ELSEVIER
SAUNDERS

Neurol Clin 25 (2007) 331–351

NEUROLOGIC
CLINICS

Anatomy and Examination of the Spine

Michael W. Devereaux, MD[a,b]

[a]*Department of Neurology, University Hospitals of Cleveland/Case Western Reserve University, 11100 Euclid Avenue, Cleveland, OH 44106, USA*
[b]*University Hospitals Health System, Richmond Heights Hospital 27100 Chardon Road, Richmond Heights, OH 44143-1116, USA*

A review of the anatomy of the spine in a few pages must, by necessity, be abridged. This article concentrates on clinically relevant anatomy. For a more expansive discussion, the reader is referred to the most recent edition of Gray's anatomy [1].

Vertebral column

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

The spinal column is composed of 7 cervical, 12 thoracic, 5 lumbar, and 5 fused sacral vertebra, along with 5 coccygeal bones. The cervical, thoracic, and lumbar vertebrae are similar in structure except for the first (atlas) and second (axis) cervical vertebrae. Each "standard" vertebra is composed of a body, two pedicles, two lamina, four articular facets, and a spinous process. The atlas is composed of a ring of bone without a body, whereas the axis has an odontoid process around which the atlas rotates. Between each pair of vertebrae are two openings, the foramina, through which pass a spinal nerve, radicular blood vessels, and the sinuvertebral nerves (recurrent meningeal nerves) (Fig. 1). Each foramen is bordered superiorly and inferiorly by pedicles, anteriorly by the intervertebral disc and adjacent vertebral body surfaces, and posteriorly by the facet joint.

E-mail address: Michael.Devereaux@UHhospitals.org

doi:10.1016/j.ncl.2007.02.003

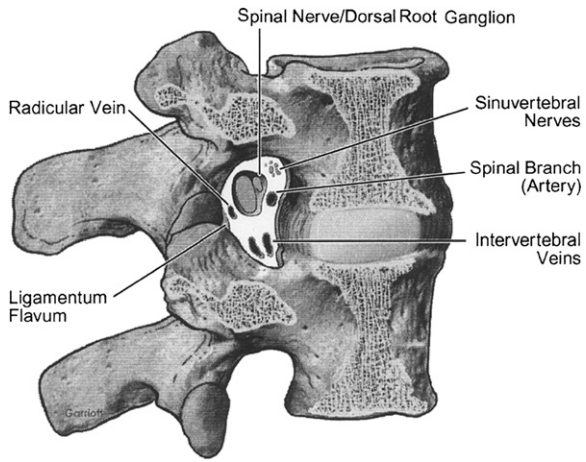

Fig. 1. The foremen. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:9; with permission.)

The spinal canal itself is formed posterolaterally by the laminae and ligamentum flavum, anterolaterally by the pedicles, and anteriorly by the posterior surface of the vertebral bodies and intervertebral discs. The midsagittal (anterior-posterior) diameter of the cervical canal from C1 to C3 is usually approximately 21 mm (range 16–30 mm), and from C4 to C7 the diameter is approximately 18 mm (range 14–23 mm). The midsagittal diameter of the cervical spinal cord is 11 mm at C1, 10 mm from C2 to C6, and 7 to 9 mm below C6. The midsagittal diameter of the cervical cord normally occupies approximately 40% of the midsagittal diameter of the cervical canal in healthy individuals. This cervical canal midsagittal diameter is decreased by 2 to 3 mm with extension of the neck, which is of clinical importance in the context of hyperextension injuries in an individual with a congenitally narrow spinal canal, especially in the presence of additional narrowing caused by cervical spondylosis. Under such circumstances an acute cervical myelopathy may result. With regard to the lumbar canal, the midsagittal diameter is approximately 18 mm. Narrowing as a result of spondylosis coupled with extension can compromise the cauda equina and the accompanying vasculature, producing the symptoms of neurogenic claudication.

The facet (zygapophyseal) joint, unlike the intervertebral disc, is a true synovial joint. Although it contributes—to a limited extent—to the support of the spinal column, this joint's main function is to maintain stability of the spinal column by guiding the direction of vertebral movement, a function that depends on the plane of the facet joint surface, which varies throughout the spinal column. The joint is subject to degenerative change that results in enlargement, which, in association with thickening of the ligamentum flavum, can contribute to canal stenosis as a component of spondylosis. It is

innervated by branches from the posterior ramus of the spinal nerve. The exact role of the facet joint in the production of back pain, particularly low back pain, remains somewhat controversial [2].

The intervertebral disc

The intervertebral disc is a cartilaginous and articulating structure between the vertebral bodies. Intervertebral discs have the dual role of providing the primary support for the column of vertebral bones while possessing enough elasticity to permit the required mobility of the spine (flexion, extension, and rotation). The aggregate of discs together accounts for 25% to 30% of the overall length (height) of the spine. Each disc is comprised of a ring of elastic collagen, the annulus fibrosus, which surrounds the gelatinous nucleus pulposus (Fig. 2). The collagen fibers of the annulus are arranged obliquely in alternating directions, in layers (lamellae), which allows for flexibility while maintaining strength. Fifteen to 25 lamellae comprise the annulus [3]. Collagen fibers continue from the annulus into the adjacent tissues, which ties this structure to each vertebral body at its rim, to the anterior and posterior longitudinal ligaments, and to the hyaline cartilage endplates superiorly and inferiorly. The cartilage endplates in turn lock into the osseus vertebral endplates via the calcified cartilage [4].

The nucleus pulposus is a self-contained, pliable gelatinous structure that is 88% water in a healthy young disc. It is essentially a hydraulic system that provides support and separates the vertebrae, absorbs shock, permits transient compression, and allows for movement. As a result of the aging process and injury to the disc, increasing amounts of fibrous tissue replace the highly elastic collagen fibers of the young, normal, uninjured disc. The older disc is less elastic, and its hydraulic recoil mechanism is weakened [4].

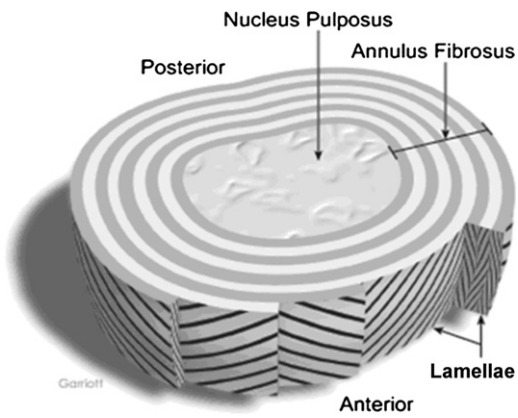

Fig. 2. The intervertebral disk. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:11; with permission.)

By the fifth decade of life, the annulus becomes fissured, with transformation into fibrous bodies separated by softer substances. Ultimately the disc deteriorates into a desiccated, fragmented, and frayed annulus fibrosus surrounding a fibrotic nucleus pulposus [4,5].

The intervertebral disc is avascular by the third decade of life, and nutrition is delivered to the disc by diffusion. The nucleus pulposus in the normal adult disc has no nerve supply. The outer lamellae of the annulus fibrosus contains nerve endings derived from the sinuvertebral nerves (recurrent meningeal nerves), however [4,6–9].

There is debate in the literature regarding nociceptive nerve supply to the intervertebral disc and what role the disc plays as a generator of back pain. Korkala and colleagues [8] showed that the nerve endings entering the annulus fibrosus do not contain substance P and are not nociceptors. The authors noted that nociceptive nerve endings are located in the posterior ligament adjacent to the disc. Palmgren and colleagues [9], in a study of normal human lumbar intervertebral disc tissue, demonstrated that nerve endings could be found at a depth of a few millimeters, whereas neuropeptide markers (eg, substance P) revealed nociceptive nerves only in the outermost layers of the annulus fibrosus. This study lends support to the concept that the normal intervertebral disc is almost without innervation.

This finding leads to the question of the mechanism of primary discogenic pain, particularly in the lumbar spine. Damage to the intervertebral disc can produce pain, but no consensus exists on the responsible mechanisms. Radial tears and fissures in the annulus fibrosus occur as the disc ages. This change has been linked to the ingrowth of blood vessels and nerve fibers, leading to the concept that the ingrowth of these nerve endings may be the pathoanatomic basis for discogenic pain [6,10]. First, if the ingrowth of nociceptive nerve fibers into the intervertebral disc may be the neuroanatomic substrate for discogenic pain, then why are most degenerative discs not a source of pain? For example, discography of degenerative discs does not uniformly induce pain [10]. Because disc degeneration per se is not the basis for discogenic pain, contributing factors must be at play. Possibly a combination of focal damage to the annulus fibrosus, inflammation, neoinnervation, and nociceptor sensitization is necessary to induce discogenic pain [11].

Ligaments of the vertebral column

Several ligaments lash the vertebrae together and, along with the intrinsic paraspinous muscles, control and limit spinal column motion. From a clinical perspective, some of the ligaments are more important than others.

The posterior longitudinal ligament stretches from the axis (named the membrane of tectoria in the ''high'' cervical spine) to the sacrum and forms the anterior wall of the spinal canal. It is broad throughout the cervical and thoracic portions of the spine. At the L1 vertebral level it begins to narrow,

however, and at L5 it is one-half its original width. It is attached firmly to each intervertebral disc by hyalin cartilage endplates, but only in the midline by a septum to the periosteum of each vertebra. The open space between the posterior longitudinal ligament and the vertebral body is the anterior epidural space, which is important in disc herniation. The narrowing of the ligament in the lumbar spine inadequately reinforces the lumbar disc, which creates an inherent structural weakness. This narrowing, coupled with the great static and kinetic stress placed on the lumbar discs, contributes to their susceptibility to injury and herniation.

The ligamenta flava is composed of a series of strong paired elastic ligaments that span the space between the laminae, attached to the anterior inferior surface of the laminae above and the posterior superior margin of the laminae below. Each component stretches laterally, joining the facet joint capsule. The ligament stretches under tension, which permits flexion of the spine. It contains few, if any, nociceptive nerve fibers. It can be clinically important because with age it can thicken and, along with other spondylotic degenerative changes, can contribute to canal stenosis, which produces myelopathy in the cervical spine and cauda equina compression in the lumbar spine.

Other ligaments that contribute to the stabilization of the spine include the anterior longitudinal ligament, the ligamentum nuchae from the occiput to the cervical vertebra, the infraspinous ligaments, and the supraspinous ligaments. The occipitovertebral ligaments are dense, broad, and strong and connect the occiput to the atlas. These ligaments permit up to 30° of flexion and extension around the atlanto-occipital joint. The stability of the atlantoaxial joints depends almost entirely on ligaments. The transverse ligament of the atlas helps to contain the odontoid process in place. It is actually stronger than the odontoid process, which means that the odontoid process fractures before the ligament is torn. The paired alar ligaments attach on either side of the apex and extend to the medial side of the occipital condyles. The main function of these ligaments is limitation of rotation, keeping in mind that in the atlas axis there is approximately 90° of the 160° of total head rotation capacity.

The paraspinous muscles

With the exception of the atlas and axis, the range and type of movement in each segment of the spine is determined by the facet joints, but spine stability and the control of spinal movement depend on muscles and ligaments. The movement itself, of course, depends on muscle.

The spinal muscles are arranged in layers. The deeper layers comprise the intrinsic, true back muscles, as defined by their position and innervation by the posterior rami of the spinal nerves. This is in contrast to the more superficial extrinsic muscles, which insert on the bones of the upper limbs and are innervated by anterior rami of the spinal nerves.

The intrinsic muscles are also divided into superficial and deep groups. The superficial layer is comprised of the paraspinous erector spinae group, which spans the entire length of the spine from the occiput to the sacrum, and the splenius muscles of the upper back and neck. This superficial group functions collectively primarily to maintain erect posture. Deep to the erector spinae is the transversospinalis muscle group, which is composed of muscles made up of several smaller muscles that run obliquely and longitudinally. In essence, they form a system of guy ropes that provide lateral stability to the spine, contribute to maintenance of an erect posture, and rotate the spine. Deepest of all are the interspinal and intertransverse muscles, which are composed of numerous small muscles involved in the maintenance of posture.

The multiple subdivisions of muscle mass, numerous connective tissue planes, and multiple attachments of tendons over small areas of vertebral periosteum help to explain the prevalence of neck and back pain while simultaneously explain the difficulty in precisely localizing the source of that pain. Taking into account this difficulty in identifying muscle and tendon injury as the source of pain and the fact that there are other generators of low back pain besides muscles (eg, fascia, ligaments, facet joint, intervertebral disc), it is no wonder that according to Deyo and colleagues [12], the source of acute low back pain cannot be identified in 85% of patients. It also should be noted that when muscle is the source of pain, the pathophysiologic pain-generating process is unclear. In the clinic, muscle spasm is often the diagnosis made. Muscle spasm is generally defined as a contraction of muscle that cannot be voluntarily released and is associated with electromyographic activity. Johnson and others [13,14] have taken issue with increased muscle activity as a source of paraspinous pain, noting a lack of electromyographic evidence indicative of muscle spasm.

Vascular supply to the spinal column and contents

The vertebral column and its contents receive blood supply from segmental medullary arteries. These arteries originate from the vertebral arteries in the cervical spine and from the posterior intracostal and lumbar arteries in the thoracic and lumbar portions of the spine, which ultimately originate from the aorta. Branches enter into the spinal column through the foramina. Some of the anterior branches are large, such as the great anterior segmental medullary artery of Adamkiewicz, and anastomose with longitudinal spinal cord vessels to form a pial plexus on the surface of the cord. The segmental spinal arteries send anterior and posterior radicular branches to the spinal cord along the ventral and dorsal roots. The importance of the segmental arterial blood supply to the spinal cord is amply demonstrated in patients with a dissection of the aorta, with resultant occlusion of paired branches feeding segmental branches to the spinal column and cord and resultant ischemic injury to the cord.

In addition to the segmental arties, the longitudinal paired posterior spinal arteries and the single anterior spinal artery originate from the distal vertebral arteries. Although they run the length of the surface of the spinal cord, they alone cannot supply the spinal cord and anastomose with segmental vessels along their entire length.

The importance of the anterior spinal artery in the cervical cord is well known to clinicians. Hyperextension injuries to the neck in association with cervical spondylosis and canal stenosis can result in occlusion of the anterior spinal artery and ischemia to the anterior two thirds of the cord. Therapeutic cervical manipulation has been associated with mechanical injury to the spinal cord and distal vertebral artery dissection, which results in posterior cerebral circulation distribution strokes [15].

The nerve supply to the spinal column and related structures

One of the most frustrating aspects of neck and back pain for the physician and patient is the difficulty in arriving at a precise cause. As in the case of acute low back pain, a definite diagnosis cannot be established in 85% of patients because of weak associations between symptoms, pathologic changes, and imaging results [12]. It is widely assumed that much nonradiating neck and low back pain is secondary to musculoligamentous injury and degenerative changes.

Localized cervical and lumbosacral pain is mediated primarily through the posterior primary ramus and the sinuvertebral (recurrent meningeal) nerves. The sinuvertebral nerves supply structures within the spinal canal. They arise from the rami communicantes and enter the spinal canal by way of the intervertebral foramina [16]. Branches ascend and descend one or more levels, interconnecting with the sinuvertebral nerves from other levels and innervating the anterior and posterior longitudinal ligaments, the anterior and posterior portion of the dura mater, and blood vessels, among other structures (Fig. 3). This system also may supply nociceptive branches to degenerated intervertebral discs.

Branches of the posterior ramus provide sensory fibers to fascia, ligaments, periosteum, and facet joints (Box 1). The source of deep somatic neck and low back pain can be the vertebral column itself, the surrounding muscle, tendons, ligaments, and fascia, or a combination thereof.

Radicular pain, unlike spondylogenic pain, is not mediated by sinuvertebral nerves or the posterior rami, but rather by proximal spinal nerves. Two major factors are involved in the generation of radicular pain: compression and inflammation. Compression of the nerve root produces local ischemia with possible alteration in axoplasmic transport and edema. Ischemia may have a particular impact on large mechanoreceptor fibers. Because of their large diameter, these fibers have greater metabolic activity and are more sensitive to reduced blood flow. This reduction can result in the loss of inhibitory pain impulses and lead to preferential nociceptive input into the spinal cord.

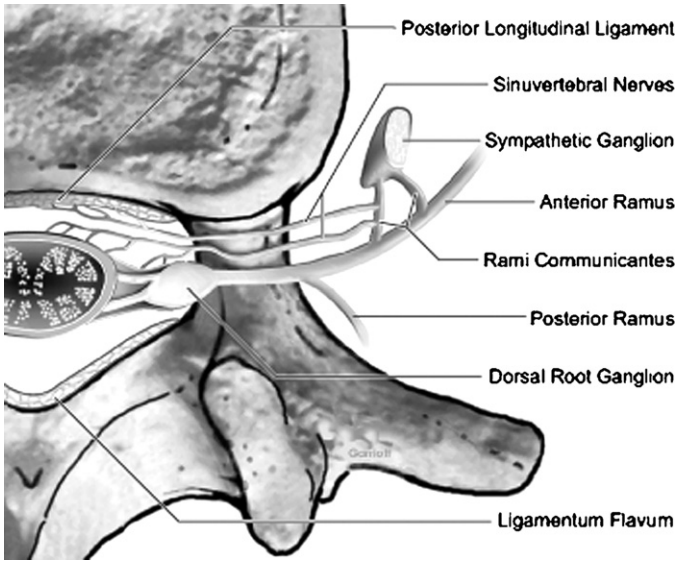

Fig. 3. The sinuvertebral nerve. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:13; with permission.)

Compression alone can produce paresthesia and some radicular pain; however, compression and traction in the presence of chronic inflammation produces more prominent radicular pain. The origin of the inflammatory response itself may be neurogenic or immunologic. In the case of disc

Box 1. Pain-sensitive tissues in the spine

Skin, subcutaneous tissue, and adipose tissue
Capsules of facet and sacroiliac joints
Ligaments: longitudinal spinal, interspinous (mainly posterior), flaval (minimal innervation – probably not clinically significant), and sacroiliac
Periosteum: vertebral bodies and arches
Dura mater and epidural fibroadipose tissue
Arterioles that supply spinal and sacroiliac joints and vertebral cancellous bone
Veins: epidural and paravertebral
Paravertebral muscles: perivascular unmyelinated nerve endings in the adventitial sheaths of intramuscular blood vessels

Data from Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:13.

herniation with nerve root compression, the normally avascular nucleus pulposus comes into contact for the first time with the immune defense mechanism, which results in autoimmune-induced inflammatory response in the region of the spinal nerve.

Radicular, radiating pain secondary to disc herniation is the product of spinal nerve compression and local inflammation. The normal disc itself may not contain nociceptive nerve fibers and is insensitive to pain. When the nucleus pulposus ruptures through the annulus fibrosus, there is little or no localized pain until nociceptive fibers of the sinuvertebral nerves in the lateral posterior ligament and the dura of the nerve root sleeves are stimulated. This stimulation generates localized back and neck pain. Understanding the role of (1) the sinuvertebral system and the posterior rami in the generation of localized spine pain and (2) the spinal nerve and the generation of radiating pain helps to explain why patients with disc herniation develop sciatica only approximately one third of the time. The following clinical situations also can be more easily understood on this basis:

- Disc herniation visualized on a neuroimaging procedure in the absence of a history of radicular pain [17,18].
- Weakness in a radicular distribution without significant radicular pain secondary to disc herniation, with compression of the ventral root only
- Nonradiating low back pain secondary to disc herniation (with radicular pain perhaps developing months or years later)

One last generator of spine pain, viscerogenic referred pain (pain that arises from organs that share segmental innervation with structures in the lumbosacral spine), sometimes eludes clinical neurologists. The quality of pain is often, but not always, different (eg, cramping in quality). Organs that can refer pain to the low back and sometimes mid-spine include the aorta, pancreas, duodenum, ascending and descending colon, rectum, kidney, ureter, bladder, and pelvic organs. The abdominal examination is important in the evaluation of the patient who is experiencing low and mid-back pain.

Neurologic history

History

The history is of critical importance in assessing patients with symptoms believed to be secondary to cervical and lumbar spine disorders, especially in persons with a nonfocal neurologic examination. The differential diagnosis is frequently based solely on the history in these patients.

Pain profile

Onset

In most instances, patients who present with a history of acute onset of neck and low back pain have a history of preceding pain, often for weeks

or months or longer. This is also the case in patients with the acute onset of radicular pain. The acute onset of cervical or lumbosacral radicular pain in the absence of any prior history of neck and low back pain is the exception rather than the rule.

Quality

Variable, nonradiating musculoskeletal back pain is often described as being deep and aching, whereas radicular pain is usually described as sharp, jabbing, or lancinating in quality.

Location

Musculoskeletal pain is usually localized to the paraspinous regions. In the neck, it is generally maximally felt in the paracervical regions, at times spreading into the shoulders and scapular regions. Lumbosacral pain tends to be maximal in the paraspinal regions, spreading at times to the flanks and into the buttocks. When cervical roots are involved, the pain generally radiates into the upper extremity. Occasionally the distribution of the pain alone may be enough to allow localization to a specific cervical root (Table 1). In the case of lumbosacral radiculopathy, the pain usually radiates into one or

Table 1
Symptoms and signs associated with cervical radiculopathy

Root	Pain distribution	Dermatomal sensory distribution	Weakness	Affected reflex
C4	Upper neck	"Cape" distribution shoulder/arm	None	None
C5	Neck, scapula, shoulder, anterior arm	Lateral aspect of arm	Shoulder abduction Forearm flexion	Biceps Brachioradialis
C6	Neck, scapula, shoulder, lateral arm, and forearm into first and second digits	Lateral aspect forearm and hand and first and second digits	Shoulder abduction Forearm flexion	Biceps Brachioradialis
C7	Neck, shoulder, lateral arm, medial scapula, extensor surface forearm	Third digit	Elbow extension Finger extension	Triceps
C8	Neck, medial scapula, medial aspect arm and forearm into fourth and fifth digits	Distal medial forearm to hand and fourth and fifth digits	Finger: abduction adduction flexors	Finger flexors

Data from Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:7–43.

both lower extremities. The distribution of the pain also can occasionally point to the specific root involved (Table 2). For example, "high" lumbar (L2, L3) radiculopathic pain does not radiate distal to the knee, whereas the pain of an L4 radiculopathy can radiate to the medial leg distal to the knee. L5 and S1 radiculopathies tend to produce pain that radiates into the posterolateral thigh and posterolateral leg and often involves the foot. Pain may be maximum in the medial (L5 radiculopathy) or lateral aspect of the foot (S1 radiculopathy).

Duration

Mechanical low back pain generally has a duration of days to weeks. Radicular pain often resolves more gradually over 6 to 8 weeks. An extensive neurodiagnostic evaluation is generally not necessary in this setting. A patient who presents with a history of chronic low back pain, however, requires a careful history to rule out a new problem superimposed over chronic symptoms that, in the proper setting, may require an immediate neurodiagnostic evaluation.

Severity

As all clinicians recognize, the severity of pain is often difficult to interpret because it can be colored by several factors, including a patient's

Table 2
Symptoms and signs associated with lumbar radiculopathy

Root	Pain distribution	Dermatomal sensory distribution	Weakness	Affected reflex
L1	Inguinal region	Inguinal region	Hip flexion	Cremasteric
L2	Inguinal region and anterior thigh	Anterior thigh	Hip flexion Hip adduction	Cremasteric Thigh adductor
L3	Anterior thigh and knee	Distal anteromedial Thigh, including knee	Knee extension Hip flexion Hip adduction	Patellar Thigh adductor
L4	Anterior thigh, medial aspect leg	Medial leg	Knee extension Hip flexion Hip adduction	Patellar
L5	Posterolateral thigh Lateral leg Medial foot	Lateral leg, dorsal foot, and great toe	Dorsiflexion foot/toes Knee flexion Hip extension	
S1	Posterior thigh and leg and lateral foot	Posterolateral leg and lateral aspect of foot	Plantar flexion foot/toes Knee flexion Hip extension	Achilles

Data from Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:7–43.

personality. Severe low back and neck pain that is not relieved when the patient is recumbent suggests metastatic cancer, pathologic vertebral fracture, or infection of a vertebra, disc, or the epidural space.

Time of day

Cervical and lumbar radiculopathy frequently present upon awakening in the morning. Nonradiating pain that tends to be dull during the day is often the result of mechanical disorders (eg, muscle strain, degenerative disc disease/spondylosis). Tumors of the spine and spinal cord often produce pain that persists and occasionally increases in the supine position; patients with lumbar and cervical tumors may have increased pain in bed at night.

Associated symptoms

Several cervical spine disorders that cause localized and radiating pain into an upper extremity also may produce symptoms secondary to an associated cervical myelopathy (eg, weakness and paresthesia in the lower extremities) and sphincter dysfunction. In the case of low back pain, the patient should be questioned about abdominal pain and intestinal or genitourinary symptoms.

Triggers

Valsalva maneuvers (eg, coughing, sneezing, and bearing down at stool) often transiently aggravate lumbosacral and cervical radicular pain. In the case of cervical radicular pain, lateral head movements to the side of the radiating pain—and sometimes to the opposite side—may aggravate the pain. Low back radicular pain is generally made worse by sitting and standing and often is relieved by lying supine. If pain persists or increases in the supine position, the possibility of spinal metastatic cancer or infection must be considered. In the case of lumbar canal stenosis, neurogenic claudication can be brought on by standing erect and walking.

Motor symptoms

In the face of pain, distinguishing between weakness and guarding by the history alone can be difficult. In the case of low back and lower extremity pain, however, weakness is suggested by a history of a foot slap when walking or of falls secondary to a lower extremity "giving way." With neck pain radiating into an upper extremity, a history of difficulty writing with the symptomatic extremity and difficulty elevating the limb may be useful clues as to the presence of true accompanying weakness. Although weakness is usually best appreciated on a neurologic examination, the history is a useful adjunct in helping to separate weakness from guarding secondary to pain.

Sensory disturbances

Patients with radiculopathy often report numbness, tingling, and even coolness in the involved extremity. At times, symptoms suggest dysesthesia

and allodynia. The distribution of a sensory disturbance by history, particularly of numbness and tingling, may be even more useful in determining the presence and localization of a radiculopathy than the sensory examination itself.

Bladder and bowel disturbances

Symptoms of a hypertonic bladder (ie, urgency, frequency, nocturia, and incontinence of bladder [or occasionally of bowels]) are often found in association with cervical myelopathy. Sphincter disturbances also may appear with cauda equina compression and, when acute, always must serve as a warning of the need for urgent surgical intervention.

Risk factors

Although various risk factors have been associated with an increased incidence of neck and low back pain, knowledge of these risk factors is not necessarily helpful in evaluating individual patients. Risk factors are better established for low back pain than neck pain, but many risk factors are common to both, including the following:

- Increasing age
- Heavy physical work, particularly long static work postures, heavy lifting, twisting, and vibration
- Psychosocial factors, including work dissatisfaction and monotonous work
- Depression
- Obesity
- Smoking
- Severe scoliosis ($>80\%$)
- Drug abuse
- History of headache

Several other factors are commonly thought to increase the risk of low back and neck pain but probably do not, including

- Anthropometric status (height, body build)
- Posture, including kyphosis, lordosis, and scoliosis $<80\%$
- Leg length differences
- Gender
- State of physical fitness (although not a predictor of acute low back pain, fit individuals have a lower incidence of chronic low back pain and tend to recover more quickly from episodes of acute low back pain than unfit individuals).

The pain patient at risk

Although most patients who present with neck and back pain do not need immediate diagnostic evaluation and initially should be treated conservatively, certain historical features should lead to the consideration of an

immediate and thorough study of the patient with new onset neck/back pain with or without radiating pain into extremity. These historical features include the following factors:

- Age >50
- Body temperatures $>38^{\circ}C$
- Neuromuscular weakness
- Significant trauma before the onset of pain
- History of malignancy
- Pain at rest in the recumbent position
- Unexplained weight loss
- Drug and alcohol abuse (increased risk of infection and possibly unremembered trauma)

Evaluation in the emergency room

Neurologists generally do not see patients early in the course of low back pain. Occasionally, however, a neurologist may be called to the emergency room. It is clear that patients who present to the emergency room with the acute onset of focal neurologic deficits, such as weakness in the lower extremities or bladder and bowel disturbances, require an immediate and detailed evaluation, usually including a neuroimaging procedure. Patients who present with severe low back pain and abdominal symptoms also should be evaluated for a leaking aortic aneurysm or other acute abdominal disorders.

Physical examination

The experienced neurologist knows that the neurologic examination of the patient with neck and low back pain can be altered by the pain itself. For example, when testing strength, guarding must be taken into account. Tendon reflexes may be suppressed as a result of poor relaxation of a limb as a consequence of pain. Preparing the patient by explaining each step of the examination in advance may reduce anxiety and encourage relaxation, thereby reducing guarding and enhancing the reliability of the examination itself.

General examination

The necessity for a general physical assessment in the patient who complains of back pain cannot be underestimated. The presence of a low-grade fever, for example, may signal infection that involves the vertebral column, the epidural space, or the surrounding muscle (eg, psoas abscess). Inspection of the skin for lesions may yield diagnostic information (Box 2). Changes in the rectal examination, including sphincter tone, anal "wink," and the bulbocavernosus reflex, may reflect changes in the spinal cord or cauda equina, whereas an abnormal prostate may lead to a diagnosis of prostate cancer with spinal metastases.

Box 2. Skin lesions and spine pain

Psoriasis—psoriatic arthritis
Erythema nodosum—inflammatory disease, cancer
Café-au-lait spots—neurofibromatosis
Hydradenitis suppurativa—epidural abscess
Vesicles—herpes zoster
Needle marks (intravenous drug abuse)—vertebral column infections
Subcutaneous masses—neurofibroma, lymphadenopathy

Data from Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:18.

The abdominal examination may be particularly important. The presence of abdominal tenderness, organomegaly, or a pulsatile abdominal mass with a bruit in a patient with low back pain should immediately direct an urgent diagnostic evaluation, which may lead to a potentially lifesaving diagnosis, such as a leaking abdominal aortic aneurysm. In patients with low back pain and claudication, evaluation of the peripheral pulses in both lower extremities is essential to help distinguish neurogenic claudication from vascular claudication.

Neurologic examination

Low back pain

Inspection of the low back can be of value. The presence of a tuft of hair over the lumbar spine suggests diastematomyelia/spina bifida occulta. Percussion may produce pain over an infected area or at the site of a malignancy. Palpation of the paraspinous muscles may demonstrate spasm as a cause, or accompaniment, of acute low back (and neck) pain. The concept of spasm itself as a cause of back pain has been challenged.

Posture while standing may be altered by a herniated lumbar disc. Splinting with list away from the painful lower extremity is seen with lateral lumbar disc herniation, whereas list toward the painful side can be seen with medial herniation. Tilting the trunk to the side opposite the list can cause additional nerve root compression, with resultant accentuation of radicular distribution pain. Patients with neurogenic claudication secondary to compression of the cauda equina may tend to stand and walk with the trunk flexed forward, which reduces compression by widening the anteriorposterior dimension of the lumbar canal. Walking with the trunk extended may accentuate the symptomatology. Lumbar spine mobility is usually reduced in patients with low back pain, but because there is such wide variability

as a result of conditioning and age, a measurement of degrees of mobility is usually not useful. Evaluation of the gait is of fundamental importance to seeking, for example, evidence of

- An antalgic gait that favors the side of a lumbar radiculopathy
- ''Foot slap'' (ie, foot drop) secondary to weakness of dorsiflexors of the foot, found with an L5 radiculopathy
- Trendelenburg gait (''drop'' of ipsilateral side of pelvis as foot is lifted), which signals proximal (unilateral or bilateral) lower extremity weakness.

Neuromechanical tests are an important adjunct to the traditional neurologic examination in patients with low back pain and sciatica.

- **Straight leg raising test:** With the patient in the supine position, the symptomatic lower extremity is slowly elevated off the examining table. The spinal nerve and its dural sleeve, tethered by a herniated disc, are stretched when the lower extremity is elevated between 30° and 70°. This movement accentuates the radiating pain (''sciatica''). Increased pain at less than 30° and more than 70° is nonspecific (Fig. 4).
- **Lasegue test:** A variation of the straight leg raising test. With the patient in the supine position, the symptomatic lower extremity is flexed to 90° at the hip and knee. The knee is then slowly extended, which produces radiating pain with L5 and S1 nerve root compression.

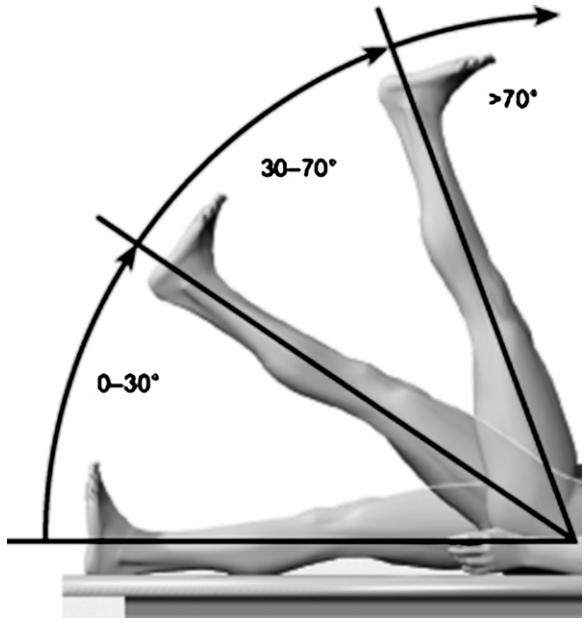

Fig. 4. The straight leg raising test. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:20; with permission.)

- **Bragard's sign (test):** After a positive straight leg raising test, the elevated extremity is lowered to the point of pain resolution. The foot is then dorsiflexed by the examiner. If this movement recreates the pain, the test is positive (Fig. 5).
- **Contralateral ("well") straight leg raising test:** Performed on the asymptomatic lower extremity, this test has specificity but low sensitivity for disc herniation.
- **Prone straight leg raising test:** With the patient in the prone position, the symptomatic lower extremity is slowly extended at the hip by the examiner. Accentuation of pain in the anterior thigh suggests a "high" lumbar (L2, L3) radiculopathy (Fig. 6).
- **Valsalva test:** This maneuver increases intrathecal pressure, which accentuates radicular pain in the presence of spinal nerve compression and inflammation.
- **Brudzinski test:** With the patient supine, the head is flexed by the examiner, which aggravates radicular pain in the presence of spinal nerve compression.
- **Patrick's (Faber) test:** The lateral malleolus of the symptomatic lower extremity is placed on the patella of the opposite extremity, and the symptomatic extremity is slowly externally rotated. Accentuation of pain favors a lesion of the hip or sacroiliac joint as the cause for the pain (Fig. 7).
- **Gaenslen test:** With the patient supine and the symptomatic extremity and buttock slightly over the edge of the examination table, the asymptomatic lower extremity is flexed at the hip and knee and brought to the chest. The symptomatic lower extremity is extended at the hip to the floor. Increased nonradiating low back and buttock pain indicates sacroiliac joint disease (Fig. 8).
- **Waddell test:** Excessive sensitivity to light pinching of the skin in the region of the low back pain suggests a functional component.

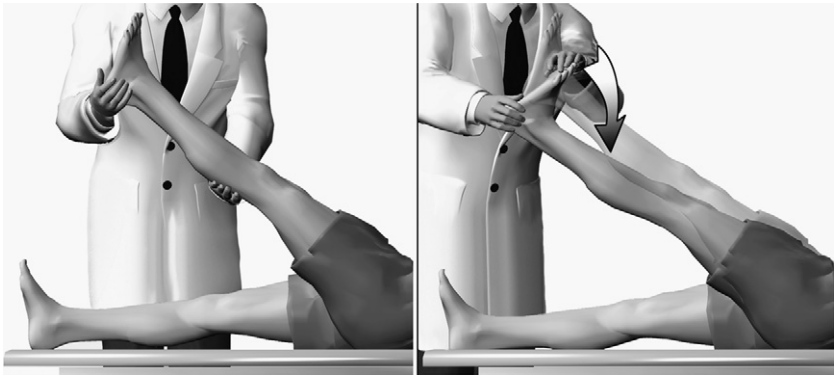

Fig. 5. Bragard's sign. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:20; with permission.)

Fig. 6. Prone straight leg raising test. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:21; with permission.)

Lumbosacral root testing is the essence of the neurologic examination in patients with back pain and a suspected lumbosacral radiculopathy. Each myotome and dermatome must be carefully evaluated (Table 2). There are several pitfalls to be avoided in this portion of the examination. Guarding secondary to pain may simulate weakness, but this is usually diffuse and not specific to a given myotome. Reflexes may be suppressed secondary to poor relaxation. The sensory examination is usually less useful than the history of the distribution of paresthesia, particularly early in the course of a radiculopathy.

Neck pain

Inspection of the head and neck, noting reduced spontaneous head movement, head tilt, and neck deformity all raise the possibility of an underlying vertebral disorder or deformity. Palpation and percussion of the neck, as

Fig. 7. Faber maneuver. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:22; with permission.)

Fig. 8. Gaenslen test. (*From* Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (NY) 2001;7:22; with permission.)

with the low back, have a low yield with regard to identifying a specific process, but paracervical tenderness or other changes such as palpation of a mass offer support of the diagnosis of a vertebral column disorder.

Gait assessment is also important in patients with neck pain, because evidence of myelopathy may appear. Unilateral or bilateral spastic, ataxic, spastic-ataxic, or Trendelenberg gait all signal a possible cervical myelopathy.

Neuromechanical tests, as with low back and lower extremity pain, are useful in the assessment of patients with neck and upper extremity pain.

- **Spurling test:** The head is inclined toward the side of the painful upper extremity and then compressed downward by the examiner. Pain and paresthesia that radiate into the symptomatic extremity strongly suggest nerve root compression, usually secondary to disc herniation. (It should be noted that lateral head movement away from the symptomatic extremity sometimes can accentuate pain and paresthesia in the symptomatic upper extremity, secondary to stretching a compressed nerve root.)
- **Traction ("distraction") test:** Lifting (traction) on the head may relieve cervical spinal nerve compression and reduce upper extremity pain and paresthesia.
- **Valsalva test:** As with low back pain, the Valsalva maneuver with resultant increased intrathecal pressure can accentuate neck and upper extremity symptoms.
- **Lhermitte's test:** In patients with myelopathy that affects the posterior columns, neck flexion can produce paresthesia, usually in the back but sometimes into the extremities. As is familiar to neurologists, Lhermitte's sign is most commonly associated with an inflammatory process, such as multiple sclerosis but it is sometimes noted with spinal cord compression.
- **Adson's and hyperabduction tests:** Long used in the evaluation of suspected thoracic outlet syndrome, these tests are nonspecific and unreliable. With the patient sitting erect and the upper extremities at the

side (Adson) or the symptomatic upper extremity abducted and extended (hyperabduction), the radial pulse is palpated. The test results are positive if the pulse disappears and paresthesia develops in the hand of the symptomatic extremity.

Cervical root and spinal cord tests

As with the evaluation of low back pain, cervical root testing is central to the neurologic evaluation of a patient with neck and upper extremity pain. In addition to cervical root involvement, the possibility of associated spinal cord compression makes the examination of the lower extremities essential.

Motor examination

In additional to evaluating the strength of each cervical myotome for evidence of a cervical radiculopathy as such, assessment of strength and tone of the lower extremities is required to rule out a cervical myelopathy.

Reflexes

Cervical radiculopathy and myopathy in combination may result in loss of a tendon reflex at the level of the lesion with heightened reflexes below the level of the lesion. All reflexes may be lost with an acute myelopathy during periods of diaschisis (''spinal shock'').

Sensation

In addition to testing for a dermatomal pattern of sensory loss, a segmented checking for ''cord'' level also should be sought. Spinal cord compression may be associated with paresthesia and sensory disturbance confined to the upper extremities as a result of a so-called *central cord syndrome* with involvement primarily of decussating anterior sensory fibers [19].

Sympathetic function

Lesions in the superior thoracic spine may affect the T2 spinal nerve and produce pain in the upper back, shoulder, and proximal upper extremity along with an ipsilateral Horner's syndrome.

Summary

A careful history and physical examination are of primary importance in the evaluation of a patient with spine pain and related symptoms. It can be the difference between sending a patient home with a conservative treatment plan and admitting the patient for an immediate evaluation and possible surgery. In this same vein, the history and physical examination can determine if an expensive evaluation is necessary immediately or whether conservative treatment is appropriate first.

References

[1] Williams A, Newell RLM. Back and macroscopic anatomy of the spinal cord. In: Standing S, editor. Gray's anatomy. 39th edition. New York: Elsevier Churchill Livingstone; 2005. p. 727–87.
[2] Jackson R. The facet syndrome: myth or reality? Orthop Clin North Am 1992;279:110–21.
[3] Boos N, Weissbach S, Rohrbach H, et al. Classification of age-related changes in lumbar intervertebral discs. Spine 2002;27:2631–44.
[4] Roberts S, Evans H, Trivedi J, et al. History and pathology of the human intervertebral disc. J Bone Joint Surg Am 2006;88(Suppl 2):10–4.
[5] Battie MC, Videman T. Lumbar disc degeneration: epidemiology and genetics. J Bone Joint Surg Am 2006;88(Suppl 2):3–9.
[6] Brisby H. Pathology and possible mechanisms of nervous system response to disc degeneration. J Bone Joint Surg Am 2006;88(Suppl 2):68–71.
[7] Levin KH, Covington EC, Devereaux MW, et al. Neck and low back pain. Continuum (N Y) 2001;7:1–205.
[8] Korkala O, Gronblad M, Liese P, et al. Immunohistochemical demonstration of nociceptors in the ligamentous structures of the lumbar spine. Spine 1985;10:156–7.
[9] Palmgren T, Gronblad M, Virri J, et al. An immunohistochemical study of nerve structures in the annulus fibrosus of human normal lumbar intervertebral discs. Spine 1999;24:2075–9.
[10] Coppes M, Marani E, Thomeer R, et al. Innervation of ''painful'' lumbar discs. Spine 1997; 22:2342–9.
[11] Lotz JC, Ulrich JA. Innervation inflammation and hypermobility may characterize pathologic disc degeneration: review of animal model data. J Bone Joint Surg Am 2006;88(Suppl 2):76–82.
[12] Deyo RA, Cherkin D, Conrad D, et al. Cost controversy crises: low back pain and the health of the public. Annu Rev Public Health 1992;12:141–55.
[13] Johnson EW. The myth of skeletal muscle spasm [editorial]. Am J Phys Med 1989;68:1.
[14] Mense S, Simons D. Muscle pain understanding is nature, diagnosis and treatment. Baltimore (MD): Lippincott, Williams & Wilkins; 2001. p. 117–8.
[15] Devereaux MW. The neuro-ophthalmologic complications of cervical manipulation. J Neuroophthalmol 2000;20:236–9.
[16] Groen GJ, Baljet B, Drukker J. Nevers and nerve plexuses of the human vertebral column. Am J Anat 1990;188:282–96.
[17] Boden SD, Davis DO, Dina TS, et al. Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. J Bone Joint Surg Am 1990; 72:403–8.
[18] Jensen M, Brant-Zawadzki M, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med 1994;331:69–73.
[19] Voskuhl R, Hinton R. Sensory impairment in the hands secondary to spondylotic compression of the cervical spinal cord. Arch Neurol 1990;47:309–11.