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Neurologic Approach to Diagnosis of Low Back Pain

INTRODUCTION

Low back pain is extremely common and has major economic significance in industrial societies. It is reported to occur in 26% of the working population each year and occurs to a disabling degree in 2 to 8%. Eighty percent of the population have at least one episode of low back pain in their lifetimes. It is the fifth leading reason for medical office visits in the United States. Low-back injury compensation accounts for 33% of all workers' compensation costs (1/3 for medical treatment, 2/3 for indemnity). Seventy-five percent of compensation payments go to back patients, although they constitute only 3% of total compensation patients (Klein et al, 1984; Hart et al, 1995).

Low back pain occurs most frequently between the ages of twenty and forty and is more severe in older patients. There is no strong association based on sex, height, body weight, or physical fitness. High-risk occupations include miscellaneous labor, garbage collection, warehouse work, and nursing, all of which are usually associated with lifting, twisting, bending, and reaching.

Prognosis

The typical attack involves 35 days (median) of pain and 9 to 21 days out of work. Those who are out of work longer than 6 months have only a 50% likelihood of returning. This drops to 25% after more than 1 year out and to nil after 2 years. After an initial episode, the probability of recurrence is increased fourfold. Treatment tends to be less successful in workers' compensation cases. The average cost for care is 4.5 times greater if the patient is represented by an attorney.

CLASSIFICATION

Low back pain is an important part of neurologic and general medical practice. Of all office visits for back pain, 56% are to family practitioners and internists, 25% are to orthopedic surgeons, 7% are to neurosurgeons, and 4% are to neurologists. These patients make up 10% of the average neurologist's caseload (Hart et al, 1995). The neurologist is usually called upon to evaluate and treat the patient with acute or subacute pain and symptoms and signs of nerve-root irritation—radiating pain, weakness, numbness, and bladder or bowel symptoms. Such patients make up a small minority of those with low back pain. In many of these cases, the neurologist is also asked to assess the presence and degree of impairment (physical defect) and disability (what the patient can or can't do as a result of this defect) (AMA, 1993).

There are a multitude of causes of low back pain. Even when there is evidence of nerve-root involvement (radiculopathy), not every patient has a herniated lumbar disc. The classifications outlined in Table 1 may be helpful in the differential diagnosis.

Key words: back pain, pain, lumbar spine, lumbar disks

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CLINICAL EVALUATION

As for any neurologic patient, the general physical and neurologic examinations and history are essential. This discussion will emphasize areas of special concern in the evaluation of back pain.

History

Particular note should be made of any preceding trauma, prior attacks of pain, prior evaluations, prior or current treatment, and the duration and progression of symptoms. The patient should be asked about weakness; numbness; dysesthesias and paresthesias; bladder, bowel, and sexual dysfunction; and any accompanying abdominal or flank pain.

Pain

Inquire as to the quality, location, and radiation of pain, and any exacerbating or relieving factors and activities. Severe, constant back pain persisting at night suggests the presence of neoplasm, infection, or lateral recess nerve-root compression. Pain fibers are present in the annulus surrounding the disc (in the spinal ligaments, facets, and joint capsules) but not in the intervertebral disc itself. Nerve-root pain is usually brief, sharp, and shooting, is often increased by coughing, straining, standing, or sitting, and is usually relieved by lying down.

Peripheral-nerve or plexus pain is usually described as burning, tingling (pins and needles), or “asleep” or numb in quality; it is usually worse when the patient is lying down at night. (See Table 2.) In painful radiculopathies and mononeuropathies, the area of pain and sensory abnormality may extend beyond the known sensory distribution of the affected peripheral nerve or beyond the dermatome of the affected-root or dorsal-root ganglion, as in postherpetic neuralgia. This phenomenon has been attributed to central nervous system plasticity. In most nerve-root syndromes, however, a precise description of radiating pain will help localize to a nerve-root level.

Physical Examination

The general physical examination can be as important as the neurologic examination and should include the vasculature (especially the pedal pulses), abdomen, inguinal areas, and rectum (especially if a cauda equina syndrome is suspected). The patient should be undressed.

Watch how the patient moves, sits, and stands. Look for atrophy (measure the calf and thigh circumferences for asymmetry), fasciculations, pelvic tilt (“bad” side is down), involuntary knee flexion (to guard against root traction), scoliosis, and café au lait spots (they might indicate neurofibromatosis). Gait testing should include walking on heels and on toes.

TABLE 1 Causes of Low Back Pain

Radicular—evidence of nerve-root involvement

Intraspinal causes:

- Proximal to the disc (conus and cauda equina)—neurofibroma, ependymoma, meningioma
- Disc level—herniated intervertebral disc, spinal stenosis (canal or lateral recess), synovial cyst of facet joint
- Vascular—arteriovenous malformation (AVM) of spinal cord, spinal dural AV fistula

Extraspinal causes:

- Pelvic—vascular, gynecological (endometriosis), sacroiliac joint, retroperitoneal neoplasms affecting the lumbosacral plexus, lumbosacral plexitis
- Peripheral nerve—mononeuropathy, polyneuropathy (diabetic and other), trauma, local neoplasm, herpes zoster (shingles)

Nonradicular—no evidence of nerve-root involvement

Traumatic causes:

- musculoskeletal strain, vertebral-compression fracture, transverse-process fracture

Chronic or subacute causes:

- spondylosis and degenerative disc disease, spondylolisthesis, sacroiliac joint disease, muscular (chronic and repeated strains), deconditioning, postural, “fibromyalgia”

Nonmechanical causes:

- Referred pain—abdominal or retroperitoneal (e.g., abdominal aortic aneurysm, pancreatic disease, endometriosis)
- Infection—bone, disc, epidural, urinary tract (especially in women)
- Neoplasm of vertebrae or epidural space—metastatic tumor, multiple myeloma, primary bone tumor
- Rheumatologic disease—ankylosing spondylitis, degenerative disease, and other arthritides
- Miscellaneous metabolic and vascular—osteopenia with compression fracture, Paget’s disease, etc.
- Psychogenic

Adapted from Macnab and McCulloch, 1990.

Palpate the lower spine, paraspinal muscles, sciatic notches, and sciatic nerve looking for tenderness, muscle spasms, and radiating pain. Muscle tenderness may be associated with nerve-root irritation (calf muscles with S1, anterior tibial muscles with L5, and quadriceps with L4).

Nerve Root Stretching

Roots may be impinged upon or tethered by herniated discs or other lesions, so that stretching the root causes pain. This should be tested by having the patient bend forward or by straight-leg raising (SLR).

TABLE 2 Root Pain and Peripheral Nerve Pain

	<i>Root Pain</i>	<i>Peripheral Nerve Pain</i>
Duration	Brief	Continual
Quality	Sharp, shooting	Burning, "asleep," numb
Worse with	Coughing, straining, standing, sitting	Lying in bed at night
Relieved by lying down	Yes	No

SLR is performed by raising the extended leg of a supine patient to determine whether this action elicits pain in the leg, buttock, or back, and, if so, at what angle from the horizontal the pain occurs. The pain is usually worsened by dorsiflexion at the ankle and relieved by flexion of the knee and hip. Positive SLR results usually indicate S1 or L5 root irritation. Pain occurring in the contralateral, symptomatic leg when the asymptomatic leg is raised is considered a positive crossed SLR test, which usually indicates the presence of a disc herniation medial to the nerve root, often with an extruded disc fragment. Reverse SLR tests detect L3 or L4 root irritation. The patient lies prone or on his side, and the thigh is extended at the hip joint. If the patient has hip or groin pain, the examiner should rotate the hip; pain on hip rotation suggests hip disease rather than radiculopathy.

Motor Examination

It is helpful to bear in mind certain features of the motor examination of the lower extremities in patients with low back pain. Since it is difficult to detect proximal leg weakness when the patient is lying down, it may be necessary to ask her to attempt to rise from a squatting position. Similarly, gastrocnemius weakness is easiest to detect with repeated rising up on the toes. Toe flexors and extensors usually become weak before the foot muscles do. If the gluteus maximus (supplied by S1) is weak, one buttock may sag; gluteus medius (L5) weakness may cause a lurching or waddling (Trendelenburg) gait. In a patient with root pain, do not test the dorsiflexors of the foot with the knee extended, since this may stretch the S1 or L5 root and increase sciatic pain. For the same reason, quadriceps strength should be tested with the patient prone. Muscles are innervated by more than one nerve root, so total paralysis implies a lesion of multiple roots or of peripheral nerves. Even if a single root has been severed, there is little weakness. Atrophy is rarely seen unless symptoms have been present for more than three weeks. Severe atrophy should raise the suspicion of an extradural neoplasm.

Sensory Examination

A dermatomal distribution of loss of pinprick and touch sensation indicates and localizes root involvement (Figure 1). Because there is a wide overlap of root distributions, a single root lesion usually causes mild hypalgesia. The examiner may not be able to detect any sensory deficit, even though the patient has sensory symptoms.

Tendon reflexes

Asymmetry of the ankle and knee jerks can be helpful in identifying the affected nerve root.

Nerve-Root Syndromes

In S1 nerve-root syndromes (see Table 3), leg pain is often worse than low back pain. Pain and paresthesias are felt in the buttock, posterior thigh, posterolateral calf and heel, and sometimes in the lateral foot and last two toes. Numbness and pinprick hypalgesia may be in the fifth toe and lateral foot, and, to a lesser degree, in the posterolateral calf and posterolateral thigh. There may be weakness of the toe flexors, the gastrocnemius, and (rarely) the hamstrings as well as toe abduction and eversion of the foot. The ankle jerk is often diminished or absent.

In L5 nerve-root syndrome, low back pain is often worse than leg pain. Pain and paresthesias radiate to the posterolateral thigh, groin, lateral calf, dorsomedial foot, and first two toes. Numbness and hypalgesia may be found in the great toe and medial foot, and, to a lesser extent, the anterolateral calf. Weakness may be noted in the extensor hallucis longus (EHL), the tibialis anterior (TA), and peroneal muscles, causing a foot drop. There is usually no reflex loss.

In L4 and L3 nerve-root syndromes, low back pain is worse than leg pain. There may be some anterior thigh pain. Numbness and hypalgesia may be present over the anteromedial thigh and knee. Weakness may be detected in the quadriceps and iliopsoas muscles. The knee jerk is often diminished or absent.

Inconsistencies

Since some patients with back pain may exaggerate their symptoms, especially in medicolegal or workers' compensation cases, it is important to detect inconsistencies in the clinical presentation (Macnab and McCulloch, 1990; Waddell et al, 1980). The history may indicate that the patient is able to engage in activities inconsistent with the severity of his complaints. Symptoms may be described in an exaggerated or histrionic manner. During the examination, tenderness may be elicited by minimal pressure or over areas where pain would not be expected. Axial loading, by pressing down on top of the head, or rotating the body at the hips or shoulders should not elicit low back

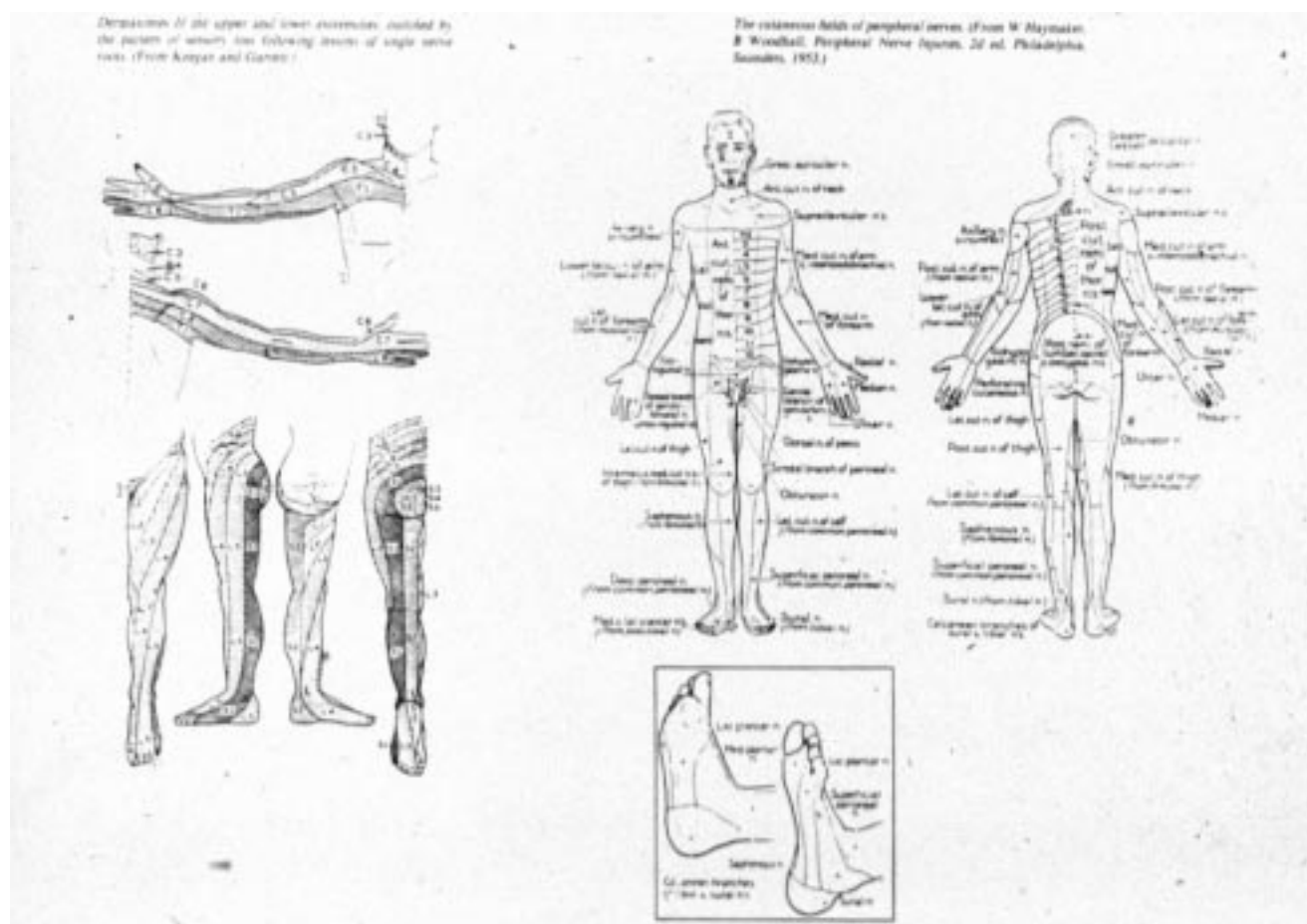


FIGURE 1 Dermatomal and peripheral nerve sensory distributions (from Keegan, JJ, Garrett, FD. *Anat. Rec.* 102:411, 1948; and Haymaker, W, Woodhall, B. *Peripheral Nerve Injuries*. 2d ed. Philadelphia. Saunders. 1953).

TABLE 3 Nerve-Root Syndromes

Root	Pain and Paresthesias	Hypesthesia	Motor Disturbance	Reflex Loss	Stretch
S1	Buttock, post. thigh, postlat. calf, heel, lat. foot, 4th and 5th toes. Leg pain worse than back pain	Lat. foot, 4th and 5th toes, (sometimes) postlat. calf and post. thigh	Toe flexors, gastrocnemius, (rarely) hamstrings and toe abductors	Ankle jerk	SLR
L5	Postlat. thigh, groin, lat. calf, dorsomed. foot, first 2 toes. Back pain worse than leg pain	First toe, medial foot, anterolat. calf	EHL, TA, peroneii (foot drop)	None	SLR
L4 L3	Anteromed. thigh, anteromed. shin. Back pain worse than leg pain	Anteromed. thigh, knee	Quadriceps, iliopsoas	Knee jerk	Reverse SLR

TABLE 4 Laboratory Evaluation of Low Back Pain

<i>Symptoms</i>	<i>Laboratory Tests Indicated</i>
Pain is chronic, incompletely relieved by lying down, or constant nocturnal or Patient is more than sixty years of age, has unexplained weight loss, or has a history of cancer (situations in which myeloma or other neoplasms should be of concern)	ESR, Glucose, IEP, PSA, Urine Bence-Jones proteins, LS spine X rays, possible bone scan
Motor, bladder, bowel, or sexual-function deficits	MRI (contrast-enhanced if there has been prior back surgery) or CT*
History suggestive of lumbar spinal stenosis	MRI or CT *
Patient has had a fusion, and instability might be causing pain	Flexion-extension LS spine X rays
Surgery is being considered (especially in cases of spinal canal stenosis, lateral recess stenosis, multiple abnormal discs, or possible neoplasm)	Lumbar myelogram, CT

*See Miller et al, 1989.

pain. It may be helpful to distract the patient, to see whether there is more mobility in spontaneous activities than elicited during the formal examination. The patient may be observed during dressing (especially shoes and socks) and getting on and off the examination table. Straight-leg raising (SLR) should be tested when she is in a sitting position (e.g., while testing the plantar responses or measuring the calf circumferences) as well as when supine. There should be no significant difference in the degree of hip flexion with sitting or supine SLR or when the patient bends forward while standing. Nondermatomal or otherwise nonanatomic distributions of sensory loss should also be noted. In the motor examination, sudden giving way, jerky movements, or weakness appearing to involve many muscle groups should raise suspicion. Note whether the patient appears to overreact during the examination, including what appears to be a disproportionate verbalization of pain on minimal provocation, dramatic facial expressions, muscle tension and tremors, collapsing when asked to bear weight, and requiring a companion for dressing and undressing. These observations must be considered in the context of the overall examination. Severe pain can cause extreme reactions in some patients.

TABLE 5 Management Guidelines

Moderate pain; no neurologic deficit or mild to moderate deficit	Pain medications, muscle relaxants, heat, back precautions; neurology consultation, if deficit; follow-up examination in 2 weeks, if deficit; if symptoms improve, refer to physical therapy (back exercises, "back school"), or back exercises at home
Severe pain; no neurological deficit or mild to moderate deficit	Bed rest up to 10 days (depending on pain severity and response to rest), pain medications, muscle relaxants, heat; neurology consultation, if deficit; Follow-up examination in 2 weeks, if deficit; if symptoms improve, physical therapy or home exercises
Severe deficit (neurologic, bladder, bowel, sexual) or Increasing neurologic deficit; severe pain not improved by 1+ week of bed rest	Neurology or neurosurgery consultation and MRI on an urgent basis; if a large-mass lesion is found, refer to a neurosurgeon or orthopedic surgeon Neurology consultation; MRI or CT; if large-mass lesion found, refer to neurosurgeon or orthopedic surgeon; if no large lesion, up to one more week of bed rest, then refer to physical therapy or home exercises
No improvement or incomplete improvement after bed rest, gradual ambulation, physical therapy, and home exercises	MRI or CT if not already done; consider referral to neurologist, neurosurgeon, or orthopedic surgeon; consider epidural steroids

LABORATORY EVALUATION

Laboratory tests are not necessary in every patient. They are important to verify the diagnosis if surgery is contemplated and are sometimes necessary to clarify the differential diagnosis. Since X rays, CT and MRI scans, myelograms, and other tests may be abnormal in asymptomatic patients, the results must be interpreted with the entire clinical presentation in mind. Some guidelines are outlined in Table 4. (For a detailed discussion, see "Laboratory Evaluation of Low Back Pain" in this issue.)

MANAGEMENT

Most patients with back pain will respond to conservative treatment. Guidelines appropriate to the primary care physician as well as to the neurologist are outlined in Table 5. (For a more detailed discussion, see "Management of Low Back Pain" in this issue.)

REFERENCES

- Klein BP, Jensen RC, Sanderson LM: Assessment of workers' compensation claims for back strains/sprains. *J Occup Med* 26:443, 1984.
- Hart LG, Deyo RA, Cherkin DC: Physician office visits for low back pain. Frequency, clinical evaluation, and treatment patterns from a US National Survey. *Spine* 20:11, 1995.
- American Medical Association. Guides to the evaluation of permanent impairment, 4th ed. Chicago: AMA, 1993.
- Macnab I, McCulloch J: *Backache*, 2nd ed. Baltimore: Williams and Wilkins, 1990.
- Waddell G, McCulloch JA, Kumel EG, et al: Nonorganic signs in low back pain. *Spine* 5:117, 1980.
- Miller GM, Forbes GS, Onofrio BM. Magnetic resonance imaging of the spine. *Mayo Clin Proc* 64:986, 1989.

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