

An Approach to the Patient with Neck Pain

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Abstract

In the United States, neck pain is an extremely common reason for seeking medical attention. While symptoms can occur abruptly, they are often more indolent in their course, usually without any temporal relationship to trauma or other inciting events. For these reasons, physicians need to be well-versed in the initial evaluation and management of these complaints. It is important to have a working knowledge of the cervical anatomy, the differential diagnosis for neck pain, and the potential mimics which may present. It is equally imperative to hone the skills necessary to effectively differentiate localized, mechanical neck pain syndromes from those of a more serious etiology. To that end, identifying “red flags” during the patient’s history and performing a focused musculoskeletal and neurological examination is critical to triaging the patient appropriately. As the U.S. health care system moves away from traditional fee-for-service reimbursement, greater emphasis will be placed on providing high-quality, cost-conscious care. Physicians must be deliberate when evaluating a patient with neck pain, rather than ordering a “one size fits all” battery of tests. Knowing when to order advanced imaging modalities or refer a patient to a specialist will be paramount in providing the best patient care while responsibly utilizing resources.

Keywords: Neck pain; radiculopathy; myelopathy; chronic pain; spondylosis

1. What is the prevalence of neck pain?

Neck pain is the fourth leading cause of disability in the United States [18]. Reduced productivity related to neck pain and other musculoskeletal disorders has been estimated to cost \$61.2 billion annually [25]. It is expected that about two thirds of Americans will experience a significant episode of neck pain at some point in their life [4,7,9]. A recent study estimated that, over a 3-month period, 9 million Americans were affected by new-onset neck pain with an additional 19 million citing both neck and back pain [26]. The incidence rises as people grow older, but plateaus in middle-age. Women are more often affected than men (chart 1) [4,6,14,20].

pain vary considerably. Abrupt, new onset pain tends to be associated with exposure to certain events (e.g. rear-end automobile accidents, sporting injuries). However, as will be discussed later, this pain is generally self-limited. There is an expanding literature exploring the subset of people in which acute neck pain does not resolve, but rather transforms into chronic pain [24,26,27]. Frequently identified risk factors include high body mass index, frequent neck extension during the day, psychologically demanding jobs, insomnia, tobacco use and a sedentary lifestyle [24,26]. A variety of psychosocial factors often seem to have an impact as well. Concurrent depression, anxiety or somatization tendencies seem to play a significant role in the evolution of chronic musculoskeletal pain [26, 27].

2. What are the risk factors for neck pain?

As one might expect, the risk factors for acute onset neck pain and chronic neck

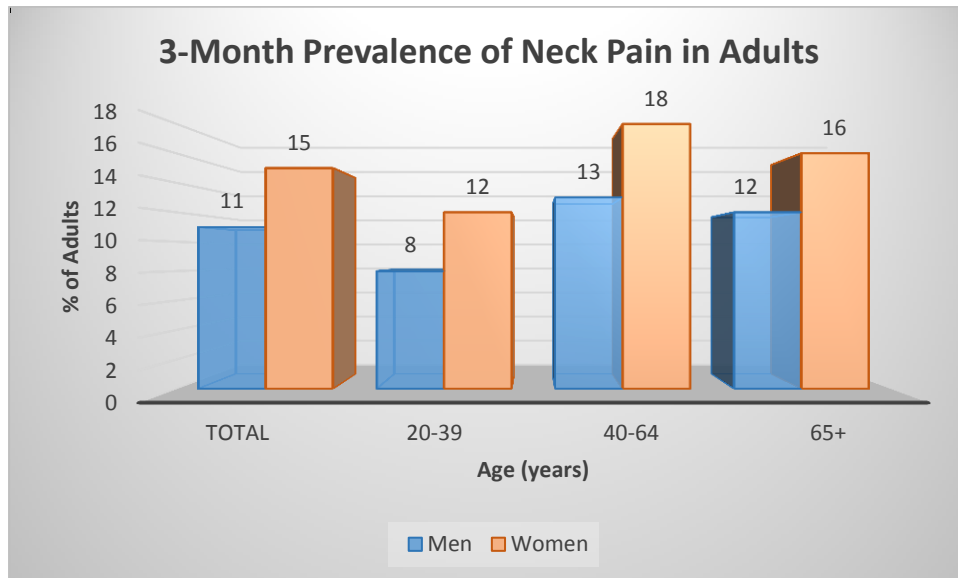


Chart 1. Source: National health interview survey, 2007

3. What is the relevant anatomy and its common pathology?

The cervical spine must be rigid enough to support the head and strong enough to protect the indwelling spinal cord, yet sufficiently flexible to allow for movement in multiple planes. This is a difficult task and one that places the region at great risk for injury. The articulation between the occiput and the first cervical vertebra is responsible for approximately one third of neck flexion and extension and one half of lateral motion in the neck. The articulation between the first and second cervical vertebra allows for 50% of rotational range of motion. The articulations between the second and seventh cervical vertebrae allow for approximately two thirds of flexion and extension, 50% of rotation, and 50% of lateral motion. It is this region where the highest proportion of cervical disease occurs. Ironically, it is the structures intended to protect the spinal cord and nerves which ultimately inflict the most damage. Uncovertebral joints (joints of Luschka) span from the C3 to the C7 vertebrae. With age, these joints hypertrophy and, as a result, narrow the intervertebral foramen which may impinge on the exiting spinal nerves. The ligamentum flavum is in actuality a series of ligaments which run the length of the posterior aspect of the spinal canal, connecting the laminae of adjacent vertebrae. This structure contributes to the alignment and integrity of the spinal column, but also has elastic components which assist the cervical musculature in maintaining a neutral head position. Over time, with repeated rupture and regeneration, these ligaments can hypertrophy and result in narrowing of the central canal (cervical spinal stenosis).

The “shock absorbers” of the spine, the intervertebral discs, are one of the more

frequent generators of neck pain. A review of MRI scans recently revealed disc extrusions in 20% of asymptomatic patients between the ages of 40 and 55 years [10]. These herniations are typically directed in a posterolateral direction due to the presence of the posterior longitudinal ligament. As a result, this pathology tends to affect the exiting nerve root, resulting in cervical radiculopathy. On occasion, the disc may be more broad-based or may extend directly posterior by way of an annular tear (disruption of the annulus fibrosis). In these cases, there may be central canal stenosis and myelopathy.

The normal cervical lordosis is known as a “secondary curvature,” not appearing until several months after birth. This is a compensatory curve which helps to shift the weight of the head directly over the lower extremities, in line with the body axis. This natural curvature can be lost or even reversed in patients with significant degenerative disease. These changes place the neck at a mechanical disadvantage, forcing other structures such as the trapezius and paraspinal muscles to play a larger role in maintaining appropriate posture. While there isn't much support in the literature, it would stand to reason that this would be a common generator of neck pain. As might be expected, these anatomical alterations tend to occur in concert, precipitating neck pain which is thus multifactorial in etiology. This results in cervical spondylosis, a diagnosis covering a broad array of arthritic and degenerative conditions that ultimately translates to “a bad neck.”

4. What are the essentials in the history that help to define the nature of the neck pain?

While neck pain onset is often insidious, special attention is required in cases of newly diagnosed pain or when there

is an abrupt change in quality or severity of established neck pain. Obtaining the history is not unlike other complaints, though certain questions are of great importance. One must assess the acuity of the symptoms and their relationship to any particular trauma or activity. What is the quality of the pain? Where is the location of greatest discomfort? For example, anterior neck pain is uncommon in musculoskeletal injury and should prompt other investigations. What time of day is it most intense? Mechanical pain is typically worse at the end of the day, while inflammatory conditions may be worse in the morning. Is it well-localized or does it radiate to another region? Are there mitigating factors? Radicular pain may be provoked by increases in intrathoracic pressure (e.g. coughing, sneezing, Valsalva). Are the symptoms alleviated by certain body positions? As with low back pain, if the pain is not reduced by recumbency, vertebral column infections and metastatic cancer should be considered [3,11,15].

The key to the interview is to screen for conditions which could potentially lead to significant morbidity if missed. Patients with prominent pain may complain of that alone. It is the responsibility of the physician to probe for other symptomatology which the patient may not readily volunteer. The examiner must inquire about disturbances of bowel or bladder function, sensory changes in the lower extremities, muscle weakness or imbalance, all of which could be the sequelae of a compressed cervical spinal cord. Keep in mind that cord compression in and of itself is not usually painful, but can occur in association with a painful cervical radiculopathy. Take care not to be preoccupied with shooting upper extremity pain and miss the so-called “long-track” symptoms of myeloradiculopathy. While the majority of neck pain is musculoskeletal or neurologic in origin, one must also be wary

of other general medical symptoms such as weight loss, persistent fevers or night sweats. A careful review of systems should be completed looking for any evidence of constitutional signs which may lead to a broader differential diagnosis.

5. What are the essentials of the physical examination?

The examination begins with inspection. If the neck pain is the result of recent significant trauma, the neck should be stabilized and imaged before commencing the physical examination (see imaging below). Once the spine is “cleared,” an examination can be performed. Findings of reduced spontaneous head movement, head tilt and deformity in the natural neck curvature all raise the possibility of an underlying vertebral column disorder. Tenderness on palpation and restricted passive range of motion are sensitive markers of cervical spine disease, but very non-specific. This may be due to guarding, associated muscle spasm/injury or even focal dystonia. A thorough neurological assessment is needed to differentiate isolated axial pain syndromes from those of a more significant cause. Manual power testing should be performed to identify focal weakness, particularly if it occurs in a myotomal distribution. Depressed upper extremity reflexes can occur with cervical radiculopathy and again are generally root-specific (table 1). Lower motor neuron findings (e.g. weakness, hyporeflexia) not limited to a specific myotomal pattern may suggest multi-root involvement (i.e. polyradiculopathy) or potentially a brachial plexopathy- which can present with neck pain as well. These entities can be difficult to distinguish on clinical examination alone and, as we will discuss below, additional investigations are often needed. Brisk reflexes in the lower extremities are

suggestive of spinal cord pathology as are a number of other pathological reflexes. Passive muscle tone or spasticity must be assessed as another valuable tool in distinguishing upper and lower motor neuron pathology. A thorough sensory examination should be completed both assessing for dermatomal loss (table 1) as well as screening for a sensory cord level on the back and chest. When suspicion for cord involvement is high, assessment of rectal tone is important. There are several special tests or “provocative maneuvers” which are particularly helpful in evaluating a patient with neck pain [15]:

- Modified Spurling’s maneuver: The head is extended, rotated toward the side of the pain and laterally flexed toward the side of pain. An axial load is then placed downward on the head. If this induces radiating pain and paresthesia into the symptomatic extremity, it is highly specific for nerve root compression, usually secondary to disc herniation.
- Traction “distraction” test: Placing vertical upward 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 impacts the posterior columns, neck flexion may 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 with multiple sclerosis, but it is sometimes noted with spinal cord compression.

Once again, these maneuvers should only be performed in non-traumatic cases or in situations where this is little chance of skeletal instability.

Table 1. Roots of the brachial plexus with their associated myotome and dermatome.

Root	Weakness	Sensory Loss	Reflex
C5	Shoulder abduction, elbow flexion	Lateral arm	Biceps, Brachioradialis
C6	Shoulder abduction, elbow flexion	Lateral forearm, first digit, second digit	Biceps, Brachioradialis
C7	Elbow extension, wrist extension, finger extension	Middle finger	Triceps
C8	Finger abduction, adduction	Medial forearm, fourth digit, fifth digit	N/A

6. What imaging modalities are used to evaluate neck pain and when should they be ordered?

Caution must be exercised with ordering imaging of the cervical spine because asymptomatic herniated discs and spondylotic changes are common and may be seen particularly in older patients with unrelated non-radiating neck and low back pain [5]. Reliance on imaging results alone may lead to additional unnecessary testing or even unsuccessful surgery. In an effort to avoid these situations, imaging should always be ordered as a confirmatory test to investigate a clinician's suspicion for a particular disease which is based on the history and physical examination. The routine ordering of imaging as a substitute for a complete history and physical examination may often reveal

“incidentalomas” which confound the workup and exhaust resources. Trauma is the exception to this general principle, as a full examination is often not possible due to concerns about cervical instability. Radiographs are affordable and useful in assessing for skeletal abnormalities such as atlantoaxis subluxation, Klippel-Feil syndrome or vertebral compression fractures. While plain films are often ordered to assess for neuroforaminal or spinal stenosis, the findings are generally very inaccurate. MRI is the modality of choice when investigating cervical radiculopathy or central canal stenosis of any etiology. CT scan is often appropriate for a patient with a suspected occult fracture (because of severe guarding or spinous process point-tenderness) and it is superior to MRI in this regard [2].

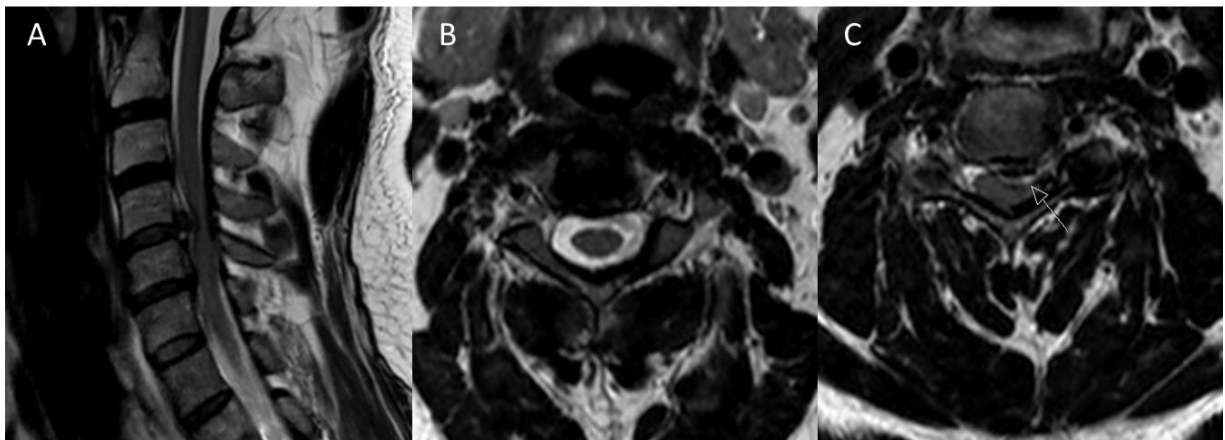


Image 1. MRI cervical spine, T2 weighted imaging. A) Midline sagittal image demonstrating multi-level central canal stenosis with spinal cord compression due to broad-based disc herniations at C4/C5 and C5/C6. B) Axial image at the level of the C3/C4 disc space. C) Axial image at the level of the C4/C5 disc space demonstrating extruded disc material (arrow) which obliterates the normal bright CSF signal and deforms the spinal cord.

7. How do cervical radiculopathies present and how are they best managed?

The most common reason for an acute cervical radiculopathy is an intervertebral disc herniation [1]. These injuries may be brought on by an identifiable event, but others seem to occur sporadically. 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, this is of particular importance when evaluating imaging studies. For example, the C6 spinal nerve exits through the foramen between the C5 and C6 vertebrae. The level of disc herniation/radiculopathy is as follows [22]:

- 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 disc herniation with radiculopathy are generally neck pain and stiffness. The pain tends to evolve, eventually radiating into the shoulder, scapula or upper extremity. The exact distribution depends on the particular root(s) involved. Other symptoms include paresthesia, hyperesthesia and weakness. Sensory disturbances are generally dermatomal in distribution, with the greatest involvement often predominately affecting the distal portion of the involved dermatome. When a disc herniation is responsible, the natural course of the condition is typically self-limited with 75-90% improving without surgery. The disk material eventually resorbs, alleviating the pressure on the adjacent nerve root. Patients without

concurrent myelopathy or significant weakness should be treated conservatively for at least six weeks. The cornerstones of conservative management are immobilization, nonsteroidal anti-inflammatory medication and physical therapy [29]. Epidural steroid injections are more controversial. A systematic review of 34 recent studies demonstrated that injections with local anesthetic and steroids can result in pain intensity reduction and improved functional status, but these interventions should be considered on a case by case basis [12]. 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 with nerve conduction studies of the symptomatic limb and ipsilateral paracervical muscles.

Surgery is typically reserved for those who have a significant functional deficit, incapacitating pain or fail to improve with medical management. Surgical options include anterior cervical discectomy alone, laminectomy with discectomy, discectomy with fusion, disc arthroplasty (implanting an artificial cervical disc) and posterior foraminotomy [29]. Comparisons of the various surgical techniques are beyond the scope of this article and best left for the surgeon to discuss with the patient. It should be noted, however, that when surgery is performed, the alteration of the spinal mechanics may place adjacent vertebral levels at risk for accelerated degenerative disease. When cervical radiculopathy is caused by osteophytes as seen in spondylosis, they are much less likely to remit without intervention. In these cases, the risks and benefits of surgery need to be carefully evaluated.

8. How do patients with cervical canal stenosis present?

In isolation, cervical myelopathy can often be painless and, early on, minimally symptomatic. Patients often present with “unsteadiness,” leg stiffness or even mild foot numbness. About 80% of people by age 50 and virtually 100% of people by age 70 have radiologic evidence of cervical spondylosis [15]. A mid-cervical canal sagittal diameter of 12 mm or less is generally associated with the development of myelopathy. Although 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 disc herniation, the most common cause is spondylosis (degeneration) [16]. Degenerative changes combining any of the following: osteophytes, herniating discs, facet joint hypertrophy, ossified posterior longitudinal ligament and thickened ligamentum flavum 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 (which is located on the outermost surface of the cord) with resultant ischemic myelopathy [13]. Crandall and colleagues [8] described five distinct cord syndromes representing relatively advanced disease, as follows:

- Brown-Sequard 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 (myeloradiculopathy), 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 clearly defined early in the course of cervical myelopathy. Symptoms are often subtle at onset and recognition is difficult. Hyperreflexia and extensor plantar responses (Babinski sign), minimal weakness in the lower extremities, and a subtle gait disturbance are common early signs [16]. Other patients may only experience mild foot numbness. These patients are often erroneously evaluated for peripheral polyneuropathy. The key to avoiding this all too common error is assessing for the presence or absence of ankle reflexes. While the presence of an Achilles reflex is a normal finding in most people, this response would not be expected in a person with a suspected large-fiber peripheral polyneuropathy. Distal lower extremity numbness in a patient with retained ankle reflexes may be suggestive of upper motor neuron pathology. Lhermitte’s sign may be present 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 [28].

9. What are the causes and treatments for non-radiating neck pain?

Once significant neurologic comorbidities can be ruled out as a cause of neck pain, the focus shifts to identifying the specific etiology of the localized neck pain. A number of conditions can present in this fashion including: neck strains, cervical facet syndrome and myofascial pain syndrome. It is often difficult to identify the precise instigator, as symptoms are often vaguely defined and overlap. In these patients, diagnostic testing is low yield. That said, after several weeks of unrelenting pain, diagnostic testing may prove necessary to rule out the unexpected. Acute, localized neck pain typically resolves within days or weeks. Evidence regarding the efficacy of individual interventions for neck pain is often contradictory because of poor quality trials and the tendency to combine therapies. Treatment of acute non-radiating neck pain is largely empiric and may include the following [15]:

- 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, non-steroidal anti-inflammatory drugs (NSAIDs), pain medication when necessary, and possibly muscle relaxants.
- Local application of heat or cold if either modality proves helpful to the patient while recognizing the lack of “science” to support either.

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 non-addictive analgesics, may lead to analgesic rebound pain. Given the increasingly convincing association between chronic neck pain and psychological comorbidities there may be a role for antidepressant medication, especially those known to have benefit in the treatment of neuropathic pain, such as duloxetine. To that same end, there is some recent evidence that cognitive-behavioral therapy (CBT) is better at pain reduction than other interventions for neck pain lasting more than three months [17]. Physical therapy and regular aerobic exercise is particularly important in management of these patients.

10. What if it's not just the neck? What associated conditions are important to be aware of?

There are a number of other conditions that may produce neck pain, but the generator may not be the cervical vertebrae or related structures. Herpes zoster can present with severe neck and/or occipital pain often within a C2-C4 dermatomal distribution. Keep in mind that the onset of pain can precede the rash eruption. Rheumatoid arthritis can produce neck pain, stiffness and impaired range of motion. In advanced cases it may erode the transverse ligament, allowing for atlantoaxial subluxation. If this goes undiagnosed, the dens may move anteriorly compressing the spinal cord producing a myelopathy and possibly respiratory failure. Ankylosing spondylitis can also produce neck pain and rarely, atlantoaxial subluxation. Any mechanical instability of the high cervical spine should be considered a surgical emergency. Locally invasive processes such as malignancy or infection should be considered under appropriate circumstances. The walls of cervical blood vessels contain

nociceptive nerve fibers, thus vertebral or carotid dissections may present with acute neck pain. For similar reasons, giant cell (temporal) arteritis or coronary artery ischemia may present as primary neck pain. Pain from the brachial plexus can often mimic pain in the spine. These disorders may be inflammatory (neurogenic amyotrophy), infiltrative (lymphoma), or compressive (Pancoast tumor). An examination that yields motor or sensory deficits affecting the upper extremity and not fitting a clear root distribution may need further investigation with contrast-enhanced MRI and electrodiagnostic studies. Lastly, pain from the shoulder joint can often radiate into the upper extremity in a “radicular” fashion. In this setting, there will be no abnormal findings on motor, sensory and reflex testing. Furthermore, passive movement of the shoulder may exacerbate symptoms.

11. Conclusion

New onset neck pain is a frequently encountered complaint in the primary care setting. Often pain begins sporadically, with no clearly identifiable precipitating event. While most cases are self-limited in their natural course, physicians must be able to efficiently and reliably recognize the patient whose symptoms are atypical and warrant further investigation. Any patient with neck pain needs a focused neurological assessment. Imaging and other diagnostic testing should be reserved to confirm neurological complications rather than search for them. As the landscape of modern medicine continues to shift, the ability to take an appropriate history and perform a competent physical examination will always be the foundation of good clinical medicine.

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