Low back pain, a leading cause of disability in the United States, has a significant economic impact not only on lost productivity but also on healthcare expenditures. Approximately a fifth of patients will see multiple physicians in their quest for relief of low back pain. Primary care physicians therefore play a crucial role in the initial approach to these patients. A thorough history and physical examination directed toward the neurologic, orthopedic, and osteopathic evaluation are essential. This article reviews the diagnosis and assessment of pain levels and a triad system of therapy involving cortical, spinal, and peripheral levels. Options include antidepressants, neuroleptics, neurostimulants, and osteopathic manipulative treatment (OMT) (cortical level); opiates, tramadol hydrochloride, and transcutaneous electrical nerve stimulators (spinal level); and nonsteroidal anti-inflammatory drugs, epidural injections, spinal blocks, antispasmodics, physical therapy, muscle relaxants, exercise, and OMT (peripheral level). By choosing a modality directed at each level, the clinician may provide the patient with a pain management program that will maximize the chosen mode of therapy and restore function and mobility.

(Key words: low back pain, neuromusculoskeletal examination, sacroiliac dysfunction, hip dysfunction, lumbosacral strain, lumbosacral sprain, radiculopathy, myopathy, neuropathy, sciatica, pain control, physical therapy, osteopathic evaluation, osteopathic manipulative treatment)

Low back pain is the second most common cause of absence from the workplace among people younger than 55 years, second only to the common cold. Among people younger than 35 years, back pain is the leading cause of disability in the United States. Pain is responsible for 50 million the leading cause of disability in the Unit-

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mastic reflex and the superficial anal reflex can indicate a significant upper motor neuron lesion corresponding to L1, L2 and S2, S3, S4, respectively. The finding of an unexplained upper motor neuron pathologic process on physical examination should be immediately correlated with head and spine magnetic resonance imaging (MRI) or computed tomography (CT) scans.

A lower motor neuron pathologic process will present with decreased reflexes and weakness on physical examination. The triceps surae reflex, or Achilles tendon reflex, corresponds to the first and second sacral level; the patellar reflex corresponds to the third and fourth lumbar segment; the medial hamstring reflex corresponds to the fifth lumbar and the first and second sacral segment.

Dural tension test, or Lasègues’s test, is a classical test to indicate a proximal nerve impingement. The straight-leg test (SLT) is performed with the patient supine, and the lower extremity is slowly raised. With a positive test, radicular symptoms will be present at less than 70 degrees of flexion, indicating irritation of the sciatic nerve. Studies show that the test is very sensitive to lumbar disk herniation but not highly specific. Nonneurologic etiologies of a positive SLT include tight hamstrings, muscle spasms, and sprained posterior longitudinal ligament sprain.

Manual muscle testing can be extremely helpful in determining weakness corresponding to a neurologic level. Weakness of great toe dorsiflexion corresponds to the fifth lumbar segment. Weakness of ankle plantar flexion corresponds to the first sacral segment. If the dorsal root component of the lower motor neuron is compressed, there will be a corresponding sensory abnormality in that sensory dermatome. Sensory abnormalities include hypoesthesia, burning, electrical sensations, or allodynia (a painful sensation to a nonpainful mechanical stimulation such as clothing or bed covers).

The evaluation of the vascular system of the patient with low back pain includes detection of any ischemic changes in the lower extremity. Palpation of the pulses in the lower extremity should be a routine part of the evaluation of the patient with low back pain. Vascular claudication presents as bilateral leg pain that begins at a fixed distance when ambulating and is relieved by standing. Pseudoclaudication, or neurogenic claudication (associated with spinal stenosis), is relieved only by sitting or forward flexion of the lumbar spine.

The orthopedic evaluation of the low back includes gait, posture, standing balance, crouching, range of motion, SLT, and fabere (flexion, abduction, external rotation, extension) sign (Patrick’s test). A complete orthopedic evaluation includes evaluation of leg-length discrepancies, segmental and intersegmental somatic dysfunctions, sacroiliac dysfunctions, and sources of referred pain from the abdomen and viscera. Each lumbar and sacral segment should be palpated for tenderness. Mobility of the spine should be evaluated in flexion, extension, sidebending, and rotation. The patient should be evaluated sitting, standing, supine, and prone. Patrick’s test, consisting of flexion, abduction, and external rotation of the hip, can evaluate both hip and sacroiliac dysfunction.

Causes of low back pain
Causes of low back pain are listed in Figure 3. Metabolic abnormalities are the most common clinical finding associated with global deep tendon hyperreflexia and upper motor neuron findings. Syndromes include a thyroid pathologic lesion, electrolyte imbalances, and drug toxicities. Parkinson’s disease can present with low back pain and leg pain. Physical findings are increased tone and shuffling gait. Primary or metastatic space-occupying lesions should always be considered, especially if there is a history of cancer. Cerebrovascular accidents (CVAs) can present with both neck and low back pain. Pain associated with CVAs is secondary to increased tone or even pain syndromes such as complex regional pain syndromes or hand-shoulder syndrome. Myelopathy, both cervical and lumbar, can result from central disk herniations and severe spinal stenosis or chronic disease processes such as the human immunodeficiency virus (HIV) infection. Diagnosis can be made with CT or MRI.

The conditions that will present with lower motor neuron symptoms are impingement syndrome of the proximal nerve root (radiculopathy), neuropathy, and myopathy. Impingement syndrome resulting from dorsal root compression of the proximal lower motor neuron complex accounts for the majority of true “sciatica.” Although no definition of sciatica is universally agreed on, most practitioners consider it a neuropathic injury to the sciatic or proximal tibial nerve. If neuropathic injury is involved, proximal tibial nerve neuropathy is probably more appropriate nomenclature. Disk herniation is a common cause of sciatica; however, not all disk herniations result in a painful process. It has been demonstrated that 20% to 30% of healthy, asymptomatic patients have CT- or MRI-identified herniated disks. The most common symptoms of radiculopathy are pain,
reflex loss, sensory changes, and weakness. Computed tomography or MRI can be diagnostic if disk disease is present. Electromyography (EMG) and nerve conduction studies (NCS) may be helpful in determining chronicity and recovery of the nerve or detection of an injury at the nerve root that is not related to disk disease or bony stenosis.

Myopathy is an abnormal breakdown of muscle and may present as pain involving the back and proximal region of the leg. It is usually bilateral and involves the proximal muscle groups. It also can be associated with decreased reflexes and pain in a myotomal distribution. Causes include disuse myopathy, inflammatory processes (that is, dermatomyositis, polymyositis, hepatitis, or HIV,14 and side effects of pharmacotherapy (“statin” drugs). If myopathy is suspected, it is recommended that serum creatine kinase levels be measured. Diagnosis can be made with EMG and NCS or muscle biopsy.

Spinal stenosis, cauda equina syndrome, and multiple sclerosis should be considered when a patient has a combination of mixed upper and lower motor neuron findings on physical examination. By definition, spinal stenosis is a narrowing of the spinal canal or foramen (or both). Patients with central canal stenosis will have bilateral neurologic symptoms, whereas patients with foraminal stenosis will have unilateral symptoms. Causes include discogenic disease, degenerative arthritis, spondylolisthesis, or a congenitally narrow spinal canal or foramen. Many times, the patient will have normal findings on neurologic examination but the history will point to pain with walking which radiates to the buttocks and legs and is relieved only by bending or sitting, that is, neurogenic claudication. Symptoms progress slowly over a course of years. Sudden onset of symptoms with urinary retention may indicate an acute cauda equina syndrome. Other symptoms include bowel incontinence, sexual dysfunction, bilateral lower extremity pain, and diminished perineal sensation. Fifty percent of acute cauda equina syndromes are due to tumors causing central canal stenosis.11(p39) Acute cauda equina syndrome is considered a surgical emergency. Diagnosis can be made with MRI or CT.

Orthopedic causes of low back pain are mechanical alterations in the spine. Patients with lumbar spondylolisthesis or degenerative disk disease will have nonradicular back pain. They describe general or point tenderness in the low back. Physical activity usually makes the pain worse, and rest relieves it. The examination will demonstrate a decreased lumbar lordosis, paraspinal tenderness, and spasm. Diagnosis can be made with a plain x-ray film. Facet (zygapophyseal) joint pain may account for up to 15% to 40% of low back pain.11(pp36-90) Patients have low back pain that may radiate to the buttocks which is made worse by extension and rotation of the lumbar spine. Radiologic studies may demonstrate facet arthrosis but most likely will show no abnormality. Lumbar spondylolisthesis is a slippage usually of the fifth lumbar segment due to a fracture of the pars interarticularis, and those affected have low back pain that radiates to the coccyx or lateral aspect of the leg.11(pp14) Plain x-ray films are all that are usually needed to make the diagnosis. Coccygodynia is the complaint of pain at the base of the spine; etiology is unknown. Trauma, intraosseous lipoma, chondroma, and giant cell tumor have been postulated as the cause.11(p92) Imaging studies usually show no abnormality.

Lumbar spinal fractures are usually the result of trauma occurring mostly in osteoporotic patients or patients with prolonged corticosteroid use. In younger patients without a history of trauma, malignancies such as multiple myeloma or metastatic disease should be considered. Pain is localized to the involved vertebrae and made worse with flexion. Plain x-ray films are usually diagnostic, but a bone scan may prove more beneficial to determine the acuteness of the fracture.

Diffuse idiopathic skeletal hyperostosis is a hypercalcification of the anterior and posterior longitudinal ligament and is found in middle-aged men. X-ray films will demonstrate large bone spurs and syndesmophytes that fuse regions of the spine. Patients complain of diffuse, nonradicular back pain. Younger male patients with ankylosing spondylitis will have gradual sacral pain that ascends to the low back. It is usually worse in the morning and improves as the day progresses. X-ray films will show sclerosis of the sacroiliac joint and bridging syndesmophytes that will eventually take on the form of a “bamboo” spine.

Lumbosacral sprain and strain is the most common diagnosis of low back pain. The etiology is not completely understood, but this condition is associated with an increased mechanical load through the low back. The abnormal forces can result in microtrauma to the ligamentous and muscular structures. The posterior longitudinal ligament and interligamentous structures are heavily innervated with pain fibers, and overstretching or tearing them can cause pain. The patient describes pain localized in the low back and may have some symptoms of radicular pain but no numbness or tingling. The physical examination will demonstrate paraspinal spasm, decreased lumbar lordosis, and pain with increased flexion but no neurologic deficits. X-ray films may show a decreased lumbar lordosis. Overall, the main sites of low back pain are the posterior longitu-
dinal ligament, the interospinous ligaments, the nerve roots and dural coverings, the facet joints, and the deep muscles.15

Osteopathic lesions may be the primary cause or a result of low back injury, both acute and chronic. Somatic dysfunctions, both segmental and intersegmental, may result in significant pain. Physical examination will demonstrate decreased mobility at that segment and resultant paraspinal tenderness. Evaluation of posture should be part of every osteopathic evaluation. According to Caille14, 80% to 90% of low back pain is related to poor posture. Piriformis syndrome is a compression of the sciatic nerve as it courses under or through the piriformis muscle. Patients complain of gluteal and hip pain that is exacerbated with flexion, adduction, and internal rotation of the hip, that is, piriformis stretch. Finally, low back pain can be the result of referred pain from the viscera, known as viscerosomatic reflexes. These reflexes can be seen with pathologic lesions in the prostate, stomach, colon, uterus, kidney, urinary bladder, liver, and spleen.

Understanding the pain complex
To understand how to treat pain symptoms, it is important to understand the pain pathway. Figure 4 is a simplification of the pain process that demonstrates the neurophysiologic pain pathway when a person stubs a toe. At the site of injury, inflammatory precursors are released, as well as free nerve-ending stimulation. Nociceptors in the toe are stimulated and ascend to the spinal cord via large-diameter and fast-conducting myelinated A delta fibers and slow-conducting unmyelinated C fibers. At the level of the dorsal horn of the spinal cord, neurotransmission occurs via a complex array of interneurons before ascending to the brain. Here is where opioid receptors (μ, κ, and δ) modulate or dampen the transmission of pain, forming a gate. If enough pain stimulation has occurred to overwhelm the system, the gate will open and pain will ascend to the brain via the spinothalamic tract to the medulla and thalamus of the brain and eventually on to the cerebral cortex. At the cortical level, modulation occurs again to dampen the effect of pain before descending back to the spinal cord. Here, the neurotransmitters serotonin, norepinephrine, γ-aminobutyric acid (GABA), dopamine, and opioids again work to dampen the effect of pain at the cortex, thalamus, and medulla.9,10,11,16 If there is enough pain stimulation to overwhelm the cortical modulation process, the pain will descend back to the spinal cord and back to the peripheral nerve where it originated. In the example of the person who stubbed a toe, the mechanoreceptors and quick A delta fibers are stimulated, quickly resulting in a reflex to move the foot. The slow C fibers go through the modulation at the spinal cord and the cortex and eventually will transmit back to the foot, accounting for that millisecond delay that is experienced after banging the toe.

Treatment strategies for low back pain: the pain triad
As previously outlined, for one to experience pain, stimulation must occur at the periphery, spinal cord, and cortex. Treatment options can be geared toward those that affect the periphery, spinal cord, and cortex, that is, the pain triad. When treating pain, especially chronic, the clinician can choose modalities from each level, giving the patient a more efficient pain management program. Figure 5 illustrates the trilevel pain management program for low back pain.

At the level of the peripheral nerve, NSAIDs and muscle relaxers, epidural and nerve and spinal blocks, physical therapy modalities, osteopathic manipulation, and physical exercise are current treatment options. At the level of the spinal cord, opiate analgesics and transcutaneous electrical nerve stimulators (TENS) have efficacy. And finally, control of pain at the cortical level can be achieved with antidepressants, neuroleptics, and neurostimulants as well as opiate analgesics and osteopathic spinal manipulation.

Peripheral pain control
NSAIDs are the most common analgesic medication used to treat low back pain. It is most beneficial where inflammatory processes are evident. It works by inhibiting prostaglandin synthesis, primarily through the cyclooxygenase pathway, which occurs in response to injury of the cell membrane. Two broad categories of NSAIDs exist:

- traditional NSAIDs, which inhibit cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2), and
- selective COX-2.

COX-2 inhibitors are reported not to interfere with protective prostaglandins in the gastric mucosa and should be used for patients who have a history of or risk factors for peptic ulcerative disease. Oral-dose corticosteroid can also be used in acute, significant inflammatory processes. Muscle relaxants such as cyclobenzaprine hydrochloride, methocarbamol, baclofen, and tizanidine (Zanaflex) can relieve muscle spasm. The purpose of these medications is to allow increased range of motion and improved tolerance for physical exercise. Side effects of these medications are extreme sedation; they should be used judiciously. The long-term effects of continued use of these agents are still unknown, and they should be used only for short periods.

Epidural steroid injections are effective in about 66% of selected patients with low back pain.17 Patients with suspected nerve root inflammation due to disk disease or lumbar stenosis may benefit. Although no clear criterion exists, patients who respond best are those who have not improved after 4 weeks of conservative care and those who have an acute flair of a chronic condition.

Other blocks include paravertebral, sacroiliac, and facet joint nerve blocks. Facet joint blocks can be both diagnostic and therapeutic in the treatment of low back pain18; as mentioned previously, radiologic studies most likely will show absolutely no abnormality in patients with facet joint–mediated pain. Injections into trigger points in isolated muscle spasm locations can be beneficial in decreasing pain symptoms. Injections are usually given with a local anesthetic. Injections into trigger points in patients with piriformis pain has been proven beneficial in relieving pain15 in both acute and chronic low back pain. Chemical neurolytic agents such as botulinum toxin are also being used, but long-term studies are still on going.

Physical therapy is critical to the recovery of patients to restore flexibility, motion, and improved function. Stretching to increase flexibility of the hip flexors, hip extensors, and hamstrings should be taught to patients, even those with acute pain. Patients with tight hamstrings will not have adequate hip range of motion and may sprain the lumbar spine with relatively minimal hip flexion. Eventually, patients should progress to more taxing exercises to strengthen the abdominal musculature and improve pelvic stability. An integral part of rehabilitating and preventing future injuries is instructing the patient on proper lifting techniques that emphasize lifting with the large muscle groups of the lower extremity, as
opposed to the weak paraspinal stabilizing musculature. Physical therapy modalities such as application of heat and cold can provide strong anti-inflammatory and analgesic effects. Patients should be instructed to heat, perform stretches, and to ice afterward. The use of low back bracing remains controversial. It may provide temporary relief in the acute pain setting, but prolonged use can promote muscular weakness and even atrophy due to disuse.

Osteopathic manipulation is a treatment for both acute and chronic low back pain. Manual muscle techniques are critical in enhancing segmental hypomobility, thus allowing uniform segmental motion and functional balance. Manual techniques have been used in most diagnoses of both acute and chronic pain syndromes. High-velocity techniques should be used with caution on patients who have an upper motor neuron pathologic process or when bone metastatic disease is known or suspected. Osteopathic manipulative medicine is the only treatment modality that has effects at all three treatment levels, having effects on peripheral, spinal, and cortical regions.

**Spinal level**

Modes of treatment geared at the spinal level are to dampen or impede pain from ascending to the brain. In addition to osteopathic manipulative medicine, opioid analgesics and TENS units have effect here. According to the World Health Organization analgesic ladder, opioids are appropriate for moderate and severe pain. In patients with mild pain, adjunctive analgesics are the mainstay of pharmacologic therapy; these agents include NSAIDs, acetaminophen, neuroleptics, and antidepressants. Opiates work at the spinal level by binding to opiate receptors at the interneuron level in the dorsal horn as mentioned earlier. There are two classes of opioid analgesics: agonists and agonist-antagonists. The pure opioid agonists include oxycodone, hydrocodone, and codeine. Stronger opioid agonists include morphine in both an immediate-release formulation (morphine sulfate immediate release, MSIR) with effects lasting 3 to 4 hours, and a sustained-release form (OxyContin, MS Contin, Roxanol SR), which provides 12 hours of relief. It is recommended to start with the lowest possible dose and titrate as needed. Never prescribe opioids on an as-needed basis but as a scheduled dose.

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**Figure 4. Diagram demonstrating the neurophysiologic pain pathway when a person stubs a toe.**

**Figure 5. Illustration of trilevel pain management program for low back pain. OMT = osteopathic manipulative treatment; TENS = transcutaneous electrical nerve stimulator; NSAIDs = nonsteroidal anti-inflammatory drugs.**
Be aware of the development of physical tolerance after prolonged use of an opioid. To decrease the risk of addictive behavior, the patient should be placed on long-acting agents with rescue doses of immediate-release medication for breakthrough pain to augment the sustained-release drugs. This decreases the euphoria usually associated with shorter-acting agents that are thought to contribute to drug-seeking behavior. Tramadol hydrochloride (Ultram) is a nonnarcotic medication that also works on opioid receptors in the central nervous system and prevents reuptake of the neurotransmitters serotonin and norepinephrine. Tramadol is a unique agent in that it can work both on the spinal and cortical levels in the pain triad.

Transcutaneous electrical nerve stimulators and dorsal column stimulators work at the spinal level by inhibition of the A delta and C fibers by utilizing A alpha or beta neural input, according to Melzac and Wall. Generally, a TENS unit works only when on and may provide significant improvement in pain and muscle spasm. Patients can be given their own unit and simply master its use. Although no clear criterion exists for surgical placement of dorsal column stimulators, they have been helpful in some patients who have failed to respond to conservative treatment and are not candidates for low back surgery.

**Cortical level**

Antidepressant and anticonvulsant medications are adjunctive analgesics that work cortically to dampen pain before descending from the cortex to the spinal cord. Antidepressant medications work on both serotonergic and noradrenergic pathways in the cortical pain centers of the brain. They can also potentiate the analgesic effects of the opioids and help to alleviate depression that is commonly seen in patients with chronic pain. Commonly used tricyclic antidepressants (TCAs) include amitriptyline hydrochloride (Elavil), nortriptyline (Pamelor), imipramine hydrochloride (Tofranil), desipramine hydrochloride (Norpramin), and doxepin hydrochloride (Sinequan). Although not studied as extensively as the TCAs, other classes of antidepressants such as trazodone hydrochloride (Desyrel) as well as serotonin-specific reuptake inhibitors (SSRIs) such as fluoxetine hydrochloride (Prozac), paroxetine hydrochloride (Paxil), and sertraline hydrochloride (Zoloft) are also used for adjunctive pain control with some clinical success.

Medications originally designed to treat seizure disorders, such as carbamazepine (Atarax, Tegretol) and gabapentin (Neurontin), have been shown to be helpful in the treatment of neuropathic and other types of pain. These medications also use neurotransmitters such as GABA, in the case of gabapentin, to dampen the pain before its descent to the spinal cord. These agents also can potentiate the opioid medications and be powerful mood-stabilizing agents. Other treatment modalities that are effective in pain control at the cortical level include tramadol, which has been previously mentioned. Although no published studies exist, osteopathic manipulative treatment with such techniques as craniosacral manipulation has also been reported to have effects at the cortical level.

**Comment**

The approach to the patient with low back pain begins with a thorough history and physical examination. The physical examination should be geared toward the neurologic, orthopedic, and osteopathic evaluation. Modes of therapy can be categorized into a triad system involving peripheral, spinal cord, and cortical levels. The clinician can choose a modality directed at each level to provide the patient with a pain management program that will maximize the chosen mode of therapy and restore function and mobility to the patient with low back pain.

**References**


