morphologic level



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Initiating Factor in Degenerative Cascade

- Injury to annulus fibrosus
- Matrix composition alteration of the nucleus pulposus
- Vascularity and permeability change of end-plate
- Primary causative agent??
- The process of disc degeneration is multifactorial



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Disc Degeneration

- Environmental factor
- Genetic predisposition
- Normal aging process

- * Biomechanical stress
 - Degeneration of soft tissue and bone
 - progressive morphologic change

Intervertebral Disc

Cellular and Biochemical Change

- Decrease proteoglycan content
- Loss of negative charged proteoglycan side chain
- Water loss within the nucleus pulposus
- Decrease hydrostatic property
- Loss of disc height
- Uneven stress distribution on the annulus



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Morphologic Changes

- bulging of the annulus fibrosus
- radial tear
- in-growth of granulation tissue in the annulus
- annular defect, cleft and fissure
- cellular necrosis → loss of distinction between the nucleus and annulus
- focal extrusion of disc material

Aging Progress

- disc become more fibrous and disorganized
- replaced by amorphous fibrocartilage
- no clear distinction between nucleus and annulus
- gas formation and vacuum disc sign

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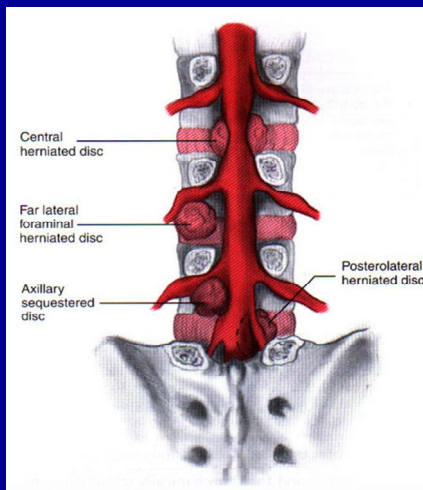
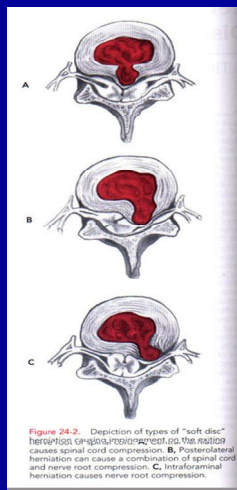
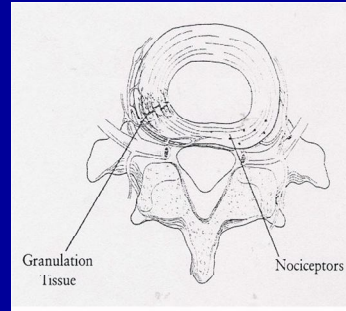
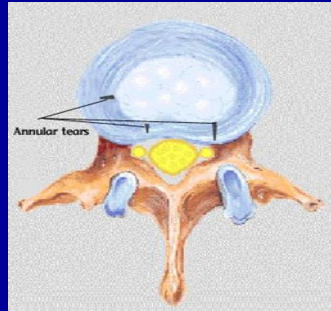
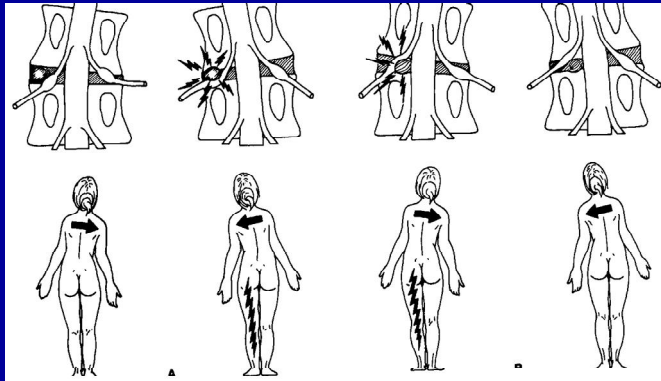


Figure 24.2. Depiction of types of "soft disc" herniations. **A.** Posteriorly directed disc herniation causes spinal cord compression. **B.** Posterolateral herniation can cause a combination of spinal cord and nerve root compression. **C.** Intralaminar herniation causes nerve root compression.

Pain and movements



Vertebral End-Plate

- Become thinner and hyalinized
- Decrease permeability
- Inhibit nucleus metabolism
- Disc space narrowing
- Osteophyte formation at the end-plate and annular junction
- Marrow change with increased axial loading
- Subluxation and instability

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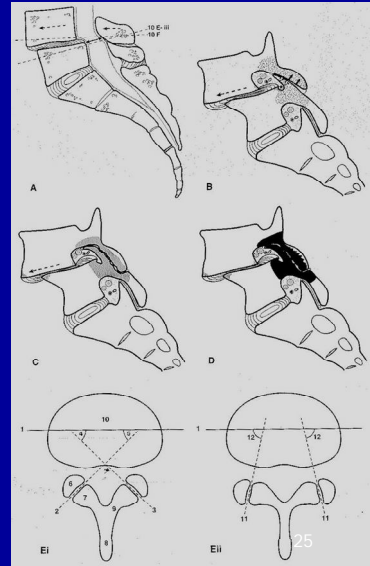
Facet Joint

Disc height reduction →

- Facet joint capsule become lax
- Increased load transfer to the facet joint
- Accelerate degeneration
- Joint subluxation, hypertrophy, osteophyte formation

- ** Primary disc degeneration
→ Secondary change in the posterior facet joint and soft tissue

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Lumbar Disc Disease

Discogenic Back Pain

- A. Internal Disc Disruption (IDD)
- B. Degenerative Disc Disease (DDD)
- C. Segmental Instability

Lumbar Disc Herniation and Radiculopathy

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Lumbar Disc Herniation

How pain is generated?

- Inflammatory
- Biochemical
- Vascular
- Mechanical compression



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Inflammation

- Central role in radiculopathy
- Olmarker(1995, spine)
 - Epidural application of autologous nucleus without any pressure
 - Nerve function impairment
 - Axonal injury with significant primary cell damage
- Nucleus is totally avascular
 - Perceived as an antigen
 - Intense inflammation response
- Application of annulus fibrosus
 - No reduction of nerve conduction velocity

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Biochemical Effect

- Nuclear herniation
 - Increase phospholipase A2, prostaglandin E2, cytokine, nitric oxide
- Disc herniation and sciatica
 - Neurofilament protein and S-100 increase in CSF
 - Axonal and Schwann's cell damage

Mechanical Compression

- Local damage and intraneural ischemia

Vascular Pathophysiology

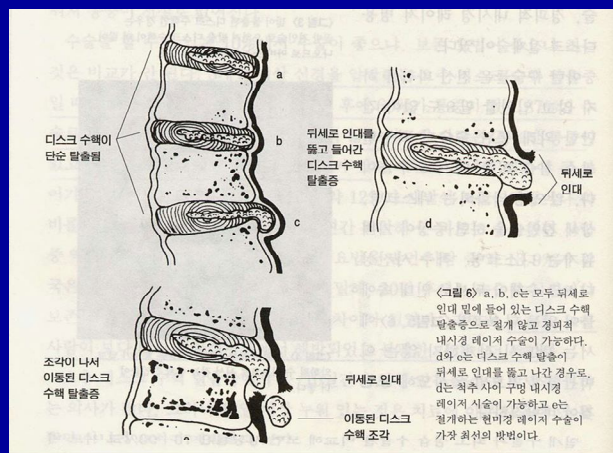
- * Application of nucleus pulposus to nerve root
 - increase endoneurial pressure
 - decrease blood flow in the dorsal root ganglia
 - compartment syndrome

Clinical Anatomy

- Disc injury
 - annular disruption, fissuring, annular defect
- Contained herniation
Noncontained herniation
Extruded
Sequestrated
- L4- 5 and L5- S1 herniation most common
 - 90% of disc herniation
 - Great axial load, lordotic shear

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History

- symptom of disc herniation : acute or gradual
- after trauma or without and inciting event
- most common 3rd and 4th decade

Chief Complain

- Pain, radiating from the back or buttock into the leg
- Numbness and weakness
- Sharp, lancinating, shooting/radiating down the leg posteriorly below the knee
- Coughing, Valsalva maneuver → increase intracecal pressure → increase pain
- Sitting position, driving → out of lordosis → increase intradiscal pressure → increase pain

Sciatica

- radiating pain down the leg

Radiculopathy

- radiating pain down the leg as a result of nerve root irritation

Back Pain

- irritation of the posterior primary ramus
 - facet capsule, local musculature
- sinuvertebral branch - posterior annulus
- change in disc loading and shape, biomechanics
- loss of viscoelasticity.
- 90% of radiating pain have long-standing prior episodic low back pain

Quality of pain and associated symptom

- dull ache or sharp, stabbing pain?
- electricity, tingling, numbness, shooting down the leg?
- any associated weakness?
- does anything make the pain better or worse?
- forward flexion or hyperextension exacerbate or relieve pain?
- standing more comfortable than sitting?

** Back pain abated when leg pain developed

→ relief of annular tensile stress, nerve root irritation

** Isolated leg pain → acute disc extrusion

Differential Diagnosis

Vascular claudication

- Vascular assessment and flow study
- Dorsalis pedis palpation

Spinal stenosis

- leg pain, dysesthesia, paresthesia, often not dermatomal
- pain d/t mechanical compression of spinal canal and foramen
- lordosis and axial loading
- symptomatic on walking, relief by sitting

Thrombophlebitis

Metabolic and peripheral neuropathy

Physical Examination

Inspection

- Old scar, muscle spasm, cutaneous stigma, spinal alignment, loss of lordosis

Palpation

- Midline, sciatic notch, iliac crest, SI joint, coccyx
- Paraspinal tenderness, rigidity
- Costovertebral angle, abdomen
- Kidney, stone, retroperitoneal abnormality

Hip pathology

- Patrick test

Skin

- Temperature and atrophic change

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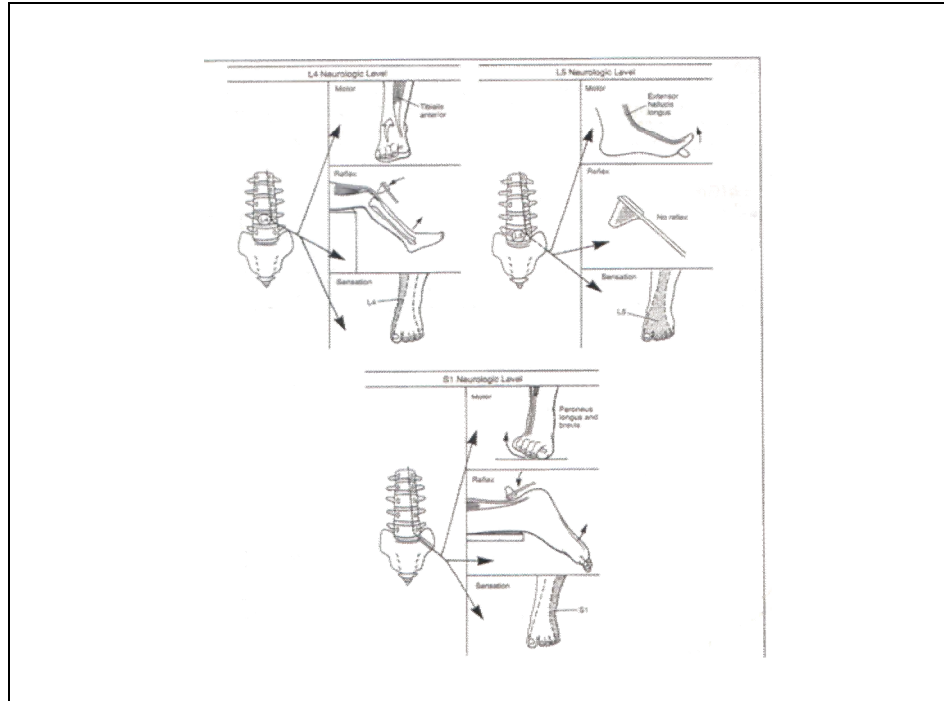
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Neurologic Examination

Root	Sensory	Motor	Reflex
L-1	Groin		—
L-2	Anterior thigh	Iliopsoas	—
L-3	Lateral thigh/knee	Quadriceps	—
L-4	Medial leg (postero-lateral thigh, across patella, anteromedial leg)	Anterior tibialis, quadriceps	Patella
L-5	First dorsal web space; medial foot (posterior thigh, anterolateral leg, medial foot, and great toe)	Extensor hallucis longus; extensor digitorum longus and brevis, gluteus medius	None (post-tibialis)
S-1	Lateral foot (posterior thigh and leg, posterolateral foot, lateral foot)	Gastrocnemius; peroneus longus and brevis, gluteus maximus	Achilles

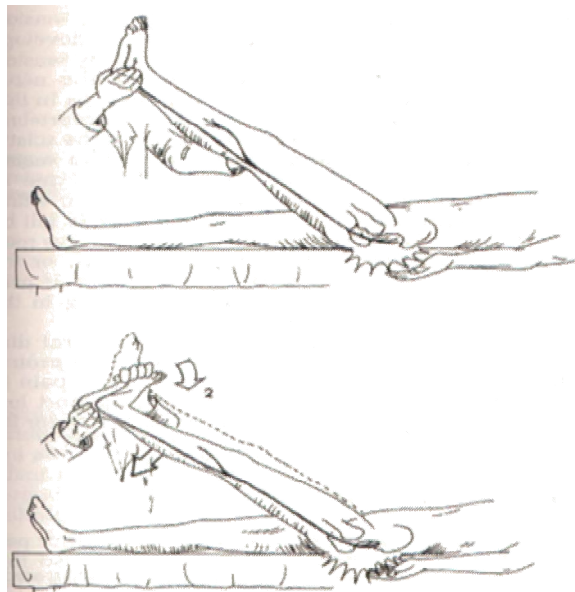
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Root Tension Signs

- Straight- leg raising : L5, S1 root
- Contralateral SLR : sequestrated or extruded disc
- Femoral stretching, reverse SLR : L3, L4 root



Diagnostic Test

Simple x-ray

- Disc space narrowing

MRI(magnetic resonance imaging)

- Disc pathology, neural structure, musculoligamentous structure
- Soft tissue edema, hematoma, intrinsic cord abnormality
- Synovial cyst, neurofibroma, perineural cyst
- 30% of asymptomatic individual have abnormal MRI

CT, Myelography



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Nonoperative Treatment

- 90% of patient improve with conservative treatment
- Short-term rest, NSAID, analgesics, antispasmodic medication, exercise
- Physical therapy
- Oral corticosteroid

**** Conservative treatment should continue for 6 weeks, before other measure are attempted**

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Indication of Surgery

Ideal candidate

- history, physical examination, radiographic finding, are consistent with one another
- when discrepancy exist, the clinical picture should serve as the principal guide.

Absolute surgical indication

- cauda equina syndrome
- acute urinary retention/incontinence, saddle anesthesia, back/buttock/leg pain, weakness, difficulty walking

Relative indication

- progressive weakness
- no response to conservative treatment

Best predictive factor

1. persistent leg pain, that fail to respond to a 6-week trial of nonoperative care
2. well - defined neurologic deficit
3. positive SLR test
4. positive imaging that correlates anatomically to clinical findings

** 3 of these factor, at least 90% success rate

Other factors

- duration of sciatica, sick leave stress, depression, level of education, work/disability