

Neck Pain

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KEYWORDS

- Neck pain • Cervicalgia • Acute neck pain
- Chronic neck pain • Cervical spondylosis
- Cervical radiculopathy • Cervical myelopathy

Neck pain due to cervical spine and related disorders, although not as common as low back pain, is nonetheless a common and often debilitating problem and an important reason for seeking medical attention. Although patients with neck pain secondary to trauma may be seen initially in an emergency department or in some cases by a specialist, such as an orthopedic surgeon, neck pain more often is spontaneous in onset without correlation with a specific activity or neck trauma.¹ Patients with neck pain frequently see their primary care physician first, which is most appropriate given that many of these patients can be treated effectively without extensive diagnostic testing or referral to a specialist. It is important that the generalist have a good working knowledge of how to evaluate patients with neck pain and the differential diagnosis of disorders of the neck. It is also important to remember that patients presenting with neck and shoulder pain, particularly when it extends into the upper extremities, may have disorders of the brachial plexus rather than a cervical radiculopathy.

WHAT IS THE PREVALENCE OF NECK PAIN?

The lifetime prevalence of neck pain is less than that of low back pain. At the same time, there are fewer epidemiologic studies on neck pain available for review.^{1,2} Studies suggest that perhaps two thirds of individuals experience neck pain at least once during their lifetime.^{3,4} Visits to a primary care physician for the treatment of neck pain, particularly in the geriatric population, is not uncommon.^{1,5} The prevalence of neck pain increases with age and is more common in women than men.⁶

WHAT ARE THE RISK FACTORS FOR NECK PAIN?

Risk factors are better established for low back pain than neck pain, but many risk factors probably are common to both (see the article on low back pain elsewhere in this issue). As noted in the discussion on whiplash to follow, gender and rear-end automobile accidents are risk factors. Older age also is a risk factor.^{7,8}

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WHAT ANATOMIC ESSENTIALS DOES A PRIMARY CARE PHYSICIAN NEED TO KNOW TO DIAGNOSE AND TREAT THE CAUSES OF NECK PAIN?

The spinal column must be rigid enough to support the trunk and the extremities, strong enough to protect the spinal cord and cauda equina and to anchor the erector spinae and other muscles, and yet sufficiently flexible to allow for movement of the head and trunk in multiple directions. The anatomic organization of the spinal column and related structures allows for all of this, but at a price because the combined properties of rigidity and mobility can lead to many problems. The spine is most flexible in the neck, permitting multiple head movements over a wide arc.

Much of the basic organization of the spine is reviewed in the article on low back pain and is not repeated here. Instead, basic differences between the cervical and lumbar spine are emphasized. The two vertebrae in the spinal column, which vary from all the rest, are the atlas and axis. The atlas is composed of a ring of bone without a body, and the axis has the odontoid process around which the atlas rotates. The vertebral arteries pass through the transverse foramina of the first six vertebrae of the highly mobile cervical spine, which permits approximately 160° of head rotation, most of which occurs between the skull and the C3 vertebra. Approximately 90° of this rotation occurs between the axis and the atlas at the level of the atlantoaxial loop of the vertebral artery. One study showed occlusion of the vertebral artery when the head is placed in full extension and rotated 90° to the side opposite of the occluded vertebral artery.⁹ It is not surprising that vertebral artery occlusion and resultant posterior circulation distribution strokes can occur as a result of a variety of athletic injuries and therapeutic “techniques,” such as chiropractic manipulation.¹⁰ The spinal canal houses the spinal cord and the proximal portion of the cervical roots. Although lumbar canal stenosis produces only radiculopathy because the tip of the conus medullaris is at the level of L1-L2, cervical canal stenosis can produce a sometimes complex picture of cervical myelopathy and radiculopathy. The midsagittal diameter of the cervical canal from C1 to C3 is usually about 21 mm (range 16 to 30 mm) and from C4 to C7 about 18 mm (range 14 to 23 mm).¹ The midsagittal diameter of the spinal cord is 11 mm at C1 and 10 mm from C2 to C6 and 7 to 9 mm below C6.¹ The midsagittal diameter of the cervical cord normally occupies about 40% of the midsagittal diameter of the cervical canal in a healthy individual. The cervical canal midsagittal diameter is decreased by 2 to 3 mm with normal head and neck extension.¹ These anatomic relationships are of clinical importance in the context of head and neck hyperextension injuries in individuals with a congenitally narrow spinal canal, especially in individuals with additional narrowing because of cervical spondylosis. The result can be acute cervical myelopathy.

In addition to the major ligaments of the spine discussed in the article on low back pain, there are several important ligaments unique to the cervical spine. The occipital vertebral ligaments are dense, broad, extremely strong ligaments that connect the occiput to the atlas. These permit 30° of flexion and extension around the atlanto-occipital joint. The stability of the atlantoaxial joint depends almost entirely on ligaments. In this regard, the transverse ligament is the most important, permitting the atlas to rotate around the odontoid process. A tear in this ligament has the same effect as a fractured odontoid process.

With regard to the relationship between the cervical roots and the cervical vertebrae, each numbered cervical root passes through the foramen above the numbered cervical vertebra (ie, the C6 spinal nerve exits through the foramen between the C5 and C6 vertebrae). In the lumbar spine, each numbered root exits below the numbered vertebra (L5 root exits through the foramen between the L5 and S1 vertebrae). This

can be a point of confusion, particularly when reviewing MRI reports. With regard to the paraspinous musculature in the neck, there are separate extensors and flexors of the head and the cervical spine, making for a complex muscular arrangement that is in all likelihood a common generator of neck pain.

WHAT ARE THE ESSENTIALS IN THE HISTORY THAT HELP TO DEFINE THE NATURE OF NECK PAIN?

Although the approach to history taking for neck pain is similar in many respects to that for low back pain there are enough differences to warrant a separate discussion (see also the article on low back pain elsewhere in this issue). Patients with cervical disorders of one type or another may present with lower extremity and bladder or bowel symptoms often with only minimal neck pain. It is important in patients with suspected neck disorders to ask about symptoms referable to lower extremities as well as bladder and bowel functions. This can include questions about the presence of paresthesia in the lower extremities, weakness of the lower extremities, gait disorders, impotence in men, and anorgasmia in women along with bladder disturbances. Sometimes differentiating a peripheral neuropathy, cauda equina syndrome, and cervical myelopathy on the basis of the history is more difficult than one might suspect.

WHAT ARE THE ESSENTIALS OF THE PHYSICAL EXAMINATION?

On inspection of the head and neck, findings of reduced spontaneous head movement, head tilt, and neck deformity all raise the possibility of an underlying vertebral column disorder or deformity. Palpation and percussion of the neck/cervical spine, as with low back pain, have a low yield with regard to identifying a specific process. However, paracervical tenderness or other changes such as palpation of a mass do offer support for the diagnosis of a vertebral column disorder.

A gait assessment looking for evidence of bilateral lower extremity weakness and possibly spasticity is important and often easily done. Watching the patient rise from a chair and walk to the examining table often suffices. The traditional neurologic examination including individual muscle group testing of the upper and lower extremities; a sensory examination concentrating on the dermatomes of the upper extremities; and a check of deep tendon reflexes searching for absent reflexes in the upper extremities and possibly heightened reflexes and Babinski signs in the lower extremities can further define and localize the problem to the cervical spine.

In patients presenting with shoulder and upper extremity pain, careful questioning is important. The presence of paresthesia with shoulder and upper extremity pain, with or without a complaint of upper extremity weakness, rules against a diagnosis of shoulder arthropathy and rotator cuff injury. Patients with brachial plexopathy can present with shoulder and upper extremity pain and weakness and paresthesia of the upper extremities. These patients frequently do not have evidence of neck pain or a history of modulation of the pain with head/neck movement. On the basis of the history alone, it can be difficult to distinguish brachial plexopathy from cervical radiculopathy. The primary care physician may reach this differential based on the history (and subsequent physical examination) and be unable to go any further. Diagnostic testing, particularly electromyography and nerve conduction study, is often necessary for ultimate localization. The essentials of the examination can be done in several minutes.

Several neuromuscular tests are particularly helpful in evaluating a patient with neck pain:¹

- Spurling's maneuver (test): The head is inclined toward the side of the painful upper extremity and then compressed downward by the examiner. If this induces radiating pain and paresthesia into the symptomatic extremity, it strongly suggests nerve root compression, usually secondary to disk herniation. It should be noted that lateral head movement away from the symptomatic extremity sometimes can accentuate pain and paresthesia in the symptomatic upper extremity secondary to stretching of a compressed nerve root.
- Traction "distraction" test: Lifting "traction" on the head may relieve cervical spinal nerve compression reducing upper extremity pain and paresthesia.
- Valsalva test: As with low back pain/sciatica, the Valsalva maneuver with resultant increased intrathecal pressure can sometimes accentuate neck and upper extremity symptoms when due to an underlying cervical radiculopathy.
- Lhermitte's test: In patients with myelopathy that affects the posterior columns, neck flexion can produce paresthesia, usually in the back, but sometimes into the extremities. The Lhermitte's sign is most commonly associated with an inflammatory process such as multiple sclerosis, but it is sometimes noted with spinal cord compression.
- Adson's and hyperabduction tests: Long used in the evaluation of suspected thoracic outlet syndrome, these tests are nonspecific and unreliable. With the patient sitting erect, the upper extremities at the side (Adson) or symptomatic upper extremity abducted and extended (hyperabduction), the radial pulse is palpated. Each test is positive if the pulse disappears and paresthesia develops in the hand of the symptomatic extremity.

WHAT IS THE CAUSE AND WHAT IS THE BEST TREATMENT APPROACH FOR PATIENTS WHO PRESENT WITH NONRADIATING NECK PAIN?

The causes and treatment approach to neck pain are similar to the causes and treatment approach to low back pain (see article on low back pain). Patients presenting with acute and chronic neck pain generally also complain of neck stiffness and reduced mobility. The pain typically is reduced when the patient is recumbent. As with low back pain, if the pain is not reduced by recumbency, vertebral column infections and metastatic cancer should be considered.^{1,11,12}

The precise generator of pain usually cannot be identified. The inability to identify a specific etiology is confirmed in part by the lack of precision and specificity of the terms used to describe the syndrome—neck strain, musculoskeletal pain syndrome, neck spasms, myofascial pain syndrome, and in the case of chronic and more widespread pain, fibromyalgia. Tendons, ligaments, paracervical muscles, and facet joints all have been implicated as a source of pain, and all may be. However, none can be determined easily in any given patient.

In these patients, diagnostic testing has a low yield, although chronic neck pain patients frequently undergo more than one battery of tests during the course of pain. Although testing has a low yield in a patient with persistent nonradiating neck pain, after several weeks of pain, diagnostic testing may prove necessary to rule out the unexpected, such as congenital malformations of the vertebral column (eg, Klippel-Feil deformity). Although spinal radiographs almost always show spondylotic changes in older patients with nonradiating neck pain, the correlation between symptoms and radiographs is poor.¹³ Even if ultimately an MRI of the cervical spine is obtained, results can be misleading because asymptomatic herniated discs are common

and may be seen particularly in older patients with unrelated nonradiating neck and low back pain.^{14–16}

As with treatment of low back pain, common sense and conservatism are the cornerstones of management. Treatment of acute nonradiating neck pain is largely empiric and may include the following:¹

- Relative quiescence/pain avoidance, which, if necessary, may include a short period of bed rest. A cervical pillow or towel rolled up and placed under the neck in bed may help. Long-term bed rest is to be avoided.
- Medications including acetaminophen, nonsteroid anti-inflammatory drugs (NSAIDs), pain medication when necessary, and possibly muscle relaxants.
- Local application of heat or cold, although not scientifically validated, can be tried and then continued if the patient finds either beneficial.
- Bracing (controversial). Short-term use of a soft cervical collar maybe of value situationally, particularly during the performing of certain necessary activities of daily living such as driving. Long-term, regular use of a collar may actually aggravate the problem by leading to paracervical muscle disuse atrophy.

The treatment of chronic nonradiating neck pain is virtually identical to the treatment of chronic low back pain and shares much in common with the treatment of other chronic pain syndromes.¹ Physical therapy, as in the case of treatment of low back pain, is particularly important. A variety of scientifically unvalidated treatments for chronic neck pain are best avoided. They include traction, spinal manipulation/manual therapy, trigger point injections, botulinum toxin injections, transcutaneous electrical nerve stimulation (TENS) therapy, electromagnetic therapy, and acupuncture.

In the case of chronic neck pain, as with any chronic pain syndrome, it is crucial to avoid the regular use of reactive pain medications, particularly potentially addictive analgesics. The overuse of analgesics, even nonaddictive analgesics, may lead to analgesic rebound pain.

HOW COMMON ARE CERVICAL RADICULOPATHIES, AND HOW DO THEY PRESENT?

Although there are many causes of cervical radiculopathy, the most common is intervertebral disk herniation and cervical spondylosis.¹⁷ People in their 40s and 50s are particularly at risk for disk herniation. According to one study, the annual incidence of disk herniation is 83.2 per 100,000, an incidence substantially lower than lumbosacral disk herniation.¹⁸ Risk factors include heavy manual jobs, operation of vibrating equipment, lifting heavy objects, frequent automobile travel, smoking, and coughing. There is an antecedent history of trauma in 14.8% of cases and a past history of lumbar radiculopathy in 41%.¹ Despite these recognized risk factors, most patients wake up with the pain in the morning with no recall of a specific trigger for the pain. A significant percentage of these patients have a prior history of episodes of neck discomfort. The level of disk herniation/radiculopathy is as follows:¹⁸

- C6–C7 compressing the C7 root: 45% to 60%
- C5–C6 compressing the C6 root: 20% to 25%
- C8–T1 compressing the C8 root: approximately 10%
- C4–C5 compressing the C5 root: approximately 10%

Initial symptoms after disk herniation with radiculopathy are generally neck pain and stiffness. The pain tends to radiate quickly into the shoulder or scapular region and upper extremity; the exact distribution depends on the particular root involved (**Table 1**). Other symptoms include paresthesia, hyperesthesia, and weakness.

Root	Pain Distribution	Dermatomal Sensory Distribution	Weakness	Affected Reflex
C4	Upper neck	"Cape" distribution shoulder/arm	None	None
C5	Neck, scapula, shoulder, anterior arm	Lateral aspect of arm	Shoulder abduction Forearm flexion	Biceps Brachioradialis
C6	Neck, scapula, shoulder, lateral arm and forearm into first and second digits	Lateral aspect forearm And hand and 1st and 2nd digits	Shoulder abduction Forearm flexion	Biceps Brachioradialis
C7	Neck, shoulder, lateral arm, medial scapula, extensor surface forearm	3rd digit	Elbow extension Finger extension	Triceps
C8	Neck, medial scapula, medial aspect arm and forearm into 4th, 5th digits	Distal medial forearm to hand and digits 4 and 5	Finger: abduction adduction flexors	Finger flexors

From Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:1–205; with permission.

Paresthesia and hyperesthesia are generally dermatomal in distribution, with the greatest involvement often centered in the distal portion of the involved dermatome.

HOW DO PATIENTS WITH CERVICAL CANAL STENOSIS PRESENT?

Cervical stenosis may be clinically silent for long periods, sometimes throughout life. Although cervical stenosis and resultant myelopathy can be caused by many pathologic processes, including trauma with resultant hyperextension in the presence of congenital stenosis (a concern in contact sports) and central disk herniation, the most common cause is spondylosis (degeneration).¹⁹ About 80% of people by age 50 and virtually 100% of people by age 70 have cervical spondylosis to some degree.¹

About a midcervical canal sagittal diameter of 12 mm or less generally is associated with the development of myelopathy; larger dimensions do not rule out the possibility of developing myelopathy under some circumstances. Degenerative change with resultant osteophytes, bulging disks, facet joint hypertrophy, and thickened ligamentum flavum, in combination with intermittent flexion-extension-mediated injury, can produce cervical myelopathy in the absence of dramatic cervical canal stenosis. In addition to myelopathy as a result of direct cord compression, there can be a compromise of perfusion in the distribution of the anterior spinal artery with resultant ischemic myelopathy.²⁰ Crandall and colleagues²¹ described five distinct cord syndromes representing relatively advanced disease, as follows:

- Brown-Séquard syndrome (as a result of hemicord injury)
- Central cord syndrome, with motor and sensory deficits more marked in the upper extremities than the lower extremities
- Motor system syndrome resembling amyotrophic lateral sclerosis by virtue of lower motor neuron changes in the upper extremities and upper motor neuron changes in the lower extremities in the absence of significant sensory deficit

- Brachialgia and cord syndrome, characterized by upper extremity radicular distribution pain and an admixture of upper and lower motor neuron weakness in the extremities
- Transverse myelopathy, the most common, appearing suddenly or evolving from one of the preceding syndromes; all ascending and descending tracts are involved and sphincter involvement is common

These syndromes generally are not defined clearly early in the course of cervical myelopathy. Symptoms and signs often are subtle early on. Hyperreflexia and extensor plantar responses (Babinski sign), minimal weakness in the lower extremities, and a subtle gait disturbance are common early signs.¹⁹ Occipital headache and cervicalgia commonly are associated with cervical spondylosis, but the cause-and-effect relationship remains uncertain because cervical spondylotic myelopathy often occurs in the absence of head and neck pain. Leg discomfort, including burning paresthesia, can occur and may be confused with sciatica. Combined cervical and lumbar spondylosis can produce overlapping symptoms and signs of cervical myelopathy and cauda equina syndrome/lumbosacral radiculopathy, leading to difficulties in diagnosis. Lhermitte's symptom/sign may be observed early in some patients. Subtle clumsiness and paresthesia in the hands may be the only initial symptoms and can be confused with median and ulnar mononeuropathies.²² Vertebral basilar transient ischemic events may occur in association with head rotation and resultant vertebral artery compromise.⁹

The differential diagnosis of cervical spondylotic myelopathy includes multiple sclerosis, transverse myelitis, progressive motor neuron disease, subacute combined degeneration, syringomyelia, and cord tumors. Generally speaking, these diagnostic possibilities can be distinguished from each other by the history, physical/neurologic examination, and selected diagnostic tests (MRI, electromyography, and nerve conduction study). "The hard part" is the initial early recognition of an evolving myelopathy.

WHAT IS WHIPLASH AND HOW SHOULD IT BE TREATED?

There are few spinal disorders that are more controversial and contentious than whiplash. Approximately 1 million whiplash (flexion-extension) neck injuries occur annually in the United States, 85% of which are the result of rear-end automobile collisions.^{1,23} In contrast to most other injuries, there is a female preponderance of 2:1.^{1,23} Some authors have speculated that this gender difference reflects a woman's smaller, less muscular, neck.^{1,23}

Whiplash can result in a variety of symptoms and neurologic signs based in part on the velocity of the impact and in part on the presence or absence of underlying cervical spine disease. Myelopathy, radiculopathy, brachial plexopathy, and upper extremity motor neuropathy all can occur. Most patients presenting for evaluation at some point after injury have less specific symptoms, however, and few "hard" signs on examination. Localized neck pain, neck stiffness, occipital headache, dizziness in all of its forms, malaise, and fatigue are common whiplash symptoms.^{1,24} Localized paracervical tenderness to palpation, reduced range of neck motion, and weakness of the upper extremities secondary to guarding are common findings.

Although most patients with myofascial symptoms recover in several months, 20% to 40% complain of debilitating symptoms for extended periods, sometimes years.¹ This "late" or chronic whiplash syndrome defined as pain beyond 6 months post-injury, often is mired in litigation.

Mechanisms suggested for chronic whiplash syndrome include subtle lesions of cervical facet joints,²⁵ cervical ligament strain,²⁶ disk protrusions, disturbance of vestibular brain stem function, and reduced cerebral perfusion.²⁴ Some studies suggest psychosocial factors, including litigation, play a major role in chronic whiplash.^{1,27,28} A prospective control study from Lithuania, where there is no legal tort system, revealed that late whiplash syndrome does not exist.²⁹

Extensive diagnostic testing generally is not necessary in patients with acute whiplash injury. Most patients receive cervical spine x-rays, particularly if taken to an emergency department after the accident. The yield from diagnostic testing for patients with chronic whiplash injury is low, and testing should be avoided in the absence of neurologic findings.

Conservative treatment for acute whiplash injury is the treatment of choice.³⁰ Rest, avoidance of pain, NSAIDs, perhaps muscle relaxants, and a short course of pain medication all can be helpful. A soft cervical collar for acute whiplash injury can be helpful; however, continuous long-term usage should be avoided.

Treatment for chronic whiplash syndrome also should be conservative. Medications used in the treatment of chronic pain, including serotonin reuptake inhibitors, may have a role. As with any chronic pain syndrome, the persistent use of reactive pain medication is to be avoided. In a subset of late whiplash patients who had facet joint-generated pain as determined by diagnostic blocks with a local anesthetic, percutaneous radiofrequency neurotomy proved beneficial.^{31,32}

WHAT IS THE ROLE OF THE FACET JOINT IN NECK (SPINE) PAIN?

The role of the facet (zygapophyseal) joint in the pathogenesis of neck (and low back) pain is controversial.^{1,33-35} The lack of localizing specificity of neck pain from the history and physical examination can contribute to this confusion. Although degeneration of the facet joint probably produces pain in some, every clinician is aware of the patient with severe degenerative spine/facet joint disease with no associated pain. Nonetheless, there still is considerable clinical evidence supporting the existence of the facet syndrome. Certainly, degenerative changes in the facet joint can contribute to spinal stenosis and resultant radiculopathy and in the case of the cervical spine, myelopathy. Traumatic capsular tears and age-related degenerative changes are thought to be the most common causes of facet joint-mediated pain.³⁵ Facet joint disruption may also be at the basis of pain for at least some patients with a whiplash injury.³⁶ The pain of the facet joint syndrome is generally localized over the affected joint. It is aggravated by extension of the spine. Although the pain is often relatively localized, on occasion it may radiate into the ipsilateral upper extremity. Therefore, the facet joint syndrome is included in the differential diagnosis of radiculopathy. A diagnostic block into the facet joint with a local anesthetic may help to isolate the source of pain. The value of therapeutic facet joint injection remains controversial.^{37,38} However, following a diagnostic block with an anesthetic, percutaneous radiofrequency neurotomy of the medial branch of the cervical posterior ramus that innervates the facet joint has been offered as a useful treatment for patients with suspect facet joint arthritis.³²

CAN FIBROMYALGIA PRESENT AS NECK AND SHOULDER PAIN?

Fibromyalgia is a chronic widespread musculoskeletal pain syndrome of unknown etiology, present by definition for at least 3 months. At its zenith, it usually is generalized to such a degree that it is not confused easily with pain secondary to a localized spine disorder. Early in the evolution of fibromyalgia, however, neck, shoulder, and low

back pain may predominate. In addition to aching pain, symptoms include depression, fatigue, malaise, stiffness, disturbed sleep, headache, paresthesia, and irritable bowel.³⁵

There is a considerable debate as to whether fibromyalgia is a specific entity or a manifestation of an underlying psychologic disorder including stress and depression.¹ Studies have shown, however, several chemical and physiologic changes suggesting a possible biologic basis, which include increased levels of substance P in serum and cerebrospinal fluid, decreased cerebrospinal fluid tryptophan level, and decreased serum and platelet serotonin levels.³⁹ The sleep electroencephalogram/polysomnogram in these patients often reveals an alpha delta sleep pattern (as also can be seen in depression), indicating reduction of the deep sleep phase during which muscle restoration ordinarily occurs.

Examination of patients with fibromyalgia shows widely distributed tender points to light palpation. Diagnosis, according to criteria established by the American College of Rheumatology, requires 11 such tender points. Fibromyalgia is a diagnosis of exclusion. Specific rheumatologic conditions, such as the spondyloarthropathies and polymyalgia rheumatica, need to be considered. Treatment options include tricyclic antidepressants, NSAIDs, a program of regular exercise, periods of rest, massage, and possibly at times the application of local heat.

HOW IS AN ACUTE CERVICAL RADICULOPATHY CAUSED BY DISK HERNIATION/SPONDYLOSIS BEST MANAGED?

Treatment approaches to cervical radiculopathy are similar to lumbar radiculopathy (see the article on low back pain elsewhere in this issue). As with nonspecific acute spine pain, acute radiculopathy is often a self-limiting disorder with recovery expected over a period of weeks.^{40,41} The initial management of acute radiculopathy need not differ from the treatment of acute nonradiating neck pain. This is particularly true in the absence of significant weakness. In this setting, relative quiescence with avoidance of activities that increase pain, medications (NSAIDs and pain medication), and the use of a soft cervical collar situationally (I often recommend wearing it reversed to allow for neck flexion and more comfort) can help. Some authors advocate a brief course of oral corticosteroids. Corticosteroids and NSAIDs should not be used in combination.

If the patient has significant weakness within a given cervical myotome, a prompt workup is usually indicated, including MRI of the cervical spine and possibly electromyography and nerve conduction study of the symptomatic upper extremity and ipsilateral paracervical muscles. Spontaneous recovery without surgery can be expected in 75% to 80% of all patients with cervical disk herniation.⁴² Spontaneous recovery is in part a result of the absorption and shrinkage of the displaced disk material with resultant reduction of nerve root impingement.^{16,42}

As with lumbar radiculopathies, determining when a patient should undergo surgical treatment for a disk herniation causing a radiculopathy is often challenging. There are no hard and fast rules. As discussed in the management of lumbosacral radiculopathies, an important indicator for surgery is the presence of significant weakness in a group of muscles (myotome) innervated by the impinged nerve root. In addition, if there is evidence of cervical myelopathy in a patient with cervical radiculopathy, immediate surgical decompression may be appropriate.

As with the management of lumbar radiculopathy, there are many widely used treatments that are not fully scientifically validated, including oral glucocorticoids, epidural blocks, selected nerve blocks, and acupuncture. Cervical manipulation, chiropractic or otherwise, is to be avoided.

IS CHIROPRACTIC MANIPULATION USEFUL IN TREATMENT OF NECK PAIN?

Chiropractic manipulation for the treatment of spine symptoms including neck pain is a common practice in the United States. Chiropractic treatment is approved by many insurance companies. Chiropractic is the invention of Daniel David Palmer in 1895. At that time, he was a dry goods grocer and part-time magnetotherapist. Chiropractic is based on a theory that all disease is a result of interference with the body's "innate intelligence" by misaligned vertebrae. Some studies have shown marginal benefit for low back symptoms.^{17,19,21} However, with regard to chiropractic manipulation of the cervical spine, there is risk with little if any reward.^{1,10,43-45} Risks include cervical myelopathy, cervical radiculopathy, and vertebrobasilar artery distribution strokes. Neck manipulation, particularly when it includes a combination of rotation and tilting, stretches the contralateral vertebral artery, producing a sheering force on the segment of the artery at the level of the atlantoaxial joint.⁹ This force may result in dissection of the vertebral artery, with resultant potential occlusion of the lumen, thrombus formation, and embolization. The frequency of vertebrobasilar artery distribution strokes is argued, but it is probably more common than reported.¹⁰ Given the risk of complications in the absence of well-documented benefit, chiropractic cervical manipulation should be avoided.^{10,46}

ARE THERE ANY OTHER COMPLEMENTARY AND ALTERNATIVE MEDICAL TREATMENTS THAT MIGHT BE OF VALUE FOR PATIENTS WITH NECK PAIN?

There are a variety of complementary and alternative medical (CAM) treatments available for most medical/neurologic conditions. With regard to the treatment of neck pain, the most common CAM treatments, other than chiropractic manipulation, are massage therapy and acupuncture. There is some evidence to suggest that therapeutic massage may be of value in patients with neck pain, in particular nonradiating neck pain.⁴⁷ Although scientific validation is limited, massage is safe and appears to reduce pain in at least some patients.

The effectiveness of acupuncture remains controversial. Some studies demonstrated that acupuncture may be more effective than no treatment or sham treatment.⁴⁷

Both massage therapy and acupuncture are generally safe.

Although there is controversy, I generally recommend massage relatively early in the course of neck pain if the patient is not improving. I generally do not recommend acupuncture.

REFERENCES

1. Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. *Continuum* (NY) 2001;7:1-205.
2. Rubin DI. Epidemiology and risk factors for spine pain. *Neurol Clin* 2007;25:353-71.
3. Cote P, Cassidy JD, Carroll L, et al. The Saskatchewan health and back pain survey: the prevalence of neck pain and related disability in Saskatchewan adults. *Spine* 1998;1689-98.
4. Croft PR, Lewis M, Papegegiou AC, et al. Risk factors for neck pain: a longitudinal study in the general population. *Pain* 2001;93:317-25.
5. Anderson G. The epidemiology of spinal disorders. In: Frymoyer JW, editor. *The adult spine: principles and practices*. New York: Raven Press; 1991. p. 107-46.

6. Cote P, Cassidy JD, Carrole J, et al. The factors associated with neck pain and its related disability in the Saskatchewan population. *Spine* 2000;25:1109–17.
7. Cassidy JD, Cote P, Carroll L, et al. The prevalence of neck pain and associated factors: a population-based study from North American. Denmark: E.C.U. Convention; 1999. p. 17–8.
8. Harder S, Veilleux M, Suissa S, et al. The effect of socio-demographic and crash-related factors on the prognosis of whiplash. *J Clin Epidemiol* 1998;51:377–84.
9. Brown B, Tatlow W. Radiographic studies of the vertebral arteries in cadavers: effects of position and traction on the head. *Neuroradiology* 1963;81:80–8.
10. Devereaux MW. The neuro ophthalmologic complications of cervical manipulations. *J Neuroophthalmol* 2000;20:236–9.
11. Deyo RA, Cherkin D, Conrad D, et al. Cost, controversy, crisis: low back pain and the health of the public. *Annu Rev Public Health* 1992;12:141–55.
12. Anonymous. Scientific approach to the assessment and management of activity-related spinal disorders: a monograph for clinicians. Report of the Quebec task force on spinal disorders. *Spine* 1987;12:S1–59.
13. Deyo RA. Plain roentgenography for low-back pain: finding needles in a hay stack. *Arch Intern Med* 1989;150:1125–8.
14. Boden SD, Davis DO, Dina TS, et al. Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. *J Bone Joint Surg Am* 1990;72:403–8.
15. Jensen M, Brant-Zawadzki M, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med* 1994;331:69–73.
16. Ahmed M, Modic MT. Neck and low back pain: neuroimaging. *Neurol Clin* 2007; 25:439–71.
17. Algren B, Garfen S. Cervical radiculopathy. *Orthop Clin North Am* 1996;27: 253–63.
18. Radhakrishnan K, Litchy W, O'Fallon W, et al. Epidemiology of cervical radiculopathy: a population-based study from Rochester, Minnesota, 1976 through 1990. *Brain* 1994;117:325–35.
19. McCormack B, Weinstein P. Cervical spondylosis: an update. *West J Med* 1996; 165:43–51.
20. Fehlings M, Skaf G. A review of the pathophysiology of cervical spondylotic myelopathy with insights for potential novel mechanisms drawn from traumatic spinal cord injury. *Spine* 1998;23:2730–7.
21. Crandall P, Batzdorf U, Conrad D, et al. Cervical spondylotic myelopathy. *J Neurosurg* 1996;25:57–66.
22. Voskuhl R, Hinton R. Sensory impairment in the hands secondary to spondylotic compression of the cervical spinal cord. *Arch Neurol* 1990;47:309–11.
23. Evans R. Some observations. *Neurol Clin* 1992;10:975–97.
24. Kasch H, Bech FW, Stengaard-Pedersen K, et al. Development pain and neurologic complaints after whiplash. *Neurology* 2003;60:743–61.
25. Winkelstein BA, Nightingale RW, Richardson WJ, et al. The cervical facet capsule and its role in whiplash injury: a biomechanical investigation. *Spine* 2000;25:1238–46.
26. Ivancic PC, Pearson AM, Pajabi MM, et al. Injury of the anterior longitudinal ligament during whiplash simulation. *Eur Spine J* 2004;13:61–8.
27. Peterson D. A study of 249 patients with litigated claims of injury. *Neurologist* 1998;4:131–7.
28. Cassidy JD, Carol LJ, Coté P, et al. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *N Engl J Med* 2000;342:1179–86.

29. Obelieiene D, Schrader H, Bovim G, et al. Pain after whiplash: a prospective controlled inception cohort study. *J Neurol Neurosurg Psychiatr* 1999;66:279–82.
30. Carette S. Whiplash injury and chronic neck pain [editorial]. *N Engl J Med* 1994; 330:1083–4.
31. Barnsley L, Lord SM, Wallis BJ, et al. Lack of effect of intraarticular corticosteroids for chronic pain in the cervical zygapophyseal joints. *N Engl J Med* 1994;330: 1047–50.
32. Lord S, Barnsley L, Wallis BJ, et al. Percutaneous radio-frequency neurotomy for chronic cervical zygapophyseal-joint pain. *N Engl J Med* 1996;335:1721–6.
33. Jackson R. The facet syndrome: myth or reality? *Clin Orthop* 1992;279:110–21.
34. Schwarzer AC, Aprill CN, Derby R, et al. Clinical features of patients with pain stemming from the lumbar zygapophyseal joints: is the lumbar facet syndrome a clinical entity? *Spine* 1994;19:1132–7.
35. Meleger AL, Krivickas LS. Neck and back pain: musculoskeletal disorders. *Neurol Clin* 2007;25:419–38.
36. Lord SM, Barnsley L, Wallis BJ, et al. Chronic cervical zygapophyseal joint pain after whiplash: a placebo-controlled prevalence study. *Spine* 1996;21:1737–45.
37. Nelemens PJ, Bie RA, de Vet HCW, et al. Injection therapy for subacute and chronic benign low back pain. *Cochrane Database Syst Rev* 2000;2:CD001824.
38. Niemisto L, Kalso E, Malmivaara A, et al. Cochrane Collaboration Back Review Group. Radio frequency denervation for neck and back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine* 2003;28:1877–88.
39. Russell I. Advances in fibromyalgia: possible role for central neurochemicals. *Am J Med Sci* 1998;316:377–84.
40. Persson J, Moritz W, Brandt L, et al. Cervical radiculopathy: pain, muscle weakness and sensory loss in patients with cervical radiculopathy treated with surgery, physiotherapy or cervical collar: a prospective controlled study. *Eur Spine J* 1997; 6:256–66.
41. Deyo RA, Weinstein JN. Low back pain. *N Engl J Med* 2001;344:363–9.
42. Fager CA. Observations on spontaneous recovery from intervertebral disc herniation. *Surg Neurol* 1994;42:282–6.
43. Hurwitz EL, Aker PD, Adams AH, et al. Manipulation and mobilization of the cervical spine. *Spine* 1996;21:1746–60.
44. Gross AR, Hoving JL, Haines TA, et al. Cervical overview group. Manipulation and mobilization for mechanical neck disorders. *Cochrane Database Syst Rev* 2006;3.
45. Ernst E, Canter PH. A systematic review of spinal manipulation. *J R Soc Med* 2006;99:192–6.
46. Barr J. Point of view. *Spine* 1996;21:1759–60.
47. Cherkin DC, Sherman KJ, Deyo RA, et al. A review of the evidence for the effectiveness, safety, and cost of acupuncture, massage therapy, and spinal manipulation for back pain. *Ann Intern Med* 2003;138:898–906.