Jen Tooher

Pathophysiology of Cervical Radiculopathy and Current Research for Evaluation and Treatment of Symptoms

Introduction

Cervical radiculopathy (CR) describes pain and/or sensorimotor deficits from the neck down the upper back, shoulder, and arm, and it occurs as a result of cervical nerve root compression.[1,2] 90% of cervical radiculopathies are attributable to herniated nucleus pulposus (HNP) of the intervertebral disc or degenerative disc disease (DDD) and its' associated pathologies,[3] including spinal stenosis, spondylosis of facet joints, osteophyte formation, ligamentous thickening, and spondylolisthesis.[1-3] In the remaining 10% of cases, compression occurs from tumors, trauma, or instability.[4,5] For the purposes of this paper, discussion will focus on treatment and management of cervical radiculopathy as a result of HNP or DDD.

Approximately 30-50% of adults experience neck pain each year, with highest incidence in people working in hospitals or offices.[1] Neck pain has been categorized into 4 grades according to the 2010 Joint and Bone Task Force, and cervical radiculopathy fits into grades II or III.[6] Prevalence of cervical radiculopathy is 3.5 per 1,000, and it is more common in males.[3] Peak incidence of herniated discs occurs in the 30s and 40s, whereas spondylosis tends to be seen in adults over 55.[3] Significant pain and disability can result.[7]

In order to understand the pathology of cervical radiculopathy, a basic understanding of cervical spine anatomy is warranted. The cervical spine is composed of bone, cartilage, and nerves. Bone acts to transmit forces,[8] whereas cartilage attenuates compressive loads,[9] and nerves send signals between the brain and the body.[10] Seven cervical vertebrae with corresponding intervertebral discs and eight spinal nerves comprise the cervical spine.[2] The ligamentum flavum and laminae border the posterior

spinal canal; vertebral bodies and intervertebral discs border the spinal canal anteriorly.[1]

Spinal foramina form openings between the vertebrae where spinal nerves and blood vessels pass obliquely at each level (Figure 1- Appendix A.)[1,2] Cervical spinal nerves divide into dorsal and ventral primary rami to supply the posterior neck, or prevertebral, paraspinal muscles, and the brachial plexus, respectively.[1] Intervertebral foramina are funnel-shaped, with a narrow entrance closest to the spinal cord,[3] whereas nerve roots are largest in the central dural sac.[3] Both intervertebral foramina and the spinal canal are largest in the upper cervical spine and decrease in size until C7-T1, causing disc pathology or narrowing to be more pronounced in the middle-lower cervical spine; nerve root compression is most common between C5 and C7 vertebral levels.[3] Current research suggests the diameter of the cervical spinal canal changes biomechanics of cervical spine mobility and may predispose individuals to pathology. In one study of 295 subjects, congenital narrowing increased degeneration and cord compression in all cervical segments except C2-3. The group with congenitally smaller canals also had significantly higher segmental mobility at C4-5 and C6-7 and lower mobility at C3-4.[11] Based on the orientation of facet joints in the cervical spine, lateral bending and axial rotation are coupled motions in the cervical spine from C2-C7.[12] Coupled extension and rotation occur from C2-C5 while coupled flexion and rotation occur in C5-C7.[13]

Intervertebral discs attenuate compressive loads throughout the spine.[9] To effectively do this, discs are composed of an inner nucleus pulposus, a surrounding annulus fibrosus, and superior and inferior cartilaginous vertebral end plates.[9] The fibers in the annulus fibrosus are made up of type 1 collagen and arranged in alternating oblique layers to improve mobility and prevent excess tensile stress.[1,9] The nucleus pulposus is

made of primarily type 2 collagen[1] and functions using fluid dynamics: where the fluid is distributed evenly throughout the disc to attenuate focal areas of stress.[9] Type 2 collagen, proteoglycans, and hyaluronan components of the nucleus pulposus are hydrophilic and promote disc hydration. These minerals decrease with age, causing the discs to dry out and start to bulge.[1,14] Bone mass also decreases with age,[15] which can prompt ligamentum flavum thickening to compensate for instability that results from bone degeneration and subsequent facet joint hypermobility.[2,16] All these age-related factors contribute to HNP, spondylosis, and the narrowing of the cervical canal.[1]

The pathophysiology of cervical radiculopathy depends on the cause of the radicular symptoms. In the case of a herniated disc, the disc bulge, protrustion, or extrusion causes pain as degeneration begins in the second decade from repetitive strain on the posterolateral annulus.[9] Circumferential tears cause fissures and allow the nucleus pulposus to extrude.[1] Pain is thought to occur as proteoglycans and phospholipases from the nucleus pulposus initiate the inflammatory cascade, directly compress nerves, and create symptoms down the arm.[1,2] Inflammation and ischemia associated with root compression appear to be contributing causes of symptoms in cervical radiculopathy.[2,3] Spontaneous recovery occurs in weeks to months in many herniated discs, and the presence of interleukins, prostaglandins, and inflammatory cytokines like MCP and the fibronectin-aggrecan complex support the theory of inflammation.[3,17,18] Adults have modest lordosis of the cervical spine, but postural and age-related changes can decrease or reverse lordosis, increasing pressure on the anterior disc to produce posterior-lateral protrusion.[1] Non-traumatic disc herniation rarely occurs at more than one level, but spondylosis can involve multiple vertebral levels.[3]

DDD causes mechanical nerve root compression in the intervertebral foramina because disc height decreases with age, the foramina and ligamentum flava hypertrophy, and annular fibrous tissue decreases.[1,9] Decreased disc height increases motion at facet joints posteriorly, alters normal bony alignment, and increases strain on supporting ligaments.[1] Poor posture provides a feedforward mechanism that further aggravates DDD.[19] Degenerative changes are most common at C5-6, followed by C6-7.[1,2] Spondylotic radiculopathy occurs after disc degeneration begins, and can cause facet joint osteophyte formation because the mechanical behavior of the nucleus pulposus doesn't adequately distribute forces and focal areas of stress cause damage.[20] Osteophytes can compress nerves and cause patients with spondylotic radiculopathy to complain of multilevel or bilateral radiculopathy.[1]

Clinical Evaluation

Patient history is essential to understanding the cause of cervical radiculopathy and forming an appropriate treatment plan.[1] Signs and symptoms of cervical radiculopathy include insidious onset of pain, numbness, and/or tingling in the neck and upper extremity, and muscle weakness.[2] Pain is the presenting symptom in 70% of patients with radiculopathy, and can range from a dull ache to burning, electrical pain.[1] Pain often radiates to the medial border of the scapula, progressing down the arm and to parts of the hand.[1] Neck movements may increase pain, especially cervical extension,[2] and coughing and sneezing can increase tingling in the arm.[1] Neck pain is usually less severe than arm symptoms.[3] Atypical pain presentations include subscapular or chest pain.[3] Cervical extension, lateral bending, or rotation with axial loading narrow the intervertebral foramina and can aggravate acute herniations.[1] Acute disc problems

typically have quick onset of very severe pain, whereas chronic disc pathology and spondylosis usually have less severe, gradual onset of pain or numbness.[2] Risk factors associated with CR include smoking, axial load bearing, high-risk occupations, and prior lumbar radiculopathy.[1] Smoking is thought to increase risk of radiculopathy due to decreased vertebral blood supply, which accelerates ischemia.[3]

Weakness is more often associated with herniated discs than with spondylosis.[1] Myotomes relate to muscles innervated by one spinal nerve, whereas dermatomes relate to sensory distribution of a dorsal root.[21] Paresthesias or anesthesias occur as often as 91% of the time along the involved dermatomes, reflexes will be diminished 70% of the time, and muscle weakness can be present in a myotomal pattern up to 65% of the time.[1,3] Using information from myotomes, dermatomes, and decreased reflex tests help identify if and where cervical radiculopathy occurs (Table 1- Appendix B.)[1,2]

After the patient history, the objective portion of the clinical exam includes range of motion, strength and myotomes, palpation, dermatomes, reflexes, and special tests.[21] Patients should rate their pain using the Verbal Rating Scale (VRS) from 0-10 in all locations to quantify pain severity.[21] Cervical range of motion using a goniometer, along with descriptions for which motions are limited due to pain, provides objective information to understand impairments from cervical pathology.[6] A stiff neck and head tilt away from the side of injury are common in cervical radiculopathy.[19] Decreased active range of motion (AROM) in lateral bending and rotation can occur towards or away from the side of the injury. If the motion is limited on the same side of the injury, it is likely because the nerve root is compressed at the formina. Limited motion on the opposite side is usually because of increased pressure on the nerve from a herniated disc.[1]

Myotome testing of the upper extremities denotes if muscle weakness exists from radiculopathy. If weakness is found, comparison between sides is essential.[1] Palpation of spinous processes and articular pillars is reliable and helps to rule out excess movement of vertebrae.[6] With muscle palpation, a clinician should find tenderness, tightness, and potential hypertonicity on ipsilateral cervical paraspinals and possibly along the muscle where radicular symptoms present.[1] Sensory exams should include both light touch and pinprick, and should be done along a dermatomal distribution to identify if anesthesia or hyperesthesia exists.[3] Pinprick sensation is more reliable than light touch in people with mild radiculopathy.[1] Asymmetry of C5, C6, and C7 deep tendon reflexes should be assessed, as they are suggestive of radiculopathy.[7] Lhermitte's sign, tested by flexing the neck and asking about patient symptoms, may cause electrical pain down the spine and possibly into the extremities for people with HNP, cervical cord involvement, severe spondylosis with myelopathy, cervical tumor, and multiple sclerosis (Figure 2- Appendix A.)[19] However, Babinski and Hoffman's should both be negative in CR; if these tests are positive, cervical myelopathy or neurological processes must be ruled out.[1]

Five provocative tests can help identify cervical radiculopathy. These tests include Spurling's test, the valsalva maneuver, the shoulder abductor sign, the upper limb tension test, and neck distraction.[2] Spurling's, shoulder abductor sign, and neck distraction have high specificity but lower sensitivity. Spurling's test is statistically best at identifying cervical radiculopathy (Figure 3- Appendix A.)[1] Based on psychometric properties, these tests should be used to confirm a diagnosis of cervical radiculopathy, not to screen for it. The upper limb tension test has 97% sensitivity, so it is the best screening tool for cervical radiculopathy (Figure 4- Appendix A.)[7] A clinical prediction rule (CPR)

for cervical radiculopathy has been developed, with 4 components: positive Spurling's test, positive neck distraction test, positive upper limb tension test, and less than 60 degrees of cervical range of motion to the involved side.[22] 3 out of 4 findings has 94% specificity for cervical radiculopathy, with a positive likelihood ratio of 6.1.[7] If all of the 4 components are found in a patient, there is 99% specificity and a positive likelihood ratio of 30.3.[22] The valsalva maneuver has insufficient psychometric testing to validate its use, but may be practical in clinical settings to screen for cervical radiculopathy.[2,3]

Differential Diagnosis

According to the APTA Guide to PT Practice, cervical radiculopathy fits into Pattern 4F: Impaired Joint Mobility, Motor Function, Muscle Performance, Range of Motion, and Reflex Integrity Associated with Spinal Disorders.[23] Because of the range of symptoms associated with cervical radiculopathy, differential diagnoses need to be considered and ruled out before continuing with treatment for cervical radiculopathy. People with severe spondylosis or spondylolisthesis[14] can develop cervical myelopathy, which presents with radicular symptoms, gait changes, bowel or bladder dysfunction, sensory dysfunction or weakness in the lower extremities. These patients need immediate referral.[1] Spondylotic myelopathy is degeneration of spine with osteophyte formation that compresses the central canal. Spinal cord compression presenting with vascular insufficiency and direct mechanical pressure can cause radicular symptoms (ie- central cord syndrome) and serious, long-term effects if not treated promptly.[1]

Multiple sclerosis (MS) can present with a variety of symptoms, including unilateral or bilateral arm paresthesias or anesthesias.[24] Length of sensory changes,

positive upper motor neuron tests (clonus, Babinski, Hoffman's), and a comprehensive history help determine if referral to neurologist is necessary to rule out MS.[25]

Carpal tunnel syndrome (CTS) presents similar to cervical radiculopathy from C6 nerve root compression, but patients with CTS often have increased pain in the morning and abductor pollicis brevis and thenar eminenance weakness. Spurling's maneuver can rule in cervical radiculopathy and rule out CTS, whereas Tinel's and wrist flexion will rule in CTS.[2] For a more comprehensive look at other diagnoses that present with neck, shoulder, and/or arm pain, weakness, or sensory changes, see Table 2 in Appendix B.

Imaging

Imaging is not indicated with grade I or II neck pain, it may be necessary in grade III neck pain.[6] Since most of the patients physical therapists see with CR will likely fit into grades II or III, understanding imaging options helps guide referral decisions. Disc herniations and stenosis are best identified using MRI (93% predictive),[2,26] and can be difficult to capture on x-rays.[27] However, MRI is not supported as a necessary routine screening tool for radiculopathy.[6] X-rays show obvious spondolytic changes or instability.[2,27] CT scans are good if an individual is trying to identify bony ossification, such as ossification of cervical ligaments or osteophytes of foramina.[2] CT with myelography combines bone and nerve root, but it is invasive and doesn't show soft tissue, making it more useful for foraminal narrowing and stenosis rather than disc pathology.[28] Electromyography (EMG) or nerve conduction studies (NCS) are useful if double crush injury is suspected. It will show decreased nerve conduction if pathology exists.[2,3,29] Unfortunately, predictive capacity might be as low as 42% for compression.[2] These tests help localize the level of the injury if other imaging has been inconclusive.[29]

Treatment

75-90% patients will have improved symptoms and pain with non-operative management of cervical radiculopathy, so understanding current evidence-based treatment options will help physical therapists effectively manage patients with this condition.[3] Urgent surgery is only indicated in small portion of patients, mostly in patients with progressive weakness or extreme, unremitting pain.[2]

Evidence regarding physical therapy interventions for cervical radiculopathy is limited.[3,7] However, most studies have shown at least moderate effects of multi-modal treatment for cervical radiculopathy.[2,30] The general focus of exercise interventions should be to restore function and decrease pain.[1] Stretching tight scalenes, trapezius, and pectoralis muscles improves postural alignment and decreases abnormal stresses on vertebral bodies.[31] Strengthening scapular stabilizers (rhomboids, middle trapezius) and deep neck muscles (longus colli, longus capitis, rectus capitis anterior) sustains improved posture and provides extrinsic support to the cervical spine.[7] Press ups and push ups with a plus at both low and high intensities are good exercises to target serratus anterior and lower trapezius muscles while keeping the upper trapezius quiet.[31] Mobilization has been shown to decreased pain and increase range of motion.

Other interventions include range of motion exercises, neck relaxation techniques, superficial heat, manual traction, TENS, cervical pillow, massage, and ultrasound.[1,3] Literature suggests a combination of range of motion, heat, postural education, and neck relaxation can successfully improve patient symptoms after six weeks.[2] Modalities (ice, heat, electrical stimulation) alone don't significantly improve symptoms but may control pain and inflammation, so can be used with other interventions to maximize treatment

effects.[1] PT has shown improved pain and sensorimotor symptoms as much as surgery or cervical collars 16 months after intervention, and it is a more cost effective intervention than surgery.[3] Strength exercises are more effective than aerobic exercises.[1]

Effective intervention conclusions are limited for individuals with cervical radiculopathy because most studies identifying conservative exercise interventions for neck pain do not separate CR from other neck diagnoses.[7] Cervical traction may provide temporary relief, reduce pain, and improve radicular symptoms for people with CR.[32] High velocity-low amplitude (HVLA) manipulation has also been studied for cervical radiculopathy with mixed results; some studies suggest improved symptoms for as long as 9 months with no complications while others suggest complications of worsening radiculopathy.[2] The proposed mechanism for improvements involves relaxing resting paraspinal muscle activity to provide relief.[33] Many studies addressing effectiveness of manipulation for neck pain exclude people with nerve root compression or radiating symptoms, suggesting the diagnosis is a contraindication to this type of treatment.[34-36] Manipulation with cervical radiculopathy has increased risk of vertebral dissection and spinal cord compression. Since superior results for manipulation haven't been found when compared to mobilization, mobilization is safer and is better technique to use for people with CR.[3,37] Neural dynamic mobilizations, cervical mobilizations, and Maitland mobilizations may provide more benefits than other types of mobilizations.[38]

A treatment-based classification system has been developed for neck pain to categorize and individualize treatments for subgroups of patients. Child's classification system categorizes cervical radiculopathy into the centralization classification. Centralization focuses on repeated active or passive neck movements, traction, and

exercises to decrease peripheralization of symptoms.[32] In order to correctly classify patients and identify exercises that will benefit people with cervical radiculopathy, identifying the pathology is essential.[3] If the cervical radiculopathy is due to a herniated disc, targeted exercises include chin retractions and repeated extension or oblique lateral extension.[32] Studies of the McKenzie method have shown moderate to strong improvements in radicular symptoms when used appropriately.[7]

If cervical radiculopathy is the result of spondylosis, the focus of treatment should be activity modification, neck immobilization, intermittent cervical traction, and isometric exercises to improve symptoms.[19] Semi-hard cervical collars control pain by minimizing motion in acute stages but shouldn't be used for long periods. Immobilization causes paraspinal atrophy and decreases intrinsic cervical stability.[3] Pain control should be the initial goal of treatment for these patients, and then strengthening and postural reeducation can begin.[19] Gentle, active interventions have found better results.[2]

Additional Treatments

CR symptoms don't improve significantly with NSAIDs or oral corticosteroids, but NSAIDs are commonly used in acute stages to control pain.[3] An artificial soluble TNF receptor significantly decreases TNF-alpha, a cytokine that causes inflammation, and may be a future solution to prevent inflammation and decrease spine pathology.[17] Epidural steroid injections are another conservative treatment option for people with cervical radiculopathy.[1,3,39,40] Epidural steroid injections might be most effective for patients who have MRI findings of central canal stenosis (approximately 60% effective.)[1,40] Studies suggest steroids provide short-term relief, but evidence about long-term impact of steroids is unclear.[2,6] Complications are also more common in epidural

steroid injections, especially transforaminal injections.[2] Interlaminar injections might be safer.[1] One cohort study of transforaminal steroid injections found satisfactory recovery in patients with CR, but another randomized controlled trial of transforaminal steroid injections did not show positive effects compared to a control.[3] Low-dose anticonvulsants or antidepressants might provide some short-term relief.[1]

Surgery has been shown to be useful in some cases with cervical radiculopathy.[6] Surgical options include anterior or posterior discectomy, arthroplasty, disc fusion, foramenotomy, and decompression.[3] Fast and long-term results have been seen with anterior cervical discectomy with and without fusion. Anterior cervical discectomy with fusion provides relief in approximately 90% of patients with CR.[41,42] Arthroplasty has shown similar results as anterior discectomy and improved results compared to cervical fusion when done on patients with cervical radiculopathy.[6,43] Accelerated spinal degenerative at adjacent levels has been seen in patients with a history of cervical fusion, so radicular symptoms may recur in patients after surgical fusion.[3,44]

Conclusion

Cervical radiculopathy is a clinical syndrome marked by neck, upper extremity pain, and possible sensorimotor deficits.[22] People with CR often have difficulty with ADLs and can lose time from work and social obligations.[7] The most difficult aspect of clinical diagnosis is identifying the vertebral level where compression is occurring.[3] Using anatomy, pathology, clinical, and treatment knowledge discussed in this paper, PTs can more effectively identify, localize, and treat cervical radiculopathy efficiently and effectively to decrease disability that often occurs as a result of cervical radiculopathy.

APPENDIX A.

Figure 1. C5 vertebra of the cervical spine

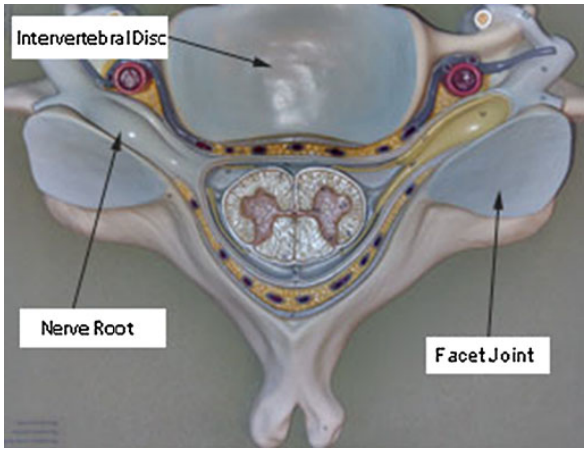
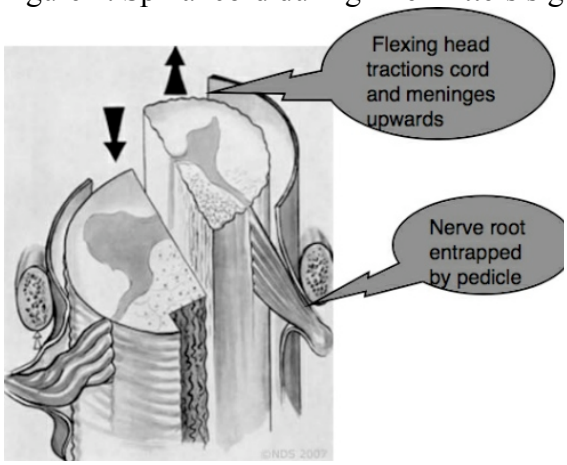


Fig. 1. Axial representation of C5 vertebra looking cephalad to caudal

Figure 2. Spinal cord during Lhermitte's sign

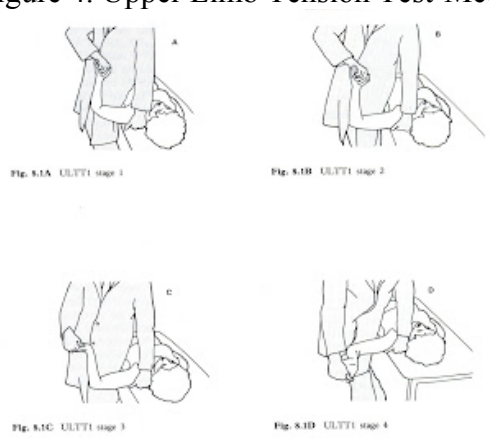


To Administer the Test:
Have patient sit and bend their neck forward. Complaints of electrical, shooting pain that radiates from their neck down their spine or to their toes denotes a positive test.

Figure 3. Spurling's Test[2,7]

To Administer the Test:
Rotate the patient's head towards the radicular symptoms, extend the neck slightly, and apply an axial load to gently compress the neck. Increased radicular symptoms denote a positive test.

Figure 4. Upper Limb Tension Test-Median Nerve[22]



To Administer the Test:
Depress and stabilize the scapula, move affected arm into 110 degrees of shoulder abduction. Then supinate the forearm, add ulnar deviation and wrist and finger extension. Externally rotate the shoulder, and then extend the elbow. Laterally bend the head away from the arm being tested. Monitor symptoms after each change before moving on to the next position. A test is positive if radicular symptoms worsen.

APPENDIX B.

Table 1.
Pain Patterns, Myotomes, Dermatomes, & Reflexes associated with Cervical Radiculopathy[2,21]

	Pain Pattern	Myotomes	Dermatomes	Reflexes
C2	Occiput, eyes	N/A	Lateral occipital protuberance	
C3	Neck, Trapezius	N/A	Supraclavicular fossa, midclavicular line	
C4	Neck, Trapezius	Shoulder Elevation	Acromioclavicular joint	
C5	Shoulder, lateral UE	Shoulder abduction or external rotation	Lateral antecubital fossa	Brachioradialis
C6	Lateral forearm, first 2 fingers	Elbow flexion or wrist extension	1st digit (thumb)	Biceps
C7	Posterior forearm, third finger	Elbow extension or wrist flexion	3rd digit (middle finger)	Triceps
C8	Medial forearm, fourth and fifth finger	Thumb extension or ulnar deviation	Medial antecubital fossa	

Table 2. Differential Diagnosis List for Cervical Radiculopathy[2,3]

Acromioclavicular pathology
Acute posterior cervical strain
Adhesive capsulitis
Aortic disease
Arachnoiditis
Arteriovenous malformation
Back pain
Bicipital tendonitis- rotator cuff tears, lateral epicondylitis
Brainstem syndromes
Calcareous tendonitis
Carpal tunnel syndrome
Cervical disk syndromes
Cervical lymphadenitis
Cervical rib
Congenital spinal lesion
Diskitis
Double crush syndrome
Elbow Epicondylitis
Epidural absess
Extrinsic neoplaia (usually metatastic)
Facet joint pain
Frozen shoulder syndromes
Glenohumeral arthritis
Gout (infrequently)
Heart Disease
Hyperabduction syndrome
Intervertebral osteoarthritis
Idiopathic brachial plexopathy
Intrinsic neoplasia
Myocardial ischemic pain
Nerve injuries
Occipital neuralgia
Osteomyelities
Osteoarthritis of apophyseal joints
Paget's disease
Pancoast's tumor
Parsonage-Turner syndrome
Pharyngeal infections
Postural disorders
Psychogenic disorders
Rheumatoid arthritis
Rib-clavicle compression
Rotator cuff injury
Scale muscle tightness
Septic arthritis
Spinal cord tumors
Sternocleidomastoid tendinitis
Subacromial bursitis
Synovial cysts
Tabes dorsalis
Thoracic disk
Thoracic outlet syndrome
Tropical spastic paraparesis
Ulnar nerve entrapment

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