

[Print](#) [Close](#)

Current Therapeutic Options for Chronic Low Back Pain: A Focus on Nonsurgical Approaches

Course Director:

Binod Prasad Shah, MD, Albert Einstein College of Medicine, Bronx, New York

Faculty:

Michael K. Schaufele, MD, Emory University, Atlanta, Georgia

The Significant Impact of Low Back Pain

Epidemiology

In recognition of the extreme burden and impact that musculoskeletal disorders have on society, the United Nations and the World Health Organization (WHO) have designated 2000 to 2010 as the Bone and Joint Decade. Musculoskeletal disorders—of which low back pain (LBP) is the most prevalent condition—are the most common cause of severe long-term pain and physical disability.³ International studies indicate that the percentage of people that experience LBP during their lifetime ranges from 58% to 84%,³ while point prevalence figures estimate that LBP affects an average of 30% of the population at any given time.⁴ Currently, there are approximately 10 million Americans disabled by LBP.⁵



The potential risk factors that increase the likelihood of developing LBP vary from smoking and obesity to jobs that require heavy lifting and job dissatisfaction.⁶ Sixty percent to 70% of back pain patients recover within 6 weeks, and by week 12, 80% to 90% of patients with back pain have recovered.⁴ However, people with low educational levels, tendencies for depressive moods and distress, obesity, and job dissatisfaction have a higher risk of having LBP develop into a chronic, disabling condition.⁷ LBP that persists for more than 3 months is considered “chronic”,² and frequent episodes of LBP are described as “recurrent”. Forty percent of patients with LBP have recurrences within 6 months.² The highest incidence of LBP is within the working population, persons aged 25 to 64 years.³ Twenty percent to 44% of the working-population patients with LBP have further episodes within a year; overall, up to 85% of LBP sufferers have lifetime recurrences.^{3, 4}

Economics

Back pain results in 19 million physician visits yearly,⁵ making LBP second only to upper respiratory complaints in symptomatic reasons for seeing a doctor.⁸ Correspondingly, the economic impact of LBP on society has been staggering. In the United States \$14 billion is spent annually on the cost of care for LBP.⁵ Beyond direct medical care expenditures, indirect costs for a patient with LBP include time away from work costs, disability payments, and diminished productivity.⁸ Inclusion of both direct and indirect expenses for back pain in the United States yields an annual expenditure of between \$20 billion and \$50 billion.^{1,9} Approximately 2% of the American workforce is compensated for back injuries every year,¹ and it is estimated that 250 million workdays are lost annually in the United States due to LBP.⁵

A randomized, controlled trial of 681 patients published in 2005 considered the cost-effectiveness of 4 different treatment groups: simple medical care (MC), medical care with physical therapy (MC+PT), straight chiropractic care (CC), and chiropractic care combined with physical modalities (CC+PT).⁹ When adjusted for covariates, average LBP outpatient costs were calculated to be \$369 for MC, \$560 for CC, \$579 for CC+PT, and \$760 for MC+PT. Patient satisfaction was rated higher for the patients given chiropractic care rather than medical treatment,⁹ possibly due to the patient’s perception of a more personal approach.¹⁰ However, there were no differences in clinical outcomes between treatment groups, suggesting that a higher utilization of chiropractic care does not seem to be the most cost-effective solution to LBP. These findings corroborated the results of 2 out of 3 previous studies that found medical care to be less expensive than alternative therapies.^{11,12} A recent

study in the United Kingdom (UK)—the UK back pain exercise and manipulation trial—reported the highest efficacy for treating LBP with a combined regimen of exercise and manual therapy; even each therapy separately was still moderately more efficacious than standard medical care.¹³ There were only moderate additional costs incurred for the physical therapies— in contrast to the study in the United States (US). This difference may be due to the study groups; the UK study specifically defined the physical therapy treatments, while the US study included a heterogeneous collection of interventions in the physical therapy group.¹⁰

Furthermore, the expensive spinal-fusion operation has become a popular treatment option for LBP that has failed nonsurgical therapies. The surgery, which incurs an average hospital bill of more than \$34,000 (excluding professional fees), has risen in number performed by 77% between 1996 and 2001.¹⁴ The rates of fusion surgery have risen, in part, because of the added indication of discogenic pain, or LBP without sciatica, in patients with degenerative discs. Since back pain and disc degeneration are a natural part of the aging process, the number of potential candidates for fusion surgery is enormous. However, the high costs associated with the operation, combined with the high rates of re-operation, high incidence of operation complications, and limited support from randomized controlled clinical trials indicating efficacy for degenerative disc disease, suggests that further evidence needs to be collected to support the use of spinal-fusion surgeries for LBP.¹⁴

Quality of Life

Typically, patients affected by chronic LBP have a background of pain with additional intermittent, debilitating flare-ups of pain. In fact, certain epidemiologic studies indicate that LBP would best be described as fluctuating over time with frequent recurrences or exacerbations, rather than simply as “acute” or “chronic”.⁷ Psychologically, this can have an impact on the activities that an LBP-afflicted individual will engage in. LBP patients can become limited by a fear of recurrence, leading to a reduction in their strenuous and leisure activities.³ The condition can also restrict mobility and long distance vehicle travel, as well as disrupt sleep.³ Although many people that suffer from LBP do not seek medical care (61% never sought medical treatment for their LBP in one telephone survey) because of the brief nature of the condition, more than half of those that do seek medical care visit a primary care physician.⁸ Since the majority of LBP sufferers are seen by primary care physicians, these physicians play a key role in determining where a patient is referred.

Etiology and Pathology of Chronic LBP

LBP has been defined as pain localized to the lumbosacral and paraspinal regions⁵ and includes both somatic and radicular etiologies. Somatic pain originates from processes including the spine and surrounding muscles, ligaments, periosteum, facet joints, blood vessels, and intervertebral discs,⁵ while radicular pain stems from neural structures. This monograph focuses on LBP that has a somatic origin.

The primary cause of low back pain is considered to be the natural aging of the discs of the spine.¹⁵ This process results in degeneration of the discs, beginning with subtle biochemical alterations, progressing to microstructural changes, and finally leading to gross structural alterations.¹⁵ The term “disc degeneration” covers a broad range of clinical, radiologic, and pathologic processes that can lead to the deterioration of the facets, ligaments, and muscles of the spine. At the cellular level, degeneration of the disc is caused by reduced production of extracellular matrix.¹⁵ Degeneration is also promoted by reduced blood flow from the end plate, and the resulting lowered nutrient supply to disc cells.¹⁵ These processes lead to macroscopic changes, which include a less distinct boundary between the nucleus and annulus, concentric fissuring and radial tears, and the loss of disc height and turgor.¹⁵ These disc changes can have the secondary effect of increasing the load on the facets, resulting in cartilage degradation there.¹⁵ Furthermore, a narrowing disc space within the lumbar vertebra has been strongly associated with LBP, more than other types of radiographic evidence.¹⁶

Overall, the prevalence of disc degeneration and facet joint arthritis increases with age.¹⁶ The degeneration of the discs of the spine can become evident as early as the mid twenties and is nearly universal by age 50 years. However, typically the physical changes associated with disc degeneration are asymptomatic in most individuals.⁶

In a healthy individual, generating the sensation of pain begins with the stimulation of specialized primary afferent nociceptors

in peripheral tissues. These neurons in turn create electrochemical impulses—or action potentials—that are transmitted to the dorsal horn of the spinal cord. At the dorsal horn, a complex cascade of events involving excitatory neurotransmitters (such as glutamate) and neuropeptides (such as substance P) cause the rapid depolarization of secondary afferent neurons. Subsequent action potentials are generated along the spinothalamic tract, up to the brain, where the signals are processed and interpreted as the perception of pain.

Symptoms and Diagnosis

Despite advances in diagnostic and interventional techniques, it is often difficult to identify the origin of LBP. While some LBP has a specific suspected pathologic cause, the majority of cases are classified as “nonspecific back pain”,³ or labeled as musculoskeletal strain or degenerative disc disease.⁶ Since the etiopathogenesis and mechanisms of the majority of chronic back pain are unknown, the therapies are often empirically based⁶ and are directed at symptomatic relief.

However, it is important to efficiently identify rare, serious causes of LBP, such as neoplasia, infection, inflammation, rheumatoid arthritis, and fractures.^{5,7} Epidemiologic research in the US indicates that of all back pain patients being seen by primary care physicians, 4% have a compression fracture; 3% have spondylolisthesis; 0.7% have a tumor or metastasis; 0.3% exhibit ankylosing spondylitis; and 0.01% have an infection.⁷ Identification of these specific causes of LBP can often be accomplished through a focused physical examination and a thorough patient history. The 1994 Clinical Practice Guideline entitled “Acute Low Back Pain Problems in Adults”, detailed the symptoms that are critical to efficiently identify and that will assist in diagnosing a rare cause of LBP. These signs and symptoms include: weight loss, fever, chills, fatigue, night sweats, increased pain while supine, and recent infections, immunosuppression, trauma, or cancer.¹⁷ Severe tenderness, bilateral neurologic deficits, saddle anesthesia, and hyperalgesia during physical examination also support the presence of a rare, underlying disorder that must be immediately identified.¹⁷

Patients presenting with LBP can be diagnosed through several modalities, beginning with taking a history, followed by clinical interviews and examinations, health questionnaires, pain inventories, and measurements of psychologic and behavioral dysfunction.⁵ There are several measurement tools to assess the extent of LBP and to track the outcome of an intervention, including the Medical Outcomes Study 36-item short-form survey (SF-36 ®), the 12-item short-form survey (SF-12 ®) Oswestry questionnaire, Roland-Morris questionnaire, and EuroQol EQ-5D.¹⁸⁻²¹ Following an initial evaluation, selective diagnostic testing can be used to further isolate an etiology; this testing commonly can include spinal x-rays, magnetic resonance imaging, computed tomography with or without myelography, discography, electromyography, and nerve conduction studies.⁵ Infections and tumors can be screened for using laboratory techniques,⁵ such as erythrocyte sedimentation rates, complete blood counts, or urinalysis.⁸

Approaches to Management of Low Back Pain

Pharmacologic

There are several types of pharmacologic therapies that are commonly administered for the relief of chronic LBP. These include nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxers, membrane-stabilizing agents, antidepressants, and opioids. The following section will briefly discuss the benefits and drawbacks of these therapies.

Nonsteroidal Anti-inflammatory Drugs

NSAIDs have analgesic, anti-inflammatory, and antipyretic activity resulting from their ability to inhibit prostaglandin synthesis.⁵ More specifically, the enzymes cyclooxygenase (COX-1 and COX-2) are inhibited by all first-generation NSAIDs, while second-generation NSAIDs (such as celecoxib) selectively inhibit the COX-2 isoform. COX-2 is inducibly expressed in the peripheral tissues; its inhibition leads to the analgesic activity of both first-generation and second-generation NSAIDs. Nonselective NSAIDs can have adverse effects that include inhibiting platelet aggregation and gastrointestinal distress. Renal insufficiency and liver damage can result from the use of either first-generation or second-generation NSAIDs.⁵

Although NSAIDs have been found to be useful in the management of chronic pain, and strong evidence supports NSAID-use for acute LBP, there is only moderate supporting efficacy data for the treatment of chronic LBP.⁵ Furthermore, a recent review of the literature revealed 51 trials that assessed NSAID use for the treatment of LBP. The studies found that short-term symptomatic pain relief could be achieved, and that there were no particular NSAIDs that exhibited superior effectiveness. However, the review concluded that the studies on chronic LBP provided insufficient evidence to suggest effectiveness.²⁷

Muscle Relaxers

Muscle relaxers are another common treatment for LBP, although there have been very few studies conducted to demonstrate effectiveness.⁵ This group of agents includes centrally acting medications, such as benzodiazepines, antihistamines, and sedatives. They can induce a sedative effect, and tend to produce more central than skeletal muscle relaxant activity after more than 2 weeks of use, sometimes leading to physical dependence. The typical adverse effects of muscle relaxants include dizziness and drowsiness.²⁸

Overall, there have been very few studies that have rigorously assessed the efficacy of muscle relaxants for chronic LBP, however a review by van Tulder and Koes reported the results of 5 different trials. Two randomized controlled trials identified in the systematic review, found that the benzodiazepine—tetrazepam—reduced pain and increased improvement compared with the control.²⁸ The review also reported the results of 3 randomized, controlled trials that demonstrated the nonbenzodiazepines—flupirtine and tolperisone—increased overall improvement after 7 to 21 days, but did not have a significant effect on pain.

Antidepressants: Tricyclic Antidepressants and Selective Serotonin Reuptake Inhibitors

Tricyclic antidepressants inhibit the reuptake of serotonin and noradrenaline. A systematic review of the evidence available in 1997 suggested that tricyclic antidepressants were not effective for chronic LBP.²⁹ However, a more recent review of the literature suggests that antidepressants can decrease pain compared with placebo.²⁸ Also, antidepressants can have a synergistic effect. Beyond analgesia, they can provide sedation—when taken at bedtime—to lessen sleep disturbance, and can relieve depression for patients with that comorbidity.⁵ The possible side effects of tricyclic antidepressants include dry mouth, drowsiness, constipation, urinary retention, and sedation.²⁸

Opioids

Opioids specifically target receptors in the central nervous system, leading to modulation of the perception of pain.⁵ Although randomized, controlled studies support the analgesic effectiveness of opioids, their use for chronic back pain is controversial. Nonetheless, both short-acting and long-acting opioids are prescribed for LBP by some clinicians. Long-acting opioids may reduce medication overuse behaviors, as they are administered on a regular schedule. Possible adverse effects of opioids include constipation, sedation, and drowsiness.^{5, 28}

The effectiveness for controlling chronic back pain of the opioid tramadol alone or in combination with the acetaminophen paracetamol was studied in 2 randomized controlled trials.²⁸ Both trials reported that the opioid significantly lowered pain and increased functional status compared with placebo.²⁸ Overall, trials that rigorously assess the use of opioids for chronic back pain indicate that this drug class can provide substantial pain relief, improve the quality of life, and ameliorate daily functioning.⁵

Approaches to Management of Low Back Pain (continued)

Nonpharmacologic

Interventional Treatments

Various interventional treatments are frequently employed for LBP relief, including spinal injections (epidural, local, and facet injection of steroids), radiofrequency denervation of facet joints, and intradiscal treatments, such as intradiscal electrothermal therapy (IDET). Also, less invasive interventional techniques such as cognitive-behavioral therapy and quota-based exercise therapy, as well as advanced interventional pain therapies including intrathecal opioid pumps and spinal cord stimulators have been studied for reducing LBP. They will all be briefly reviewed in the following section.

Epidural steroid injection is one of the most common nonsurgical interventions for LBP with associated radiculopathy; its usage was reported as early as the 1920s.³⁰ In theory, the treatment reduces inflammation, in part by inhibiting lymphokines and the arachidonic acid pathway.³⁰ However, there have been few investigations of the efficacy of epidural therapy and they are often poorly designed.³⁰ Although the reports on epidural steroid injection efficacy are inconsistent, certainly some patients with radicular pain have benefited from the treatment, but with limited duration.³⁰ The therapeutic effect of epidural steroid injections for discogenic LBP without radicular symptoms is controversial. In contrast, a randomized, controlled trial of facet joint injection therapy reported no difference between steroid and saline injections after 1 month and 3 months.²⁸ Furthermore, adverse effects included infection, hemorrhage, chemical meningitis, and neurologic damage.²⁸ Local injections of steroids have also not been reported to have a significant impact on pain relief.²⁸

Radiofrequency denervation of facet joints has been a common treatment for chronic LBP for the past 2 decades. Pain is reduced or eliminated by the application of radiofrequency energy to temporarily denervate the painful facet joints. A multicenter, randomized, controlled trial assessed the efficacy of this treatment and found no significant difference between the experimental and control group, as assessed by the visual analog score; however, there was a significant perceived benefit.³¹

IDET and other similar intradiscal techniques (nucleoplasty, annuloplasty) are other alternative therapies for chronic LBP, although they are much newer technologies. In the case of IDET, the intervention involves the x-ray-guided-percutaneous insertion of a thermal resistance probe to induce controlled heating of the affected intervertebral disc.³² This may induce disc shrinkage, annular denervation, and reduction in cytokine expression, resulting in concomitant pain reduction.³² A randomized, controlled trial of IDET reported that 40% of patients treated achieved greater than 50% relief of their pain.³³ Thus far, IDET therapy is reported to be a safe procedure.³²

Implanting an opioid pump into the intrathecal space is an advanced interventional pain therapy that has been studied in select patients with chronic nonmalignant pain syndromes.³⁴ A prospective study by Kumar and colleagues considered the efficacy of an implanted morphine pump for pain relief in 16 patients over a 13-to-49 month period.³⁴ The strict selection criteria for inclusion in the study included: 1) a known organic, benign cause of pain that is not directly treatable; 2) poor pain control despite pharmacotherapy and other pain therapy measures including transcutaneous electrical nerve stimulation (TENS) or spinal cord stimulation, if indicated; 3) a favorable response to a double-blind intrathecal morphine test. Patients with psychiatric illness and/or medicolegal issues were excluded from the study. The mean morphine dose administered initially was 1.11 mg/day, increased to 3.1 mg/day after 6-months, and elevated to more than 10 mg/day when taken for over 2 years. At the 6-month follow-up to surgery, the average pain intensity value had decreased from 91.8±2.8 (preoperative) to 24.3±4.3—a 67.5% reduction in average pain score. At the last follow-up, the average pain score was 34.2 ±13.2, a 57.5% reduction from preoperative values. Ten of the 16 implanted patients required supplementary non-narcotic oral analgesics for optimal pain control.³⁴ Furthermore, spinal cord stimulators (SCS) are another advanced interventional therapy that has been investigated for LBP relief. Although the first article on SCS was published in 1967, the neurophysiological mechanism of action for the therapy has not been elucidated. SCS may activate a pain inhibition system or block facilitating pain circuits.³⁵ A systematic synthesis of the literature through June 1994 analyzed the long-term risks and benefits of SCS for patients with failed back surgery syndrome. The included analyses—39 case studies—reported that an average of 59% patients attained ≥ 50% pain relief at follow-up (an average of 16 months following surgery).³⁵ At the time, the dearth of randomized trials inhibited the comparison of SCS to other therapies, placebo, or no treatment.³⁵ Still, in 2004, another systematic review of the literature was able to identify only 1 randomized trial on the effectiveness and safety of SCS for patients with failed back surgery syndrome or complex regional pain syndrome (CRPS).³⁶ The authors of the trial reported a clinically modest, but statistically significant reduction in pain at 6 and 12 months following intervention by SCS and physical therapy, compared to physical therapy alone. On average, 34% of the patients in this study had an adverse event.³⁶ Again, the lack of randomized, controlled trials prohibits making strong conclusions about the benefit of SCS for LBP.

Intense, non-pain-contingent exercise therapy has become more widely-accepted over the last 2 decades for treatment of LBP.³⁷ These quota-based exercise regimens begin with a quantification of back function through the assessment of trunk and lower extremity flexibility, trunk strength, lifting capacity, and cardiovascular endurance.^{37,38} This serves as a baseline level of function and assists clinicians in selecting an appropriate level of exercise for the initiation of therapy.³⁷ Therapy usually is completed within 8 weeks and includes behavioral techniques to help eliminate pain behaviors, alter fears and beliefs about pain, and promote overall wellness.³⁸ A prospective study of 85 patients treated with this aggressive, quota-based approach reported reduced back and leg pain scores by Pain Intensity (0-10) Scale and Oswestry Back Pain Index.³⁸

As studies indicate that psychological and psychosocial factors can be predictive of the likelihood of developing LBP-induced disability, as well as the probability of a positive treatment response,³⁹ it follows that addressing these factors during therapy may improve the likelihood of success. LBP, along with other chronic illnesses, may most accurately be viewed as an “ongoing, long-term fluid condition with reciprocal interplay between the patient’s biological, psychological, and social factors.”³⁹ Based on these theories, studies have been conducted to investigate cognitive-behavioral therapies in concert with other physical methods of rehabilitation for LBP. Gatchel and colleagues conducted a prospective study of 152 patients with chronic LBP disability.⁴⁰ The patients underwent a 3-week comprehensive functional restoration program that included physical reconditioning, as well as a psychological component that consisted of: behavioral stress management training, cognitive-behavioral skills training, individual and group counseling (emphasizing a crisis intervention model), and family counseling.⁴⁰ The outcome measured was return-to-work status, 1 year after discharge from the program. The study demonstrated a comorbidity of chronic mental health and physical disability conditions, and found that functional restoration can be an effective method for treating individuals with LBP.⁴⁰ Also, a systematic review of randomized, controlled trials compared multidisciplinary bio-psycho-social rehabilitation with non-multidisciplinary control interventions.⁴¹ This comprehensive search of the Cochrane Library found 10 trials that met the search criteria, with a total of 1964 chronic LBP patients included. The studies provided evidence that intensive multidisciplinary bio-psycho-social rehabilitation with a functional restoration approach can reduce pain and improve function.⁴¹

Approaches to Management of Low Back Pain (continued)

Physical Medicine Interventions

A large variety of modalities is commonly used for the treatment of chronic LBP, including bed rest, activity modification, exercise therapy, and several physical interventions—heat, ice, electrical stimulation, ultrasound, TENS, and traction-based therapies. Overall, many of these treatments do not have rigorously conducted clinical trial evidence supporting their use,²⁹ but the available data supporting several of these physical medicine interventions are briefly discussed in the following section.

Exercise Therapies — Exercise therapy is considered a widely accepted method for the reduction of chronic LBP.⁴² This type of therapy is intended to help recondition, increase the range of motion, and improve muscle strength and length.⁵ However, the term “exercise therapy” encompasses a heterogeneous collection of exercises that lack definition of intensity, duration, and frequency.⁴² There is no consensus on the most effective approach, nor is there a clear mechanism explaining why exercise is of benefit to LBP sufferers, to provide guidance towards defining an approach.⁴² A meta-analysis of 43 randomized, controlled trials of chronic LBP patients indicated that exercise therapy can effectively reduce pain and functional limitations in the lower back, although the low quality of some of the studies limits conclusive interpretations.⁴³ In particular, studies reported that individually designed strengthening or stabilizing programs were effective in a healthcare setting.⁴³

Transcutaneous Electrical Nerve Stimulation — More than 30 years ago TENS was developed as an alternative to pharmacologic treatments for chronic pain.⁴⁴ Despite its widespread use, there is no clinical evidence to support its efficacy for the relief of chronic LBP. A review of 5 clinical trials suggests that the technique is not effective for the treatment of chronic LBP.^{5,45} The clinical trials showed no significant difference between the TENS group and the placebo group.^{44, 45}

Acupuncture Therapy — Acupuncture therapy involves the application of needles placed at specific locations on the skin.⁴⁶ It has been proposed that acupuncture may provide pain relief by the gate control theory of pain (where the sensory input of needling inhibits the sensation of other inputs, ie, LBP) and by inducing the production of endorphins, serotonin, and acetylcholine within the central nervous system.⁴⁶ Data from 33 randomized, controlled trials were considered to assess the

effectiveness of acupuncture at relieving LBP.⁴⁷ Measurements of pain relief, functional status, overall improvement, time to return to work, and analgesic consumption were included as outcomes. The authors conclude that acupuncture effectively relieves chronic LBP better than no treatment or sham treatment, but no more than other active therapies.⁴⁷

Massage Therapy — Massage therapy is directed at reducing muscle spasms and tension, and improving circulation.²⁸ The soft tissues are manipulated by hand or a mechanical device, and vary in types from Shiatsu, Swedish, and friction, to trigger point and neuromuscular. Studies indicate that massage is more effective at reducing chronic nonspecific LBP than sham treatment, but comparisons with conventional therapies have been inconclusive.⁴⁶

Spinal Manipulation Therapy — Spinal manipulation therapy is a common treatment for LBP that involves a high-velocity thrust to a joint beyond its restricted range of movement.⁴⁸ The rationale for this type of rehabilitation involves reducing bulging discs, correcting the internal displacement of disc fragments, and freeing adhesions around a prolapsed disc or facet joint.⁴⁸ Furthermore, other goals include relaxing entrapped synovial folds or plica, relaxing hypertonic muscles, and unbuckling motion segments that have undergone displacement.⁴⁸ Spinal manipulation has been shown to be more effective than the control comparison and just as effective as conventional therapies—such as analgesics, physical therapy, and exercise—at providing pain relief of the lower back.^{46, 49}

Approaches to Management of Low Back Pain (continued)

Traditional Traction Therapy — The primary rationale for traction therapy is to increase intervertebral space and the relaxation of spinal muscles. Harnesses are fitted on the lower rib cage and iliac crest for lumbar traction therapy. Force can be administered by the therapist (as in manual traction), by a motorized pulley (for motorized traction), or less commonly, by gravity (for inverted suspension), or by a pulley and weights (in bed-rest traction).⁵⁰ It has been proposed that a traction force of at least 26% of the body weight is required to overcome friction and to begin to induce spinal elongation.⁵¹ Forces below this value are often used as a placebo in controlled studies,⁵⁰ while traction therapies that exceed 50% of the body weight can have adverse effects.⁵⁰

A systematic review of the literature for studies conducted before February 1995 on traction therapy for the relief of back pain indicated that most studies were not rigorously conducted enough to demonstrate efficacy, and that many studies lacked power, due to small sample sizes.⁵⁰ The authors suggested that it was not possible to make a conclusive determination about the efficaciousness of traction. Further, properly designed trials were recommended to assess the effectiveness of traction therapy for LBP.

A more recent review of randomized, controlled trials on the efficacy of traction therapy identified and analyzed 13 LBP studies. Nine of these studies reported negative findings while 3 reported positive findings, and a pilot study was inconclusive.⁵²

The one recognized high-quality study⁵² was a randomized, controlled trial conducted by Beurskens and colleagues, which considered the efficacy of motorized, continuous high-dose traction for the reduction of nonspecific, chronic back pain. One hundred fifty-one patients were divided between the treatment group and a low-dose traction (control) group. The trial was not able to demonstrate a benefit in high-dose traction, when compared with the sham group.^{53, 54}

Spinal Decompression Therapy — Vertebral axial decompression (VAX-D®) therapy has evolved from the basic rationales and methods originally used in traction therapy. This therapy uses spinal distraction to reduce intradiscal pressure by inducing disc and nerve root decompression. Ultimately this can lead to back pain relief.

During VAX-D® therapy, patients lie prone on a split table, with the upper body over the stationary portion of the table.^{55, 56} Patients are required to actively participate by grasping pegs at the table's edge. Distraction tensions are applied through a pelvic harness attached to a tensionometer and by separation of the movable part of the table.^{55, 56} It has been shown in clinical studies to create negative intradiscal pressures, promoting the relief of chronic LBP for patients with associated leg

pain.⁵⁷ Two randomized, controlled clinical studies have reported the efficacy of VAX-D® for the relief of LBP. Seven hundred seventy-eight patients undergoing VAX-D® treatment and diagnosed with herniated disc, degenerative disc, or facet syndrome were studied in data collected from 22 medical centers for one study.⁵⁸ VAX-D® therapy was successful for 71% of the patients, where success was defined as a reduction in pain to 0 or 1 on a 5-point scale.⁵⁸ Although VAX-D® has widely been promoted as an effective and safe treatment for degenerated and herniated lumbar intervertebral discs, there has been a report of a single case of exacerbated radicular pain and further enlargement of disc protrusion following VAX-D® therapy.⁵⁹ This required urgent surgical intervention to lessen the potential for sudden deterioration.⁵⁹

Spinal Mobilization Therapy (Computer Monitored) — Intervertebral Differential Dynamics Therapy (IDD Therapy ®) was developed in the late 1990s to reproducibly mobilize and distract isolated lumbar segments. A computer-directed patient harness system delivers manipulative forces to the patient lying supine. The device induces a negative pressure state that has a mobilizing effect on the disc. The pull angle can be adjusted to accommodate lumbar lordosis and allow for the greatest patient comfort while targeting the affected intervertebral segment. Furthermore, specifically applied waveform adjustments at varying angles promote intermittent force release, reducing the incidence of posttreatment flare-up and allowing for higher maximum treatment force (where target treatment force is 50% of the body weight + 10 lbs). Also, standardized IDD Therapy ® includes the application of heat and ice, pretreatment and posttreatment, as well as instruction in lumbar stabilization exercises. Overall, a negative hydrostatic pressure within the affected disc is reported to induce physiologic changes that ultimately relieve LBP.⁶⁰ The original clinical data on IDD Therapy ® indicated that 86% of ruptured intervertebral disc patients achieved “good” to “excellent” results with IDD Therapy ® (where “good” is 50% to 89% improvement and “excellent” is 90% to 100% improvement).⁶⁰ The active control group—which received classic traction therapy—had 55% of the patients achieve “good” results and no patients report a rating of “excellent”.⁶⁰

More recently, a review of results from 10 clinics confirmed the initial IDD Therapy ® findings,⁶¹ as did a retrospective study of 33 patients.⁶² Although 54% of the patients in the cohort had previous failed therapy for LBP, the mean improvement following IDD Therapy ® was 5.23 points on the Neuropathic Pain Scale. The authors recommend further clinical trials to confirm these findings.⁶²

Surgical

Screening a patient's history is an important step in the selection process to determine appropriate patients for surgical treatments of LBP. Patients with untreated mood disorders, opioid dependency, and/or legal claims such as workers compensation LBP should not be treated surgically for LBP. Patients with radiculopathy that exhibit progressive motor deficits and/or cauda equina syndrome *should* be treated with surgery to avoid long-term neurological deficits.⁶³ However, the majority of surgeries are performed because of lack of improvement with nonsurgical measures. Even successful surgeries have a 15% relapse rate within 4 years.⁶³ Some authors believe that as many as 50% of spinal surgeries are considered unnecessary and fail to provide long-term pain relief.¹ Despite this, the rate of surgical intervention for back pain in the US is at least 40% higher than in other countries.⁴

For appropriately selected patients, lumbar fusion surgery can result in pain relief. A randomized, controlled, multicenter study of 294 patients with radiologic evidence of disc degeneration compared lumbar fusion therapy with physical therapy.⁶⁴ Fritzell and colleagues reported that the surgical group had a 33% reduction in back pain, compared to 7% in the non-surgical group ($P=0.0002$). Pain improved the most during the initial 6 months following surgery then gradually deteriorated. Also, the disability of the surgical group was reduced by 25% compared to 6% in the nonsurgical patients ($P=0.015$), measured by Oswestry Disability Index. The net back-to-work rate was 36% in the surgically-treated patients, compared to 13% in the non-surgical group ($P=0.0002$). The surgical group had an early complication rate of 17%.⁶⁴ In contrast, a single-blind, randomized trial compared the effectiveness of lumbar fusion with cognitive intervention and exercises for patients with chronic LBP, and found similar effectiveness between the therapies.⁶⁵ Oswestry Disability Index scores were significantly reduced following surgery from 42 to 26, compared to 42 to 30 with the non-surgical intervention group—a mean difference of 2.3 ($P=0.33$). Improvements in back pain, analgesic use, emotional distress, life satisfaction, and return-to-work were similar between the 2 therapy groups. An independent observer rated the success rate following surgery at 70% and following cognitive intervention/exercises at 76%. The early complication rate in the surgical group was 18%.⁶⁵

A Multidisciplinary Approach to the Management of LBP

A comprehensive approach to pain management involving both pharmacologic agents and physical therapy methods will likely prove to be the most effective way for controlling chronic LBP. The Multidisciplinary Pain Center (MPC) is an interdisciplinary approach to the diagnosis and management of chronic pain. This model can provide a comprehensive program that addresses clinical symptoms and associated distress, dysfunction, and disability.⁶⁶ The MPC model can successfully provide pain relief, while incurring smaller long-term expenditures. A meta-analysis of the efficacy of 65 multidisciplinary treatments for chronic LBP indicated that actively treated patients functioned 60% better than control subjects on the short term.⁶⁷ Furthermore, the patients undergoing a multidisciplinary approach remained 55% better than controls on the long term and were twice as likely to return to work.⁶⁷ Comprehensive pain clinics and spine centers can provide both pharmacologic approaches and physical therapy methods to manage chronic LBP. Physical medicine approaches as adjuncts to pharmacologic therapies will likely achieve the highest success in providing relief for chronic LBP. Surgical treatments for chronic LBP should be reserved for patients with identifiable pain generators, significant functional limitations, and a favorable psychosocial profile.

IDD Therapy® is a registered trademark of North American Medical Corporation.

SF-12® and SF-36® are registered trademarks of Medical Outcomes Trust, Inc.

VAX-D® is a registered trademark of VAX-D Medical Technologies.

References

1. Pai S, Sundaram LJ. Low back pain: an economic assessment in the United States. *Orthop Clin North Am.* 2004;35:1-5.
2. Licciardone JC. The unique role of osteopathic physicians in treating patients with low back pain. *J Am Osteopath Assoc.* 2004;104:S13-18.
3. Woolf AD, Pfleger B. Burden of major musculoskeletal conditions. *Bull World Health Organ.* 2003;81:646-656.
4. Andersson GB. Epidemiological features of chronic low-back pain. *Lancet.* 1999;354:581-585.
5. Grabois M. Management of chronic low back pain. *Am J Phys Med Rehabil.* 2005;84:S29-41.
6. Atkinson JH. Chronic back pain: searching for causes and cures. *J Rheumatol.* 2004;31:2323-2325.
7. van Tulder M, Koes B, Bombardier C. Low back pain. *Best Pract Res Clin Rheumatol.* 2002;16:761-775.
8. Atlas SJ, Deyo RA. Evaluating and managing acute low back pain in the primary care setting. *J Gen Intern Med.* 2001;16:120-131.
9. Kominski GF, Heslin KC, Morgenstern H, Hurwitz EL, Harber PI. Economic evaluation of four treatments for low-back pain: results from a randomized controlled trial. *Med Care.* 2005;43:428-435.
10. Carey TS, Freburger J. Prudence, nihilism, and the treatment of low-back pain. *Med Care.* 2005;43:425-427.
11. Carey TS, Garrett J, Jackman A, et al. The outcomes and costs of care for acute low back pain among patients seen by primary care practitioners, chiropractors, and orthopedic surgeons. The North Carolina Back Pain Project. *N Engl J Med.* 1995;333:913-917.
12. Shekelle PG, Markovich M, Louie R. Comparing the costs between provider types of episodes of back pain care. *Spine.* 1995;20:221-226; discussion 227.

13. United Kingdom back pain exercise and manipulation (UK BEAM) randomised trial: cost effectiveness of physical treatments for back pain in primary care. *Bmj*. 2004;329:1381.
14. Deyo RA, Nachemson A, Mirza SK. Spinal-fusion surgery - the case for restraint. *N Engl J Med*. 2004;350:722-726.
15. Benoist M. Natural history of the aging spine. *Eur Spine J*. 2003;12 Suppl 2:S86-89.
16. Manek NJ, MacGregor AJ. Epidemiology of back disorders: prevalence, risk factors, and prognosis. *Curr Opin Rheumatol*. 2005;17:134-140.
17. Bach HG, Lim RD. Minimally invasive spine surgery for low back pain. *Dis Mon*. 2005;51:34-57.
18. Deyo RA, Battie M, Beurskens AJ, et al. Outcome measures for low back pain research. A proposal for standardized use. *Spine*. 1998;23:2003-2013.
19. Resnik L, Dobrykowski E. Outcomes measurement for patients with low back pain. *Orthop Nurs*. 2005;24:14-24.
20. Resnik L, Dobrzykowski E. Guide to outcomes measurement for patients with low back pain syndromes. *J Orthop Sports Phys Ther*. 2003;33:307-316; discussion 317-308.
21. Schaufele MK, Boden SD. Outcome research in patients with chronic low back pain. *Orthop Clin North Am*. 2003;34:231-237.
22. Roland M, Morris R. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. *Spine*. 1983;8:141-144.
23. Fairbank JC, Pynsent PB. The Oswestry Disability Index. *Spine*. 2000;25:2940-2952; discussion 2952.
24. Ware J, Jr., Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care*. 1996;34:220-233.
25. Ware JE, Jr., Sherbourne CD. The MOS 36-item short-form health survey (SF-36®). I. Conceptual framework and item selection. *Med Care*. 1992;30:473-483.
26. EuroQol—a new facility for the measurement of health-related quality of life. The EuroQol Group. *Health Policy*. 1990;16:199-208.
27. van Tulder MW, Scholten RJ, Koes BW, Deyo RA. Nonsteroidal anti-inflammatory drugs for low back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine*. 2000;25:2501-2513.
28. van Tulder M, Koes B. Low back pain (chronic). *Clin Evid*. 2004:1659-1684.
29. van Tulder MW, Koes BW, Bouter LM. Conservative treatment of acute and chronic nonspecific low back pain. A systematic review of randomized controlled trials of the most common interventions. *Spine*. 1997;22:2128-2156.
30. McLain RF, Kapural L, Mekhail NA. Epidural steroid therapy for back and leg pain: mechanisms of action and efficacy. *Spine J*. 2005;5:191-201.
31. van Wijk RM, Geurts JW, Wynne HJ, et al. Radiofrequency denervation of lumbar facet joints in the treatment of chronic low back pain: a randomized, double-blind, sham lesion-controlled trial. *Clin J Pain*. 2005;21:335-344.

32. Welch WC, Gerszten PC. Alternative strategies for lumbar discectomy: intradiscal electrotherapy and nucleoplasty. *Neurosurg Focus*. 2002;13:E7.
33. Pauza KJ, Howell S, Dreyfuss P, et al. A randomized, placebo-controlled trial of intradiscal electrothermal therapy for the treatment of discogenic low back pain. *Spine J*. 2004;4:27-35.
34. Kumar K, Kelly M, Pirlot T. Continuous intrathecal morphine treatment for chronic pain of nonmalignant etiology: long-term benefits and efficacy. *Surg Neurol*. 2001;55:79-86; discussion 86-78.
35. Turner JA, Loeser JD, Bell KG. Spinal cord stimulation for chronic low back pain: a systematic literature synthesis. *Neurosurgery*. 1995;37:1088-1095; discussion 1095-1086.
36. Turner JA, Loeser JD, Deyo RA, Sanders SB. Spinal cord stimulation for patients with failed back surgery syndrome or complex regional pain syndrome: a systematic review of effectiveness and complications. *Pain*. 2004;108:137-147.
37. Cohen I, Rainville J. Aggressive exercise as treatment for chronic low back pain. *Sports Med*. 2002;32:75-82.
38. Rainville J, Sobel J, Hartigan C, Monlux G, Bean J. Decreasing disability in chronic back pain through aggressive spine rehabilitation. *J Rehabil Res Dev*. 1997;34:383-393.
39. Gatchel RJ, Gardea MA. Psychosocial issues: their importance in predicting disability, response to treatment, and search for compensation. *Neurol Clin*. 1999;17:149-166.
40. Gatchel RJ, Polatin PB, Mayer TG, Garcy PD. Psychopathology and the rehabilitation of patients with chronic low back pain disability. *Arch Phys Med Rehabil*. 1994;75:666-670.
41. Guzman J, Esmail R, Karjalainen K, et al. Multidisciplinary bio-psycho-social rehabilitation for chronic low back pain. *Cochrane Database Syst Rev*. 2002:CD000963.
42. Hayden JA, van Tulder MW, Tomlinson G. Systematic review: strategies for using exercise therapy to improve outcomes in chronic low back pain. *Ann Intern Med*. 2005;142:776-785.
43. Hayden JA, van Tulder MW, Malmivaara AV, Koes BW. Meta-analysis: exercise therapy for nonspecific low back pain. *Ann Intern Med*. 2005;142:765-775.
44. Brosseau L, Milne S, Robinson V, et al. Efficacy of the transcutaneous electrical nerve stimulation for the treatment of chronic low back pain: a meta-analysis. *Spine*. 2002;27:596-603.
45. Milne S, Welch V, Brosseau L, et al. Transcutaneous electrical nerve stimulation (TENS) for chronic low back pain. *Cochrane Database Syst Rev*. 2001:CD003008.
46. van Tulder MW, Furlan AD, Gagnier JJ. Complementary and alternative therapies for low back pain. *Best Pract Res Clin Rheumatol*. 2005;19:639-654.
47. Manheimer E, White A, Berman B, Forsy K, Ernst E. Meta-analysis: acupuncture for low back pain. *Ann Intern Med*. 2005;142:651-663.
48. Koes BW, Assendelft WJ, van der Heijden GJ, Bouter LM. Spinal manipulation for low back pain. An updated systematic review of randomized clinical trials. *Spine*. 1996;21:2860-2871; discussion 2872-2863.
49. Margo K. Spinal manipulative therapy for low back pain. *Am Fam Physician*. 2005;71:464-465.

50. van der Heijden GJ, Beurskens AJ, Koes BW, et al. The efficacy of traction for back and neck pain: a systematic, blinded review of randomized clinical trial methods. *Phys Ther.* 1995;75:93-104.
51. Judovich BD. Lumbar traction therapy and dissipated force factors. *Lancet.* 1954;74:411-414.
52. Harte AA, Baxter GD, Gracey JH. The efficacy of traction for back pain: a systematic review of randomized controlled trials. *Arch Phys Med Rehabil.* 2003;84:1542-1553.
53. Beurskens AJ, de Vet HC, Koke AJ, et al. Efficacy of traction for nonspecific low back pain. 12-week and 6-month results of a randomized clinical trial. *Spine.* 1997;22:2756-2762.
54. Beurskens AJ, de Vet HC, Koke AJ, et al. Efficacy of traction for non-specific low back pain: a randomised clinical trial. *Lancet.* 1995;346:1596-1600.
55. Ramos G. Efficacy of vertebral axial decompression on chronic low back pain: study of dosage regimen. *Neurol Res.* 2004;26:320-324.
56. Ramos G, Martin W. Effects of vertebral axial decompression on intradiscal pressure. *J Neurosurg.* 1994;81:350-353.
57. Sherry E, Kitchener P, Smart R. A prospective randomized controlled study of VAX-D® and TENS for the treatment of chronic low back pain. *Neurol Res.* 2001;23:780-784.
58. Gose EE, Naguszewski WK, Naguszewski RK. Vertebral axial decompression therapy for pain associated with herniated or degenerated discs or facet syndrome: an outcome study. *Neurol Res.* 1998;20:186-190.
59. Deen HG, Jr., Rizzo TD, Fenton DS. Sudden progression of lumbar disk protrusion during vertebral axial decompression traction therapy. *Mayo Clin Proc.* 2003;78:1554-1556.
60. Shealy CN, Borgmeyer V. Emerging Technologies: Preliminary Findings. *American Journal of Pain Management.* 1997;7:63-65.
61. Shealy CN. Intervertebral differential dynamics therapy. *Practical Pain Management.* 2005.
62. Wesemann MM, Koladia NK, Shealy N. Long term effect-analysis of IDD Therapy® in low back pain: A retrospective clinical study. *Am J Pain Man.* 2005.
63. Margo K. Diagnosis, treatment and prognosis in patients with low back pain. *Am Fam Physician.* 1994;49:171-179, 183-174.
64. Fritzell P, Hagg O, Wessberg P, Nordwall A. 2001 Volvo Award Winner in Clinical Studies: Lumbar fusion versus nonsurgical treatment for chronic low back pain: a multicenter randomized controlled trial from the Swedish Lumbar Spine Study Group. *Spine.* 2001;26:2521-2532; discussion 2532-2524.
65. Brox JI, Sorensen R, Friis A, et al. Randomized clinical trial of lumbar instrumented fusion and cognitive intervention and exercises in patients with chronic low back pain and disc degeneration. *Spine.* 2003;28:1913-1921.
66. Turk DC, Stacey BR. Multidisciplinary pain centers in the treatment of chronic back pain. In: Frymoyer JW, ed. *The Adult Spine: Principles and Practice.* Philadelphia, Pa: Lippincott-Raven; 1997:253-274.
67. Flor H, Fydrich T, Turk DC. Efficacy of multidisciplinary pain treatment centers: a meta-analytic review. *Pain.* 1992;49:221-230.

Powered by Mediwire Network™

© 2005 Advanstar Communications. All rights reserved.
Reproduction in whole or in part is prohibited.