"Oh, My Aching Back!"
An Evidence-Based Review of One of Mankind's Oldest Afflictions

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David M. Lemonick, MD, FAAEP, FACEP

Abstract

A majority of adults suffer from at least one episode of low back pain (LBP), which is second only to upper respiratory infection as a reason to visit a physician. Most of these episodes are due to a variety of muscular and ligamentous strains and sprains, but in a minority of cases, LBP heralds a life threat or a spinal cord threat. Identifying the few serious conditions that lead to LBP among all the benign causes is a daunting task.

The evaluation and treatment of LBP is also quite expensive. Total costs of low back pain in the United States exceed $100 billion per year, with two-thirds of these costs indirect, due to lost wages and reduced productivity.

In order to effectively and efficiently evaluate LBP in the ambulatory care setting, a practitioner needs an organized approach to risk stratification, testing, and treatment. This article strives to present such an approach. By using history and physical examination to identify "red flags" for serious disease in patients with LBP and then utilizing testing and imaging selectively, both the patient and the practitioner will be well served.

Evidence-based treatment of LBP is reviewed, and several well-entrenched myths regarding pharmaceutical and physical therapies for LBP are discussed.

"Back pain is the price mankind paid for the hubris of walking erect."

—Anonymous

Introduction

Acute low back pain (LBP) is a huge clinical challenge in the United States. More than 6 million cases of LBP occur annually in United States. Mechanical LBP is the most common cause of work-related disability in persons younger than 45 years of age in the United States. It is also the most expensive cause of work-related disability. In a 2002 survey, back pain accounted for approximately 2.5% of medical visits in the United States, contributing approximately 15 million office visits. It is second only to upper respiratory illness as a reason for primary care office visits. The annual incidence of LBP in adults is 5%, and the lifetime prevalence of an episode of acute low back pain, defined as pain lasting less than six weeks, ranges from 60% to 90% for the adult population.

Eighty-five percent of back pain cases do not have a clear etiology, often receiving a nonspecific diagnosis of "acute lumbosacral strain." This lack of diagnostic precision is a reflection of the diagnostic challenge and lack of pathognomonic tests for low back pain. In one large study, 14% of survey respondents in the US had back pain, and 2% had back pain with sciatica, lasting at least two weeks. In the 2002 US National Health Interview Survey (NHIS), with over 30,000 respondents, 26.4% reported experiencing back pain lasting at least a whole day in the prior three months.[1, 2]

Diagnostic uncertainty exists even for those with back symptoms and well-described findings on scan, as these findings are common even in subjects without back pain, and may be unrelated to the symptoms. As an example, herniated disks can be identified in significant numbers of CT or MRI low-back studies in subjects with no back pain.

Among all primary care patients with low back pain, less than 5% will have serious systemic pathology. There is also widespread professional uncertainty regarding the optimal therapy for LBP. Wide variations exist in the use of pain medications, physical measures, injections, and surgery for back pain. There is an overuse of imaging studies and surgery for back pain in the US, and there have been rapid increases in the use of imaging, opioids, injections, complementary and alternative medicine, and surgery. Despite this increase, there has been no clear evidence of improved functional status or declining work disability for sufferers of LBP.

Risk Factors

Among the risk factors identified in LBP are: heavy lifting, twisting, sedentary work, bodily vibration, obesity, poor conditioning, smoking, psychological factors, somatization disorder, anxiety, depression, older age, female gender, psychologically strenuous work, low educational attainment, workers' compensation insurance, and job dissatisfaction. It is also noted that LBP is common even without risk factors.

Huge Costs

The diagnosis and care of LBP is also very expensive. United States healthcare expenditures in 2008 were $2,394.3 billion, or 16.6% gross domestic product (GDP). By current estimates, by 2017, these will swell to $4,277.1 billion, or 19.5% of GDP. By extension, some studies have estimated that twenty percent of Medicare spending ($58 billion) appears to provide no benefit in terms of survival nor is it likely that this extra spending improves the quality of life.[3] The total costs of low back pain in the United States exceed $100 billion per year. Two-thirds of these costs are indirect, due to lost wages and reduced productivity. Seventy-five percent of the total cost is attributable to fewer than 5% of the patients with low back pain.[4]

Prognosis

Seventy-percent of patients with LBP feel better within one week, 80%, in two weeks, and 90% in one month. Only 10% of all patients with low back pain have long-term problems. Recurrence of LBP is observed in up to 40% of patients within six months. Ninety percent of patients with low back pain in primary care did not seek care after three months. However, most patients were still experiencing LBP, for which they did not seek care, one year after the initial episode.[5] The likelihood of developing chronic low back pain (i.e., LBP>12 months after initial presentation) is associated with increasing age, female gender, having a prior episode of low back pain, and pre-existing psychosocial factors.[6]

Low back pain has also been found to have a huge impact on life style and quality of life. One US survey found that 72% of those who sought treatment for back pain gave up on exercising or sports-related activities. Sixty percent said they were unable to perform some daily activities, and 46% said they had given up sex because of their back condition.[7] Physicians who care for patients with acute LBP are in an unenviable position. They are frequently accused of ordering unnecessary diagnostic tests, prescribing unnecessary bed rest and medications, and over-referring to specialists. At the same time, they are in danger of missing the "red flags" of back pain history and physical examination that suggest serious etiologies. In light of the overwhelming preponderance of benign LBP etiologies, the serious conditions are truly "a needle in a haystack."

A Reasonable Approach to LBP in Four Questions

A reasonable approach to LBP is encompassed by asking four clinically-related questions: (1) Is there a life threat? (2) Is there a spinal cord threat? (3) How much workup is enough (but how much is too much, in terms of imaging, rehabilitation, and surgical referral)? And (4) If there are no immediate threats, what interventions have been shown to alleviate symptoms and to improve recovery?

Immediate life threats that may present as LBP include ruptured abdominal aortic aneurysm, thoracic aortic dissection, pulmonary embolism, and myocardial infarction. Immediate spinal cord threats include epidural mass effect from tumor, infection, hematoma, and massive intervertebral disc herniation. Other urgent conditions that may cause LBP include endocarditis; renal disease, such as pyelonephritis, infected stone, and renal artery dissection; and gynecologic etiologies, including abruptio placenta. Other serious conditions presenting as LBP include vertebral osteomyelitis, tuberculosis (Pott's disease), tumor, fracture, discitis and herniated disc, and ankylosing spondylitis as well as pelvic inflammatory disease. Less serious etiologies of LBP include renal colic; gynecologic conditions, such as pregnancy, endometriosis, ovarian conditions, and dysmenorrheal; lumbosacral strain; and varicella zoster.

The etiology of LBP can also be divided into mechanical low back and leg pain, referred visceral pain syndromes, and non-mechanical spinal etiologies. Mechanical low back or leg pain constitutes almost 97% of cases of acute LBP that present to primary care physicians in ambulatory care settings.[8] Of these, lumbar strain and sprain contributes 70%, degenerative processes of disks and facets (usually age-related) represent 10%, herniated disk and osteoporotic compression fracture 4% each, spinal stenosis 3%, spondylolisthesis 2%, and traumatic fracture and congenital disease, less than 1% each.[8]

Visceral etiologies that refer pain to the low back account for approximately 2% of LBP and include diseases of pelvic organs, renal disease, aortic aneurysm, and gastrointestinal disease.

Nonmechanical spinal etiologies, representing 1% of LBP, include: neoplasia (0.7%), infection (0.01%), inflammatory arthritis (often associated with HLA-B27) (0.3%), inflammatory bowel disease, Scheuermann's disease (osteochondrosis), and Paget's disease of bone.

Because they represent the overwhelming majority of LBP in ambulatory care, the salient features of lumbosacral strain, spinal stenosis, sciatica, spinal cord compression, and cauda equina syndrome will be presented.

Lumbosacral Strain

Lumbosacral strain (LSS) represents the majority of patients presenting with low back pain. The pain of LSS is well localized to the lower back and upper buttocks. There is tenderness

over the paravertebral musculature and no neoplastic, infectious, or inflammatory causes exist. There is usually a history of overuse or low-energy trauma, and the pain is worsened by activity and relieved by rest. In lumbosacral strain, there is no neurological complaint or deficit noted.

Sciatica

In sciatica, which represents approximately 1% of LBP, the pain is in the distribution of a lumbar or sacral nerve root. There is sharp or burning pain radiating down the posterior or lateral aspect of the leg, usually to the foot or ankle that is associated with numbness or tingling.[9] Pain radiating below the knee is more likely to represent true radiculopathy than proximal leg pain. The key to the diagnosis is radicular pain below the knee. Sciatica is usually caused by a herniated intervertebral disc, and the pain usually increases with coughing, sneezing, or performance of Valsalva maneuver. Physical examination often reveals a positive straight leg raise test (SLR).

Cauda Equina Syndrome

The cauda equina syndrome is a medical emergency that is most commonly caused by tumor or a massive midline disk herniation. It presents with bowel or bladder dysfunction, and urinary retention with overflow incontinence is typically present. Saddle anesthesia, bilateral sciatica, and leg weakness are typical. Thus, both neurological complaints and neurological deficits are present, and an emergent MRI is indicated.

Spinal Cord Compression

Acute spinal cord compression usually presents with relatively mild LBP that is accompanied by progressive weakness in both lower extremities. The cause is often a tumor, a large central disc herniation, or trauma. Neurological complaints and neurological deficits are found, and emergent MRI is indicated.

Spinal Stenosis

In spinal stenosis there is narrowing of the spinal canal (congenital or acquired), nerve root canals, or intervertebral foramina. A thickened ligamentum flavum and osteophyte growth contribute to the stenosis and nerve root impingement. This narrowing results in back pain, transient tingling in the legs, and ambulation-induced pain localized to the calf and distal lower extremity that resolves with rest. This pattern of exacerbation with exercise and improvement with rest has been termed "pseudoclaudication" because of its similarity to the symptoms of peripheral vascular insufficiency. Thus, the pain of spinal stenosis can be distinguished from vascular claudication by the presence of normal arterial pulses in the former condition. More commonly, spinal stenosis presents with back and leg pain that are relieved by sitting or other spine flexion. Disc bulging and spondylolisthesis may also contribute to s pinal stenosis.[10]

Spinal Infection

With a gradual and prolonged onset, often over a course of weeks to months, spinal infections are usually caused by Staph aureus, Staph epidermidis, Streptococcus, and by urinary pathogens. Patients are typically immunosupressed or are intravenous drug users. Symptoms include fever, night sweats, weight loss, and unrelenting pain. In this group of patients, the erythrocyte sedimentation rate (ESR) is a sensitive, though nonspecific, test for spine infection. The history and physical examination in LBP are directed at answering the first two questions in our four-question approach: (1) is there a life threat and (2) is there a cord threat?

History

The history in LBP patients is designed to answer several essential questions that relate to the presence of life- and cord-threatening conditions: Is there systemic disease? Is there neurologic compromise? Are there psychological factors that amplify or modify the clinical picture? "Red flags" in the history relate to details of the patient's age and past medical his-

Table 1: Red Flags for Potentially Serious Conditions

Possible Fracture	Possible Tumor or Infection	Possible Cauda Equina Syndrome
FROM MEDICAL HISTORY		
Major trauma, such as vehicle accident or fall from height. Minor trauma or even strenuous lifting (in older or potentially osteoporotic patient).	Age over 50 or under 20. History of cancer. Constitutional symptoms, such as recent fever or chills or unexplained weight loss. Risk factors for spinal infection: recent bacterial infection (e.g., urinary tract infection); IV drug abuse; or immune suppression (from steroids, transplant, or HIV). Pain that worsens when supine; severe nighttime pain.	Saddle anesthesia. Recent onset of bladder dysfunction, such as urinary retention, increased frequency, or overflow incontinence. Severe or progressive neurologic deficit in the lower extremity.
FROM PHYSICAL EXAMINATION		
		Unexpected laxity of the anal sphincter. Perianal/perineal sensory loss. Major motor weakness: quadriceps (knee extension weakness); ankle plantar flexors, evertors, and dorsiflexors (foot drop).

Adapted from: Acute Low Back Pain Problems in Adults: Assessment and Treatment Quick Reference Guide for Clinicians. Clinical Practice Guideline #14. US Agency for Health Care Policy and Research (1994).[20]

tory and the duration, acuity, character, and location of the pain, and any associated symptoms (Table 1). Age of greater than 50 years raises concern for AAA, malignancy, and fractures. Age less than 18 increases the likelihood of spondylolysis, spondylolisthesis, discitis, spinal infections, tumors, and developmental disorders. Trauma and chronic steroid risk suggest fracture. Weight loss, fever, night sweats, injection drug use, and unrelenting pain may be due to infection. Night pain is characteristic of ankylosing spondylitis, malignancy, and infection. Epidural compression syndrome should be considered in the presence of incontinence, saddle anesthesia, and bilateral neurological deficits. In contrast, unilateral neurological deficit suggests a herniated intervertebral disc.

Physical Examination

As a complement to the history, the physical examination is a systematic evaluation of potential life and cord threats (Table 1). This evaluation includes vital signs, an observation of walking, changing positions, posture, and spinal motion. Evaluation of peripheral pulses is appropriate in older patients with leg symptoms. A focused neurological examination includes the testing of L4-5 and S1 nerve roots and straight leg raising in patients with leg symptoms. Appropriate detailed examination is directed towards any red flags found in the history. Anatomically "inappropriate" signs of pain amplification are noted, and the examination should include inspection of the back for evidence of rash or trauma.

Fever suggests infection or malignancy. Anal sphincter laxity, motor weakness, saddle anesthesia, and absent or diminished reflexes suggest epidural compression. Disc herniation may present with motor weakness or positive straight leg raise (SLR) and crossed straight leg raise (CSLR) tests (Figures 1 and 2). Bone tenderness suggests infection, trauma, and malignancy. Positive Babinski's sign implies upper motor neuron disease or cord compression.

Fourth lumbar nerve (L4) compression presents with pain distributed to the anterolateral thigh and leg and numbness in the distal anterior thigh (Figure 3). There is motor weakness in quadriceps extension and impaired squat and rise. The knee jerk is diminished. Compression of L5 presents with pain in the posterolateral thigh and leg and numbness of the lateral calf. There is weakness of dorsiflexion of the great toe and foot, tested by heel walking, but no reliable reflex can be tested for L5. In the first sacral nerve root (S1) there is pain in the posterior thigh and leg and numbness in the posterior calf and plantar aspect of the foot. Motor weakness is tested by toe walking and manifests as weak plantar flexion of great toe and ankle. The ankle jerk reflex is also diminished.

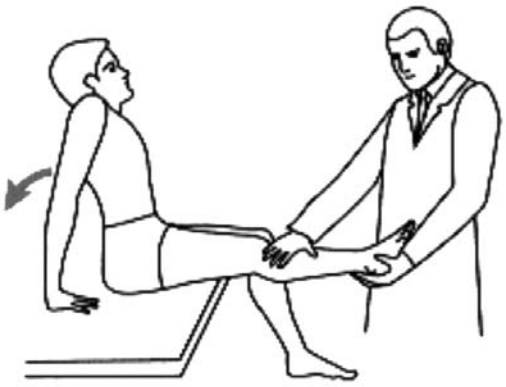
In the straight leg raise test (SLR), the patient is asked to lie as straight as possible on a table in the supine position. With one hand placed above the knee of the leg being examined, exert enough firm pressure to keep the knee fully extended. The patient is asked to relax. With the other hand cupped under the heel, the straight limb is slowly raised. The patient is reassured

Figure 1: The Straight Leg Raise Test

Instructions for the Straight Leg Raise Test

1. Ask the patient to lie as straight as possible on a table in the supine position.
2. With one hand placed above the knee of the leg being examined, exert enough firm pressure to keep the knee fully extended. Ask the patient to relax.
3. With the other hand cupped under the heel, slowly raise the straight limb. Tell the patient, "If this bothers you, let me know, and I will stop."
4. Monitor for any movement of the pelvis before complaints are elicited. True sciatic tension should elicit complaints before the hamstrings are stretched enough to move the pelvis.
5. Estimate the degree of leg elevation that elicits complaint from the patient. Then determine the most distal area of discomfort: back, hip, thigh, knee, or below the knee. Limb can also increase the tension on the sciatic nerve roots.
6. While holding the leg at the limit of straight leg raising, dorsiflex the ankle. Note whether this aggravates the pain. Internal rotation of the limb can also increase the tension on the sciatic nerve roots.

Figure 2: The Sitting Knee Extension Test

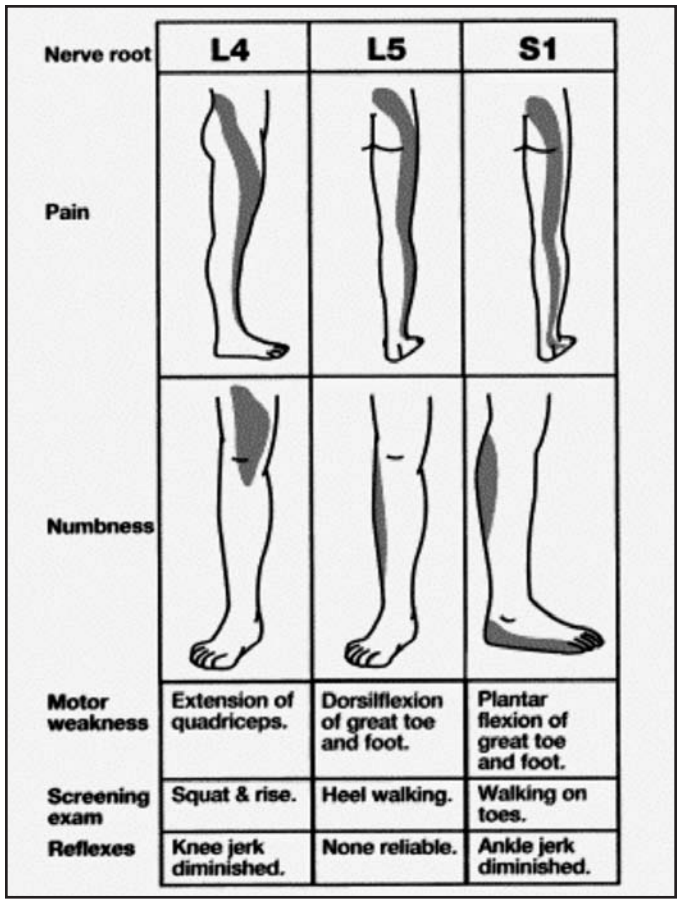


Instructions For Sitting Knee Extension Test

With the patient sitting on a table, both hip and knees flexed at 90°, slowly extend the knee as if evaluating the patella or bottom of the foot. This maneuver stretches nerve roots as much as a moderate degree of supine SLR.

Adapted from Bigos S, Bowyer O, Braen, et al. Acute low back problems in adults. Clinical Practice Guideline No. 14. AHCPR Publication No. 95-0642. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services. December 1994.

Figure 3: Testing for Lumbar Nerve Root Compromise



Adapted from: Bigos S, Bowyer O, Braen, et al. Acute low back problems in adults. Clinical Practice Guideline No. 14. AHCPR Publication No. 95-0642. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services. December 1994.

that, "If this bothers you, let me know, and I will stop." The examiner monitors the patient for any movement of the pelvis before complaints are elicited. True sciatic tension should elicit complaints before the hamstrings are stretched enough to move the pelvis. An estimate is made of the degree of leg elevation that elicits complaint from the patient. Then it is determined where is the most distal area of discomfort: back, hip, thigh, knee, or below the knee. While holding the leg at the limit of straight leg raising, the ankle is dorsiflexed, and it is note whether this aggravates the pain. Internal rotation of the limb can also increase the tension on the sciatic nerve roots.

In the crossed straight leg raise test, the patient sits on edge of the examination table with both hips and knees flexed at 90 degrees. The leg is slowly extended by the examiner at the knee as if evaluating the patella or bottom of the foot. This maneuver stretches nerve roots as much as a moderate degree of supine SLR.[11] A positive straight leg test is 80% sensitive, but it is only 40% specific for herniated disc. The crossed straight leg test is less sensitive (25%) for herniated disks, but it is 90% specific.[12, 13]

In another study, the sensitivity of the supine SLR test was .67 sensitivity of .41 of the seated SLR test (P=.003) in patients presenting with signs of and symptoms consistent with lumbar radiculopathy and MRI evidence of nerve root compression.[14]

The Waddell criteria were designed to detect nonorganic causes of LBP, and these criteria are: excessive tenderness, simulation, distraction, overreaction, and regional disturbance. He proposed that most patients with proven organic back pain had only one or none of these criteria, while patients with three or more signs were likely to have non-organic disease.[15]

Excessive tenderness includes tenderness that is superficial (e.g., significant pain to light touch or pinch), and that is non-anatomic (e.g., tenderness to palpation over thoracic and lumbar spine and pelvis). Simulation is tested by axial loading, (e.g., low back pain with light pressure to skull while standing) and by rotation (e.g., increase of low back pain with passive rotation of the shoulders and pelvis in the same plane, in the standing position). Distraction may be elicited by SLR, with inconsistent findings in sitting vs. supine straight leg tests. Regional Disturbance is elicited by: weakness (generalized giving way or cog-wheeling resistance when testing strength in the lower extremities), sensory (stocking sensory loss, non-dermatomal distribution). Overreaction is the most important Waddell criteria. This includes disproportionate pain response, bracing both limbs supporting weight while seated, clutching or grasping affected area for more than three seconds, or dramatic grimacing or sighing, with shoulders rising and falling.[16]

The practitioner is strongly cautioned to use extreme care before ascribing back pain to psychological causes or malingering. This mind set can and does lead to a failure to consider serious or life-threatening conditions that must not be overlooked. Because an increase in signs is associated with age, these criteria are not recommended for use in the elderly. It should also be emphasized that behavioral signs can occur with organic findings and that the presence of these signs does not exclude organic findings. Further, isolated behavioral signs are not clinically significant.[17]

Question #3: How Much Workup Is Enough (and how much is too much)?

Many authors have debated question #3: how much workup is appropriate in acute LBP, and how much is too much for responsible resource stewardship and cost containment?[8, 18]

Among the factors that contribute to the discussion are patient expectations, fear of missing important diagnoses and of the potential for resulting litigation, and the expense, inconvenience, and discomfort of diagnostic tests and their variable sensitivity and specificity for particular causes of LBP. Among the tests commonly used in LBP are blood tests, urinalysis, and imaging, (e.g., plain x-ray, CT Scan, MRI, and bone scan).

In the presence of red flags for infection or tumor, it is recommended that CBC, ESR with or without CRP be performed.[19] Indications for plain x-rays include: age >50 years (concern for malignancy or pathologic fractures), a history of malignancy or

of unexplained weight loss (concern for malignancy), history of fever, immunocompromised status, or injection drug use (concern for spinal infection), recent trauma or chronic corticosteroid use (concern for fracture), significant neurological deficit or sphincter dysfunction (concern for cauda equina syndrome), and symptom duration >4 to 6 weeks (concern for fracture, malignancy, infection, and cauda equina).[20, 21] It has been noted that lumbar radiography in primary care patients with mechanical low back pain (without sciatica), however, is not associated with improved patient functioning, severity of pain, or overall health status.[22] Imaging of LBP is not necessary during the first four to six weeks in the absence of progressive neurological findings, constitutional symptoms, a history of traumatic onset or of malignancy, age ≤18 years or ≥50 years, infectious risk (e.g., IV drug use, immunosuppression, indwelling urinary catheter, prolonged steroid use, skin or urinary tract infection), or osteoporosis.[8,18] It should also be noted that greater than a 50% calcium loss is required to visualize an osteoclastic metastasis of the vertebrae on plain film.

CT and MRI are more sensitive than plain x-ray for detecting infection and cancer and can show herniated discs and spinal stenosis. Findings may be incidental and unrelated to the etiology of low back pain abnormalities on CT or MRI and are common among people without back pain. Bulging discs are present on MRI or CT in >50% of asymptomatic subjects.[23, 24] CT and MRI are indicated when there are progressive neurological deficits and when there is a high suspicion of cancer or infection. They should also be considered for patients with more than 12 weeks of persistent LBP. MRI is preferred over CT scan for better visualization of soft tissue and absence of radiation exposure. In the case of suspected spinal epidural abscess, it should be remembered that only 15% of patients have fever, back pain, and neurological deficits, and that this triad is the exception rather than the rule. Radionuclide bone scan is helpful if metastatic spread of cancer to the vertebrae is suspected, but no neurological deficit exists. Bone scan is also fairly sensitive for stress fractures of the spine but is not as sensitive as MRI for infection.

Referral to a neurologist, orthopedic surgeon, or neurosurgeon is indicated for cauda equina, suspected cord compression, or progressive neurological deficits. Referral should also be arranged for a patient with neurological deficits persisting for four to six weeks of conservative therapy in the presence of positive straight leg raising sign, consistent clinical findings, and favorable psychosocial circumstances (e.g., realistic expectations and the absence of depression, substance abuse, or excessive somatization).

A Suggested Clinical Approach to the Evaluation of Acute LBP

At this point we have answered the first three questions in our evaluation and treatment of LBP: (1) is there a life threat, (2) is there a cord threat, and (3) what diagnostic tests shall be employed? It is now possible to integrate the answers into a coherent algorithm that reflects the best evidence currently available on the subject.

The overwhelming majority of LBP (approximately 97%) consists of localized pain without sciatica.[19] In the absence of sciatica, about 60% of the total LBP are "simple LBP," (i.e., under age 50, no signs or symptoms of systemic disease, and no history of cancer). In this group, the likelihood of a musculoskeletal cause is approximately 99%. If these patients have a satisfactory improvement in symptoms in four to six weeks, the evaluation stops. If symptoms persist, then the patient is considered to have "complicated LBP," which is described in the next paragraph.

Another 37% of patients with LBP without sciatica are considered to be "complicated LBP," (i.e., age over 50, or signs or symptoms of systemic disease, or risk factors that include fever, weight loss, history of cancer, intravenous drug use, hematuria, or adenopathy). Despite the increased probability of serious cause of LBP in this group, 95% still will have a musculoskeletal cause. A plain film and an ESR or CRP is reasonable and, if these are normal, the patient is treated as in "simple LBP." If either test is abnormal, then a CT or MRI should be considered.

The final categories of LBP patients are those with sciatica and are divided into those with radiculopathy, who represent almost 3% of all LBP patients, and those who present with urgent signs and symptoms, which comprise less than 1% of the total. Radiculopathy consists of nerve root impingement syndromes without bowel or bladder involvement, saddle anesthesia, or bilateral signs or symptoms of progressive motor weakness. Plain films and ESR or CRP are performed in this group and, if either is positive, CT or MR are considered. Four to six weeks of conservative therapy is appropriate if these studies are normal.

Finally, in the <1% of LBP who are urgent, there is acute radiculopathy with bowel or bladder involvement, saddle anesthesia, bilateral signs or symptoms, or progressive motor weakness. Such patients get urgent CT or MRI and immediate consultation for possible cauda equina syndrome or cord compression.

Question #4: What has been shown to alleviate symptoms?

A variety of medications, exercises, injections, activity restrictions, and physical interventions have been employed for the amelioration of LBP symptoms, with varying success. Among the common therapies are oral drugs including acetaminophen, nonsteroidal anti-inflammatory (NSAIDs), opioids, other analgesics, muscle relaxants, and steroids. Injected medications include trigger point and facet joint injections and prolotherapies. Among the physical interventions are stretching and strengthening exercises, corsets and braces, yoga, acupuncture, and spinal manipulation. Anecdotal reports about the efficacies of these interventions abound, but little evidence-based advice about therapies for LBP exists.

Nonsteroidal anti-inflammatory drugs are the most common class of medications prescribed for LBP. A Cochrane systematic review was comprised of six randomized controlled trials (RCTs), with sample size of 727 participants. The review found that NSAIDs were associated with a statistically significant improved relative risk for decrease in pain, and that NSAIDs did not produce greater adverse effects than placebo.[25]

Opioid agonists are widely acknowledged to be among the therapeutic options for low back pain, but there are limited data on their efficacy and safety for this indication. Most recent studies focus on chronic back pain. As to which, if any, opioid is superior, six studies found no difference between different opioids. A 1997 study found oxycodone better than acetaminophen with codeine (e.g., Tylenol No. 3). In the absence of definitive data, use of opiates for low back pain is a matter of clinical judgment. Adverse effects and abuse and misuse potential limit the utility of this class of medications. It is reasonable to consider short-acting opioids if NSAIDs and acetaminophen fail.[26] No studies have been conducted that compare acetaminophen to placebo. Three drug company–sponsored trials found that tramadol was better than placebo.[27] Acetaminophen is a reasonable option for most patients with acute low back pain, with perhaps less efficacy than NSAIDs.[28]

Muscle relaxants have been investigated for LBP. A meta-analysis of the efficacy of benzodiazepines consisted of two pooled studies with sample size of 222 participants, and it found that pain was improved with benzodiazepine. Six of seven studies of nonbenzodiazepine muscle relaxants (e.g., cyclobenzaprine) found that these agents are better than placebo, but they also found a greater risk for adverse effects. The main adverse effects observed were nausea, dizziness, and somnolence.

Studies have found that one muscle relaxant is not better than another, except one study which found that carisoprodol (trade name soma) was superior to other agents (but carisprodol was compared to low dose of diazepam).[29] Benzodiazepines should probably not be first line agents because of scanty evidence to support their use, because of their side effects, and because of concern about their abuse potential. When used, muscle relaxants should generally be limited to relatively short-term therapy (e.g., one to three weeks). Muscle relaxants are soporifics and, therefore, are best used at night.

There is a paucity of data regarding the utility of steroids in LBP, and there are no reliable RCTs or meta-analyses regarding their use. In one study 86 ED patients with acute nonspecific LBP were randomized to IM methylprednisolone or IM saline, independent of other treatment received in the ED. No difference in the pain score could be shown at short-term follow-up (one week and one month after visit). The methodology of the study precluded the conclusion that steroids do not have an effect on LBP. Better research is clearly needed.[30] A small randomized placebo-controlled trial of parenteral methylprednisolone in ED patients with non-traumatic LBP and a negative SLR found no benefit of steroids. The 2007 joint guidelines from the ACP and APS recommend against use of systemic glucocorticoids because of lack of proven benefit over placebo.[31]

Trigger points are discrete, focal, hyperirritable spots located in a taut band of skeletal muscle. Trigger point injections with a variety of local anesthetic agents have been studied in LBP. Only one placebo-controlled study of 63 outpatients could be found, and it showed that trigger point therapy was not any better than sham injection.[32] Convincing evidence for trigger point injection for acute LBP is lacking, and additional trials are needed.[33] Results of randomized trials using epidural steroid injections in LBP are conflicting, but these show at least temporary symptom relief for some patients with sciatica. Epidural steroids, however, have not been shown to reduce rates of disc surgery, nor is there evidence to suggest that they are effective for patients with back pain alone.

It is also unclear if epidural steroids are more effective than systemic steroids.[34] Similarly, the role for facet joint injections is unclear, and there is ongoing controversy regarding their efficacy. Randomized trials comparing corticosteroid injections of the facet joints with saline injections generally suggest no advantage to corticosteroid injection. And, if evidence supporting facet injection is sparse, evidence for sacroiliac joint injections is even sparser.[32] Comparing medications, the existing literature shows that NSAIDs, opioids, tramadol, and muscle relaxants are all superior to placebo in relieving LBP. There is no credible evidence that one medication is superior to another medication in the same class. Neither is there evidence that one class of medications is superior to another class. No conclusion is possible about parenteral steroids and trigger point injections, because the data are insufficient.

Physical interventions for LBP have been investigated. Back exercises are not helpful in relieving acute LBP, and they are marginally helpful in chronic LBP. Bed rest has been shown to be detrimental to recovery. While there is modest evidence for heat in LBP, no data are available regarding the use of cold. Transcutaneous electrical nerve stimulation (TENS) units are not recommended for acute LBP.[35, 36]

Spinal manipulation is comparable in efficacy to conventional medical therapy in both acute and chronic low back pain. No evidence shows that spinal manipulative therapy is superior to other standard treatments for patients with acute or chronic low-back pain. If used, there is little evidence to guide the duration of manipulation therapy, nor is there evidence that manipulation reduces the risk of recurrence of back pain.[37, 38]

Massage and yoga result in the same symptom response rate at five weeks compared to usual care, but they are associated with higher patient satisfaction. Similarly, acupuncture is a safe intervention, with reasonable data for efficacy in chronic back pain, and non-definitive but positive studies in acute back pain. As such, acupuncture may be a reasonable option for interested patients with access to an acupuncturist.[39]

Traction, corsets, and braces provide no significant benefit in short- or long-term outcomes for low back pain patients with or without sciatica. There is little evidence to suggest that corsets or braces have therapeutic value for most patients.[40, 41]

Conclusions

Back pain is common, recurrent, and expensive to individuals and to society. A specific etiology cannot be established for most patients, and less than 5% have a serious systemic pathology. The practitioner should advise short-term treatment with either an NSAID or acetaminophen. A CT or MRI is indicated for progressive neurologic deficits, a high suspicion of cancer or infection, and after 12 weeks of persistent low back pain. Urgent referral should be obtained for suspected cauda equina syndrome, for spinal cord compression, and for patients with progressive or severe neurologic deficits. To be considered are the use of a non-benzodiazepine muscle relaxant. Manipulation and acupuncture are options for discussion. Combination therapy (muscle relaxant plus NSAID) is encouraged, using muscle relaxants before bedtime and NSAIDs through the day. Opioids may occasionally be indicated, and these should be prescribed for short-term use on a fixed schedule, rather than on a p.r.n. basis. Do not advise bed rest, prescribe systemic steroids, or refer patients for PT in the initial two to three weeks of LBP. Do not refer patients for trigger point injection, facet joint injection, prolotherapy (irritant solution is injected into affected areas), or botulinum injections, and do not order imaging studies routinely in the first four to six weeks.

It should be kept in mind that in the natural course of low back pain, pain levels fluctuate and that the final pain scores are not necessarily the result of treatment effect. Also, even though the result of a clinical study is statistically significant, it is not necessarily clinically significant. Consider the needs of the individual patient when choosing therapy, since marginal improvements obtained from specific therapy may not outweigh the risks of a therapy for a particular patient. Finally, LBP is usually benign and self-limited, but occasionally it is dangerous and progressive. Thus, LBP is a medical-legal danger zone, and the practitioner must stay alert for red flags. The approach to LBP must be organized, addressing any red flags in the history and physical examination. By limiting diagnostic testing and therapy to that which is evidence based, the comfort and safety of both the patient and the practitioner will be enhanced.

David M. Lemonick, MD, FAAEP, FACEP, is Attending Physician, Emergency Department, Armstrong County Medical Center in Kittanning, PA.

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References

1. Deyo RA. Descriptive epidemiology of low-back pain and its related medical care in the United States. Spine 1987; 12:264.
2. Deyo RA, Mirza SK, Martin BI. Back pain prevalence and visit rates: estimates from U.S. national surveys, 2002. Spine 2006; 31:2724.
3. Skinner JS, Fisher E, and Wennberg JE. The Efficiency of Medicare (July 2001). NBER Working Paper No. W8395. Available at SSRN: http://ssrn.com/abstract=277305.
4. Katz, JN. Lumbar disc disorders and low-back pain: socioeconomic factors and consequences. J Bone Joint Surg Am 2006; 88 Suppl 2:21.
5. Croft, PR, Macfarlane GJ, Papageorgiou AC, et al. Outcome of low back pain in general practice: a prospective study. BMJ 1998; 316:1356.
6. Thomas E, Silman AJ, Croft PR, et al. Predicting who develops chronic low back pain in primary care: a prospective study. BMJ 1999; 318:1662.
7. American Academy of Orthopedic Surgeons. In Press Release. 2006.
8. Deyo RA, Kent DL. What can the history and physical examination tell us about low back pain? JAMA 1992; 268:760.
9. Morris EW, Di Paola M, Vallance R, et al. Diagnosis and decision making in lumbar disc prolapse and nerve entrapment. Spine 1986; 11:436.
10. Katz JN, Dalgas M, Stucki G, et al. Degenerative lumbar spinal stenosis. Diagnostic value of the history and physical examination. Arthritis Rheum 1995; 38:1236.
11. Bigos S, et al. Acute low back problems in adults: clinical practice guidelines, quick reference guide number 14. US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research, Rockville, MD 1994.
12. McCombe PF, Fairbank JC, Cockersole BC, et al. 1989 Volvo Award in clinical sciences. Reproducibility of physical signs in low-back pain. Spine 1989; 14:908.
13. Miller G, Hockberger R. Low back pain. In Markovchick VJ and Pons PT. Emergency Medicine Secrets 4th ed. 2006, Philadelphia, Mosby, p.114.
14. Rabin A, Gerszten PC, Karausky P, et al. The sensitivity of the seated straight-leg raise test compared with the supine straight-leg raise test in patients presenting with magnetic resonance imaging evidence of lumbar nerve root compression.1: Arch Phys Med Rehabil. 2007 Jul;88(7):840-3.
15. Waddell G, McCulloch JA, Kummel E, Venner RM. Nonorganic physical signs in low-back pain. Spine 1980; 5:117.
16. Polatin PB, Cox B, Gatchel RJ, Mayer TG. predictive. Spine (Phila Pa 1976). 1997 Jul 15;22(14):1618-21.
17. Main CJ, Waddell G. Behavioral responses to examination: A reappraisal of the interpretation of "nonorganic signs." Spine 1998;23(21):2367-2371.
18. Jarvik et al. Diagnostic evaluation of low back pain with emphasis on imaging. Ann Intern Med 2002 Oct 1;137(7):586-97.
19. Wheeler SG, Wipf JE, Staigler TO, et al. Approach to the diagnosis and evaluation of low back pain in adults 17.1: January 2009. Reference online. Available at: http://www.uptodate.com/online/content/topic.do?topicKey=spinaldi/9262&selectedTitle=1~100&source=search_result. Accessed: July 21, 2009.
20. Della-Giustina D, Kilcline BA, Denny M. Back Pain: cost-effective strategies for distinguishing between benign and life-threatening causes. 2000. Emergency Medicine Practice. Journal online at: http://ebmedicine.net/getpdf.cfm?sid=ls8p45ardwcu68005x7e99qq4l0wdh6dw85lpd7m2g454ywdg3115010557&spid=2&issueid=128. Accessed July 22, 2009.
21. Bradley WG, Seidenwurm DJ, Brunberg JA, et al. ACR Appropriateness Criteria low back pain. Expert Panel on Neurologic Imaging. Low back pain. [online publication]. Reston (VA): American College of Radiology (ACR); 2005.
22. Kendrick D Fielding K, Bentley E, et al. Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. BMJ 2001 Feb 17;322(7283):400-5.

23. Battie, MC, Videman, T. Lumbar disc degeneration: epidemiology and genetics. J Bone Joint Surg Am 2006; 88 Suppl 2:3.
24. Jensen MC, Brant-Zawadzki MN, Obuchowski N, et al. Magnetic resonance imaging of the lumbar spine in people without back pain._N Engl J Med. 1994 Jul 14;331(2):69-73.
25. Roelofs PD, Deyo RA, Koes, et al. Non-steroidal anti-inflammatory drugs for low back pain. Cochrane Database Syst Rev. 2008 Jan 23;(1):CD000396.
26. Sterenfeld, EB. From the 31st Semi-Annual Family Practice Review, sponsored by Temple University School of Medicine, Philadelphia, PA, and Lancaster General Hospital, Lancaster, PA Audio-Digest Family Practice. Volume 55, Issue 30. August 14, 2007.
27. Nguyen AP, What is the best evidence for the management of acute back pain? Contemporary Concepts in Clinical Emergency Medicine, held June 6-8, 2007, in New York, NY, and sponsored by the New York University School of Medicine, Department of Emergency Medicine.) Audio-Digest Emergency Medicine. Volume 24, Issue 24. December 21, 2007.
28. Towheed TE, Maxwell L, Judd MG, et al. Acetaminophen for osteoarthritis. Cochrane Database Syst Rev. 2006 Jan 25;(1):CD004257.
29. van Tulder MW, Touray T, Furlan AD, et al. Muscle relaxants for non-specific low back pain. Cochrane Database Syst Rev. 2003;(2):CD004252.
30. Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007; 147:478.
31. Friedman BW, Holden L, Esses D. Parenteral corticosteroids for Emergency Department patients with non-radicular low back pain. J Emerg Med. 2006 Nov;31(4):365-70.
32. Nelemans PJ, deBie RA, deVet HC, et al. Injection therapy for subacute and chronic benign low back pain. Spine (Phila Pa 1976). 2001 Mar 1;26(5):501-15.
33. Institute of Medicine. Committee on Pain, Disability, and Chronic Illness Behavior. Osterweis, M, Kleinman, A, Mechanic, D (Eds), Pain and disability: Clinical, behavioral, and public policy perspectives, National Academy Press, Washington, DC 1987.
34. Koes BW et al. Efficacy of epidural steroid injections for low-back pain and sciatica: a systematic review of randomized clinical trials Pain 1995 Dec;63(3):279-88.
35. Hayden JA, van Tulder MW, Malmivaara A, et al. Exercise therapy for treatment of non-specific low back pain. Cochrane Database Syst Rev. 2005 Jul 20;(3):CD000335.
36. French SD, Cameron M, Walker BF, et al. Superficial heat or cold for low back pain.Cochrane Database Syst Rev. 2006 Jan 25;(1):CD004750.
37. Assendelft WJ, Morton SC, Yu EI , et al. Spinal manipulative therapy for low back pain. Cochrane Database Syst Rev. 2004;(1):CD000447.
38. Cherkin DC, Deyo RA, Battié M et al. A comparison of physical therapy, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with low back pain N Engl J Med 1998 Oct 8;339(15):1021-9.
39. Eisenberg DM, Post DE, Davis RB et al. Addition of choice of complementary therapies to usual care for acute low back pain: a randomized controlled trial. Spine (Phila Pa 1976). 2007 Jan 15;32(2):151-8.
40. Clarke JA; Traction for low-back pain with or without sciatica. Cochrane Database Syst Rev. 2005 Oct 19; (4):CD003010.
41. Van Tulder. Lumbar supports for prevention and treatment of low back pain. Cochrane Database Syst Rev 2000; CD001823.