

INTERVENTIONS IN CHRONIC PAIN MANAGEMENT

Interventions in Chronic Pain Management. 2. Diagnosis of Cervical and Thoracic Pain Syndromes

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ABSTRACT. Freedman MK, Overton EA, Saulino MF, Holding MY, Kornbluth ID. Interventions in chronic pain management. 2. Diagnosis of cervical and thoracic pain syndromes. *Arch Phys Med Rehabil* 2008;89(3 Suppl 1):S41-6.

This self-directed learning module highlights approaches to the investigation of common cervical and thoracic conditions. It is part of the chapter on chronic pain management in the Self-Directed Physiatric Education Program for practitioners in physical medicine and rehabilitation. The differential diagnosis of cervical and thoracic syndromes as well as cervicogenic headache is reviewed. The need for a comprehensive history and physical examination is emphasized. Indications for diagnostic tests including magnetic resonance imaging, computed tomography scan, bone scan, diskography, radiographs, diagnostic injections, and electrodiagnostic studies are discussed with the idea that testing should be performed and interpreted with the specific clinical presentation in mind.

Overall Article Objective: To discuss the differential diagnoses for and investigation of common cervical and thoracic conditions and cervicogenic headache.

Key Words: Headache; Pain; Rehabilitation.

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2.1 Clinical Activity: To differentiate the investigation for cervical radiculopathy from other upper-extremity syndromes in a 35-year-old patient with trapezius pain and radiation of pain into the upper extremity and scapula.

INNERVATED STRUCTURES in the vertebral column (facet, outer annulus of the intervertebral disk, dura mater, anterior or posterior longitudinal ligament, or paravertebral muscles) can cause localized or radiating pain. Differential diagnoses include cervical radiculopathy, cervical facet syndrome, cervical spondylosis, cervical stenosis, myelopathy, cervical herniated nucleus pulposus (HNP), myofascial pain or fibromyalgia, shoulder pathology including rotator cuff or joint pathology, scapular and humeral fractures, or visceral disease. Peripheral nerve lesions that can lead to cervical or trapezius pain include brachial plexopathy, dorsal scapular, suprascapular, spinal accessory, and axillary and long thoracic nerve lesions.

Cervical radiculopathy is a dysfunction of the cervical spinal nerve root caused by irritation or compression. The cervical nerve root may be compressed by an HNP, foraminal stenosis from osteophyte formation or facet hypertrophy, tumor, or infection. Inflammatory factors include phospholipase A₂, prostaglandin E₂, interleukin-6, matrix metalloproteinases, and nitric oxide. Cervical radiculopathies account for 5% to 36% of all radiculopathies.¹ The incidence of cervical radiculopathy by level is as follows: 70% originate at C7, 9% to 25% at C6, 4% to 10% at C8, and 2% at C5.²

The examination should include observation and inspection of the trapezius and scapular musculature as well as palpation; range of motion (ROM) of the cervical spine; neurologic examination including testing of strength; deep tendon reflexes; and a sensory examination for pain, vibration, and light touch. Provocative maneuvers such as the Spurling test may help to reproduce symptoms arising from the cervical spine and differentiate radiculopathy from peripheral joint or nerve problems. Shoulder ROM must be evaluated. Furthermore, provocative shoulder testing should be evaluated to see if all or a portion of the pain is related to the cervical spine or the shoulder. The biceps tendon, subdeltoid bursa, and acromioclavicular joint must be palpated to look for secondary shoulder pathology. The medial and lateral epicondyle must be palpated; wrist flexors and extensors must be activated and stretched to evaluate for medial or lateral epicondylitis that can be confused with cervical radiculopathy or weak musculature associated with a peripheral nerve lesion. Percussion made over the median or ulnar nerve may be done to try to provoke the Tinel sign, which is a tingling or painful sensation in the distribution of the specific nerve that is being evaluated if that nerve is producing symptoms. This may help to rule out peripheral nerve lesions, which can be confused with cervical radiculopathy. The lower-extremity neurologic examination should be performed to rule out myelopathy, and the cranial nerves should be evaluated for upper motoneuron pathology arising from the brain. Peripheral pulses in the upper extremities should be evaluated to rule out vascular insufficiency.

Magnetic resonance imaging (MRI) is the study of choice to evaluate patients for a disk herniation; however, the presence of a disk herniation may or may not be significant. Boden et al² found MRI abnormalities in 19% of asymptomatic patients. Fourteen percent of patients who are younger than age 40 have MRI abnormalities. Twenty-eight percent of patients over age 40 have MRI abnormalities.²

Cervical provocative diskography remains a controversial diagnostic test for patients with axial pain when other diagnostic studies fail to identify the patient's pain generator. Provocative diskography may help to identify the painful disk, but the procedure is technically demanding; potential complications include diskitis, subdural hematoma, spinal cord injury, vascular injury, and prevertebral abscess.³

Electromyography is used to evaluate patients with suspected radiculopathies, plexopathies, peripheral neuropathies, or myopathies. The nerve conduction study evaluates the motor

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nerves by evoking a compound muscle action potential (CMAP), and sensory nerve action potential (SNAP) evaluates the sensory nerve. Patients with suspected radiculopathy rarely exhibit prolonged distal latency or reduced amplitudes in the SNAP. Lesions proximal to the cell body of the sensory nerve proximal to the dorsal root ganglia do not affect the SNAP; however, a lesion distal to the dorsal root ganglion will cause a decrement in the amplitude of the SNAP. In a suspected plexopathy, one should evaluate whether the sensory nerve emanates from a different trunk or cord to separate nerve root from plexus disease. Unlike the sensory nerve, the motor nerve's cell bodies (anterior horn cells) are located in the gray matter of the ventral horn; therefore, the amplitude of the CMAP may be reduced if axonal degeneration has occurred with damage to the nerve root or distal to the nerve root. The sensitivity of needle electromyography to diagnose cervical radiculopathy is 61% to 67% compared with the criterion standards of clinical evaluation, myelogram, and intraoperative impression.⁴

In cervical spondylosis, the intervertebral disk, facet joints, and ligamentous structures degenerate, a situation that leads to central and foraminal stenosis. Predisposing factors include age, trauma, work activities, and family history. Patients may exhibit signs of myelopathy and/or radiculopathy. In addition, pain may be referred to the trapezius, base of skull, or shoulders. Acquired cervical stenosis may develop as a result of osteophyte formation, disk protrusion, ligamentum hypertrophy, or facet hypertrophy. Congenital cervical stenosis is most often associated with short pedicles.

Patients diagnosed with central cervical stenosis may exhibit neurologic changes including gait disturbance, bowel or bladder dysfunction, impotence, hand weakness, leg weakness, spasticity, clonus, or hyperreflexia. The diameter of the spinal canal in the cervical spine ranges from 15 to 25mm with a mean of 17mm,^{5,6} whereas the average cervical spinal cord diameter ranges from 5 to 11.5mm with a mean of 10mm.⁷ A cervical spinal canal less than 13mm in diameter is considered stenotic,⁸ and a cervical canal less than 10mm is an absolute radiographic indicator of cervical spinal stenosis.⁹

Cervical facet syndrome is often characterized by neck, head, shoulder, and proximal upper-extremity pain and is associated with nondermatomal pattern. Aprill and Bogduk¹⁰ reported the prevalence of cervical zygapophyseal joint pain with intractable neck pain to be between 25% and 63%.

Referral patterns of the facet joints, which are innervated by the medial branches of the dorsal rami, were mapped out initially by Dwyer et al¹¹ and later Aprill et al¹² who anesthetized the medial branch above and below the symptomatic joint. Dwyer showed that C2-3 facet disease provoked occipital head pain; C4-5, C5-6, and C6-7 provoked shoulder pain; and C7-T1 provoked intrascapular pain. Windsor et al¹³ showed that stimulation of each medial branch created distinct referral patterns that are different from the mapping of the facet joints. The criterion standard for the diagnosis of pain generated by the zygapophyseal joint is diagnostic injection to the zygapophyseal joint or to the medial branch of the dorsal root since history, physical and imaging studies are unreliable.

Intra-articular injections are technically challenging in proportion to posttraumatic arthropathy, age, and surgical changes. An increased risk of false-positive results exists if the patient has extra-articular leakage into the interlaminar space or the interspinous, cervical epidural, or paraextradural space. Therefore, it is important that the local anesthetic always reaches the target nerve but does not affect any other diagnostically important structures. The patient must have between 50% and 80% pain relief with provocative pain maneuvers on 2 different

occasions in order to diagnose the facet as the pain generator. Single uncontrolled injections carry a 27% false-positive rate.¹⁴ Comparative injection considerations include placebo versus local anesthetic or short-acting versus long-acting anesthetic. Comparative blocks are more specific but less sensitive than placebo-controlled blocks.¹⁵

Myofascial pain is often defined as a regional pain syndrome of soft-tissue origin. Patients with suspected myofascial pain may experience a hyperirritable spot in the skeletal muscle, which is classically defined as a trigger point. Active trigger points produce clinical complaints when digitally compressed, whereas latent trigger points do not produce spontaneous pain but can produce other effects characteristic of trigger points. Trigger points may refer pain in a peripheral, central, or local referral pattern.

The diagnosis of fibromyalgia is based on a history of widespread pain, defined as pain present bilaterally, in the upper and lower body, and in the axial skeleton. Furthermore, applying pressure to the specific tender point sites should reveal excessive tenderness at 11 of those 18 sites. By definition, these tender points become painful at 4kg of pressure.¹⁶ It is reported that 72% of patients with fibromyalgia have active trigger points and that 20% of patients with myofascial pain syndrome also have fibromyalgia.¹⁷ Two other criteria for the diagnosis of fibromyalgia are sleep disturbance and fatigue. Minor symptoms include paresthesias, anxiety, headache, and irritable bowel syndrome.

There are no universally accepted tests that are diagnostic for the identification of a trigger point. Several studies have investigated the use of electromyography as a diagnostic test, but the results remain controversial. Both Weeks and Travell¹⁸ and Hubbard and Berkoff¹⁹ identified high-frequency potentials in trigger points while the remainder of the muscle was electrically silent. Simons et al²⁰ identified high-amplitude endplate spike potentials in addition to low-voltage endplate potentials after increasing the amplification 5-fold and the sweep speed 10-fold. Electromyographers often identify these same findings as normal endplate potentials or endplate noise.

2.2 Clinical Activity: To investigate the reason for burning midthoracic pain that is followed by a rash in a 55-year-old woman.

History and Physical Examination

The intensity, quality, and distribution of the pain should be documented for diagnostic purposes and to track the patient's response to therapeutic interventions. Provoking and alleviating factors should be assessed. Bowel and bladder incontinence should be investigated. After an acute traumatic injury to the spinal cord or cauda equina, the patient may present with constipation or urinary retention. A patient with an upper motor lesion may be in "spinal shock" initially postinjury, and spasticity may subsequently develop. Thus, initial incontinence is often from overflow. A thorough review of systems should be performed to rule out underlying visceral pathology. Fever raises concerns about vertebral osteomyelitis or visceral infection. Unexplained weight loss, nocturnal pain, history of previous malignancy, and age greater than 50 years should make malignancy a consideration.²¹ Pain from visceral pathology can present as thoracic pain or perhaps have ramifications on specific treatments even when the pain is from another source. Psychiatric disease, as well as substance abuse, must be noted and treated concurrently with organic disease and does not preclude secondary pathology. Intravenous drug use or history of recent skin or urinary infection may be risk factors for spinal

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