MRI

TR  Repetition Time
TE  Echo Time

Signal Intensity
High signal – bright
Low signal – dark

T1-Weighted Image (T1WI)
Short TR (300-600 msec)
Short TE (10-45 msec)

T1WI
Water – low signal (dark)
Fat – high signal (bright)

T2-Weighted Image (T2WI)
Long TR (1500-300 msec)
Long TE (56-100 msec)

T2WI
Water – high signal (bright)

Balance (Proton-Density) Image
Long TR (1500-3000 msec)
Short TE (20-45 msec)

Fat Saturation

Certain scanning techniques result in the suppression (reduction) of signal intensity arising from fat. The two main techniques are inversion recovery imaging and frequency-selective fat suppression.

Inversion Recovery

Commonly known as STIR (short tau inversion recovery). This technique results in excellent fat suppression and high signal intensity from areas of fluid or edema. It is extremely sensitive for detecting many types of acute pathology.
MRI with Enhancement

Gadolinium - rare earth metal. Chelated to diethylenetriamine pentaacetic acid (DTPA). Magnevist (gadopentetate dimeglumine).

Paramagnetic contrast augments signal intensity from the magnetized tissue. When paramagnetic ions are placed in a strong external magnetic field, their large magnetic dipole moments influence nearby protons by changing their relaxation times. The most pronounced effect of decreased relaxation is seen in the T1 signal. When paramagnetic ions are introduced into tissues with very similar relaxation times, the relaxation times will not be equally affected. Signal differences become more pronounced and there will be more contrast between the tissues.

Reduces hydrogen proton relaxation times. Induces increased signal intensity and enhancement of T1-weighted images. Aids in the visualization of lesions with abnormal vascularity or those thought to cause an abnormality in the blood-brain barrier. Administered intravenously. Rapidly excreted by the kidneys. Most common-adverse reactions: Headache and injection-site coldness.

Enhancement is determined by three factors:
1. A blood supply
2. A leaky capillary endothelium
3. An extravascular space
**Pain of Somatic Origin**

Anatomic structures supplied by nociceptive nerve endings (nerve endings sensitive to tissue damage). Nociceptors may be stimulated by mechanical, thermal, or chemical means.

**Structure innervated by the ventral ramus**
- Referred pain from structures innervated by nerves of the lumbar plexus
- Psoas muscle
- Quadratus lumborum muscle
- Intertransversarii muscles (lateral divisions)

**Structures innervated by the dorsal ramus**

Medial branch
- Deepest back muscles
- Zygapophyseal joints
- Periosteum of posterior vertebral arch
- Interspinous, supraspinous, and intertransverse ligaments, ligamentum flavum
- Skin (upper cervical, middle cervical, and thoracic dorsal rami)

Lateral branch
- Erector spinae muscles
- Splenius capitis and cervicis muscles (cervical region)
- Skin

**Structures innervated by the recurrent meningeal nerve**
- Periosteum of posterior aspect of vertebral bodies
- Internal vertebral (epidural) veins and basivertebral veins
- Epidural adipose tissue
- Posterior aspect of IVD
- PLL
- Anterior aspect of spinal dura mater

**Structures innervated by nerves associated with the sympathetic trunk and the gray rami communicantes**
- Periosteum of the anterior and lateral aspects of the vertebral bodies
- Lateral aspects of IVD
- Anterior aspects IVD
- ALL

Somatic Referred Pain

Nociception generated by a skeletal or related structure (muscle, ligament, zygapophyseal joint), which is felt in an area distant to the structure generating the nociception.

Distinguishing Features of Somatic Referred Pain:
- Dull ache
- Difficult to localize
- Rather constant in nature

Radicular Pain

Pain arising from the dorsal root or the dorsal root ganglion; usually causes pain to be referred along a portion of the course of the nerve or nerves formed by the affected dorsal root. This is known as a dermatomal pattern.

Structures and conditions that can irritate the dorsal roots (or ganglia):
- Disc lesion
- Abscess (osteomyelitis and tuberculosis)
- Tumor of the spinal canal
- Spondylolisthesis
- Malforamtion of the vertebral canal
- Malforamtion of the spinal nerve root and its sheath
- Miscellaneous diseases of bone
- Histamine-like chemicals released from degenerating intervertebral disc

Mechanism of Radicular Pain:
- Pressure on dorsal root or dorsal root ganglion,
- Edema within the nerves
- Further edema and hemorrhage within the dorsal root ganglion
- Ischemia of neural elements Ischemia perceived as PAIN

Distinguishing Features of Radicular Pain:
- Sharp, shooting type of pain along the distribution of the nerve(s) supplied by the affected dorsal root
- Long radiation into the upper or lower extremity (although this does not necessarily have to be the case)
- Pain coursing along a fairly thin band
- Pain accompanied by paresthesia, hypesthesia, and decreased reflexes
- Pain may be accompanied by motor weakness (as a result of compromise of the ventral roots)
Lumbar Sympathetic Afferents and Low Back Pain

Pain from a lower lumbar disc is transmitted nonsegmentally by visceral sympathetic afferent fibers, mainly from the L2 spinal nerve root. This results in referred pain in the L2 dermatome. Convergence projection theory is based upon the idea that visceral and somatic afferent fibers both synapse in the posterior horn.

Discogenic Pain

There is anatomic evidence that the disc can be a source of pain (nociceptor) because of the innervation that exists along the outer annulus from the ventral nerve roots that provide branches anteriorly (grey ramus communicans) and posteriorly (sinuvertebral nerve). However, there are many other structures in and around the spine that may be nociceptors and is often difficult for the clinician to differentiated these potential sources of pain.

Degenerative discs are thought to cause pain in several ways including mechanical instability (stretching of pain fibers) compressive impingement on adjacent nerves (radiculopathy) and biomechanical irritation via release of inflammation mediators.

Internal disc disruption describes pathologic changes of the internal structure of the disc. Internal disc disruption and degeneration involve a physiochemical change in the glycosaminoglycans of the NP, which act to bind water; over time this water-binding capacity diminishes. Disc degeneration is usually heralded by loss of hydration and thus decreased T2 signal on MR imaging.

Focal T2 bright areas reflecting annular tears indicate fragmentation of the outer collagenous annulus fibrosus. Hyper-intense zone (HIZ) is a term that has been coined to denote this finding on T2-weighted MR images. The presence of an HIZ correlates with an annular tear and an approximately 85% chance that there will be concordant pain reproduced at discography. An HIZ may enhance after contrast administration reflecting the fibrovascular ingrowth into the area of the annular tear.


Pathomechanics of Radicular Pain

When the nerve root or DRG is involved, mechanical changes such as compression are produced. These changes are easily followed by circulatory changes because of mechanical and vascular vulnerability of the nerve root. Inflammatogenic materials may leak from the degenerative disc or facet into the nerve root, causing chemical radiculitis. Disturbance of the CSF flow due to compression or fibrosis in malnutrition of the nerve root. Defective fibrinolytic activity may prolong these changes. Nerve fiber and cell changes may follow in due course.

The changes include blockage of axonal flow and demyelination. They may cause electrophysiologic changes, such as ectopic discharges and crosstalk. Pharmacological changes, including disturbed or enhanced synthesis and transport of neuropeptides, can also be elicited. Furthermore, some sympathetic influence may be added.

These morphologic, circulatory, biochemical, pathological, electrophysiologic and pharmacological changes may finally result in sensitization of both the central and the peripheral nerve systems, causing radicular pain. The important role of the impulses from the periphery is postulated in the pathomechanism of pain production.
Peripheral Annular Tear (High-intensity zone or HIZ)

Partial-thickness annular tear. Irritated or inflamed annular tear. High signal on T2-weighted images indicates the presence of fluid. Localized inflammation and neovascularization.

Chemical Radiculitis

Leaking nucleus pulposus through an annular tear contains chemicals that are inflammatory, neurodegenerative, and in the acute stages, neuroexcitatory. Result in chemical stimulation of small unmyelinated nerve fibers in the annulus or nearby neural elements. Inflammation-induced nociception stimulation and pain resulting from annulus fissure or disc herniation.

Painful Disc Herniations

Symptomatic disc herniations have both a mechanical and a chemical component. The chemical component results in sensitization of the nerve root as a result of a series of complex biologic reactions. A pro-inflammatory compound, tumor necrosis factor alpha (TNF-alpha), appears to play a central role in the biologic reactions that result in nerve damage and symptoms following a disc herniation.

Anti-TNF Therapy

Use of tumor necrosis factor alpha inhibitors to disrupt the cascade of damaging inflammatory reactions. Painful disc herniations, AS and other spondylarthropathies. Drugs - infliximab (Remicade) and etanercept (Enbrel) for RA and Crohn's disease.


DiscCure

Trademarked pharmacologic approach to sciatica and back pain. Injection of the tumor necrosis factor-alpha inhibitor etanercept (Enbrel, Amgen). Etanercept and infliximab (Remicade, Centocor) appear to block the activity of TNF-alpha, a potent pro-inflammatory compound.

Korhonen T et al., Treatment of sciatica with infliximab, a monoclonal chimaeric antibody against TNF-alpha, presented at the annual meeting, International Society for the Study of the Lumbar Spine, Cleveland, 2002; as yet unpublished.
Modic Changes

Signal intensity changes in vertebral body marrow adjacent to the endplates of degenerated discs.

Modic Type I: Low T1 signal and high T2 signal. Disruption and fissuring of the endplate with vascularized fibrous tissues within the adjacent marrow.

Modic Type II: High-T1 and high T2 signals. Disruption and fissuring of the endplate with yellow marrow replacement in the adjacent vertebral body. Type I changes often progress to Type II change over time.

Modic Type III: Low T1 and T2 signal. Extensive bony sclerosis.
Lower Motor Neuron System

Anterior horn cell, its axon, neuromuscular junction and innervated muscle fibers.

Diseases that affect the motor unit cause weakness, a decrease in muscle tone and a decrease in or loss of reflexes.

Upper Motor Neuron System

Diseases affecting descending motor pathways above anterior horn cells cause weakness and spasticity with increased muscle tone, increased tendon reflexes, abnormal reflexes and clonus.

<table>
<thead>
<tr>
<th>Muscles</th>
<th>LMN</th>
<th>UMN</th>
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<tbody>
<tr>
<td>Wasting</td>
<td>Individual</td>
<td>Absent</td>
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<tr>
<td>Fasciculations</td>
<td>Present, often marked</td>
<td>Absent</td>
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<tr>
<td>Tone</td>
<td>Flaccidity</td>
<td>Spasticity</td>
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<tr>
<td>Tendon reflexes</td>
<td>Decreased or absent</td>
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<tr>
<td>Clonus</td>
<td>Absent</td>
<td>Present</td>
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<tr>
<td>Plantar responses</td>
<td>Flexor</td>
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<tr>
<td>Superficial abdominal and cremasteric reflexes</td>
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<td>Absent</td>
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<tr>
<td>EMG</td>
<td>Abnormal</td>
<td>Normal</td>
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Cauda Equina Syndrome

Saddle anesthesia, recent onset of bladder dysfunction (urinary retention, increased frequency, or overflow incontinence), and severe or progressive neurologic deficit in the LE.

Physical examination: Laxity of anal sphincter, perianal/perineal sensory loss, and major motor weakness.

Motor Weakness: Quadriceps (knee extension), ankle plantar flexors, evertors, and dorsiflexors* (foot drop).
Cervical Disc Herniation

Lifting, straining, motor vehicle accident or sports injury. Neck pain and muscle spasm, together with arm pain and sensory symptoms. C5-6 and C6-7 are the most common levels of involvement (C5-6, C6-7, C4-5, C3-4 and C7-T1).

Symptoms (Henderson): 1. Arm pain (99%); 2. Neck pain (80%); 3. Scapular pain (50%); 4. Chest pain and headache are less common. 20-50 years of age. Direction of herniation: Posterolateral - radiculopathy; lateral-radiculopathy; central - less common and may present with less specific neurological findings such as neck pain or intermittent signs of radiculopathy or myelopathy.

Cervical spondylosis: Patients may present with neck pain and a combination of radicular or myelopathic findings often bilateral and usually involving multiple levels. The history of symptoms is usually longstanding. Lower extremity signs and symptoms frequently predominate over those of the upper extremity.

Cervical Nerve Root Syndromes

C3 Nerve Root, C2-3 Disc
Pain and numbness in the back of the neck, particularly around the mastoid process and pinna of the ear. No upper extremity weakness or reflex change.

C4 Nerve Root, C3-4 Disc
Pain and numbness in the back of the neck, radiating along the levator scapulae muscle and occasionally down the anterior chest. No upper extremity weakness or reflex change.

C5 Nerve Root, C4-5 Disc
Pain radiating from the side of the neck to the shoulder top; numbness over the middle of the body of the deltoid muscle (axillary nerve distribution), weakness of extension in the arm and shoulder, particularly above 90 degrees; atrophy of the deltoid muscle; no reflex change.

C6 Nerve Root, C5-6 Disc
Pain radiating down the lateral side of the arm and forearm, often into the thumb and index finger; numbness of the tip of the thumb or on the dorsum of the hand over the first dorsal interosseous muscle. Weakness of the biceps muscle; depression of the biceps reflex.

C7 Nerve Root, C6-7 Disc
Pain radiating down the middle of the forearm, usually middle finger, although the index and ring fingers may involved. Weakness of the triceps muscles; depression triceps reflex.

C8 Nerve Root, C7-T1 Disc
Pain radiating down the medial aspect of the forearm to the ring and small finger; numbness can involve the small finger and the medial portion of the ring finger. Numbness rarely extends above the wrist. Weakness of the triceps and small muscles of the hands; no reflex change.
Cervical Radiculopathy

True radicular pain has an intense, stabbing, and burning quality. It is sharp, often well localized to a specific dermatome, and characteristically made worse by maneuvers that increase intrathoracic pressure that, in turn, increases intraspinal canal pressure. Sneezing, coughing or any type of Valsalva maneuver will, therefore, accentuate the pain. Paresthesias may be present and often precede the pain. Characteristically, paresthesias tend to be more prominent distally while the pain tends to be more prominent proximally. Root lesions may also result in weakness in the appropriate myotome distribution. If the process is long-standing, atrophy and fasciculations may become apparent. Pain in the posterior and lateral aspect of the neck is present in almost all cases.

Specific distribution of symptoms in the arm and hand is important in localizing root involvement in cervical disc protrusion.

C5: Pain did not occur distal to the elbow and paresthesias were absent from the hand.

C6: Pain was localized to the radial forearm and the thumb was paresthetic (index finger less often).

C7: Pain was more diffuse in both dorsal and volar forearm and the middle finger was numb (index finger less often).

C8: Pain was localized to the ulnar forearm and paresthesias were experienced in the little and ring fingers of the hand.

- Paresthesias in the thumb strongly suggest C6 root involvement.
- Paresthesias in the middle finger strongly suggest C7 root involvement.
- If paresthesias are reported in the little finger, C8 root involvement is likely.

Weakness, in the corresponding myotome, may or may not be present. With C5 root compression, weakness may be experienced in the supra and infraspinatus, deltoid, biceps, and the rhomboid muscles. In C6 root involvement, the biceps and brachioradialis are involved and pronation of the forearm is compromised. With C7 lesions, strength in the triceps and wrist or finger extensors may be weak and pronation may be impaired.
**Thoracic Disc Herniation**

Can occur at any level but are more common at T8-9, T9-10, T10-11 and T11-12.

**Fourth and Fifth Decades**

The prevalence of degenerative disk disease, both thoracic and lumbar, is higher in subjects who have Scheuermann’s disease.

Generally, when the spinal canal is fairly large and has no exaggerated kyphosis, small herniations can exist without manifestations; a marked kyphosis or stenosis predisposes the cord to earlier and more severe injury.

T1 root compression syndrome consists of pain in the neck, medial border of the scapula, anterior chest, and medial aspect of the upper arm and forearm. There may be hypesthesia along the ulnar aspect of the forearm, weakness in the intrinsic hand muscles, and Horner’s syndrome.

The discs from T2 to T9 may be associated with pain around the scapula and tip of the shoulder as well as the chest wall that is at times mistaken for gallbladder disease, disorders of other abdominal organs, or diseases of the scapula or shoulder joint. Most patients have a slowly evolving myelopathy.

Signs of thoracic cord compression consist of the following: contraction of the paravertebral muscles, sensory disturbances, and motor weaknesses.

Those with T11-12 and T12-L1 herniation may also have a conus syndrome (i.e., lumbar neuropathy with bladder and rectal sphincter disturbances).

**Lumbar Disc Herniation**

Level of disc herniations: L5-S1(50-54%), L4-5(38-45%), L3-4(5-8%)

Direction of lumbar disc herniation: 85-90% posterolateral (paramedian or paracentral), 5-12% central (posterior midline), 5% lateral into the intervertebral canal (IVF).

Central disc herniation typically compresses the thecal sac while sparing the individual nerve root. This leads to low back pain due to sensory innervation to the meninges, posterior longitudinal ligament and outer layers of the annulus fibrosus.

Posterolateral disc herniations may cause nerve root compression leading to back pain that progresses and radiates into the buttock, thigh and leg in the distribution pattern of the involved nerve.

Lateral disc herniations extend into or beyond the neural foramen. It may compress a nerve root as the nerve exits the neural foramen.
**Bulging Disc (Annulus)**

Uniform, generalize protrusion of the annulus fibrosus beyond the vertebral margin. Gradual desiccation of the nucleus pulposus leads to decreased turgor, permitting a decrease in disc space height. In addition, the annulus fibrosus develops fissuring, hyalin degeneration and increased pigmentation. The annulus loses elasticity and bulges in a generalized fashion beyond the adjacent body margins.

**Conjoined Nerve Roots**

Two nerve roots emerge from a common dural sheath. L5 and S1 are most frequently conjoined and are often mistaken for an extruded disc fragment. Conjoined nerve roots have been reported in 1% of lumbar disc operations and 8% in anatomic specimens.

**Classification of Discs**

Normally, no disc extension beyond the interspace (endplates). Bulge - circumferential symmetric extension of the disc beyond the interspace (around the endplates). Protrusion - focal or asymmetrical extension of the disc beyond the interspace with the base against the disc of origin broader than any other dimension of the protrusion. Extrusion - more extreme extension of the disc beyond the interspace with the base against the disc of origin narrower than the diameter of the extruding material itself or with no connection between the material and the disc of origin.

*Jensen, The New England Journal of Medicine, 331:2, 1994*
Contained discs  Noncontained discs

EXTRUDED DISC
SEQUERTERED (FREE FRAGMENT)

NORMAL

BULGING ANNULUS  SUBLIGAMENTOUS DISC HERNIATION

EXTRUDED DISC - ATTACHED  EXTRUDED DISC - FREE FRAGMENT
**Lumbosacral Nerve Root Syndromes**

**L1 Nerve Root**  
Back to trochanter and groin. Hip flexion. Associated reflex - cremasteric

**L2 Nerve Root**  
Back. Anterior thigh to the level of the knee. Hip flexion and adduction. Associated reflex - cremasteric and adductor.

**L3 Nerve Root**  

**L4 Nerve Root**  

**L5 Nerve Root**  

**SI Nerve Root**  
Tears of the Annulus Fibrosus

The outer annulus fibrosus is characterized by layers of dense fibrous tissue in which the predominant component is collagen and the predominant cell type is the fibroblast. The outer portion of the disc has a low signal intensity in MRI.

The inner annulus fibrosus and the nucleus pulposus both contain fibrocartilage, which is a chondroid matrix composed in part of collagen fibers and chondrocytes. In the normal intervertebral disc, the MRI signal intensity reflects the net effect of the low signal intensity of the collagen and the high signal intensity of the chondroid content of the disc. The proteoglycan-containing chondroid matrix in the central disc gives the central disc a higher signal intensity than the peripheral annulus on T2-weighted MRI. A band of low signal intensity in the equator of the disc is referred to as the "intranuclear cleft", representing a region of the disc with higher collagen and reticular fiber content.

Concentric Tears

Concentric tears (circumferential/crescentic) represent a delamination of the lamellae of the annulus fibrosus. Concentric tears result from rupture of short transverse fibers that connect adjacent lamellae without rupture of the longitudinal fibers within the lamellae themselves. After rupture of the short fibers, the lamellae separate from each other, allowing fluid or mucoid material to fill the hiatus within the annulus. Concentric tears are common features of the intervertebral disc after the third decade of life and occur in 58% of the intervertebral discs classified as having "normal aging" on the basis of gross morphological examination. They are found frequently at the L3-4 and L4-5 disc levels. Concentric tears are not readily identified on CT or MRI. Concentric tears may accelerate the development of radial tears.

Transverse Tears

Transverse tears (rim lesions) are avulsions of the fibers of the peripheral annulus fibrosus from their insertion onto the ring apophyses of the vertebral bodies. They are thought to result from excessive torsional force. The tears appear as transversely oriented cavities or voids within the superior or inferior aspect of the outer annulus fibrosus and contain mucoid material or sometimes gas. Transverse tears are present in all age groups after the first decade of life but are more prevalent after the age of 30 years. They have been reported to occur in 57% of intervertebral discs classified as having signs of normal aging. Transverse tears may accelerate the formation of radial tears leading to eventual degeneration of the intervertebral disc. On T2-weighted images, transverse tears appear as zones of increased signal intensity in the normally low-signal outer annulus fibrosus. Transverse tears that show increased signal on contrast-enhanced MRI are thought to contain fibrovascular or granulation tissue, suggesting a repair process after traumatic injury.
Radial Tears

Radial tears are clefs or fissures that extend radially from the central nucleus pulposus to the periphery of the intervertebral disc, significantly disrupting the integrity of the annulus fibrosus. The radial tear may affect the anterior portion of the disc only, the posterior portion of the disc only, or extend across the full equator of the disc with disruption of both the anterior and posterior lamellae of the annulus fibrosus. Radial tears have been demonstrated in almost all discs with other features of degeneration, but rarely in normal adult discs. Radial tears are most prevalent at the L4-5 and L5-S1 disc levels. On T2-weighted images, the radial tear appears as a linear area of high signal intensity within the low signal intensity of the annulus fibrosus. Because vascularized granulation tissue may be found in radial tears, radial tears may also appear as linear or globular zones of abnormal contrast enhancement within the annulus.

Role of the Radial Tear and Disc Degeneration

Theory: The radial tear may be the initial event that leads to secondary degenerative changes within the intervertebral disc. The normal metabolism of the chondrocytes and fibroblasts of the disc depend on diffusion of solutes within the disc. These solutes are needed for synthesizing new glycosaminoglycans and collagen to replace the tissue degraded by proteases and collagenses within the disc. The radial tear impedes the diffusion of solutes within the disc and could lead to intervertebral disc degeneration.

The biochemical changes in the nucleus pulposus that are found in degrading discs include diminished water content, diminished proteoglycan content, and a diminished chondroitin sulfate:keratan sulfate ratio.

Alternate Theory:

Progressive degeneration of the nucleus pulposus could lead to mechanical failure of the annulus fibrosus with a progressive increase in the number of fissures and tears. Dehydration and degeneration of the nucleus pulposus reduce the nuclear volume and nuclear elasticity, resulting in a loss of disc height and outward bulging of the annulus fibrosus. The bulging increases the tension in the lamellar collagen fibers of the annulus, leading to separation between the lamellae of collagen (concentric tears) and then to radial tears. These radial tears are a necessary precondition for herniation of the nucleus pulposus and may precede herniation of the nuclear pulposus: by a period of months.
Discogenic Pain

Clinically, the signs and symptoms in patients with a radial tear may be indistinguishable from those observed in patients with herniations of the disc. Radial tears are identified regularly in patients with low back pain or sciatic pain, even though radial tears may be identified in asymptomatic individuals. Demonstration of a radial tear on MRI is an excellent predictor of a disc that may prove to be painful at discography. Intradiscal injection of contrast medium or saline produces the patient's pain in only 3% of normal intervertebral disc, but in 75% of those with annular disruption. The sensitivity of a annular tear or "high intensity zone" for predicting pain produced at discography has ranged from 81% to 99%.

Possible Mechanisms for the Pathogenesis of Pain by Radial Tears

Irritating substances or lactic acid could leak from the damaged intervertebral disc into the epidural space to inflame the spinal nerves causing pain. The pH in degenerating intervertebral discs is low secondary to anaerobic glycolysis. Inflammatory changes have been observed in spinal nerves adjacent to degenerating intervertebral discs.

Granulation tissue could grow into the torn intervertebral disc and transform the disc from a noninnervated structure to one with nociceptors. Nerve endings in the granulation tissue represent a potential source of discogenic pain.

The radial tear may cause spinal nerve and nerve root compression directly or indirectly. The tear may destabilize the mechanical response of the motion segment, leading to abnormal tension in other innervated connective tissues leading to pain. Radial tears are associated with a decrease in the stiffness or stability of the involved motion segments. The loss of stability increases the risk that a load applied to the spine causes nerve root entrapment or compression. Critical changes in the anatomic relationships within the neural foramen secondary to physiological loading have been observed in motion segments associated with a radial tear but not in those with normal intervertebral discs. The potential risks of subsequent occult lateral spinal stenosis and compression of the nerve roots are greater when a radial tear is present. Even normal upright posture and normal daily physical activity may result in narrowing of the neural foramina that are not narrowed under unloaded conditions.

Discs with radial tears exhibit greater bulging of the annulus, both when not load bearing and when subjected to axial compression. Greater increases in the cross-sectional area of the intervertebral disc, greater decreases in the cross-sectional areas of the neural foramina and spinal canal, and greater decreases in the diameters of the neural foramina occur in discs with radial tears in comparison to normal discs. These changes in the anatomic relationships within the neural foramen may cause compression of the nerve roots.
Chronic or intermittent compression of a spinal nerve root produces demyelination. While graded pressure on myelinated nerve axons does not trigger pain impulses in normally myelinated nerves, graded pressure does trigger pain in demyelinated nerves. Demyelination changes the electrogenic properties of the axons so that graded pressure produces repetitive firing of the nerve. In a demyelinated nerve root, weak mechanical stimuli, hypoxia, chemical mediators, inflammation, and sympathetic efferent activity may elicit nerve impulses ("ectopic hyperexcitability"). Some injured afferent nerves may fire spontaneously; neuron-to-neuron cross excitation can also occur. Evidence suggests that cytokines may initiate or propagate hypersensitivity in chronically irritated nerves.

The effect of any load is increased in motion segments with radial tears. Normal physiologic loads may cause critical narrowing of a neural foramen that is not significantly narrowed under unloaded conditions. A nerve root that does not appear to be compressed in the neuroforamen of a recumbent patient may be actually compressed when the patient resumes upright, weight-bearing posture.

Radial tears significantly affect the mechanical properties of the disc. A radial tear of the annulus also affects the load-bearing and kinematic properties of motion segments, and alters their stiffness (resistance to motion). Motion segments with radial tears exhibit only a fraction (3% to 50%) of the stiffness of motion segments with normal intervertebral discs. Segments with radial tears show significantly increased motion after flexion and rotational forces. By changing the normal range of motion, the radial tear may increase the stress in the anterior and posterior longitudinal ligaments, the facet joint capsules, and other connective tissues of the spine causing back pain.

Failed Back Surgery Syndrome

The most common causes: recurrent disc herniation, lateral spinal stenosis, central spinal stenosis, arachnoiditis, epidural fibrosis, residual disc herniation, meningocele formation, nerve injury, wrong-level surgery, and remote phenomena that are unrelated to the spine itself.

Recurrent disc herniation vs epidural fibrosis

Surgical removal of a scar usually has a poor outcome, often resulting in further scarring, while surgery for recurrent disc herniation has a good prognosis. Epidural fibrosis usually enhances early on post-enhanced MRI. Recurrent herniated disc does not enhance early or enhances around the periphery only. Utilized pre- and post-contrast MRI to evaluate FBSS.

Failed Back Surgery Syndrome.

- Less than one week following surgery: postoperative hemorrhage, residual disc herniation, and lateral recess stenosis.
- One week to one month: recurrent disc herniation, spondylitis or meningomyelitis.
- More than one month (chronic): recurrent disc herniation, adhesive arachnoiditis and perineural scarring with or without chronic, sterile neural inflammation.

Postoperative pseudomeningocele

Caused by a small dural tear at the time of surgery. Allows progressive herniation of the arachnoid membrane through the vent. Or, produces a CSF leak into the soft tissues which eventually develops a fibrous capsule. The actual vent usually cannot be defined with CT or MRI. Do myelography with follow-up CT (CTM).
Lumbar Spinal Stenosis
Narrowing of the spinal canal, whether on a congenital, developmental or degenerative basis, is referred to as spinal stenosis.

Congenital stenosis
- Idiopathic
- Anchondroplastic

Acquired Stenosis
- Degenerative
  - Central portion of the spinal canal
  - Lateral portion of the spinal canal
  - Degenerative spondylolisthesis
- Combined - any combination of congenital, degenerative and disc herniations
- Spondylolytic, spondylolisthetic, iatrogenic
  - Postlaminectomy
  - Postfusion
- Postchemonucleolysis
- Post-traumatic
- Miscellaneous
  - Paget's disease
  - Fluorosis

Central (canal) stenosis
The cauda equina or spinal cord is involved. The patient will most often be male with a history of gradually progressive, chronic back pain prior to developing leg pain. The symptoms can be relieved by sitting, bending forward (flexion sign) (stoop test), squatting or lying down with hips flexed.

Neurogenic Intermittent Claudication
Extension of the spine during walking increases circumferential mechanical compression of the cauda equina. The subarachnoid space is obliterated and congestion of neural blood vessels leads to intraneural edema. This leads to an increase in pressure within the spinal nerves and degenerative of nerve cells. The increase in pressure within the nerves may result in a compartment syndrome causing ischemia of the cauda equina and the onset of neurogenic claudication. Relieved by assuming flexed or hunched posture while walking or by sitting down.

Compression of cauda equina. The patient will complain of leg pain, paresthesias, numbness and weakness initiated or aggravated by standing or walking. They may describe the sensations of numbness, tingling, burning, or a feeling of "heavy, tired" legs and, frequently, anterior thigh pain. There may also be pain at rest, night cramps and restless legs.

Vascular Intermittent Claudication
Decreased blood flow, hypoxia of muscles, cramping and pain.
Lateral stenosis
Nerve root involvement:
  - Subarticular (lateral) recess
  - Intervertebral canal (foraminal, nerve exit canal)
  - Far lateral (extraforaminal, "far out")

Lateral spinal stenosis will affect one nerve root level. Pain in the buttock, in the region of the greater trochanter and down the back of the thigh to the knee will be described. Sometimes the pain is down the back or lateral part of the calf to the ankle. Pain is exacerbated by activity and is relieved by rest. The leg feels heavy or weak.

Subarticular stenosis can be fixed or dynamic.

Central Spinal Stenosis Imaging Findings
  - Decreased AP diameter of the spinal canal
  - Short, thick pedicles
  - Thickened laminae
  - Vertebral osteophytes
  - Hypertrophy of the articular process
  - Hypertrophy of the ligamentum flavum
  - Generalized disc bulging
**Myelopathy**
Functional disturbances and/or pathological changes in the spinal cord. Spinal cord compression and ischemia. Cervical myelopathy is a consequence of pressure, tension, and torsion of the spinal cord.

Myeloradiculopathy: Combination of Myelopathy and Radiculopathy,

MR is initial screening exam for myelopathy. Use CTM for presurgical assessment in difficult cases.

**Causes of Myelopathy**
Compression, spinal stenosis, OPLL, disc herniation with congenitally narrow spinal canal, tumor, radiation, AIDS, vascular malformations, toxic/metabolic (alcoholism, vitamin B12 deficiency), congenital spinocerebellar degeneration syndromes, infection, post-vaccination, autoimmune (SLE, MS).

Myelopathic symptoms originating at a cervical level include any combination of spastic gait and incoordination, bilateral upper and lower extremity weakness, paresthesias with diminished sensation in the hands, increased DTRs, and upgoing toes. In clinical practice, cervical spondylosis causing extrinsic cord compression is the most common etiology. Less common causes include demyelinating disease, degenerative disease, and inflammatory, infectious, vascular, and neoplastic diseases.

Cervical myelopathy may result when large degenerative spurs or, less commonly, a central HNP occurs in combination with a small cervical canal. The classical clinical triad in these patients includes 1) painful stiff neck, 2) brachialgia, and 3) spastic leg weakness with ataxic gait. There is poor correlation between the presence and severity of these symptoms and the anteroposterior dimension of the spinal canal. There is also an incomplete correlation between the presence of cord compression at MR and myelopathic symptoms.

Electric sign of Lhermitte (neck bending sign or barber's chair sign): Flexion of neck results in sensation of shock, electricity, and paresthesia with pain and tingling running down the spine or into arms and legs.

Myelomalacia: Cord gliosis and edema. Increased signal intensity within the spinal cord due to demyelination or myelomalacia secondary to chronic compression.
Cervical Spondylytic Myelopathy

There are five basic syndromes:

1. Lateral cord and root compression presenting as nerve root symptoms: Pain, numbness, and neurologic deficit in a nerve root distribution.

2. Myelopathic syndrome is characterized by numbness, gait abnormalities, and bilateral upper-extremity weakness. This is caused by central cord compression.

3. Myeloradicular syndrome is a combination of symptoms produced by central cord and root compression. The syndrome is characterized by gait abnormalities, bilateral lower extremity involvement, and unilateral upper-arm deficits.

4. Vascular syndrome is the least common (zone of ischemia). The sensory and motor deficits are of variable patterns making level localization difficult.

5. Anterior syndrome presents as upper motor weakness without pain. There is no lower extremity involvement. This represents anterior column compression by tenting of the cord over the anterior elements during flexion causing only localized anterior column pathology.

Spastic weakness in the legs results because of compression of the descending pyramidal tracts while paresthesias in the feet, legs, and even trunk evolves from similar compression of the long ascending sensory pathways. Puzzling combinations of lower and upper motor neuron signs may appear in the arms.
Chiari Malformations.

Chiari 1: Inferior displacement of the cerebellar tonsils into the cervical spinal cord. Mild ectopia (less than 3mm below, the foramen, magnum) is usually of no clinical significance. When tonsils extend more than 5mm below the foramen magnum, the incidence of clinical symptoms rises.

Causes of:

1. Dysgenesis, the cerebellar tonsils are large. Associated anomalies: occipitalization of the atlas, Klippel-Feil anomalies.

2. Tonsillar ectopia as a result of intrauterine hydrocephalus (tonsillar herniation).

3. Acquired deformities of the foramen magnum (platybasia and basilar invagination).

Syringohydromyelia: Cavitation of spinal cord with accumulation of fluid. 20-25% of Chiari I malformations have.

Syrinx: Cavity in the spinal cord.

Hydromelia: Dilatation of the central canal of the spinal cord.

Syringomyelia: Cavitation of the spinal cord.

Treatment: Suboccipital craniectomy and upper cervical laminectomy, cord cavity drained.

Cysts associated with spinal cord tumors

1. Tumoral cyst: degeneration, necrosis, and liquefaction within the neoplasm. Contains a mixture of differing elements, such as protein, old hemorrhage, and necrotic tumoral tissue.

2. Rostral or caudal cysts: occur above and/or below the tumor. contain either hemorrhagic or xanthochromic fluid.

3. Reactive dilatation of the central canal, most likely related to partial obstruction of the central canal.
Atypical Idiopathic Scoliosis

Atypical clinical or radiographic features: Early onset or rapid progression of scoliosis, presence of pain or Other neurologic symptoms or signs, kyphosis, pedicle thinning, convex left thoracic or thoracolumbar curve, and associated syndromes. Approximately one third of these cases had abnormalities demonstrated on MR studies. Hydrosyringomyelia and Chiari I malformation were the most common findings. Pain is a frequent early presenting symptom in children with intramedullary spinal cord symptoms.

Young patient (less than 11 years of age). Severe or rapidly progressing thoracic/thoracolumbar scoliosis (particularly left-sided). No family history of scoliosis. Unusually rigid scoliosis in a young patient. Scoliosis which is associated with pain and is unresponsive to conservative treatment.

Abnormal neurological findings: Muscular weakness and/or atrophy, sensory loss (particularly pain and temperature), bladder/bowel dysfunction, unexplained/painless swollen joint (Charcot's joint), abnormal superficial abdominal wall reflex, abnormal deep tendon reflexes (increased or decreased), and cranial nerve abnormality.

Abnormal radiographic findings: Segmentation defects, platybasia, basilar impression, increased spinal canal diameter, posterior body scalloping, and pedicle widening.