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.


Convergence Theory of Conscious Somatotopic Localization and Pain Referral
Peripheral afferent fibers from visceral sources and somatic sources converge at the level of the spinal cord on the same cord neuron, producing a cerebral perception of pain located to the initiating sources (vertebra), as well as within the site of referral (lower extremity).

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 differentiate 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.


Peripheral Annular Tear (High-Intensity Zone or HIZ)
Partial-thickness annular tear (fissuring). 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 nocioception stimulation and pain resulting from annulus fissure or disc herniation.

**Painful Disc Herniations**
Symptomatic disc hemiations 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.

Herniated disc material attracts macrophages, fibroblasts and lymphocytes – inflammatory chemicals are produced by these cells or the disc material itself.

These chemicals include: phospholipase A2, metalloproteinases, prostaglandin E2, leukotriene B4, thrombaxane, nitric oxide, interleukin – 6, 8, 12 and tumor necrosis factor alpha (TNF-alpha).

**Inflammation – Role In Pain**
Awareness that chemical factors are required for production of pain. This changes prior perception of mechanical compression and structural dysfunction as sufficient causes. Lesion size, therefore, need not always correlate directly with extent of pain in discogenic pain production. Patients with significant findings and complaints of lumbar radiculopathy may be found on scans or surgery to have minimal neural compression because symptoms are of an inflammatory etiology. Patients frequently improve well in advance of anticipated or documented morphologic disc change because of improvement in chemical factors.

**Spontaneous Herniated Disc Resorption**
Disc herniation, intervertebral disc cells produce proinflammatory cytokine TNF-alpha (tumor necrosis factor-alpha) and IL-1beta (interleukin-1beta). Initiators of inflammatory response and are, therefore, referred to as “proinflammatory cytokines” or “alarm cytokines”. Stimulate the production of MCP-1 in the intervertebral disc cells resulting in macrophage infiltration in herniated discs. The infiltrating macrophages also produce MCP-1, amplifying the macrophage infiltration into the discs. Finally, infiltrating macrophages absorb disc materials due to their powerful phagocytic activity and the release of neutral metalloproteinases.
**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.

**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*

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**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.
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
- Spondylytic, 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
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.
Zygapophyseal Joint Pain After Whiplash
Neck pain due to post-traumatic arthropathy. Headache from referred pain.

Cervical Zygapophyseal Joints
Innervated by articular branches derived from the medial branches of the dorsal rami. Ascending branch innervates joint above and descending branch innervates joint below (dual innervation)
**Whiplash: S-shaped curve**

The cervical spine does not undergo smooth, even extension during whiplash. The spine is subject to an S-shaped curve during the early phase of the collision. Grauer reported that the whiplash motion was not simply extension, but a complex combination of compression, flexion of the upper cervical spine, and excessive extension of the lower cervical spine: Ono found that a subject's torso shows the ramping-up motion by the inclined seatback during rear-end impact. As the head remains in its original position due to inertia in the initial phase of impact, an axial compression force is apt to be applied to the cervical spine, which in turn moves upward and the flexion occurs at about the same time. The lower vertebral segments (C6, C5 and C4) are extended and rotated earlier than the upper vertebral segments. Those motions are beyond the normal physiological range of motion. It is found that by comparing the motions during crash with the normal extension motions of the same subject that the rotational angle pattern is reversed by the pattern of the normal state around 100ms. The lower the vertebral segment, the larger the rotational angle becomes. That is, the rotational angle between the fifth and sixth vertebral segments is the largest of all. This is a non-physiological motion of the vertebral segments.

Normally, the facets slide over each other, allowing smooth, equal movement of the motion segments. When the spine is compressed, however, the mechanics of facet movement changes dramatically. Researchers have found that the Instantaneous Axis of Rotation (IAR) - or the point which the vertebrae rotate around - actually moves, as the following illustrations shows:

The result of this abnormal motion? The facets of the vertebrae (see arrows), rather than sliding over each other smoothly, are jammed into each other, as shown in the illustration on the right. Such abnormal motions are believed to result in joint injury - a lesion that would not be detectable with modern imaging techniques, but one that could cause chronic pain.

New research by Kaneoka found that at approximately 100 milliseconds into the collision, the cervical spine undergoes an S-shaped curve, where the upper spine experiences flexion and the lower cervical spine undergoes extension. This S-shaped curve is caused by the simultaneous compression of the spine when the occupant's body moves up the seat back, and the forward motion of the torso when the car seat pushes into the occupant. This motion was determined by high-speed x-ray video of the whiplash motion in a test subject at approximately 5 mph.

Yoganandan analyzed the whiplash motion in a human cadaver specimen and confirmed that there is indeed an S-shaped curve that occurs at approximately 100 milliseconds into the collision. Furthermore, Yoganandan et all were able to show the detailed motion between the 5th and 6th cervical vertebrae. They write, "... the lower cervical spine facet joint kinematics demonstrate varying local compression and sliding. While both the anterior-and posterior-most regions of the facet joint slide, the posterior-most region of the joint compresses more than the anterior-most region. These varying kinematics at the two ends of the facet joint result in a pinching mechanism."
The following is a summary of the mechanics in order of occurrence:

0 ms—Normal cervical curve
50 ms—Cervical spine straightens and experiences compression.
100 ms—Lower cervical spine experiences abnormal extension while head remains in same position.
150 ms—Spine undergoes normal C-shape extension.

Harrison P. The prevalence of zygapophysial joint disease as a cause of chronic neck pain/headache. World Congress on Whiplash-Associated Disorders 1999; 120.


**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)

**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.

**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 surround 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.

**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

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**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.

Chiari Malformations
Chiari malformation is a complex congenital anomaly of the posterior fossa accompanied by cerebellar ectopia. The severity of the malformation is dependent on the extent of cerebellar herniation through the foramen magnum. Three types of Chiari malformation are described. Chiari Type I: herniation of the cerebellar tonsils below the foramen magnum. Chiari Type II: caudal displacement of the cerebellar tonsils, inferior vermis, fourth ventricle, and medulla oblongata, with subsequent cervical cord kinking. Hydrocephalus and syringohydromyelia are commonly associated anomalies. Chiari Type III: herniation of the cerebellum into a cervical-occipital encephalocele.

Chiari I: Inferior displacement of the cerebellar tonsils into the cervical spinal canal. 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. Low-lying, pointed (triangular-shape, not round), “peg-like” tonsils. Up to 50% asymptomatic. May mimic multiple sclerosis. Chiari I "spells": cough/headache/sneeze/syncope. Symptomatic brain compression and symptomatic
syringohydromyelia.

**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. Syrinx may be primary or secondary. Primary: idiopathic or in conjunction with Chiari I malformation. Secondary: Tumor, inflammatory, or trauma.

**Hydromyelia:** Dilatation of the central canal of the spinal cord.

**Syringomyelia:** Cavitation of the spinal cord.

Treatment would include 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.

**White Matter Diseases in Adults**

Multiple sclerosis (MS) and variants
- Acute disseminated encephalomyelitis
- Hurst hemorrhagic leukoencephalitis

Vascular disorders
- Ischemic arteriolar disease (mycroangiopathy)
- Boundary zone ischemia (unilateral carotid disease)
- Arteritis (e.g., systemic lupus erythematosus (SLE), sarcoidosis

Infectious/immune disorders
- Acquired immune deficiency syndrome (AIDS) related disorders
- Progressive multifocal leukoencephalopathy
- Lyme disease

Vasogenic edema
- Traumatic shear injury
- Radiation injury/necrosis

Metabolic disorders
- Central pontine myelinolysis
- Marchiafava-Bignami disease
- Adult leukodystrophies

Multiple sclerosis is the most common white matter disease and is related to focal areas of demyelination with reactive gliosis in the white matter of the brain, spinal cord, and the optic nerves.

Clinical Presentation: Exacerbations and remissions of multifocal neurologic deficits.

Impaired or double vision. Fatigue, weakness, numbness, tingling, and gait disturbances. Loss of sphincter control, blindness, paralysis and dementia.

Onset 20-40. More common in persons of Western European lineage who live in temperate zones.

Individuals who migrate in early childhood from a low-risk to a high-risk area have the same risk of developing MS as those in the area they move to. If the same move is made after adolescence, the risk remains low.

- Familial incidence.
- HLA antigens: HLA-A3, HLA-B7, HLA-DR2.
- Cerebrospinal Fluid: Mild lymphocytosis, slightly elevated protein, and oligoclonal immunoglobulin bands of IgG on immunoelectrophoresis.
- Visual, auditory and somatosensory evoked responses.
- Lhermitte’s Sign: Electric-like shocks spreading down the body on forceful flexion of the head and neck.
Neoplasms of Nerve Roots, Dura and Spinal Cord

- Extradural
- Intradural extramedullary
- Intramedullary

Incidence of Intraspinal Neoplasms

- Schwannomas 30%
- Meningiomas 26%
- Gliomas 23%
- Sarcomas 11%
- Hemangiomas 6%
- Other 4%

Schwannomas

The most common primary intraspinal tumor is the schwannoma. Schwannomas are extramedullary-intradural tumors composed of Schwann's cells, which arise from spinal nerves at any level (cervical, thoracic, lumbar or cauda equina) and most often arise from a posterior (sensory) nerve root. The most common initial symptom therefore is pain in a radicular distribution. Schwannomas grow slowly and pain may be present for years before the correct diagnosis is made, especially when the schwannoma is in the relatively spacious lumbosacral region. Schwannomas of nerve roots in the relatively tight cervical region compress the spinal cord early in their course. Neurofibromas are composed of Schwann's cells and fibroblasts.

Meningiomas

Meningiomas are the second most common primary intraspinal tumor. Spinal meningiomas have a marked propensity to occur in the thoracic spinal cord, and they are rare below the midlumbar level. Like schwannomas, meningiomas are slow-growing, extramedullary-intradural tumors. The clinical picture usually evolves over months to years before the diagnosis is made. Thoracic area, fifth and sixth decades, women 80%.
Astrocytomas and Ependymomas
The most common intramedullary spinal tumors are the gliomas, which comprise mainly ependymomas and astrocytomas. The ependymomas predominate in the cauda equina and lumbar region; the astrocytomas in the cervical region. The clinical syndrome produced by these tumors is usually indistinguishable from that produced by extramedullary tumors. Spinal gliomas are slow-growing tumors, and a history of deficits of several years duration is common. Children - astrocytomas 60%, ependymomas 30%. Adults - ependymomas 55-60%, astrocytomas 30-40%

Ependymoma: Lower cord, conus and filum. Third to sixth decades. Myxopapillary tumor of filum (young adults).
Astrocytoma: More common in children (60% of intramedullary tumors). Cervical or thoracic areas (rostral more common in children)

Approximately 60% of intramedullary spinal tumors are associated with syrinx.

MR ANGIOGRAPHY
MRA capitalizes on creating intensity differences between flowing tissue and stationary tissue. By suppressing background stationary tissue and focusing only on the high-signal flowing blood, one can obtain a data set that depicts only vascular structures. If employed with contiguous sections or three-dimensional volumetric acquisitions, one can produce very thin section MR angiograms that can be rotated in space to visualize the circle of Willis or the carotid bifurcation. MR is capable of detecting atherosclerotic narrowings or intracranial aneurysms in three-dimensional space.

MR Diffusion Imaging
Almost immediately after the onset of ischemia, the apparent diffusion coefficient of brain water drops by approximately 30 to 50%, resulting in hyperintensity in DWI. The most likely cause is the redistribution of water from the interstitial to the diffusion-restricted intracellular space (cytotoxic edema).

Carotid Injury
The CCA bifurcates into the ICA and external carotid artery at approximately the level of the fourth cervical vertebral body near the superior border of the thyroid cartilage. Although the external carotid artery is initially anteromedial to the ICA, and ICA quickly courses medially at approximately the C1 or C2 level before entering the skull base. Unlike the lower cervical vertebrae, the lateral articular processes and pedicles of the first through third cervical vertebrae project more anteriorly, with the distal portion of each cervical ICA lying in close proximity just anterior to these. The ICA enters the carotid canal as the petrous segment, where it is firmly fixed within the petrous bone. With extension of the neck, the carotid canal is elevated stretching and partially fixing the cervical ICA against the lateral masses of the upper cervical spine. Rotation, which largely occurs at the atlantoaxial joint, forces the contralateral lateral mass of C1 anteriorly, further stretching the ICA. Because of this relationship, the cervical ICA is the typical site of injury associated with hyperextension-rotation of the head and neck.
Type I injuries result from a direct blow to the neck. This is the characteristic mechanism in elderly persons with advanced atherosclerotic disease. Impingement of the ICA between the mandible and the cervical spine with acute hyperflexion would injure the vessel in a similar fashion and may account for some of the injuries seen in victims of motor vehicle accidents. Type II injuries are due to hyperextension and contralateral rotation of the head and neck and are the characteristic injuries seen in victims of motor vehicle accidents. Damage occurs as the ICA is stretched over the lateral masses of the first and second cervical vertebrae. This mechanism accounts for over 90% of blunt injuries to the ICA and tends to affect young patients more frequently, perhaps due to the protection afforded the elderly by their tortuous vessels and less mobile cervical spine. Type III injuries result from intraoral trauma and are typically seen in a child who has fallen with a hard object, such as a pencil, in their mouth. Type IV injuries result from associated basilar skull fractures.

**Carotid Artery Dissection**
The disorder occurs in hypertensive individuals who have no evidence of atherosclerotic vessel disease. Other risk factors include smoking and fibromuscular dysplasia. Dissection may occur with hyperextension and lateral flexion of the neck as the artery is stretched over the transverse processes of the upper cervical vertebrae. Focal unilateral headache is the most common symptom in association with dissection. The headache is steady, non-throbbing, of variable intensity, and is located in the frontal, auricular, or periorbital area. Neurologic manifestations may include stroke (resulting in contralateral hemiparesis, paresthesias, aphasia, ipsilateral blindness, or abducens paralysis) or oculosympathetic palsy with ptosis and miosis without anhidrosis. Focal neurologic deficits may follow the onset of headache or neck pain within minutes of hours. Bruits may be heard over the carotid.

**Vertebral Artery Dissection**
Vertebral artery dissection occurs most commonly in middle-aged women. People with hypertension or fibromuscular dysplasia are at greater risk of dissection. Pain in the occiput or posterior neck is the presenting symptom in 80% of patients, preceding ischemic symptoms by minutes to 30 days. Most patients present with a completed stroke, with a minority presenting with transient ischemic attacks. The lateral medullary syndrome (pain, numbness, ipsilateral face [trigeminal], ataxia, vertigo, nystagmus, Horner’s syndrome [descending sympathetic tract], dysphagia, numbness of ipsilateral appendages) is the most common neurologic manifestation. Severe cases may have basilar artery involvement with associated quadriparesis, dysphagia, diplopia, with preserved sensation.

**Symptoms of TIA**
Carotid Territory – paresthesia/weakness of hand, arm, and face; aphasia (dominant hemisphere); dysarthria; unilateral neglect.

Lacunar – hemibody sensory loss of paresthesia; pure motor hemiparesis.

Vertebrobasilar – dysarthria; vertigo, ataxia; diplopia; visual field loss; perioral paresthesias; acute confusional state; profound general weakness.

**Vertebrobasilar Injuries**
Mechanisms of vertebral artery injury: cervical hyperextension, excessive contralateral rotation, and hyperextension and rotation.
Familial history of stroke or cardiovascular disease, hypertension, smoking, cervical spondylosis/arthrosis, bleeding disorders, medication, and/or anatomical anomaly/pathology.

Rotation of C1 on C2 between 30 and 45º causes the vertebral artery at the atlantoaxial junction to become compressed on the opposite side of head rotation, subsequently reducing blood flow to the basilar artery. In the normal patient, this diminution of blood flow caused by positional change of the cervical spine will not cause any neurological symptoms, such as dizziness, nausea, tinnitus, faintness, or nystagmus. This lack of symptoms is a result of the normal flow of collateral circulation by the opposite vertebral artery, common carotid arteries, and a communicating cerebral arterial circle (Circle of Willis).

Seven areas of possible compression: 1) between C1-2 transverse processes, where the vertebral arteries are relatively fixed at the C1 and C2 transverse foramina; 2) C2-3 at the level of the superior articular facet of C3 on the ipsilateral side to head rotation; 3) the C1 transverse process and the internal carotid artery; 4) the atlanto-occipital aperture by the posterior arch of atlas and the rim of foramen magnum, or anteriorly by folding of the atlanto-occipital joint capsule and posteriorly by the atlanto-occipital membrane; 5) C4-5 or C5-6 levels because of arthrosis of the joints of von Luschka with compression on the ipsilateral side to head rotation; 6) at the transverse foramina of the atlas or axis between the obliquus capitis inferior and intertransversarii during rotatory movements; 7) before entering the C6 transverse process by the longus colli muscle or by tissue communicating between the longus colli and scalenus anterior muscles.

Vertebral artery pathological alterations: intimal disruption, subintimal hematoma, dissection, pseudoaneurysm, and thromboembolism.

Wallenberg's Syndrome: ipsilateral loss of cranial nerves V, IX, X, and XI cerebellar ataxia, Horner's syndrome, and contralateral loss of pain and temperature sensation. Sudden death, quadriplegia, and the “locked-in” syndrome (quadriplegia with loss of all lower cranial nerves).

Risk factors: age, hypertension, hyperlipidemia, family history of stroke or heart attacks, diabetes, smoking, heart and peripheral vascular disease, young adult females on birth control pills, cervical spondylosis, and cervical spine injury (hyperextension injury).

Clinical evaluation: blood pressure (both arms), palpate radial pulses (normal, feeble or absent), palpate carotid pulses, auscultate carotid arteries (bruit, hissing or squirting sound), and auscultate subclavian arteries. If pulsations or bruits are present at the carotid or subclavian arteries, do not perform the functional maneuver. A difference of 10 mm Hg between the two systolic blood pressures and a feeble or absent radial pulse is suggestive of subclavian artery stenosis.
**Functional Maneuver for Vertebrobasilar Artery Insufficiency**

Test procedure: Examiner passively moves patient’s head and neck into extension and lateral flexion, then rotation, holding for 30 seconds. Patient must keep eyes open.

Positive finding: Vertigo, dizziness, visual blurring, nausea, faintness, and nystagmus.

Symptoms and signs of insufficiency: dizziness, giddiness, drop attacks, syncope, stroke, diplopia, blurred vision, visual hallucination, auditory hallucination, tinnitus, flushing, sweating, lacrimation, rhinorrhea, scotomatoa, hiccups, myotonic jerks, tremor and rigidity, disorientation, vertigo, photophobia, numbness and tingling, quadriparesis, dysphagia, dysarthria, photopsia, visual anosognosia, nystagmus and ataxia.
Zygapophyseal Joint Pain After Whiplash
Neck pain due to post-traumatic arthropathy. Headache from referred pain.

Cervical Zygapophyseal Joints
Innervated by articular branches derived from the medial branches of the dorsal rami. Ascending branch innervates joint above and descending branch innervates joint below (dual innervation)
Whiplash: S-shaped curve

The cervical spine does not undergo smooth, even extension during whiplash. The spine is subject to an S-shaped curve during the early phase of the collision. Grauer reported that the whiplash motion was not simply extension, but a complex combination of compression, flexion of the upper cervical spine, and excessive extension of the lower cervical spine: Ono found that a subject's torso shows the ramping-up motion by the inclined seatback during rear-end impact. As the head remains in its original position due to inertia in the initial phase of impact, an axial compression force is apt to be applied to the cervical spine, which in turn moves upward and the flexion occurs at about the same time. The lower vertebral segments (C6, C5 and C4) are extended and rotated earlier than the upper vertebral segments. Those motions are beyond the normal physiological range of motion. It is found that by comparing the motions during crash with the normal extension motions of the same subject that the rotational angle pattern is reversed by the pattern of the normal state around 100ms. The lower the vertebral segment, the larger the rotational angle becomes. That is, the rotational angle between the fifth and sixth vertebral segments is the largest of all. This is a non-physiological motion of the vertebral segments.

Normally, the facets slide over each other, allowing smooth, equal movement of the motion segments. When the spine is compressed, however, the mechanics of facet movement changes dramatically. Researchers have found that the Instantaneous Axis of Rotation (IAR) - or the point which the vertebrae rotate around - actually moves, as the following illustrations shows:

The result of this abnormal motion? The facets of the vertebrae (see arrows), rather than sliding over each other smoothly, are jammed into each other, as shown in the illustration on the right. Such abnormal motions are believed to result in joint injury - a lesion that would not be detectable with modern imaging techniques, but one that could cause chronic pain.

New research by Kaneoka found that at approximately 100 milliseconds into the collision, the cervical spine undergoes an S-shaped curve, where the upper spine experiences flexion and the lower cervical spine undergoes extension. This S-shaped curve is caused by the simultaneous compression of the spine when the occupant's body moves up the seat back, and the forward motion of the torso when the car seat pushes into the occupant. This motion was determined by high-speed x-ray video of the whiplash motion in a test subject at approximately 5 mph.

Yoganandan analyzed the whiplash motion in a human cadaver specimen and confirmed that there is indeed an S-shaped curve that occurs at approximately 100 milliseconds into the collision. Furthermore, Yoganandan et all were able to show the detailed motion between the 5th and 6th cervical vertebrae. They write, "... the lower cervical spine facet joint kinematics demonstrate varying local compression and sliding. While both the anterior- and posterior-most regions of the facet joint slide, the posterior-most region of the joint compresses more than the anterior-most region. These varying kinematics at the two ends of the facet joint result in a pinching mechanism."