

### AUTHOR INFORMATION

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### INTRODUCTION

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#### Background:

Cervical disc disorders encountered in physiatric practice include herniated nucleus pulposus (HNP), degenerative disc disease (DDD), and internal disc disruption (IDD). HNP implies extension of disc material beyond the posterior margin of the vertebral body. Most of the herniation is made up of the annulus fibrosus. DDD involves degenerative annular tears, loss of disc height, and nuclear degradation. IDD describes annular fissuring of the disc without external disc deformation. Cervical radiculopathy can result from nerve root injury in the presence of disc herniation or stenosis, most commonly foraminal stenosis, leading to sensory, motor, or reflex abnormalities in the affected nerve root distribution.

Understanding cervical disc disease requires basic knowledge of anatomy and biomechanics. The intervertebral disc absorbs shock, accommodates movement, provides support, and separates vertebral bodies to lend height to intervertebral foramina. The disc consists of an eccentrically located nucleus pulposus and a surrounding annulus fibrosus separating each segmental level between the C2-T1 vertebrae. No disc exists between C1 and C2, and only ligaments and joint capsules resist excessive motion. Disc degeneration and/or herniation can injure the spinal cord or nerve roots and result in stenosis and/or myofascial pain.

#### Pathophysiology:

Manifestations of HNP are divided into subcategories by type (i.e., disc bulge, protrusion, extrusion, sequestration). Disc bulge describes generalized symmetric extension of the disc margin beyond the margins of the adjacent vertebral endplates. Disc protrusion describes herniation of nuclear material through a defect in the annulus, producing a focal extension of the disc margin. Extrusion applies to herniation of nuclear material resulting in an anterior extradural mass attached to the nucleus of origin, often via a pedicle. Disc sequestration refers to separation of material from the disc, which ultimately comes to lie in the spinal canal.

Herniation typically occurs secondary to posterolateral annular stress. Herniation rarely results from a single traumatic incident. Acute traumatic cervical HNP serves as a major etiology of central cord syndrome (CCS). The C6-C7 disc herniates more frequently than discs at other levels.

Acute disc herniation causes radicular pain through chemical radiculitis in which proteoglycans and phospholipases released from the nucleus pulposus mediate chemical inflammation and/or direct nerve root compression. Interleukin 6 and nitric oxide are also released from the disc and play a role in the inflammatory cascade. The chemical radiculitis is a key element in the pain caused by HNP as nerve root compression alone is not always painful unless the dorsal root ganglion is also involved. Herniation may induce nerve demyelination with resulting neurologic symptoms. Cervical HNP may be resorbed during the acute phase. Indeed, studies documenting frequent herniation resorption and correlating herniation regression with symptom resolution support conservative treatment of cervical radicular pain.

A rare trauma-induced high cervical (C2-C3) HNP syndrome manifests nonspecific neck and shoulder pain, perioral hypesthesia, more radiculopathy than myelopathy, and more upper limb motor and sensory dysfunction than lower limb symptomology. Decreased middle and/or lower cervical spine mobility from spondylosis, with consequent overload at higher segments, may precipitate high cervical disc lesions in older patients. A retro-odontoid disc may result from an upwardly migrating C2-C3 HNP. Some case reports describe cervical HNPs causing Brown-Séquard syndrome, as well as atypical nonradicular symptoms in patients with congenital insensitivity to pain.

Cervical radiculopathy results from mechanical nerve root compression or intense inflammation (i.e., chemical radiculitis). Specifically, nerve root compression may occur at the intervertebral foraminal entrance zone at the narrowest segment of the root sleeve anteriorly by disc protrusion and uncovertebral osteophytes and posteriorly by superior articulating process, ligamentum flavum, and periradicular fibrous tissue. Decreased disc height, as well as age-related foraminal width decrease from inferior z-joint hypertrophy, may impinge subsequently on nerve roots. The cervical region accounts for 5-36% of all radiculopathies encountered. Incidence of cervical radiculopathies by nerve root level is as follows: C7 (70%), C6 (19-25%), C8 (4-10%), and C5 (2%).

The most common cause of cervical radiculopathy is foraminal encroachment (70-75%). The cause is multifactorial, including degeneration of the discs and the uncovertebral joints of Luschka and the zygapophyseal joints. In contrast to lumbar spine disorders, HNP in the cervical spine is responsible for only 20-25% of radiculopathies.

Cervical degenerative disc disease (DDD) most commonly is due to age-related changes, but the condition also is affected by lifestyle, genetics, smoking, nutrition, and physical activity. Degenerative disc changes observed on radiographs may reflect simple aging and do not necessarily indicate a symptomatic process.

The disc begins to degenerate in the second decade of life. Circumferential tears form in the posterolateral annulus after repetitive use. Several circumferential tears coalesce into radial tears, which progress into radial fissures. The disc then disrupts with tears passing throughout the disc. Loss of disc height occurs with subsequent peripheral annular bulging. Proteoglycans and water escape through fissures formed from nuclear degradation, resulting in further thinning of the disc space. Vertebral sclerosis and osteophytic formation ultimately follow.

Internal disc disruption (IDD) describes pathological annular fissuring within the disc without external disc deformation. This disorder results from trauma-related nuclear degradation, cervical flexion/rotation-induced annular injury, or whiplash. The innervated outer disc annulus serves as a major pain generator. DDD ultimately may progress to IDD.

### Frequency:

- **In the US:**  
NP may be observed with MRI in 10% of asymptomatic individuals aged younger than 40 years and 5% of those older than 40 years. Degenerative disc disease (DDD) may be observed with MRI in 25% of asymptomatic individuals aged less than 40 years and 60% of those aged more than 40 years. The true incidence and prevalence of cervical radiculopathy is uncertain; however, 51% of adults experience neck and arm pain at some time. In a population-based study in Rochester, Minn, the

annual incidence of documented cervical radiculopathy for men and women from all causes was 107.3 and 63.5 cases per 100,000 population, respectively.

- **Internationally:**

A study from Italy in 1996 reported a prevalence of cervical spondylotic radiculopathy as 3.5 cases per 1000 people.

**Mortality/Morbidity:**

Occasionally, an acute HNP can herniate centrally and cause a myelopathy. This can manifest as hyperreflexia, positive pathologic reflexes such as Babinski and Hoffman signs, and sphincter disturbances. If left untreated, the effects can be irreversible.

**Sex:**

Kelley suggests that the male-to-female incidence of cervical disc herniation is approximately 1:1. Marchiori and Henderson cite women as reporting higher disability with increasing levels of DDD than men.

**Age:**

HNP typically affects younger patients (i.e., <40 y). DDD, part of natural aging, typically affects older patients (i.e., >40 y).

**History:**

- Pertinent history should include the following information:
  - Information about pain onset (e.g., abrupt onset suggests acute injury)
  - Time since injury
  - Mechanism of injury
  - Percentage of axial versus peripheral pain (e.g., 90% neck pain versus 10% upper limb)
  - Review of systems to uncover possible systemic illness (e.g., fever suggests infection, weight loss suggests malignancy).
- Discogenic pain without nerve root involvement typically is vague, diffuse, and distributed axially.
  - Pain referred from disc to upper limb usually is nondermatomal.
  - Activities that increase intradiscal pressure (e.g., lifting, Valsalva maneuver) intensify symptoms. Conversely, lying supine provides relief by decreasing intradiscal pressure.
  - Vibrational stress from driving also exacerbates discogenic pain.
- Depending on whether primarily motor or sensory involvement is present, radicular pain is deep, dull, and achy or sharp, burning, and electric.
  - Such radicular pain follows a dermatomal or myotomal pattern into the upper limb.
  - Cervical radicular pain most commonly radiates to the interscapular region although pain can be referred to the occiput, shoulder, or arm as well.

- Neck pain does not necessarily accompany radiculopathy and frequently is absent.
- Patients may present with distal limb numbness and proximal weakness in addition to pain. Atrophy may be present.
- A recent study demonstrates cervical HNP-induced thermal changes (i.e., thermatomes) in specific upper extremity distributions.
- Mechanical stimulation of cervical nerve roots has shown that the distribution of referred radicular symptoms (i.e., dynatome) may be different from sensory deficits outlined by traditional dermatomal maps.

### Physical:

- The patient with radicular pain also displays decreased cervical range of motion (ROM).
  - Pain is exacerbated from neck extension and rotation or from spurling maneuver (patient's neck is extended, laterally bent, and held down) designed to elicit radicular symptoms.
  - Pain improves with neck flexion or abduction of the symptomatic upper limb over the top of the head (abduction sign).
  - Decreased sensation to pain, light touch, or vibration may be present in the distal upper limb. Proximal limb weakness manifests when significant motor root compromise exists but must be differentiated from pain-related weakness.
  - Diminished or absent reflexes corresponding to the root level may be present.
  - Increased upper and lower limb reflexes or other upper motor neuron signs suggest myelopathy and mandate aggressive diagnostic evaluation.
- The patient with discogenic pain without nerve root involvement demonstrates decreased cervical ROM, normal neurologic examination, and possible pain exacerbation with axial compression and pain alleviation with distraction.
- Myofascial tender or trigger points, which may be primary in origin or secondary to other pathologic processes, commonly, are palpable.
- Tenderness with posteroanterior (PA) mobilization may suggest disc pathology.

### Causes:

- HNP results from repetitive cervical stress or, rarely, from a single traumatic incident. Increased risk may accrue because of vibrational stress, heavy lifting, prolonged sedentary position, whiplash accidents, and frequent acceleration/deceleration.
- DDD is part of natural aging, but it is also a consequence of poor nutrition, smoking, atherosclerosis, job-related activities, and genetics.
- IDD can result from cervical trauma, including whiplash, cervical flexion/rotation injury, and repetitive use.
- Cervical radiculopathy results from nerve root compression secondary to herniated disc material, stenosis, or proteoglycan-mediated chemical inflammation released from discs. Smoking and certain occupational activities also predispose patients to cervical radiculopathy.

- Brachial Neuritis
- Cancer and Rehabilitation
- Cervical Myofascial Pain
- Cervical Spondylosis
- Cervical Sprain and Strain
- Complex Regional Pain Syndromes
- Fibromyalgia
- Neoplastic Brachial Plexopathy
- Osteoarthritis
- Osteoporosis (Primary and Secondary)
- Paget Disease
- Psoriatic Arthritis
- Radiation-Induced Brachial Plexopathy
- Rheumatoid Arthritis
- Rotator Cuff Disease
- Scheuermann Disease
- Thoracic Outlet Syndrome
- Traumatic Brachial Plexopathy

**Other problems to be considered:**

- **Mechanical etiologies**
  - Cervical stenosis
  - Cervical zygapophyseal (facet) arthropathy
- **Infectious etiologies**
  - Discitis
  - Epidural, subdural, or intradural abscess
- **Metabolic etiologies**
  - Osteomalacia
  - Parathyroid disease
- **Rheumatologic etiologies**
  - Polymyalgia rheumatica (PMR)
  - Ankylosing spondylitis
  - Reiter syndrome
  - Enteropathic arthritis
  - Diffuse idiopathic skeletal hyperostosis (DISH)

**Lab Studies:**

- Consider performing rheumatologic workup to evaluate for possible rheumatoid arthritis, ankylosing spondylitis, Reiter syndrome, and polymyalgia rheumatica. These tests include the following:
  - Rheumatoid factor (elevated in rheumatoid arthritis)

- Human leukocyte antigen (HLA)-B27 (positive in ankylosing spondylitis)
- Erythrocyte sedimentation rate (elevated in polymyalgia rheumatica)
- Consider performing infection workup to evaluate for possible discitis, epidural abscess, and vertebral osteomyelitis including the following tests:
  - White blood cell count with differential (elevated with a left shift in bacterial infection)
  - Blood cultures (positive for the infecting organism)
  - Erythrocyte sedimentation rate (elevated in infection, but may be a nonspecific finding)

### Imaging Studies:

- Imaging studies evaluate anatomy, rather than function, and are prone to false positive and negative results. For example, Boden's cervical magnetic resonance imaging (MRI) study cites abnormalities in nearly 20% of asymptomatic subjects. Consequently, results of imaging studies must be interpreted within the context of each clinical case.
- Plain radiographs
  - Plain cervical spine radiographs evaluate chronic degenerative changes, metastatic disease, infection, spinal deformity, and stability.
  - Cervical spine trauma films use 7 views, including anteroposterior (AP), lateral, bilateral oblique, open-mouth, flexion, and extension.
    - Flexion-extension views identify subluxations or cervical spine instability.
    - Open-mouth views evaluate the odontoid process and C1-C2 stability.
    - AP views identify tumors, osteophytes, and fractures.
    - Lateral views assess stability and spondylosis (i.e., spurring, disc space narrowing).
    - Oblique views reveal DDD, as well as foraminal encroachment by uncovertebral or z-joint osteophytes.
- Computed tomography
  - CT scan delineates cervical spine fracture and is used extensively in trauma cases.
  - Helical or spiral CT scan generates an infinite number of images after data acquisition, providing more information for detailed fracture evaluation than conventional CT scan.
- CT-myelography
  - A myelogram followed by CT scan may be obtained prior to cervical decompressive spinal cord or nerve root surgery.
  - This study evaluates the spinal canal, its relationship to the spinal cord, and nerve root impingement from disc, spur, or foraminal encroachment.
  - CT-myelography, still the criterion standard, remains superior to MRI in detecting lateral and foraminal encroachment, despite greater expense and morbidity. Consequently, CT-

myelography is not the initial imaging study to evaluate cervical spine and is reserved for complicated cases.

- MRI remains the imaging modality of choice to evaluate cervical HNP due to its low morbidity.
  - Advantages include soft tissue definition (eg, cervical discs, spinal cord), cerebrospinal fluid (CSF) visualization, noninvasiveness, and lack of patient radiation exposure.
  - Newer MRI pulse sequences and higher field magnets provide faster and more detailed imaging.
  - Unfortunately, some sequences (eg, spin echo) depict pathology larger than actual size and obscure other abnormalities. Other disadvantages include expense, inability of claustrophobic patients to tolerate the procedure, dependence on patient cooperation to minimize artifact, high false-positive rate, and insensitivity compared to CT scan in evaluating bony structures.
  - Furthermore, MRI appears inferior in differentiating cervical disc prolapse (i.e., soft cervical disc) from spondylitic osteophytic compression (i.e., hard cervical disc).
  - Contraindications to MRI include patients with embedded metallic objects, such as pacemakers, surgical clips, spinal cord stimulators, or prosthetic heart valves that may be dislodged by MRI magnets.
  
- Provocative cervical discography has been controversial since its introduction in 1957 by Smith.
  - This imaging procedure involves sterile-technique placement of spinal needles into cervical intervertebral discs.
  - At least 2 different techniques exist for performing this procedure.
    - The paravertebral technique uses digital palpation to retract vital soft tissue structures (e.g., trachea, carotid artery, esophagus).
    - The oblique approach obviates the need for digital palpation. After spinal needles are placed within the center of the nucleus pulposus, contrast is injected to determine internal disc architecture and any pain response provoked.
  - Provocative discography is the only procedure that can determine whether a disc serves as the pain generator.
  - Discomfort and invasiveness render this procedure less desirable than cervical MRI, which provides much of the anatomical information that provocative discography does.
  - Provocative cervical discography identifies symptomatic disc(s), assisting in evaluation of patients with inconclusive diagnostic tests and presurgical fusion planning.
  - Contraindications to provocative discography include large disc herniation and mid sagittal spinal canal diameter less than 12 mm.
  - Complications include discitis, epidural abscess, quadriplegia, stroke, pneumothorax, nerve injury, and spinal cord injury. The reported rate of cervical discitis is 0.37%.
  - Discography should be performed at all accessible cervical levels, given the high frequency of multilevel symptomatic cervical discs.
  - Provocative discography may identify poor surgical candidates, thereby improving fusion outcomes.

#### **Other Tests:**

- Electrodiagnostic studies continue to be standard for evaluating neurologic function of the cervical spine.
  - Advantages of these tests include limited expense and low morbidity.
  - Nerve conduction studies (NCSs) and electromyography (EMG) studies provide physiological assessment of cervical nerve root and peripheral nerve function.
    - Needle EMG can detect acute, subacute, and chronic radicular features if motor nerve fiber pathology exists.
    - A diagnosis of radiculopathy is apparent when needle EMG reveals abnormal spontaneous potentials and/or certain changes in motor unit action potentials, in 2 or more muscles innervated by the same nerve root but different peripheral nerves. Ideally, EMG abnormalities also should be demonstrated in the paraspinal muscles to confirm the diagnosis of radiculopathy.
    - Compound motor action potential (CMAP) amplitude drop of 50% or more indicates significant axonal loss. This assessment is made via NCS of motor axons.
    - NCS/EMG is especially helpful to differentiate cervical radiculopathy from confounding neuropathic conditions (e.g., ulnar nerve entrapment, carpal tunnel syndrome, peripheral neuropathy, plexopathy).
    - Unfortunately, cervical radiculopathies involving exclusively sensory axons (i.e., without involvement of motor axons) rarely are detected by electrodiagnostic studies, which is a shortcoming of this diagnostic modality. In addition, routine motor NCSs do not evaluate the C6 and C7 nerve roots, which are most commonly involved, nor the levels above.
    - Unlike needle EMG (which involves intramuscular evaluation and is a well-accepted diagnostic test), surface EMG generally is not considered to have an accepted role in the diagnosis of radiculopathy.
  - Somatosensory evoked potentials (SEP) evaluate sensory conduction peripherally and centrally.
    - Lower limb SEPs involving tibial and peroneal nerves, which assess spinal cord conduction, are more sensitive in diagnosing myelopathy than upper limb median and ulnar SEPs.
    - Dermatomal evoked potentials have been used to detect cervical radiculopathy but are of questionable value.

### Rehabilitation Program:

#### Physical Therapy:

For most cervical disc disorders, studies support conservative treatment, such as the McKenzie approach and cervicothoracic stabilization programs, combined with aerobic conditioning.

The McKenzie system identifies 3 mechanical syndromes that cause pain and compromise function. The postural syndrome provokes pain when normal soft tissues are loaded statically at end range of motion (ROM); pathology need not be present. Treatment aims to correct posture. The dysfunction syndrome produces pain when the patient, upon attempting full movement, mechanically deforms contracted scarred

soft tissue. Consequently, therapy involves stretching and remodeling of such contracted tissue. The derangement syndrome produces intermittent pain when certain movements or postures occur. Specifically, pain may become centralized or peripheralized because of theoretical activity-dependent displacement of intradiscal material. Therapy attempts to correct derangement by promoting activity that centralizes pain.

The McKenzie theory recognizes that, although patients may demonstrate similar signs and symptoms, one movement (eg, cervical extension) nevertheless may help some patients and aggravate symptoms in others. Indeed, McKenzie therapy does not use only extension-biased exercise. Consequently, treatment individualization and patient education play key roles.

Cervicothoracic stabilization limits pain, maximizes function, and prevents further injury. Such stabilization includes cervical spine flexibility, postural training, and strengthening. This program emphasizes patient responsibility through active participation.

Restoring flexibility prevents further repetitive microtrauma from poor movement patterning. Pain-free ROM is determined by placing the cervical spine in positions that produce and relieve symptoms. Initially, stabilization commences within established pain-free ROM and then progresses outside this ROM as pain diminishes. Soft tissue or joint restriction inhibiting ROM is treated quickly. Anterior and posterior neck muscles are stretched. Indeed, such spine and soft tissue mobilization, passive ROM, self-stretching, and correct posturing collectively restore ROM.

Postural training commences with the patient, supervised by a therapist, in front of a mirror. The patient performs various transfer maneuvers while maintaining a neutral spine (ie, correct posturing) with feedback from the mirror and the therapist. Patient goals include maintenance of neutral spine and demonstrating correct posture during daily activities.

These proprioceptive skills, implemented during strengthening exercises, facilitate stable, safe, and pain-free cervical posture during strenuous activity. Indeed, cervicothoracic stabilization requires strengthening and coordination of neck, shoulder, and scapular muscles. Cervical muscles include extensors, flexors, rectus capitis anterior, rectus capitis lateralis, longissimus cervicis, and longissimus capitis. Primary thoracic stabilizers include abdominals, lumbar paraspinal extensors, and latissimus dorsi. Scapular muscles include the middle and lower trapezius, serratus anterior, and rhomboids. Chest muscles include the pectoralis major and minor. Successful stabilization also requires the training of the lumbar spine and lower extremities, which provide a foundation for the cervicothoracic spine.

Stabilization exercises proceed systematically from simple to complex. Isometric and isotonic resistive exercises employ elastic bands, weight machines, and free weights. Such conditioning distributes forces away from the cervical spine. Exercise repetition ultimately encodes an engram that commands immediate, automatic cervicothoracic stabilization during everyday activity.

Butler's therapy techniques treat radicular symptoms by mobilizing the involved nerve. First, the therapist identifies "adverse neural tension," defined as pathological mechanical and physiological responses elicited from a nerve when its stretch properties and ROM are evaluated. Specifically, the therapist performs neurodynamic testing to evaluate a nerve's mechanical properties (e.g., its mobilization around neighboring intervertebral discs) and physiological characteristics (e.g., its response to ischemia, inflammation). Having tested the nerve in question, the therapist then may institute treatment consisting initially of passive mobilization to provide CNS input without inciting a stress response and neurogenic massage to reduce perineural swelling. Later, the therapist progresses to active neuromobilization because, according to Butler, recovering nervous tissue (like other connective tissue) requires movement to promote healing and restoration of optimum mechanical properties.

Butler admits that limited evidence suggests that neurodynamic mobilization improves clinical outcomes. However, he believes that optimizing tissue health and cardiovascular fitness, as well as minimizing negative beliefs and environmental factors, can be beneficial.

Functional restoration programs assist patients disabled by chronic cervical pain overcome obstacles to recovery. Such obstacles include deconditioning, secondary gain, poor motivation, and psychopathology. An occupational or physical therapist, athletic trainer, or nurse instructs the patient in cervical anatomy, biomechanics, pathology, and ergonomics. Patients employ preventive measures prohibiting further injury

during all daily activities. These medically directed interdisciplinary programs have been successful at enabling workers' compensation patients to return to work. Furthermore, Wright and colleagues report lower rates of recurrent injury, new surgery, and need for health care services for patients with chronic cervical pain who successfully complete functional restoration.

### **Medical Issues/Complications:**

An intervertebral disc compressing the spinal cord can provoke myelopathy with associated weakness, hyperreflexia, and neurogenic bowel and bladder dysfunction. Radiculopathy can manifest significant upper limb weakness or numbness. Intractable axial or radicular pain may result from cervical disc disorders.

### **Surgical Intervention:**

Studies indicate that cervical HNP with radiculopathy can be managed conservatively. Surgery is warranted when neurogenic bowel or bladder dysfunction, deteriorating neurologic function, or intractable radicular or discogenic neck pain exists. Specifically, cervical spine surgical outcomes are most favorable for radicular pain, spinal instability, progressive myelopathy, or upper extremity weakness. Recent literature demonstrates favorable cervical spine fusion outcomes for chronic discogenic axial neck pain when the presurgical evaluation incorporated provocative cervical discography. Provocative discography identified the painful segment(s) and confirmed adjacent pain-free levels. Fusion can increase intradiscal pressure and other stress at adjacent unfused levels, thereby accelerating postsurgical spinal degeneration.

### **Consultations:**

- Internal medicine consultation is indicated when neck pain suggests an underlying systemic illness (e.g., malignancy, infection, metabolic bone disease).
- Consider rheumatology consultation when neck pain suggests a rheumatological condition (e.g., polymyalgia rheumatica).
- Surgical consultation for cervical disc disorders is warranted for resulting neurogenic bowel/bladder dysfunction, deteriorating neurological status (e.g., myelopathy), segmental instability, and/or intractable radicular or discogenic pain.

### **Other Treatment (injection, manipulation, etc.):**

- Physical modalities should be used to reduce pain only in the acute phase. Once past the acute phase, modalities are used sparingly on an as-needed basis.
  - Superficial heat modalities relax muscle and relieve soft tissue pain.
  - Conversely, deep-heating modalities (e.g., ultrasound) should be avoided in acute cervical radiculopathy since they augment inflammation and, consequently, exacerbate radicular pain and nerve root injury.
- Cervical traction may relieve radicular pain from nerve root compression. Traction does not improve soft tissue injury pain. Hot packs, massage, and/or electrical stimulation should be applied prior to traction to relieve pain and relax muscles.
  - Traction regimens include heavy weight-intermittent or light weight-continuous. The neck is flexed 15-20° (i.e., not extended) during traction. In the cervical spine, approximately 10 lb of force is necessary to counter gravity and 25 lb of force is necessary to achieve separation of the posterior vertebral segments.
  - Light weight-continuous home traction is cost effective and provides the patient more autonomy.
  - Pneumatic traction devices afford greater patient comfort and, consequently, increased compliance.

- A soft cervical collar is recommended only for acute soft tissue neck injuries and for short periods of time (i.e., not to exceed 3-4 days' continuous use). Risks include limiting cervical ROM and losing neck strength if the collar is worn continuously for longer periods.
  - When worn for radiculopathy caused by foraminal stenosis, the wide part of the collar is placed posteriorly and the thin part anteriorly to promote neck flexion, discourage extension, and open the intervertebral foramina.
  - Collars can be worn during certain activities, such as sleeping or driving, for longer periods.
  - Although not used commonly, a Philadelphia collar can be worn at night to position the neck rigidly in flexion, thereby maintaining open foramina.
- Spinal manipulation and mobilization may restore normal ROM and decrease pain; however, no clear therapeutic mechanism of action is known. Some believe that zygapophysial joint adjustment improves afferent signals from mechanoreceptors to peripheral and central nervous systems.
  - Normalization of afferent impulses improves muscle tone, decreases muscle guarding, and promotes more effective local tissue metabolism. These physiologic modifications subsequently improve ROM and pain reduction.
  - Studies document short-term improvement in the acutely injured patient and in those with cervicogenic headache and radiculopathy secondary to disc herniation.
  - No evidence exists that manipulation confers long-term benefit, improves chronic conditions, or alters the natural course of the disorder.
- Cervical epidural, spinal nerve (or root), z-joint, and sympathetic injections serve diagnostic and therapeutic roles. These procedures can be instrumental in determining the anatomical pain generator (e.g., nerve root, facet) and providing aggressive conservative treatment.
- Therapeutic cervical epidural injections treat radicular pain, although some literature has demonstrated reduced axial pain as well.
  - An anesthetic and corticosteroid mixture may be injected into the epidural space (interlaminar) or along the nerve root (transforaminal) after precise radiologic contrast-enhanced fluoroscopic localization.
  - The anesthetic can relieve sympathetically mediated pain.
  - The corticosteroid provides long-term relief if pain results from an intense inflammatory component.
  - Such injections provide a pain-free window of opportunity for more aggressive physical therapy.
- Diagnostic selective spinal nerve or ventral ramus blocks inject a small anesthetic volume extraforaminally at a single spinal segmental level (e.g., C5 versus C6), and, consequently, they are more precise than the "gun shot" interlaminar approach in identifying the symptomatic nerve.
  - Precise symptomatic nerve identification permits the physician to design a more focused treatment protocol.
  - Patients record pain changes in a pain diary following the injection to confirm diagnostic accuracy.
  - A double injection paradigm previously reported in the literature for facet injections can provide information to the physician in determining diagnosis of radicular pain and help confirm the symptomatic nerve level. This paradigm identifies patients who have tested false-positive or may have a tendency to respond to a placebo by determining whether, on separate injection

days, they receive short- term relief with a short-acting anesthetic (e.g., lidocaine) and long-term relief with a long-acting anesthetic (e.g., bupivacaine).

- Adverse effects include those from anesthesia, corticosteroids, and radiologic contrast dye.
  - Blood clotting parameters should be drawn prior to injection in patients with suspected bleeding diathesis. Indeed, spinal cord compression could result if bleeding occurs in the presence of relative spinal stenosis (i.e., mid-sagittal diameter less than 12 mm) in which little room exists to accommodate an epidural hematoma.
  - Nonsteroidal anti-inflammatory drugs (NSAIDs), including aspirin, should be discontinued prior to the procedure in accordance with their half-life and hematological profile.
  - Other potential risks include seizure, vertebral artery spasm, infection, temporary quadriparesis from anesthetic and respiratory arrest.
  - One study, however, suggests that selective cervical nerve blocks carry low morbidity when performed under contrast-enhanced fluoroscopic guidance.
  - In any event, proper patient monitoring and emergency equipment always should be present.
- Recent reports of serious CNS complications following cervical transforaminal steroid injections have gained the attention of many practitioners. These include both spinal cord injuries and strokes. The mechanism of the injury is believed to be related to the introduction of particulate matter within the corticosteroid preparations, causing occlusion of a vessel.
  - Hodges and colleagues describe 2 case reports in which intrinsic spinal cord damage resulted from cervical epidural steroid injection despite fluoroscopic guidance; the patients, because of intravenous sedation, were unable to perceive and report pain and paresthesias from needle-induced spinal cord trauma during the procedure.
  - Furman et al demonstrated a relatively high incidence of entering the intravascular space with transforaminal epidural steroid injections. They also showed that attempting to use a flash of blood in the needle hub to predict intravascular compromise was 97% specific, but only 45.9% sensitive. This article underscored the importance of using fluoroscopy and contrast dye to ensure proper placement of the therapeutic agents. Using a flash of blood in the hub without fluoroscopy cannot reliably predict intravascular compromise.
  - Brouwers et al reported a fatal case of spinal cord infarction following a cervical transforaminal steroid injection.
  - Baker et al demonstrated that a radicular artery supplying the cervical spinal cord can be infiltrated by a transforaminal epidural steroid injection. In this report, prior to steroid injection for a left C6-C7, contrast was administered. Using digital subtraction technique, it was clear that a radicular artery filled with contrast; the procedure was aborted without adverse effects. This report reveals a potential access point for an injection-related spinal cord infarction.
  - The potentially catastrophic complications that can follow a cervical transforaminal epidural steroid injection cannot be underestimated. While these procedures are perceived as posing less of a risk than surgery, they still carry substantial hazards. They should be performed by skilled practitioners and under fluoroscopic guidance. Baker et al further suggest the use of digital subtraction because intravascular compromise may be missed on routine spot films.

NSAIDs are first-line pharmacologic intervention for most cervical conditions. NSAIDs reduce pain at low doses and decrease inflammation at high doses. Patients require a therapeutic NSAID plasma level to achieve anti-

inflammatory effect. NSAIDs with once-a-day dosing improve compliance and increase probability of achieving therapeutic levels. Controlling inflammation is paramount when treating cervical radiculopathy.

Aspirin rarely is recommended because it binds irreversibly to cyclo-oxygenase and incites gastritis, requiring large doses to reach anti-inflammatory effect. Traditional NSAIDs provoke multiorgan toxicity, including peptic ulcer disease, renal insufficiency, and hepatic dysfunction. The recently released cyclo-oxygenase isomer type 2 (COX-2) NSAID inhibitors confer the same analgesic/anti-inflammatory benefits without multiorgan toxicity. All NSAIDs have a dose-related ceiling point for analgesia above which higher doses fail to provide additional pain relief. The same precautions should be observed with COX-2 NSAIDs, despite their reduced risk of organ toxicity.

Use muscle relaxants to potentiate the NSAID analgesic effect and not necessarily to control muscle spasm. Muscle relaxants primarily sedate by relaxing muscle with subsequent relaxation of the patient.

Oral corticosteroids treat inflammatory cervical radiculopathy. No documented case of avascular necrosis exists in the literature when the total prednisone dose or corticosteroid equivalent stayed under 550 mg. Some providers use a methylprednisolone dose pack (tapers from 24 to 0 mg over 7 days); however, concern exists regarding adequate dosing to treat radiculopathy. A prednisone dose schedule outlined below stays within the 550-mg limiting amount.

Tricyclic antidepressants (TCAs) decrease pain and reduce nonrestorative sleep. Side effects include dry mouth, constipation, and weight gain. Selective serotonin reuptake inhibitors (SSRIs), despite lacking side effects associated with TCAs, are inferior to TCAs in treating diabetic peripheral neuropathic pain, and their efficacy in relieving neck and back pain compared to other antidepressants remains unknown. Additional medications include membrane-stabilizing agents (eg, gabapentin, carbamazepine). Gabapentin has demonstrated efficacy in treating diabetic peripheral neuropathic pain. Other analgesics (acetaminophen, tramadol) provide pain relief without inflammation control.

Opioids may be prescribed orally, transdermally, rectally, or sublingually on a scheduled basis. Patients on opioids should sign a medication contract restricting them to a single physician and pharmacy, scheduled medication use, no unscheduled refills, and no sharing or selling medication. Patients prescribed opioids long term with a previous history of alcoholism or other addiction are at risk for dependence. Therefore, consider recommending co-treatment of these patients with a psychologist or other addiction specialist.

Lastly, many short-acting opioid preparations contain acetaminophen, which may be toxic in doses above 3 g/d. Consequently, patients should be counseled to avoid toxicity by avoiding other pharmaceuticals containing acetaminophen.

**Drug Category: Corticosteroids** -- Used to treat inflammatory cervical radiculopathy. Have anti-inflammatory properties and cause profound and varied metabolic effects. Corticosteroids modify the body's immune response to diverse stimuli.

<b>Drug Name</b>	Prednisone (Deltasone, Orasone Sterapred) -- Decreases inflammation by inhibiting polymorphonuclear leukocyte and fibroblast migration, stabilizing lysosomes, and decreasing capillary permeability.
<b>Adult Dose</b>	70 mg/d PO initial; subsequently decrease 10 mg/d for total course of 200-300 mg
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity, psychosis, idiopathic thrombocytopenia, acute glomerulonephritis, fungal infections, amebiasis, nonasthmatic bronchial disease, TB, and AIDS
<b>Interactions</b>	Activity may increase with estrogens, salicylates, oral contraceptives, ketoconazole, indomethacin, and macrolide antibiotics; decreased action with barbiturates, cholestyramine, ephedrine, phenytoin, colestipol, theophylline, rifampin; increased side effects with salicylates, cyclosporin, digitalis, alcohol, indomethacin, diuretics, and amphotericin B; decreased effects of antidiabetics with neostigmine, vaccines, somatrem, anticoagulants, anticonvulsants,

	ambenonium, isoniazid, toxoids, and anticholinesterases
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	Abrupt discontinuation of glucocorticoids may cause adrenal crisis; hyperglycemia, edema, osteonecrosis, myopathy, peptic ulcer disease, hypokalemia, osteoporosis, euphoria, psychosis, myasthenia gravis, growth suppression, and infections may occur with glucocorticoid use

<b>Drug Name</b>	Methylprednisolone dose pack (Solu-Medrol, Medrol, Depo-Medrol) -- Decreases inflammation by inhibiting polymorphonuclear leukocyte and fibroblast migration, stabilizing lysosomes, and decreasing capillary permeability.
<b>Adult Dose</b>	24 mg PO initially; taper to discontinue over 7 d
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity, psychosis, idiopathic thrombocytopenia, acute glomerulonephritis, fungal infections, amebiasis, nonasthmatic bronchial disease, TB, and AIDS
<b>Interactions</b>	Coadministration with digoxin may increase digitalis toxicity secondary to hypokalemia; estrogens may increase levels of methylprednisolone; phenobarbital, phenytoin, and rifampin may decrease levels of methylprednisolone (adjust dose); monitor patients for hypokalemia when taking medication concurrently with diuretics
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	Hyperglycemia, edema, osteonecrosis, peptic ulcer disease, hypokalemia, osteoporosis, euphoria, psychosis, growth suppression, myopathy, and infections are possible complications of glucocorticoid use

**Drug Category: Anticonvulsants** -- Use of certain anti-epileptic drugs, such as the GABA analogue Neurontin (gabapentin), has proven helpful in some cases of neuropathic pain. Have central and peripheral anticholinergic effects, as well as sedative effects, and block the active reuptake of norepinephrine and serotonin. The multifactorial mechanism of analgesia could include improved sleep, altered perception of pain, and increase in pain threshold.

<b>Drug Name</b>	Gabapentin (Neurontin) -- Has anticonvulsant properties and antineuralgic effects; however, exact mechanism of action is unknown. Structurally related to GABA but does not interact with GABA receptors.
<b>Adult Dose</b>	900-1800 mg/d PO in 3 divided doses; may start 300 mg the first day, 300 mg bid on second day, and 300 mg tid on third day May increase up to 1800 mg/d by adding 300 mg on following days Can titrate to effect over several days (300 mg on day 1, 300 mg bid on day 2, and 300 mg tid on day 3)
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity
<b>Interactions</b>	Antacids may reduce bioavailability of gabapentin significantly (administer at least 2 h following antacids); may increase norethindrone levels significantly
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	Renal disease, hepatic disease, breastfeeding, and advanced age; caution in severe renal disease

<b>Drug Name</b>	Carbamazepine (Tegretol) -- May reduce polysynaptic responses and block posttetanic potentiation. Inhibits nerve impulses by decreasing influx of sodium ions into cell membrane.
<b>Adult Dose</b>	100 mg PO bid with meals May increase 100 mg PO q12h until pain diminishes; not to exceed 1.2 g/d

	Maintenance dose 200-400 mg PO bid
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity, bone marrow depression, and concomitant MAOI use
<b>Interactions</b>	Serum levels may increase significantly within 30 d of danazol coadministration (avoid whenever possible); do not co-administer with MAOIs; cimetidine may increase toxicity especially if taken in first 4 wk of therapy; carbamazepine may decrease primidone and phenobarbital levels (their coadministration may increase carbamazepine levels)
<b>Pregnancy</b>	D - Unsafe in pregnancy
<b>Precautions</b>	Do not use to relieve minor aches or pains; caution with increased intraocular pressure; obtain CBC counts and serum-iron baseline prior to treatment, during first 2 mo, and yearly or every other year thereafter; can cause drowsiness, dizziness, and blurred vision; caution while driving or performing other tasks requiring alertness

**Drug Category: Analgesics** -- Pain control is essential to quality patient care. Analgesics ensure patient comfort and have sedating properties, which are beneficial for patients who experience pain.

<b>Drug Name</b>	Acetaminophen (Tylenol, Feverall, Aspirin Free Anacin) -- DOC for pain in patients with documented hypersensitivity to aspirin or NSAIDs, with upper GI disease, or who are taking oral anticoagulants.
<b>Adult Dose</b>	325-650 mg PO q4h prn; not to exceed 4 g/d
<b>Pediatric Dose</b>	10-15 mg/kg PO q4h
<b>Contraindications</b>	Documented hypersensitivity; intolerance to saccharin, table sugar, alcohol, or tartrazine (yellow dye #4)
<b>Interactions</b>	Increases effects of chloramphenicol; effects of acetaminophen increase with caffeine and diflunisal; effects of acetaminophen decrease with colestipol, anticholinergics, oral contraceptives, and cholestyramine; acetaminophen increases potential for hepatotoxicity with alcohol; severe hypothermia may occur with phenothiazines
<b>Pregnancy</b>	B - Usually safe but benefits must outweigh the risks.
<b>Precautions</b>	Hepatotoxicity possible in chronic alcoholics following various dose levels; severe or recurrent pain or high or continued fever may indicate a serious illness; APAP is contained in many OTC products and combined use with these products may result in cumulative APAP doses exceeding recommended maximum dose

<b>Drug Name</b>	Tramadol (Ultram) -- Inhibits ascending pain pathways, altering perception of and response to pain. Inhibits also reuptake of norepinephrine and serotonin.
<b>Adult Dose</b>	50-100 mg PO q4-6h prn; not to exceed 400 mg/d
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity; acute CNS depressant medication intoxication
<b>Interactions</b>	Tramadol levels decrease with carbamazepine; coadministration with opiates, hypnotics, sedatives, and alcohol increases CNS depression
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	Can cause dizziness, nausea, constipation, sweating, and pruritus; additive sedation with alcohol and TCAs; abrupt discontinuation can precipitate opioid withdrawal symptoms; adjust dose in liver disease, myxedema, hypothyroidism, hypoadrenalism; pregnancy, breastfeeding; seizure; development of tolerance or dependency with extended use; caution with norepinephrine and serotonin reuptake inhibitors, and MAOIs

**Further Outpatient Care:**

- Most cervical spine disorders are treated successfully with conservative measures on an outpatient basis. Refer to the physical therapy and other treatment sections for discussion.

**Deterrence/Prevention:**

- Maintaining proper cervical posture and cervicothoracic strength and flexibility, avoiding cervical trauma and repetitive cervical stress, and adhering to a healthy lifestyle which advocates proper nutrition, physical activity, and smoking cessation, might help prevent cervical disc disease.

**Complications:**

- Complications of cervical spine disorders may include the following:
  - Intractable axial or radicular pain
  - Myelopathy with associated weakness, hyperreflexia, and neurogenic bowel/bladder dysfunction
  - Radiculopathy with associated upper extremity weakness and numbness

**Patient Education:**

- Educate patients to avoid aggravating factors (e.g., vibrational stress from driving, cervical flexion, Valsalva maneuvers) that may exacerbate discogenic pain.
- Additionally, instruct patients on correct posture and a home exercise program (e.g., cervicothoracic stabilization, aerobic conditioning).
- For excellent patient education resources, visit eMedicine's Back, Ribs, Neck, and Head Center and Muscle Disorders Center. Also, see eMedicine's patient education articles Shoulder and Neck Pain, Neck Strain, Whiplash, Chronic Pain, and Pain Medications.

**Medical/Legal Pitfalls:**

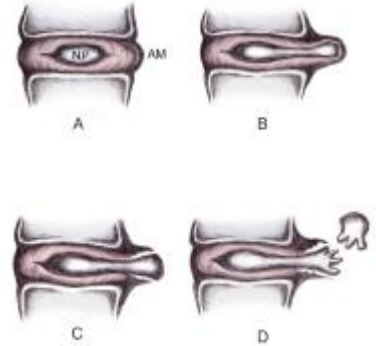
- The clinician may fail to diagnose neck pain as a manifestation of serious systemic illness (eg, malignancy, infection). Consequently, neck pain evaluation demands a careful history with review of systems, physical examination, and judicious use of imaging to avoid this medicolegal pitfall.
- Additionally, the clinician may fail to diagnose serious sequelae of cervical spine disorders (eg, myelopathy with resulting quadriplegia, radiculopathy with profound upper limb neurologic deficit) in a timely manner. Thus, neck pain evaluation requires a thorough neurologic history and physical examination.
- Interventional cervical spine procedures (eg, epidural injections, discography) carry potential for complications, including epidural hematoma with subsequent spinal cord compression, infection, respiratory arrest, vertebral artery injury, and intrinsic spinal cord damage. Competency in such

procedures, proper patient monitoring, and preparatory skills in managing such complications are mandatory.

**Picture 1**

Picture Type: Image

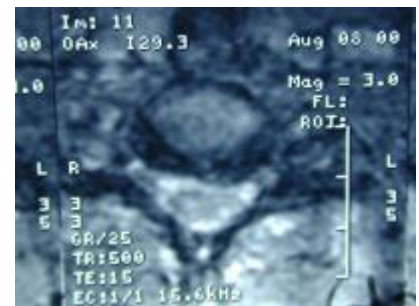
Caption: Disc herniation classification. A: Normal disc anatomy demonstrating nucleus pulposus (NP) and annular margin (AM). B: Disc protrusion with NP penetrating asymmetrically through annular fibers but confined within the AM. C: Disc extrusion with NP extending beyond the AM. D: Disc sequestration with nuclear fragment separated from extruded disc.



**Picture 2**

Picture Type: MRI

Caption: Axial MRI (C3-C4) demonstrating left-sided posterolateral protrusion of the nucleus pulposus with compression of the cerebrospinal fluid.



**Picture 3**

Picture Type: MRI

Caption: Sagittal MRI demonstrating cervical intervertebral disc protrusions at C3-C4 and C7-T1.



**Picture 4**

Picture Type: X-RAY

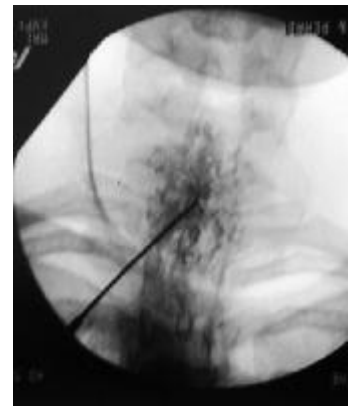
Caption: Right C7 cervical transforaminal epidural steroid injection demonstrating epidural and radicular spread of radiologic contrast dye.



### Picture 5

Picture Type: X-RAY

Caption: Cervical epidural steroid injection at the C7-T1 interlaminar space.



### Picture 6

Picture Type: X-RAY

Caption: Cervical discography. Anteroposterior fluoroscopic image.



### Picture 7

Picture Type: X-RAY

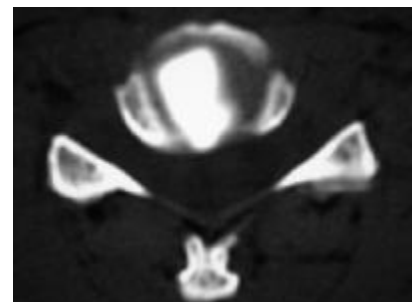
Caption: Cervical discography. Lateral fluoroscopic image.



### Picture 8

Picture Type: CT

Caption: Postdiscography axial CT scan demonstrating right posterolateral subligamentous protrusion.



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