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# Selected Instructional Course Lectures

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# Radiculopathy and the Herniated Lumbar Disc

## Controversies Regarding Pathophysiology and Management

BY JOHN M. RHEE, MD, MICHAEL SCHAUFELE, MD, AND WILLIAM A. ABDU, MD

*An Instructional Course Lecture, American Academy of Orthopaedic Surgeons*

Lumbar disc herniations remain among the most common diagnoses encountered in clinical spine practice. The incidence of symptomatic lumbar disc herniations in the American population has been estimated to be 1% to 2%[1], for which approximately 200,000 lumbar discectomies are performed annually[2]. Yet despite the frequency with which lumbar disc herniation occurs, there is substantial controversy regarding its pathophysiology and treatment. For example, from the standpoint of basic science, mounting evidence suggests that biochemical factors—in addition to the mechanical effects of the disc material on the nerve root—underlie the development of symptomatic radiculopathy, but those factors remain to be clearly elucidated. On the clinical end of the spectrum, large (five-to-fifteen-fold) variations[3] in the rates of lumbar surgery in geographically adjacent areas suggest radical heterogeneity in the application of surgical criteria to this diagnosis. In this lecture, we examine the available basic science regarding the anatomy and pathophysiology of lumbar disc herniations as well as the clinical evidence supporting nonoperative compared with surgical management of this common, yet surprisingly poorly understood, orthopaedic disorder.

### Anatomy of Lumbar Disc Herniation

Structurally, the lumbar disc has three components: the anulus fibrosus, which forms the circumferential rim of the disc; the nucleus pulposus, which composes its central core; and the cartilaginous end plates on the adjacent vertebral bodies. The anulus has a multilayer lamellar architecture made of collagen fibers. Within each layer, the collagen is oriented at approximately 30° to the horizontal. Each successive layer is oriented at 30° to the horizontal in the opposite direction, leading to a "criss-cross" type pattern. This composition allows the anulus, and in particular the outer anulus, which has the highest tensile modulus, to resist torsional, axial, and tensile loads. The nucleus pulposus provides resistance to axial compression and is the principal determinant of disc height because of its unique composition consisting of large, highly charged proteoglycan macromolecules within a collagen matrix. These macromolecules are hydrophilic and are contained within the confines of the anulus peripherally and the end plates above and below. Thus, imbibed water causes the nucleus to swell and to generate large hydrostatic pressures within the disc. A healthy nucleus consists of approximately 70% water. The nucleus also contains a cellular component of both fibroblast-like and chondrocyte-like cells. These cells maintain the matrix in which they exist, and they also receive metabolic nutrients that diffuse through the matrix.

The intervertebral disc is anatomically unique for several reasons. First, it is mostly avascular—it is the largest avascular structure in the body. Blood vessels lie on the surface of the anulus but penetrate only a very short distance into the outer portions of the anulus. Similarly, blood vessels from the vertebral body lie against the cartilaginous end plates but do not enter the central regions of the disc. As a result, disc cells derive their nutrition from diffusion through the end plates and connective-tissue transport from one part of the matrix to the other. Second, the disc is only minimally innervated. Nerve endings are present only on the surface of the disc, and they penetrate a very short distance into the outer anulus. The normal inner anulus and nucleus completely lack innervation. In contrast, the posterior and anterior longitudinal ligaments are innervated. The anterior longitudinal ligament receives nerve branches from the segmental ventral ramus and sympathetic trunk. The posterior longitudinal ligament is innervated by a branch of the dorsal root ganglion known as the *sinuvertebral nerve.* Experimental studies of patients undergoing discectomy with local anesthetic have demonstrated that surgical stimulation of the posterior longitudinal ligament can cause low-back pain[4]. Thus, stimulation of the sinuvertebral nerve may be one mediator of the low-back-pain component associated with lumbar disc herniations and anular tears.

The terminology used to describe the spectrum of lumbar disc herniations varies and is often confusing; however, when properly applied, it provides useful descriptive information. One useful method of classifying disc herniations is according to whether the herniated fragments are "contained" or "noncontained" by the anulus[5]. A protrusion is a focal bulging of nuclear material contained by the anulus (i.e., the anular fibers remain continuous and attached to the vertebral bodies). Subanular extrusions occur when the anulus remains intact but the fragment has migrated behind the body either above or below the disc while maintaining continuity with the disc. Transanular extruded disc herniations occur when the fragments have ruptured through the anulus but maintain continuity with the disc space of origin. A sequestration arises when the material has not only broken through the anulus but has also migrated away from the disc space of origin and is no longer in contact with it. Any of the three components of the intervertebral disc (the nucleus, anulus, or end plate), alone or in combination, may be the offending material when a disc herniates.

## Pathophysiology of Lumbar Disc Herniation

The origins of the modern era of lumbar disc surgery can be traced to the seminal work of Mixter and Barr[6], seventy years ago. Those authors found that sciatic pain could be relieved by removing herniated disc material compressing a nerve root. Logically, the association was made between lumbar disc herniation and the clinical entity of sciatica. That finding led to the general assumption that mechanical compression of the nerve root is the primary pathogenic factor inducing radiculopathy. Several lines of evidence support this notion. First, the structure of the nerve root renders it relatively poorly resistant to compression. Like peripheral nerves, nerve roots have an endoneurium. However, the layers equivalent to the perineurium and epineurium are cerebrospinal fluid and dural lining, respectively. Thus, the nerve root is a comparatively delicate structure that is not well insulated to resist compressive forces. Second, because nerve roots are tethered to the vertebral body at their takeoff from the common dural sac and to the subjacent pedicle within the foramen by ligamentous attachments, a disc herniation ventral to the root is poised to generate high tensile forces. The situation is analogous to the tension generated in a bowstring by the pull of an archer's hand. Third, animal models of cauda equina compression have demonstrated that compression of a nerve root impairs its nutrition. In a series of experiments, Olmarker et al. showed that mechanical compression on nerve roots within the porcine cauda equina led to decreased nutrient delivery by reducing both blood flow and nutrient diffusion from cerebrospinal fluid[7,8]. Histologically, compressed nerve roots demonstrate evidence of intraneural edema, which can directly lead to nerve fibrosis and injury. Alternatively, intraneural edema can secondarily lead to an intraneural "compartment syndrome," as pressures within the nerve root overcome perfusion pressures, resulting in nerve root ischemia and injury[9].

Although the above and other studies suggest that the mechanical effect of a herniated disc is the main factor in the genesis of radiculopathy, other lines of evidence indicate that mechanical compression alone may not be a sufficient cause for the radiculopathy associated with herniated discs. First, magnetic resonance imaging studies have shown that nerve root compression is often asymptomatic (Figs. 1-A and 1-B). Boden et al.[10] found that, in a group of people who had never had radicular pain, 20% of those under the age of sixty years and 36% of those over the age of sixty years had evidence of a herniated disc on magnetic resonance imaging. Second, other reports have suggested that, while irritated nerve roots demonstrate susceptibility to mechanical compression, normal (nonsensitized) roots do not. Smyth and Wright[11] found that, when sutures placed around nerve roots at the time of surgery were tugged postoperatively, only those roots that had been noted at surgery to be compressed by a herniated disc generated a radicular pain response. When there had been no intraoperative evidence of root compression by a herniated disc, tugging on the suture and creating tension in the root postoperatively did not elicit radicular symptoms. Kuslich et al.[4] performed lumbar discectomy, using local anesthesia, on awake patients and found that surgical stimulation of compressed roots caused pain 90% of the time, whereas manipulation of normal roots provoked pain only 9% of the time. These observations suggest that a nerve root needs to be sensitized in order to be mechanically susceptible and that mechanical compression alone is not the sine qua non of radiculopathy.

A growing body of evidence has implicated bioactive molecules within the disc as important in sensitizing nerve roots and participating in the pathogenesis of radiculopathy. Olmarker et al.[12] reported that, when autogenous nucleus pulposus was applied to the porcine cauda equina, physiologic and anatomic evidence suggestive of radiculopathy was noted, even in the absence of any nerve root compression. Compared with control animals in which autologous fat had been applied epidurally, those in which nucleus pulposus had been applied demonstrated a decrease in nerve conduction velocity and histologic evidence of nerve fiber degeneration (axonal swelling and demyelination). This study suggested that nuclear material could itself lead to neural injury in the absence of mechanical compression. Authors of later studies attempted to determine whether the observed changes in nerve structure and electrophysiology correlated with a clinical syndrome of radiculopathy. In one such study, rats underwent laminectomy and were randomized to one of three groups: (1) incision of the disc such that nuclear contents could come into contact with, but not displace, the nerve root; (2) displacement of the nerve root by placement of a pin into the vertebral body that deflected the course of the root but no incision of the disc; or (3) incision of the disc and displacement of the nerve root with the vertebral body pin[13]. Only the animals that had both disc incision and root displacement displayed behaviors consistent with pain and radiculopathy.

The picture emerging is that disc herniation-associated radiculopathy is both a biochemical and a mechanical disorder. A number of bioactive molecules known to be present in the nucleus pulposus, including interleukins and other inflammatory factors[14-16], have been purported to be biochemical "sensitizers" capable of making nerve roots susceptible to the mechanical effect of the herniated mass. Tumor necrosis factor-alpha (TNF-α) has received considerable attention in this regard. In the porcine model, TNF-α caused reductions in nerve conduction velocity similar to those seen with autologous nucleus pulposus, whereas interleukin 1-beta (IL-1β) and interferon gamma (INF-γ) did not[17]. Furthermore, TNF-α inhibition with infliximab (a monoclonal antibody to TNF-α) blocked the pain behaviors noted above after the performance of disc incision and root displacement in rats[18]. Overall, the roles of TNF-α and other bioactive agents in herniated-disc-associated radiculopathy remain poorly undiagnosed.

## Epidemiology and Natural History

Various studies have shown that the lifetime prevalence of a major episode of low back pain ranges from 60% to 80%, but only 10% of these episodes are accompanied by sciatica. Sciatica lasting longer than two weeks is even less common, with a lifetime prevalence of 1.6%[19]. The highest prevalence (23.7 per 100 persons) is in individuals between the ages of forty-five and sixty-four years old[20]. A sedentary lifestyle, frequent driving, chronic cough, pregnancy, smoking, and frequent lifting of heavy objects are considered risk factors[19,21,22].

It is commonly agreed that lumbar disc herniation has a favorable natural history (i.e., the clinical course of the disease without therapeutic intervention). Hakelius examined essentially a natural history cohort, in that the patients were treated with only bed rest and a corset for two months, and he observed a marked reduction in pain and improvement in function over time: 80% of the patients had major im-

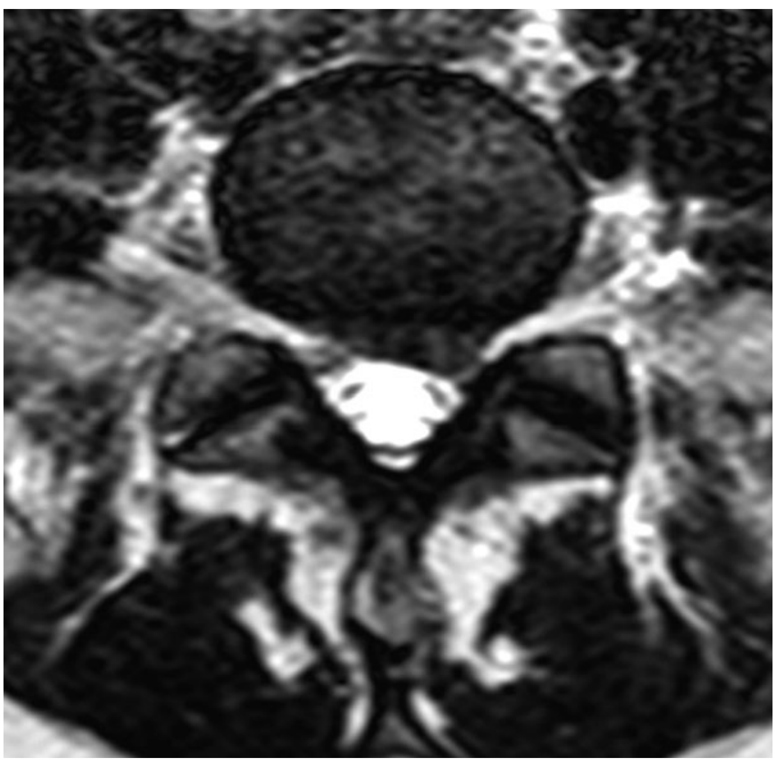

Fig. 1-A

Fig. 1-B

Asymptomatic posterolateral disc herniation in a forty-five-year-old woman with pain in the right leg. She had absolutely no symptoms in the left leg, despite the axial (Fig. 1-A) and sagittal (Fig. 1-B) T2-weighted magnetic resonance imaging scans demonstrating an extruded left L5-S1 disc herniation. Asymptomatic disc herniations are not uncommon, underlying the need to treat symptoms rather than magnetic resonance imaging findings per se.

provements after six weeks; 90%, after twelve weeks; and 93%, after twenty-four weeks[23]. Other studies have revealed less favorable results in that, although most patients without surgical treatment had improvement, 30% had persistent pain and restrictions at work and leisure activities after one year[24]. The majority of disc herniations diminish in size over time, with 80% decreasing by >50% in one study[25]. Larger disc herniations tend to regress more, most likely because of their higher water content. A positive correlation has been noted between regression of lumbar disc herniations and resolution of symptoms[25,26], and regression is thought to occur as the herniated tissue dehydrates and immunological responses help to resorb the disc material. In terms of motor function, Hakelius did not find a significant advantage to surgical treatment of patients with stable motor deficits (excluding those with cauda equina syndrome): 45% of such patients had improvement with nonoperative treatment, and 53% had improvement after surgery.

## Evidence Regarding Nonoperative Treatment

Currently accepted indications for nonoperative treatment of lumbar disc herniations include the absence of a progressive neurological deficit or cauda equina syndrome. Thus, nonoperative treatment is the initial "default" pathway for the majority of patients with lumbar radiculopathy due to disc herniation. It is not clear, however, whether nonoperative treatment offers improvement over the natural history of the disorder. Although there have been numerous studies of nonoperative treatments for low-back pain, there have been few randomized controlled trials specifically comparing the various nonoperative regimens (e.g., physical therapy, medications, traction, manipulation, immobilization, and spinal injections) with the natural history (no treatment at all). However, predictors of favorable outcomes of nonoperative care have been reported to include a negative result on the crossed straight-leg-raising test, absence of leg pain with spinal extension, absence of stenosis on imaging studies, favorable response to steroids, return of neurological deficits within twelve weeks, a motivated physically fit patient with more than twelve years of education, no Workers' Compensation claim, and a normal psychological profile[27]. Nonoperative treatment of noncontained disc herniations may also have a favorable outcome[28].

### *Medications*

Commonly used medications for pain associated with lumbar disc herniations include nonsteroidal anti-inflammatory drugs, corticosteroids, muscle relaxants, and opioid pain medications. Nonsteroidal anti-inflammatory drugs have been shown to be helpful for the management of acute low-back pain[29], but a meta-analysis of the literature demonstrated that they had no benefit in the treatment of radiculopathy compared with controls (odds ratio = 0.99)[30]. Corticosteroids are administered orally or by injection. While oral steroids are commonly prescribed in clinical practice, we found only one study on their use for the treatment of lumbosacral radicular pain[31]. In that study, dexamethasone was not superior to a placebo for either early or long-term relief of lumbosacral radicular pain, but it helped patients who had presented with a positive result on the straight-leg-raise test. The use of intramuscular corticosteroid injections for acute sciatica was examined in two randomized controlled trials. One trial showed no benefit (odds ratio = 0.8)[32], and the other trial showed a modest benefit (odds ratio = 2.0)[33]. We are not aware of any randomized clinical trials that tested the effectiveness of opioid analgesics for patients with lumbar disc herniations, although such analgesics are commonly used in clinical practice for the treatment of acute and chronic radiculopathies. Muscle relaxants have been shown to be effective for the treatment of acute low-back pain[34], but we found no data from well-controlled studies of their use for pain associated with lumbar disc herniations. Antiepileptics such as gabapentin and tricyclic antidepressants such as amitriptyline are commonly used to treat the neuropathic pain component associated with lumbar disc herniations. Again, we are not aware of any controlled trials of the use of those medications for patients who have lumbar disc herniation with radiculopathy, but one open, uncontrolled trial of lamotrigine showed a significant improvement in patients with chronic sciatica ($p < 0.05$)[35].

### *Physical Medicine*

When a patient has incapacitating pain, a period of bed rest is often unavoidable. Immobilization presumably diminishes inflammation around an irritated nerve root. However, there are no data to suggest that bed rest alters the natural history of lumbar disc herniations or improves outcomes. Because of the potentially harmful effects of prolonged bed rest, it is best to advise patients to limit bed rest to a short term only and to resume activities as soon as possible[36]. Bracing is another method of immobilizing the lumbar spine, but there is a lack of good evidence to support the use of braces and corsets for patients with lumbar disc herniations. These devices have not been shown to be effective for primary or secondary prevention of low-back pain; however, the Cochrane review found "limited" evidence favoring lumbar supports compared with no treatment[37].

Traction remains of unproven benefit in the treatment of lumbar disc herniations. A meta-analysis of pooled data from four randomized controlled trials showed some benefit of traction therapy compared with a placebo (odds ratio = 1.2)[38]. In one controlled trial, traction with physical therapy resulted in a greater reduction in the sizes of disc herniations than did physical therapy alone[39]. Vertebral axial decompression therapy (VAX-D) was developed according to the principles of traction and is popular among chiropractors. One randomized clinical trial, in which one of the authors was the medical director for a VAX-D manufacturer, demonstrated >50% relief of chronic low-back pain in 68.4% of patients treated with vertebral axial decompression therapy compared with 0% of patients treated with transcutaneous electrical stimulation[40]. However, we are not aware of any studies of vertebral axial decompression therapy for patients with isolated lumbar disc herniation. The Cochrane Review in 2005 concluded that "traction is probably not effective" on the basis of the finding that neither continuous nor intermittent traction was more effective for decreasing pain, disability, or work absence than were placebo, sham, or other treatments for patients with low-back pain, with or without sciatica[41].

Physical therapy in general has not been proven to be beneficial for patients with acute low-back pain, but it may be helpful for those with chronic low-back pain[42]. We are not aware of any randomized trials examining the outcomes of physical therapy alone for the treatment of lumbar radiculopathy. Active exercises are more appropriate than passive modalities, particularly for patients with subacute or chronic pain[27]. Hofstee et al. showed that bed rest and physiotherapy are not more effective for the treatment of acute sciatica than is continuation of activities of daily living[43]. McKenzie therapy is commonly advocated for treatment of lumbar disc herniations. However, in one study on the management of low-back pain, there was no difference among the results of McKenzie therapy, manipulation, and providing the patient with an educational booklet[44]. Another common physical therapy technique involves the spectrum of lumbar stabilization exercises. Although randomized trials have not been performed, to our knowledge, outcomes of nonoperative treatment have been better in studies employing active lumbar stabilization exercises[45] than they were in older controlled trials that employed passive treatment modalities[46].

Acupuncture is another physical medicine modality that might be applied to the treatment of lumbar disc herniations. Although anecdotal stories of success are extant in popular culture, the available literature has not demonstrated the efficacy of acupuncture in treating low-back pain[47]. Manipulation and chiropractic are similarly unproven. Burton et al. compared chemonucleolysis with manipulation in the treatment of symptomatic lumbar disc herniations in a controlled trial[48]. After twelve months, there was no significant difference in overall outcome between the treatments, but manipulation did result in a greater decrease in back pain and disability during the first weeks. At a minimum, manipulation is relatively unlikely to cause harm: it has been estimated that less than one in 3.7 million treatments with spinal manipulation results in clinical worsening of disc herniation[49]. Other modalities that are commonly used in clinical practice include massage therapy, transcutaneous electrical stimulation, and biofeedback. These methods have not been evaluated for the treatment of lumbar disc herniations and radiculopathy in well-controlled trials. Cognitive behavioral therapy has shown efficacy for the treatment of chronic low-back pain[50] , but we are not aware of any studies of its effectiveness for patients with lumbar disc herniation.

***Epidural Steroid Injections***

Epidural steroid injections have been used for decades for the treatment of spinal pain, particularly radiculopathy. In a review of four older randomized trials, epidural steroid injections were found to be more beneficial than the control treatment, especially with respect to short-term outcomes, for the treatment of acute radiculopathy (odds ratio = 2.2)[38]. A more recent study of interlaminar epidural steroid injections demonstrated a transient decrease in sciatic symptoms at three weeks but no sustained benefits in terms of pain relief, function, or avoidance of surgery[51]. Epidural steroid injections also do not appear to change the rate at which lumbar disc herniations regress[52].

Fluoroscopically guided transforaminal injection techniques, which have the theoretical advantage of delivering the injectate to the site of the disc herniation in the anterior epidural space, have been more commonly used in modern studies. Although the traditional, more dorsal interlaminar approaches may allow the injectate to flow to the site of the lesion by seeping around the thecal sac and into the ventral epidural space, a transforaminal route is presumably more reliable for delivering the steroid to the affected area, where the herniated disc comes into contact with the nerve root. One study showed transforaminal injections to be superior to trigger-point injections, with "successful" outcomes following 84% of the former procedures and 48% of the latter[53]. Other studies have suggested that transforaminal epidural steroid injections may actually change the natural history of radiculopathy by decreasing the need for surgery. In one study of fifty-five patients with lumbar radiculopathy who were all considered surgical candidates, 71% of those who received a steroid nerve-root injection and 33% of those who received a control injection of local anesthetic only decided not to have surgery[54]. Another study compared pain scores on a visual analog scale and the need for surgery between patients who had received transforaminal steroid injections and those who had received interlaminar epidural steroid injections[55]. The patients treated with the transforminal injections had a 46% reduction in the pain score, and 10% went on to need surgery. In contrast, the patients treated with the interlaminar injections had a 19% reduction in the pain score, and 25% required surgery. These findings indicated that the short-term outcomes were better following transforaminal injections.

It is impossible to directly compare the literature on outcomes of surgical discectomy with reports on outcomes of epidural injections because of the numerous differences in surgical technique (e.g., open, "minimally invasive," microdiscectomy, and aggressive disc-space curettage); injection technique (e.g., transforaminal or interlaminar, fluoroscopically guided or not); dose, timing, and type of steroid delivered; and patient selection criteria (e.g., in many studies, those with severe pain or progressive neurological deficits were not considered candidates for nonoperative treatment). In a recent study that provided level-I evidence, 100 patients who had had failure of six weeks of noninvasive treatment of a disc herniation measuring ≥25% of the cross-sectional area of the spinal canal were randomized to be treated with interlaminar epidural injections or surgical discectomy[56]. The success rates, which were 92% to 98% in the surgically treated group and 42% to 56% in the group treated with epidural injection, were significantly different. Twenty-seven patients crossed over from the epidural-injection group to the surgical group because of persistent pain, but their outcomes were not adversely affected by the delay in surgery due to the trial of the epidural injection. Whether the transforaminal approach would have led to better outcomes of the epidural injections remains unclear, but, on the basis of this study, surgery appears to be more effective than injections, at least in patients with large disc herniations. Although informative, this finding does not change the commonly accepted indications for surgery, as surgery is associated with not only greater benefit but also with higher risk than epidural injections. Data from the United States National Institutes of Health-funded multicenter randomized trial comparing surgical with nonoperative treatment of lumbar disc herniations, spinal stenosis, and spondylolisthesis (the Spine Patient Outcome Research Trial [SPORT] study) will hopefully provide clearer guidelines when they become available.

*Novel Treatments*

On the basis of the understanding that the mechanisms underlying herniated disc-associated radiculopathy are both biochemical and mechanical, novel treatments have been developed to attenuate biochemical sensitization of the nerve root by factors within the nucleus pulposus. As mentioned above, TNF-α appears to play a role in the pathogenesis of radiculopathy associated with disc herniations. In a very small pilot trial in which ten patients received a single intravenous injection of infliximab (a monoclonal antibody to TNF-α) for treatment of acute sciatica (lasting two to twelve weeks) due to disc herniation, eight patients had no leg pain at twelve months, compared with 43% of a historical control population who had received saline solution nerve-root blocks[57]. However, when a subsequent randomized controlled trial was performed by the same authors on the basis of these promising pilot data, they noted no difference in the reduction of leg pain or the need for surgery at three months between patients who had received a single dose of infliximab and controls[58]. Other authors reported success with medical ozone injections into the disc and around the nerve root[59], although the study lacked a control group treated without ozone. Various percutaneous, intradiscal treatments, such as electrothermal disc decompression, percutaneous disc decompression, and nucleoplasty, have been developed and marketed by manufacturers. However, the efficacy of these methods is yet to be demonstrated in properly controlled trials.

*Overview of Nonsurgical Treatment*

The available literature indicates that effective nonoperative treatments for lumbar disc herniations include observation only as the condition has a favorable natural history, and probably epidural steroid injection, at least for short-term relief. Intramuscular injections of steroids may provide some benefit. Nonsteroidal anti-inflammatory drugs are effective for low-back pain only, and traction is probably not effective. There are insufficient data to provide recommendations regarding the role of oral steroids, physical therapy, transcutaneous electrical stimulation, corsets, and manual therapy. On the horizon are medications to suppress reactive nerve-root inflammation and medications to inhibit cytokine production, which may improve the pharmacological treatment options for lumbar disc herniations.

## Evidence Regarding Surgical Treatment

Despite the facts that more than $90 billion per year is spent on the management of spine conditions and more than seven decades have passed since Mixter and Barr reported on the surgical management of disc herniations, there remains little level-I evidence regarding the effectiveness of surgery for symptomatic lumbar disc herniations. Although many retrospective studies have suggested a benefit, these studies have the common weakness of inadequate design. The lack of level-I data leads to widespread uncertainty with regard to the selection of patients for surgery, as reflected in the varied rates of disc excision surgery: there was a nearly twentyfold difference between high and low surgery-rate regions in an otherwise controlled population analysis in the United States[60,61]. However, these statements should not be misconstrued as a condemnation of discectomy surgery. Much to the contrary, under the right circumstances, surgery clearly "works" very well: any surgeon who has seen a patient suffer for months prior to surgery and then wake up from a discectomy with immediate relief of leg pain, numbness, and weakness can attest to that fact. Instead, the question that remains unanswered by the available literature is that, given that not every patient has an excellent result from surgery, surgery has the potential for complications, and the natural history of lumbar disc herniation tends to be favorable in the majority of patients, when and in whom should surgery be recommended?

*Neurological Variables*

Although one might expect surgery to be superior to nonoperative care of patients with stable neurological deficits, this has not been supported by the available literature. Hakelius[23], Weber[46], the Maine Lumbar Spine Study[60], and Saal[27,45] demonstrated that stable radicular weakness resolves equally well regardless of treatment. In a more recent pilot study of sixty patients with stable paresis associated with lumbar disc herniation, Dubourg et al.[62] also found no difference between neurological recovery following surgical management and that following medical management. This finding is in contrast to the situation for a patient with a progressive

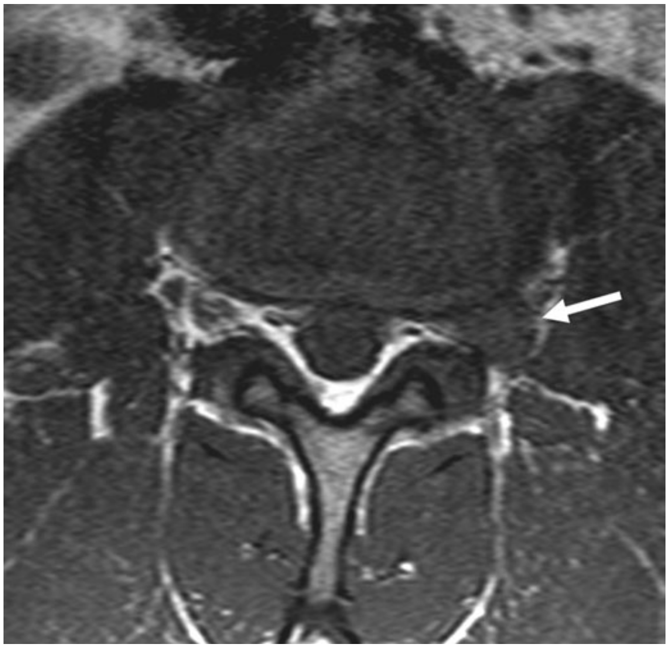

Fig. 2-A

Fig. 2-B

Axial T1-weighted (Fig. 2-A) and T2-weighted (Fig. 2-B) magnetic resonance imaging scans of a patient with an extraforaminal disc herniation (arrows). This type of herniation is more readily removed by an extraforaminal approach rather than from inside the spinal canal. Extraforaminal herniations are often better seen on T1-weighted images and are more likely to be missed on T2-weighted images. Posterolateral disc herniations, in contrast, are generally well demonstrated on T2-weighted images (see Figs. 1-A and 1-B).

neurological deficit and cauda equina syndrome, for whom, the evidence suggests, urgent decompression provides the best functional improvement[63].

*Surgical Volume*

Increased surgical volume has been correlated with improved outcomes of several operations, including joint arthroplasty, cardiac surgery, and cancer surgery, suggesting that a surgeon's experience and skill as well as the hospital's overall experience play important roles in outcomes. Paradoxically, the same may not be true for lumbar discectomy. The Maine Lumbar Spine Study showed that patients who had surgery in the highest utilization regions actually had worse outcomes than those treated in the lowest utilization area[64]. The authors concluded that these paradoxically inferior results may be related to the application of more stringent criteria for surgery in regions with lower surgical rates and expanded indications beyond what might be considered the standard in the higher-rate regions. Thus, patient selection and patient factors may have a greater influence than surgical technique on the results of spine surgery, for which the indications are not as clear-cut as they are for the types of surgery listed above.

Fig. 2-C

The sagittal T1-weighted image of a different patient demonstrates that the exiting nerve root is pinned cephalad against the undersurface of the pedicle by the foraminal portion of the disc herniation.

*Anatomic Features of the Herniation*

Although the size of a disc herniation correlates poorly with pain, there is evidence to suggest that other anatomic characteristics of the herniated disc may be predictive of clinical outcome following discectomy. In one study[65], patients who had extruded disc fragments with a largely intact anulus and those who had extruded fragments contained within an intact anulus had the best postoperative outcome scores and the lowest reherniation rates. The scores were poorer for those who had extruded disc fragments with a massive anular defect as well as for those who had no identifiable fragment within an intact anulus. Those with a massive anular defect had the highest rate of reherniation, whereas those without identifiable fragments had a high rate of persistent symptoms postoperatively despite the absence of clear structural abnormalities to account for them. This last group of patients had a clinical profile (e.g., with regard to compensation status and psychometric abnormalities) that was similar to that of patients with chronic low-back pain: both had pain behavior that was out of proportion to the anatomic pathological findings.

Contrary to popular opinion, the size of a disc herniation does not appear to correlate with the need for eventual surgical intervention. Natural history studies[27,66-68] have shown that the largest disc herniations actually demonstrate the greatest degree of resorption, whereas contained herniations demonstrate the least. Thus, the disc abnormality that seems to be best suited for surgery—a large extruded fragment—also has the greatest likelihood of natural regression; this means that size cannot be used reliably as a criterion for surgery.

*Variations in Surgical Technique*

Although various surgical techniques have been used to decompress symptomatic nerves, the data suggest that the choice of surgical technique is less critical to a good outcome than proponents of various techniques have suggested[69]. To date, there has been no proven difference in outcomes regardless of whether the root is decompressed by means of a traditional ("large incision") laminotomy-discectomy, an endoscopic discectomy, or a microdiscectomy (Figs. 2-A, 2-B, and 2-C).

The optimal amount of disc that should be removed during surgery is not clear. Although it has been established that the primary goal of surgery is neural decompression, competing considerations remain: too little removal seemingly raises the specter of increased recurrence, whereas too much removal raises concerns about accelerated disc degeneration and increased back pain. The available evidence, based largely on case reports and retrospective studies, suggests no benefit to more aggressive disc removal in terms of recurrence; to the contrary, it may be deleterious with respect to the later development of back pain[70-73]. Lumbar disc herniations recur at about equal rates (approximately 5%) regardless of treatment, and neither surgical intervention nor medical management can prevent reherniation.

*Patient Factors Affecting Surgical Outcome*

Although, traditionally, spinal research has focused on physician-determined outcomes—such as a physician's assessment of relief of symptoms (e.g., according to Odom's criteria)[74], radiographic evidence of a successful fusion, and the magnitude of deformity correction—it has become increasingly evident that the most useful measure of a given operation's success is whether the patient perceives it to be successful, regardless of what the physician-determined outcomes may demonstrate. The patient's perception of successful treatment, in turn, appears to be influenced at least as much by psychosocial and other patient factors as it is by the specific type of disc lesion or the design and execution of a proper operation by the treating surgeon. Patient-reported health surveys, which allow self-evaluation of function, are useful tools for assessing these patient factors preoperatively and postoperatively[75-77]. The health surveys include both condition-specific surveys, such as the Roland-Morris Disability Questionnaire and the Oswestry Disability Index (ODI), and the commonly used general health survey, the Short Form-36 (SF-36)[78-80]. Although the outcomes measured by the SF-36 are not specific to the spine, it is useful for measuring the outcomes of spine surgery because spinal disorders have been shown to impart a substantial negative effect on self-reported physical function. As demonstrated in one study by the Physical Component Score (PCS) of the SF-36, spinal stenosis had a greater negative effect on physical function than all other medical conditions studied with use of the SF-36[81], including cancer, chronic obstructive pulmonary disease, and congestive heart failure.

Studies of self-reported health surveys have identified a number of patient factors with significant effects on patient-determined outcomes after spine surgery. In one such study, smoking had a significant negative effect ($p < 0.05$) on self-reported function at baseline as measured by all eight subscales of the SF-36[82]. One year after spinal surgery, smokers did not have significant improvement in the scores on any subscale of the SF-36, whereas their nonsmoking counterparts had significantly improved scores on six subscales ($p < 0.05$). In another study, a low education level was an independent predictor of poor self-reported function at baseline as measured with both the condition-specific ODI and the SF-36 general health survey[83]. It was also noted that the major drivers of physical function as measured by the SF-36 and ODI were psychosocial variables rather than traditional medical conditions. In addition to low education level, these other variables included poor self-reported health, work and disability status, legal status, body mass index, and smoking. In a related study, self-reported health was also found to be an independent predictor of functional outcome following surgery[84]. Of 1833 patients who had undergone surgical intervention for lumbar disc herniation, those reporting "good" health and "poor" health both had improvement following surgical intervention. However, there was a significant difference ($p < 0.05$) between the groups with respect to scores on the SF-36 bodily pain (BP) and physical function (PF) components as well as the ODI scores, with those reporting good health faring better. Other studies have also demonstrated a negative effect of patient factors such as depression, frequent headaches, compensation status, low education level, and unemployment on both the ODI and the PCS of the SF-36[85].

Taken together, these studies demonstrate that proper selection of patients for lumbar discectomy should include a thorough assessment of patient factors as such factors have important effects on function and on the response to treatment independent of the specifics regarding the disc lesion. Identifying these factors can assist providers and patients in decision-making as well as guide reasonable expectations from surgery. In certain patient populations, the effect of low-back problems may be a greater reflection of psychosocial distress than anatomic dysfunction, which may explain why the traditional surgical model of treating spinal problems fails in many patients. If patients and surgeons are not aware of this association between patient factors and functional outcome, both may be disappointed with the results.

### *Medical Comorbidities Affecting Surgical Outcome*

The presence of comorbidities also has a significant effect on surgical outcomes. In one study, the presence of four comorbidities was noted to significantly ($p < 0.05$) and independently lower the improvement in the ODI score by almost 5 points and the improvement in the SF-36 score by more than 4 points at one year after lumbar spine surgery[86]. In another study, of 15,974 patients, obesity had a negative influence on self-reported function, as measured by both the SF-36 and the Oswestry Disability Index, and obese patients reported a greater degree of pain than nonobese patients[87].

### *Surgical Compared with Nonsurgical Treatment*

There is a dearth of level-I evidence comparing surgical with nonsurgical management of lumbar disc herniations. In 1983, Weber's classic work, "Lumbar Disc Herniation. A Controlled, Prospective Study with Ten Years of Observation"[46], included a randomized trial (the first randomized trial in spinal surgery) in which sixty patients had surgery and sixty-six continued to be treated with conservative measures. Weber found that those treated with surgery had a significantly better result at one year postoperatively ($p < 0.05$). At four years postoperatively, the surgically treated patients had a trend toward better results, but that difference was not present at ten years. The surgically treated patients had far fewer relapses than the nonoperatively treated group in the first four years. Motor weakness improved equally in both groups, as did sensory dysfunction. Thirty-five percent of the patients, equally distributed in the two groups, had demonstrable sensory dysfunction ten years after the hospitalization for the herniated lumbar intervertebral disc. Although it was a breakthrough study, it did have flaws. Not all of the patients were randomized: sixty-seven additional patients had "symptoms and signs that beyond doubt required surgical therapy" and eighty-seven others were treated "conservatively as there was no indication for operative intervention." Furthermore, a large number of nonoperatively treated patients crossed over into the surgical group, the study lacked adequate statistical power, the outcome assessment was not blinded, and the outcome measurement was relatively insensitive[88].

More recently, the Maine Lumbar Spine Study, an observational (nonrandomized) study of 507 patients (with follow-up data available for 400 of them), compared the ten-year results of operative and nonoperative treatment[60]. As would be expected with an observational study, the surgically treated patients had had worse baseline symptoms and functional status than the nonoperatively treated patients. Despite that fact, over the ten-year period, the proportion of patients who reported that their low-back pain and leg pain were greatly decreased or completely gone was larger in the surgically treated group than in the nonoperatively treated group (56% compared with 40%, $p = 0.006$), and more surgically treated patients than nonoperatively treated patients were satisfied with their current status (71% compared with 56%, $p = 0.002$). The greatest improvement in the surgically treated group occurred in the first two years after the operation. There was smaller but continued improvement in both groups through the ten-year period.

### *Overview of Surgery for Lumbar Disc Herniations*

Regardless of treatment, lumbar disc herniations usually have a favorable natural history with improvement over time, but it may take one to two years for functional improvement to plateau. In the absence of a cauda equina syndrome or progressive weakness, the best indication for surgical management is refractory radicular pain. Surgical decision-making should not be based on the size of the disc herniation, as large extruded herniations tend to resolve more predictably, or on either stable motor weakness or numbness, as the ultimate resolution of weakness and sensory deficits is similar following either nonoperative or surgical management, although surgery hastens the process. When intractable radicular pain is the strict indication for surgery, surgical intervention provides substantial and more rapid pain relief than does nonoperative treatment. The specific method of surgical intervention probably contributes little to the overall success of the intervention as long as the root is properly decompressed.

The treatment should be chosen by the patient—after proper education through a process of shared decision-making—rather than reflect the "surgical signature" of the surgeon. Health surveys can provide additional assessment of psychosocial comorbidities that are not otherwise evident during the usual clinical evaluation. Such comorbidities should be identified preoperatively as they are not likely to resolve with surgical intervention but may have greater impact than the discal pathoanatomy on the ultimate outcome.

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