Neck Pain, Cervical Radiculopathy, and Cervical Myelopathy: Pathophysiology, Natural History, and Clinical Evaluation

Raj Rao


This information is current as of March 27, 2006

**Reprints and Permissions**

Click here to [order reprints or request permission](http://jbjs.org) to use material from this article, or locate the article citation on [jbjs.org](http://jbjs.org) and click on the [Reprints and Permissions] link.

**Publisher Information**

The Journal of Bone and Joint Surgery  
20 Pickering Street, Needham, MA 02492-3157  
[www.jbjs.org](http://www.jbjs.org)
Selected Instructional Course Lectures
The American Academy of Orthopaedic Surgeons

Printed with permission of the American Academy of Orthopaedic Surgeons. This article, as well as other lectures presented at the Academy’s Annual Meeting, will be available in March 2003 in Instructional Course Lectures, Volume 52. The complete volume can be ordered online at www.aaos.org, or by calling 800-626-6726 (8 A.M.-5 P.M., Central time).

DONALD C. FERLIC
EDITOR, VOL. 52

COMMITTEE
DAVID L. HELFET
CHAIRMAN
JAMES H. BEATY
DONALD C. FERLIC
TERRY R. LIGHT
VINCENT D. PELLEGRINI JR.

EX-OFFICIO
DEMPSEY S. SPRINGFIELD
DEPUTY EDITOR OF THE JOURNAL OF BONE AND JOINT SURGERY
FOR INSTRUCTIONAL COURSE LECTURES
JAMES D. HECKMAN
EDITOR-IN-CHIEF,
THE JOURNAL OF BONE AND JOINT SURGERY
Cervical spondylosis is a common and occasionally disabling condition, occurring as a natural consequence of aging in the vast majority of the adult population. A clinical approach to symptomatic cervical spondylosis can be simplified by dividing the findings at presentation into the categories of axial neck pain, radiculopathy, myelopathy, or some combination of these three. While the pathogenesis of radiculopathy and myelopathy in cervical spondylosis is better understood, the source of neck pain remains controversial. The aim of this lecture is to review the pathophysiology and natural history of each of these conditions and to describe the pertinent clinical features of cervical disc pathology.

**Pathophysiology**

**Neck Pain**

In a substantial number of patients, axial neck pain is a result of muscular or ligamentous factors related to posture, poor ergonomics, stress, and/or chronic muscle fatigue. Neck muscle pain can develop secondarily as a result of postural adaptations to a primary source of pain in the shoulder, the craniovertebral junction, or the temporomandibular joint. The pathophysiology of this pain process in the involved muscles is unclear. Patients with chronic myofascial pain have been shown to have a lower level of high-energy phosphates in the involved muscle tissue. It is unclear whether this causes the pain or is a result of the pain. Unencapsulated free nerve endings in muscle serve as chemonociceptive and mechanonociceptive units. Chemonociceptive nerve endings may respond to metabolites that accumulate during anaerobic metabolism in fatigued muscle, or they may respond to non-neurogenic pain mediators released by injury or ischemia, such as bradykinin, histamine, serotonin, and potassium ions. Mechanonociceptive nerve endings respond to stretch or pressure. Sensitization of these nerve endings may be a primary source of muscle pain.

Attributing axial neck pain to degenerative changes in the cervical discs or facet joints is a source of controversy, primarily because of the ubiquitous nature of such changes in the spine. Nevertheless, it does appear that cervical discs and facet joints can be pain generators. Nerve fibers and nerve endings found in the peripheral portions of the disc offer a possible mechanism by which degenerated cervical discs can produce pain directly. The disc is innervated by the sinuvertebral nerve, formed by branches from the ventral nerve root and the sympathetic plexus (Fig. 1). Once formed, the nerve turns back into the intervertebral foramen along the posterior aspect of the disc, supplying portions of the annulus, the posterior longitudinal ligament, the periosteum of the vertebral body and pedicle, and the adjacent epidural veins. A recent review of the findings of cervical discography performed over a twelve-year period suggested that reliable patterns of pain are produced by stimulation of each cervical disc (Fig. 2). The authors reported a high percentage of patients in whom multiple discs were concurrently responsible for axial neck pain.

The facet joint is also recognized as a source of axial neck pain. Provocative injections into the facet joints in asymptomatic volunteers produce a reproducible pattern of axial neck pain and pain in the shoulder girdle.
Neck Pain, Cervical Radiculopathy, and Cervical Myelopathy

(Fig. 3). This pattern of facet-joint-induced pain can be accurately treated by anesthetic injections into the facet joint, or by blocking the dorsal primary rami, further suggesting that the facet joints do indeed play a role in the development of axial neck pain.

Patients with degenerative arthritis in the upper cervical joints can present with severe suboccipital pain that radiates down into the neck or to the back of the ear. Injection of the atlantooccipital and atlantoaxial joints results in a reproducible pain pattern in this region. Wächli et al. reported unilateral headaches and atypical facial pain as a result of degenerative changes at the second and third cervical level. In some patients, suboccipital headaches are presumed to be a result of irritation of the greater occipital nerve, which originates from the posterior rami at the second, third, and fourth cervical levels. Another potential source of suboccipital pain is the sinuvertebral nerves ascending cephalad to innervate the atlantoaxial ligaments, the meningeal membranes, and the dura mater of the upper cervical cord and posterior cranial fossa.

Cervical Radiculopathy and Myelopathy

Neurologic symptoms in cervical spondylosis are the result of a cascade of degenerative changes that most likely begin at the cervical disc. Age-related changes in the chemical composition of the nucleus pulposus and annulus fibrosus result in a progressive loss of their viscoelastic properties. The disc loses height and bulges posteriorly into the canal. With this loss of height, the vertebral bodies drift toward one another. Posteriorly, there is infolding of the ligamentum flavum and facet joint capsule, causing a decrease in canal and foraminal dimensions. Osteophytes form around the disc margins and at the uncovertebral and facet joints. The posteriorly protruded disc material, osteophytes, or thickened soft tissue within the canal or foramen results in extrinsic pressure on the nerve root or spinal cord.

Mechanical distortion of the nerve root may lead to motor weakness or sensory deficits. The exact pathogenesis of radicular pain is unclear, but it is generally thought that, in addition to the compression, an inflammatory response of some kind is necessary for pain to develop. Within the compressed nerve root intrinsic blood vessels show increased permeability, which secondarily results in edema of the nerve root. Chronic edema and fibrosis within the nerve root can alter the re-

**TABLE I Chemical Mediators of Spinal Pain**

<table>
<thead>
<tr>
<th>Neurogenic</th>
<th>Non-Neurogenic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substance P</td>
<td>Bradykinin</td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Serotonin</td>
</tr>
<tr>
<td>Cholecystokinin-like substance</td>
<td>Histamine</td>
</tr>
<tr>
<td>Vasoactive intestinal peptide</td>
<td>Acetylcholine</td>
</tr>
<tr>
<td>Calcitonin gene-related peptide</td>
<td>Prostaglandin E1</td>
</tr>
<tr>
<td>Gastrin-releasing peptide</td>
<td>Prostaglandin E2</td>
</tr>
<tr>
<td>Dynorphin</td>
<td>Leukotrienes</td>
</tr>
<tr>
<td>Enkephalin</td>
<td>DiHETE</td>
</tr>
<tr>
<td>Gelatin</td>
<td></td>
</tr>
<tr>
<td>Neurotensin</td>
<td></td>
</tr>
<tr>
<td>Angiotensin II</td>
<td></td>
</tr>
</tbody>
</table>
response threshold and increase the sensitivity of the nerve root to pain. Neurogenic chemical mediators of pain released from the cell bodies of the sensory neurons and non-neurogenic mediators released from disc tissue may play a role in initiating and perpetuating this inflammatory response (Table I). The dorsal root ganglion has been implicated in the pathogenesis of radicular pain. Prolonged discharges originate from the cell bodies of the dorsal root ganglion as a result of brief pressure. In addition to the chemicals produced by the cell bodies of the dorsal root ganglion, the membrane surrounding the dorsal root ganglion is more permeable than that around the nerve root, allowing a more florid local inflammatory response.

Certain positions of the arm may decrease stress within the nerve root and relieve radicular pain. Davidson et al. described the shoulder abduction sign—i.e., relief of severe radicular pain when the patient rests the hand on the top of the head (Table I). Davidson et al. theorized that, in addition to decreasing tension within the nerve root, this position may lift the sensory root or dorsal root ganglion directly cephalad or lateral to the source of compression, and decompression of epidural veins may contribute to pain relief.
Cervical spondylotic myelopathy is the manifestation of long-tract signs resulting from a decrease in the space available for the cervical spinal cord. In addition to the spondylotic processes that contribute to the extrinsic pressure (Fig. 4), certain factors are thought to be important in the development of myelopathy; these include the anterior-posterior diameter of the spinal canal, dynamic cord compression, dynamic changes in the intrinsic morphology of the spinal cord, and the vascular supply of the spinal cord.

A congenital decrease in the anterior-posterior diameter of the spinal canal can play a role in the development of cervical myelopathy. The anterior-posterior diameter of the subaxial spine in normal adults measures 17 to 18 mm, and the diameter of the spinal cord is approximately 10 mm in this region. Individuals with an anterior-posterior diameter of the spinal canal of <13 mm are considered to have congenital cervical stenosis. There is a strong association between flattening of the cord within the narrowed spinal canal and the development of cervical myelopathy. Penning et al.13 believed that symptoms of cord compression occurred when the transverse area of the cord was <60 mm². Houser et al. thought that the shape and degree of flattening of the spinal cord could be an indicator of neurologic deficit; 98% of their patients with severe stenosis and a banana-shaped spinal cord had clinical evidence of myelopathy.14 Ono et al. described an anterior-posterior cord-compression ratio that was calculated by dividing the anterior-posterior diameter of the cord by the transverse diameter of the cord15 (Fig. 5). Patients with substantial flattening of the cord, suggested by an anterior-posterior ratio of <0.40 tended to have worse neurologic function. Ogino et al. thought that an increase in this ratio to ≥0.40 or an increase in the transverse area to >40 mm² was a strong predictor of recovery following surgery.16

Dynamic factors in the cervical spinal column affect the degree of cord compression. Hyperextension narrows the spinal canal by shingling the laminae and buckling the ligamentum flavum. Translation or angulation between vertebral bodies in flexion or extension can result in narrowing of the space available for the cord. Patients who do not have cord compression statically may compress the cord dynamically, leading to the development of myelopathic symptoms.17 Retrolisthesis of a vertebral body can result in pinching of the spinal cord between the inferior-posterior margin of a vertebral body and the superior edge of the lamina caudad to it (Fig. 6). This compression may be aggravated in extension, and it may be relieved in flexion as the retrolisthesis tends to reduce. Forward slippage of a vertebral body may cause compression of the spinal cord between the superior-posterior margin of the vertebral body below and the lamina above. This is aggravated by flexion of...
the spinal column. Hypermobility cephalad to a degenerated and stiffened segment is commonly seen at the third and fourth cervical levels in elderly individuals, and it may result in myelopathy. Morphologic changes also occur within the spinal cord itself with flexion and extension. Breig et al. showed that the spinal cord stretches with flexion of the cervical spine and shortens and thickens with extension. Thickening of the cord in extension makes it more susceptible to pressure from the infolded ligamentum flavum or lamina. In flexion, the stretched cord may be prone to higher intrinsic pressure if it is abutting against a disc or a vertebral body anteriorly.

In 1924, Barre was apparently the first to propose that vascular factors play a role in the development of cervical myelopathy. Acute or subacute compression of the spinal cord is thought to be pathognomonic in these patients. There is some experimental evidence to support a role for vascular factors in the pathogenesis of myelopathy. Cervical cord ischemia superimposed on compression of the cord resulted in dramatic neurologic findings in two separate experiments involving canines. The effects of compression and ischemia were thought to be additive and responsible for the clinical manifestation of myelopathy. In a separate study, obstruction of the peripial arterial plexus in dogs caused structural changes within the spinal cord. The classic study by Breig et al. showed that blood flow through the anterior spinal artery and anterior radicular arteries may be reduced when those vessels are tented over a disc or a vertebral body, but flow through the tortuous posterior spinal arteries is not substantially affected in this position. Vessels that are thought to be most prone to reduced flow are the transverse intramedullary arterioles arising from the anterior sulcal arteries. These vessels perfuse the gray matter and adjacent lateral columns.

Severe compression results in degenerative changes in the spinal cord. The central gray matter and the lateral columns show the most changes, with cystic cavitation, gliosis, and demyelination most prominent caudad to the site of compression. The posterior columns and posterolateral tracts show Wallerian degeneration cephalad to the site of compression. These irreversible changes may explain why some patients do not recover following decompressive surgery.

Natural History
There are few population-based studies on the prevalence of neck pain. A recent study of a Saskatchewan adult population showed that neck pain is more prevalent than commonly perceived: 66% of adults experienced neck pain during their lifetime, with 54% having experienced it during the past six months and 5% highly disabled by it. Another population study demonstrated a 9% point prevalence of neck and shoulder pain. The prevalence of neck pain appears to be higher in better educated individuals with a history of injury, headaches, or low-back pain.

DePalma et al. found that most patients with axial symptoms from cervical spondylosis do reasonably well. They reported that, following three months of nonoperative care, 21% of patients had complete relief of symptoms, 49% had partial relief, and 22% had no relief. Rothman and Rashbaum found that 23% of a similar group of patients remained partially or totally disabled at the end of five years. In the same study, they found no substantial difference between that group of nonoperatively treated patients and another group of patients with “predominantly” axial neck pain who had undergone surgery. They recommended nonoperative management for axial symptoms.

In 1963, Lees and Turner reported on the natural history and prognosis of cervical spondylosis. Fifty-one patients in their “non-myelopathic group” had pain in the neck, shoulders, arms, or hands. Patients were treated with a collar, exercises, traction, manipulation, and rest. Of ten patients who were followed for ten to nineteen years, three had no symptoms after the first few months of treatment, three continued to have mild symptoms, and four had more troublesome symptoms. Of forty-one patients who were followed for two to ten years, nineteen had no symptoms, twelve had intermittent symptoms, and ten had moderate disability. Myelopathic symptoms did not develop in any of the patients during the course of the nineteen-year follow-up period. It appears that, while approximately 45% of patients with non-myelopathic symptoms have good resolution of those symptoms shortly after onset, the remaining 55% continue to have minor or moderate long-term morbidity. Nonoperative treatment appeared to alleviate symptoms without influencing the eventual outcome.

Gore et al. carried out a clinical and radiographic study of 205 patients who had presented with axial neck pain or radicular symptoms in the upper extremities. One hundred and sixty-one of these patients were treated with...
rest, traction, a collar, medications, or combinations of these modalities. At the time of follow-up at ten to twenty-five years, 43% had complete resolution of pain, 25% had mild residual pain, and 32% had moderate or severe residual pain. Patients with radicular symptoms or findings had a less favorable prognosis. Treatment did not appear to influence the eventual outcome. The pain could not be correlated to the degenerative changes seen on radiographs. Gore et al. concluded that many patients with this condition have long-term symptoms that may be moderately disabling.

The true natural history of cervical myelopathy may be difficult to determine because in the vast majority of cases the symptoms are attributed to age or other neurologic conditions. Thus, knowledge of the natural history of this condition has been derived from a select population in whom the disease was already diagnosed and possibly was well established. In 1952, Spillane and Lloyd reported that the course of cervical myelopathy in their patients appeared to be one of progressive disability. In a 1956 report on 120 patients with cervical spondylotic myelopathy, Clarke and Robinson stated their belief that, once the disorder was recognized, neurologic function never returned to normal. Of their patients, 75% had episodic progression, 20% showed slow steady progression, and 5% had a rapid onset of symptoms followed by a lengthy period of stability. Sensory and bladder changes tended to be transient, but motor changes tended to persist and to progress over time. A soft collar helped to decrease nerve-root symptoms and improve gait for 50% of their patients.

Lees and Turner reported on a group of forty-four patients who had symptoms of myelopathy for three to forty years. They thought that long periods of nonprogressive disability were the rule and progressive deterioration was the exception. Neither the age at the onset of symptoms nor treatment with a collar or surgery appeared to influence the eventual prognosis. Nurick reported similar findings: he observed that the disability was established early in the course of the disease and was followed by static periods lasting many years. The prognosis was better for patients who presented with mild disease, and disability tended to progress in patients older than sixty years of age. Symon and Lavender reviewed the results reported by Lees and Turner and found that, when disability was used as a criterion, only 18% of patients showed improvement. In their own series, they reported steady, progressive deterioration in 67% of patients with cervical spondylotic myelopathy. Phillips also thought that the prognosis was poor; only one-third of his patients obtained improvement from treatment with a soft collar, and patients who had had symptoms for more than two years showed no improvement. A recent multicenter, nonrandomized study by the Cervical Spine Research Society suggested a similarly poor outcome of nonsurgical management of cervical myelopathy. In that study of forty-three patients, twenty underwent surgery and twenty-three received medical treatment. The surgically treated patients had decreased neurologic symptoms and overall pain and improved functional status, but the nonsurgically treated patients had a decrease in their ability to perform activities of daily living with worsening of neurologic symptoms.

**Clinical Evaluation**

**Axial Neck Pain**

Neck pain is an extremely common but nonspecific presenting symptom. The pain or soreness is usually in the paravertebral neck muscles posteriorly, with radiation toward the occiput or into the shoulder and periscapular regions. The patient reports stiffness in one or more directions, and headaches are common. The neck pain may be accompanied by radiating “referred” pain in the shoulder or arm that does not follow a dermatomal distribution. Referred pain can be associated with a sensation of warmth or tingling and with autonomic phenomena such as piloerection and sweating. Localized areas of tenderness in the muscle may be present. Deep palpation of some of these areas, known as trigger points, results in reproducible patterns of referred pain.

In the absence of radicular symptoms or findings, determining the source of neck pain can be a diagnostic challenge. Identifying a position of maximal discomfort may provide a clue to the underlying pathology. Anterior neck pain along the sternocleidomastoid muscle belly that is aggravated by rotation to the contralateral side is most often a result of muscular strain. Pain in the posterior neck muscles that is worsened by flexion of the head suggests a myofascial etiology. Pain in the posterior aspect of the neck that is aggravated by extension, especially with rotation of the head to one side, suggests the possibility of a discogenic component. Patients who present with severe pain in the suboccipital region often have pathological changes in the upper cervical spine. The pain in these patients may radiate to the back of the ear or the caudal part of the neck. Rotation of the neck is often markedly restricted.

Pain in the neck and shoulder girdle can develop as a result of adaptations stemming from an initial source of pain. The initial source of pain may resolve, but postural adaptations and compensatory overuse of normal tissues in the neck and shoulder girdle can result in new pain patterns. It is important to obtain an accurate history regarding how the pain initially presented and how it might have changed with time.

Pathological changes in the shoulder can present with localized pain or pain referred to the neck. The pain may radiate down the anterior or lateral aspect of the arm. Careful examination of the shoulder will help to differentiate this cause from pathological changes in the neck. Pain in the neck and shoulder girdle can also be referred from the heart, lungs, viscera, and temporomandibular joint. A detailed and accurate history and examination will help to rule out the possibility that pain in the neck is being referred from another region. Morning stiffness, poyparticular involvement, rigidity, or cutaneous manifestations suggest an in-
flammary or arthritic component. Fever, weight loss, or nonmechanical neck pain may point to an infectious or neoplastic process.

**Cervical Radiculopathy**

Cervical radiculopathy refers to symptoms in a specific dermatomal distribution in the upper extremity. Patients describe sharp pain and tingling or burning sensations in the involved area. There may be sensory or motor loss corresponding to the involved nerve root, and reflex activity may be diminished.

Patients typically have severe neck and arm pain that prevents them from getting into a comfortable position. They may hold the arm over the head, typically resting the wrist or forearm on top of the head (the shoulder abduction sign) and sometimes tilting the head to the contralateral side. The symptoms are usually aggravated by extension or lateral rotation of the head to the side of the pain (the Spurling maneuver). Aggravation of the symptoms by neck extension often helps to differentiate a radicular etiology from muscular neck pain or a pathological condition of the shoulder with secondary muscle pain in the neck. It is also important to remember that multiple sources of pain in the neck and upper extremity are common and that the nerve structures may be compressed at more than one site.

Patients with metabolic disorders, such as diabetes, who have neuropathy may be more susceptible to radiculopathy and compressive neuropathy. Adaptations to the initial radiculopathy may result in secondary pathological changes in the shoulder, carpal tunnel syndrome, or ulnar nerve irritation, which may persist long after the initial radiculopathy has resolved. Henderson et al. reviewed the clinical presentations of cervical radiculopathy in 736 patients: 99.4% had arm pain, 85.2% had sensory deficits, 79.7% had neck pain, 71.2% had reflex deficits, 68% had motor deficits, 52.5% had scapular pain, 17.8% had anterior chest pain, 9.7% had headaches, 5.9% had anterior chest and arm pain, and 1.3% had left-sided chest and arm pain (cervical angina). Neurologic deficits corresponded with the offending disc level in approximately 80% of patients.

Radiculopathy of the third cervical nerve root results from pathological changes in the disc between the second and third cervical levels and is unusual. The posterior ramus of the third cervical nerve innervates the suboccipital region, and involvement of that nerve causes pain in this region, often extending to the back of the ear. An isolated motor deficit from radiculopathy of the third cervical nerve root cannot be detected clinically.

Radiculopathy of the fourth cervical nerve root may be an unexplained cause of neck and shoulder pain. Numbness extending from the caudal aspect of the neck to the superior aspect of the shoulder may be present. Diaphragmatic involvement may result from involvement of the third, fourth, and fifth cervical nerve roots. Motor deficits in the diaphragm manifest as paradoxical respiration, and they may be confirmed by fluoroscopic evaluation of the abdomen.

Radiculopathy of the fifth cervical nerve root can present with numbness in an "epaulet" distribution, beginning at the superior aspect of the shoulder and extending laterally to the midpoint of the arm. The deltoid muscle is innervated primarily by the fifth cervical nerve, and involvement of that nerve can result in profound weakness of this muscle. The absence of pain with a range of motion of the shoulder and the absence of impingement signs at the shoulder help to differentiate radiculopathy of the fifth cervical nerve root from a pathological shoulder condition. The biceps reflex is innervated by the fifth and sixth cervical nerves and may be affected.

Radiculopathy of the sixth cervical nerve root presents with pain radiating from the neck to the lateral aspect of the biceps, to the lateral aspect of the forearm, to the dorsal aspect of the web space between the thumb and index finger, and into the tips of those digits. Numbness occurs in the same distribution. Motor deficits are best elicited in the wrist extensors, but they also may be elicited by elbow flexion and forearm supination. The brachioradialis and biceps reflexes may be lost or diminished. The sensory symptoms may mimic carpal tunnel syndrome, which typically involves the radial three and a half digits and causes weakness in the thenar musculature.

The seventh cervical nerve root is the most frequently involved by cervical radiculopathy. The patient has pain radiating along the back of the shoulder, often extending into the scapular region, down along the triceps, and then along the dorsum of the forearm and into the dorsum of the long finger. The patient usually pronates the forearm while trying to describe the location of the symptoms, and this is a useful observation when the physician is trying to differentiate the hand symptoms from those of sixth cervical radiculopathy and carpal tunnel syndrome. Motor weakness is best appreciated in the triceps, wrist flexors, and finger extensors. The triceps reflex may be lost or diminished. Entrapment of the posterior intersosseus nerve may be mistaken for the motor component of seventh cervical radiculopathy and presents with weakness in the extensor digitorum communis, extensor pollicis longus, and extensor carpi ulnaris. Sensory changes are absent, however, and the triceps and wrist flexors show normal strength.

Radiculopathy of the eighth cervical nerve root usually presents with symptoms extending down the medial aspect of the arm and forearm and into the medial border of the hand and the ulnar two digits. Numbness usually involves the dorsal and volar aspects of the ulnar two digits and hand and may extend up the medial aspect of the forearm. The patient reports difficulty using the hands for routine daily activities. It is important to differentiate eighth cervical radiculopathy from ulnar nerve weakness. The function of the flexor digitorum profundus in the index and long fingers and of the flexor pollicis longus in the thumb can be affected by eighth cervical radiculopathy, but they are not affected by ulnar nerve entrapment. With the exception of the adductor pollicis, the short thenar muscles are spared with ulnar...
nerve involvement but involved with eighth cervical or first thoracic radiculopathy. Entrapment of the anterior interosseous nerve may masquerade as eighth cervical or first thoracic radiculopathy, but it does not cause the sensory changes or have thenar muscle involvement.

Patients occasionally present with symptoms that simulate radiculopathy but result from nonspondylotic pathological changes (Table II). Schwannomas usually arise from the intradural portion of the sensory root and may cause severe pain in a dermatomal distribution. Meningiomas can similarly cause radicular or myelopathic symptoms, depending on their size and precise location. Benign or malignant vertebral body tumors usually present with nonmechanical neck pain that progresses to severe radiculopathy, and even myelopathy, as the amount of bone destruction increases. A Pancoast tumor of the apical lung can involve the caudal cervical nerve roots and, additionally, involve the sympathetic chain. Idiopathic brachial plexus neuritis is thought to be viral in nature and presents with severe arm pain that resolves and leaves behind polyradicular motor deficits. Polyradicular involvement may also be seen with epidural abscesses. Reflex sympathetic dystrophy occasionally occurs following trauma to the upper extremity, and it presents as diffuse burning pain or paresthesias accompanied by discoloration, edema, or other autonomic phenomena.

**Cervical Spondylotic Myelopathy**

Cervical spondylotic myelopathy is the most common cause of acquired spastic paraparesis in adults. The patient may present with subtle findings that have been present for years or with quadriparesis that developed over the course of a few hours. Perhaps the most unique feature of the condition is its subtle and varied presentation, and the fact that its diagnosis requires a high index of suspicion.

The clinical picture varies, depending on the anatomic portion of the cord that is primarily involved. Crandall and Batzdorf described five broad categories of cervical spondylotic myelopathy: (1) transverse lesion syndrome, in which the corticospinal, spinothalamic, and posterior cord tracts were involved with almost equal severity and which was associated with the longest duration of symptoms, suggesting that this category may be an end stage of the disease; (2) motor system syndrome, in which corticospinal tracts and anterior horn cells were involved, resulting in spasticity; (3) central cord syndrome, in which motor and sensory deficits affected the upper extremities more severely than the lower extremities; (4) Brown-Séquard syndrome, which consisted of ipsilateral motor deficits with contralateral sensory deficits and which appeared to be the least advanced form of the disease, and (5) brachialgia and cord syndrome, which consisted of radicular pain in the upper extremity along with motor and/or sensory long-tract signs.

Ferguson and Caplan divided cervical spondylotic myelopathy into four syndromes: (1) medial syndrome, consisting primarily of long-tract symptoms; (2) lateral syndrome, consisting primarily of radicular symptoms; combined medial and lateral syndrome, which is the most common clinical presentation; and (4) vascular syndrome, which presents with a rapidly progressive myelopathy and is thought to represent vascular insufficiency of the cervical spinal cord.

The findings in cervical spondylotic myelopathy vary from patient to patient. Patients typically present with the insidious onset of clumsiness in the hands and lower limbs. They may report worsening handwriting in the past few months or weeks, difficulty with grasping and holding, or diffuse numbness in the hands. They frequently have had increasing difficulty with balance that they attribute to age or arthritic hips, and relatives may volunteer that their gait has become increasingly awkward. Nurick developed a system for grading the disability in cervical spondylotic myelopathy on the basis of gait abnormality (Table III). On physical examination, the findings that establish the diagnosis are brisk reflexes, clonus, or pathological reflexes confirming an upper-motor-neuron lesion. Myelopathy resulting from a region of the cord cephalad to the third cervical level may result in a hyperactive scapulohumeral reflex—i.e., tapping of the spine of the scapula or acromion results in scapular elevation and/or abduction of the humerus. This is thought to be a stretch reflex of the trapezius muscle. Superficial reflexes such as the abdominal or cremasteric reflex are often diminished or absent in the presence of upper-motor-neuron lesions. The pathological reflexes that are typically elicited are the inverted radial reflex, the Hoffmann reflex, and the extensor plantar reflex. Muscle weakness and wasting in the lower extremi-
ties with superimposed loss of proprioception result in an unsteady, broad-based gait.

Sensory findings in cervical spondylotic myelopathy vary. Pain, temperature, proprioception, vibratory, and dermatomal sensations may all be diminished, depending on the exact area of the cord or the nerve-root that is compromised. Sphincter disturbances are not usually presenting symptoms. Patients may complain of urinary urgency, hesitation, and frequency and rarely of urinary incontinence or retention. Fecal incontinence is unusual. In a study of sixty-two patients with cervical spondylotic myelopathy by Crandall and Batzdorf, neck pain was present in fewer than half of the patients and associated radicular pain was present in 38%. The Lhermitte sign with shock-like sensations in the torso and limbs resulting from quick flexion or extension of the neck was present in 27% of the patients, and sphincter disturbances were present in 44%.

Hand dysfunction in cervical spondylisis has, in the past, been attributed primarily to radicular pathology. Several recent reports have shown findings specific to the “myelopathy hand.” Diffuse numbness in the hands is extremely common and is often misdiagnosed as peripheral neuropathy or carpal tunnel syndrome. Clumsiness of the hands results in an inability to carry out fine motor tasks. Marked wasting of the intrinsic hand muscles is usually present. Ono et al. described two specific signs of myelopathy hand: (1) the finger-escape sign (when the patient is asked to fully extend the digits with the palm facing down, the ulnar digits tend to drift into abduction and flexion) and (2) the grip-and-release test (weakness and spasticity of the hand result in a decreased ability to rapidly open and close the fist). Many neurologic conditions can mimic cervical spondylotic myelopathy (Table IV). Multiple sclerosis, a demyelinating disorder of the central nervous system, causes both motor and sensory symptoms, but it typically has remissions and exacerbations, involvement of the cranial nerves, and characteristic plaques that can be seen on magnetic resonance imaging of the brain and spinal cord. Amyotrophic lateral sclerosis results in upper and lower motor-neuron symptoms, with no alteration in sensation. Subacute combined degeneration seen with vitamin B12 deficiency results in corticospinal tract and posterior tract symptoms, with greater sensory involvement in the lower extremities. Patients with metabolic or idiopathic peripheral neuropathy have sensory symptoms that mimic those of myelopathy.

<table>
<thead>
<tr>
<th>TABLE IV Differential Diagnoses of Cervical Spondylotic Myelopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral polyneuropathy</td>
</tr>
<tr>
<td>Motor neuron disease</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
</tr>
<tr>
<td>Syringomyelia</td>
</tr>
</tbody>
</table>

Atypical Presentations of Cervical Spondylisis

Cervical angina, a symptom constellation mimicking ischemic heart disease but produced by cervical radiculopathy, is a well-known entity. Women with pain of cervical radicular origin may present with chronic breast pain. Facial pain or paresthesias may be secondary to involvement of the spinal nucleus of the trigeminal nerve and may result from pressure on the cephalad and middle parts of the cervical spinal cord. Marked spurring along the anterior aspects of the vertebral bodies as a result of proliferative degenerative changes may manifest as dysphagia, dyspnea, or dysphonia as a result of pressure on the esophagus, larynx, or trachea. Similar hypertrophic spurs resulting from the uncovertebral joints and facet joints can occlude the vertebral artery in its foramen and result in thrombosis of the vertebral artery. If the thrombosis spreads to the posterior inferior cerebellar artery, it may lead to palsy of the ipsilateral V, IX, X, and XI cranial nerves, Horner syndrome, cerebellar ataxia, and possibly death—a constellation known as Wallenberg syndrome. Sympathetic chain involvement can result in atypical symptoms such as dizziness, blurring of vision, tinnitus, retroocular pain, facial pain, or jaw pain. Cervical spondylotic myelopathy has also been reported to present with hemiparesis.

Radiculopathy occasionally presents in association with myelopathy. It is important not to let overriding radicular deficits obscure an underlying myelopathy. Patients with cervical myelopathy occasionally may have concomitant peripheral neuropathy or lumbar stenosis, which might mask the lower-extremity hyperreflexia typically expected from the myelopathy.

Overview

Degenerative cervical disc disease is a common condition that is, for the most part, asymptomatic. When symptoms do develop, they can be easily grouped into axial neck pain, radiculopathy, and myelopathy. An understanding of the pathophysiology of these conditions suggests that patients with axial symptoms usually are best treated without surgery, although some patients with radiculopathy continue to be disabled by pain and may be candidates for operative treatment. Patients with myelopathy are unlikely to have substantial improvement and, in most cases, show stepwise deterioration. Surgical decompression and stabilization should be considered for these patients.

Raj Rao, MD
Department of Orthopaedic Surgery, Medical College of Wisconsin,
9200 West Wisconsin Avenue, Milwaukee, WI 53226

The author did not receive grants or outside funding in support of his research or preparation of this manuscript. He did not receive payments or other benefits or a commitment or agreement to provide such benefits from a commercial entity. No commercial entity paid or directed, or agreed to pay or direct, any ben-
efits to any research fund, foundation, educational institution, or other charitable or nonprofit organization with which the author is affiliated or associated.

Printed with the permission of the American Academy of Orthopaedic Surgeons. This article, as well as other lectures presented at the Academy's Annual Meeting, will be available in March 2003 in Instructional Course Lectures, Volume 52. The complete volume can be ordered online at www.aaos.org, or by calling 800-626-6726 (8 A.M.-5 P.M., Central time).

References