

Discogenic Back Pain

Introduction

Although widely recognized, discogenic back pain remains a disease that at times is difficult to unequivocally diagnose. The multiplicity of etiologies responsible for low back pain and the mixed bag of symptoms associated with it are two of the main reasons why the exact diagnosis of this disease entity remains difficult. The accurate workup of a patient with discogenic back pain requires a logical stepwise progression through the diagnostic process. This chapter discussed the pathophysiology, diagnostic algorithm and treatment options for patients with presumed lumbar discogenic pain.

Incidence

Low back pain is an essentially ubiquitous phenomenon. The annual incidence of back pain is projected to be 5% per year, with an associated prevalence of 60% to 90%. Only medical visits to primary care physicians for upper respiratory infections outnumber those for back pain. Back pain has been reported in more than 50% of people engaged in light physical activity and in more than 60% of those performing heavy labor; it is the most common reason for limited activity in individuals younger than 45 years. Back pain is the third most common cause for disability in patients 45 to 65 years of age. The potential for return to work following disabling back pain correlates directly with the length of time away from work; patients who are away from work more than 6 months have a 50% rate of returning to work, those away more than 1 year have a 25% rate, and those away more than 2 years have less than a % chance of returning to work. Despite the aforementioned statistics, only 7% of patients have low back pain that persists more than 2 weeks, and only 1% require long-term treatment. Less than 1% of patients with low back pain ultimately require surgery. Surprisingly, approximately 85% of patients who report back pain cannot be given a pathoanatomic diagnosis. This common medical condition results in an estimated medical cost of \$30 to \$50 billion annually in the United States.

Etiologies

Obesity is a known risk factor for numerous diseases, including degenerative disc disease (DDD). The mechanical load on the disc space increases in obese patients and may lead to premature degeneration through several progressive pathophysiologic processes. Smoking also has been associated with an increased incidence of both DDD and disc herniation. Nicotine has been shown to limit oxygenation to tissues leading to accelerated degeneration by inhibiting the daily restorative properties at the cellular level within the intervertebral disc. This process may cause hyalinization and necrosis of the nucleus pulposus. Nicotine inhibits osteoblastic function, bone metabolism and cellular exchange within the disc space, leading to disc degeneration. One study showed a 32% increased rate of pseudoarthrosis in smokers who underwent a two-level lumbar spinal fusion. The overall success rate for surgical and nonsurgical treatments of low back pain has been shown to be significantly lower in patients who smoke.

Genetics has always been considered an important factor in DDD. MRI investigations have found a 74% heritability of DDD in twins. Molecular biology studies have attempted to identify some of the genetic loci for DDD and have found that genes encoding polypeptide chains for collagen IX, which is produced in the disc space, may play an important role in the development of DDD.

Other studies have attempted to determine the role of heavy labor or weight-bearing exercise (such as body-building or weight lifting) as possible risk factors for DDD. One study addressed the question of whether long-term compressive loading on the intervertebral disc predisposed an individual to disc degeneration; no increased incidence of DDD was found. However, alterations in the intradiscal distribution of proteoglycan and collagen were identified. A recent 5-year prospective study for the assessment of DDD in 41 asymptomatic patients that examined physical job characteristics, participation in sports activities and various morphologic changes from baseline MRI found that nonparticipation in

sports activities, night-shift work and a previously injured disc on initial MRI were significant risk factors. Despite the logical conclusion that athletes who use their lumbar spines to perform weight-bearing exercises over a long period will increase their risk for DDD, studies have determined that exercise alone is not a factor in DDD and may be protective in preventing DDD.

The incidence of DDD in athletes is likely to be increased not from the mechanical loading associated with weight lifting (which in most instances is a static exercise in which the joints are loaded in a fixed physiologic manner, limiting the risk for injury), but rather the athlete is at increased risk during sports competitions when the spine is loaded in a nonphysiologic manner and in a mechanically more vulnerable position. A 2004 study analyzed a large cohort of identical twins and determined that multiple social factors and work experience had little influence on the development of DDD. It was concluded that genetics played the most important role, accounting for up to 74% of the variance in the adult population.

Anatomy and Physiology

The discs contribute approximately 30% of the height to the lumbar spine. The disc consists of cartilaginous endplates, the annulus fibrosus and nucleus pulposus. Hyaline cartilage connects the endplates to the vertebral body. The lamina cribrosa, a calcified layer within the cartilaginous endplate, has a porous surface that permits diffusion of nutrients and plays an important role in the pathophysiologic cascade of DDD.

The annulus fibrosus has collagen fibrils in concentric laminae. Each lamina has parallel fibrils obliquely oriented to the endplates; the direction of the fibrils alternate between laminae, analogous to the belts of a radial tire. Sharpey's fibers, which are predominantly type I collagen, are the outer most layer of fibrils attaching directly to the epiphyses of the vertebral body. The inner layer of fibrils is type II collagen, which are also attached to the cartilaginous endplates.

The nucleus makes up approximately 40% of the intervertebral disc volume and is located in the posterior central portion of the disc. The nucleus is more gelatinous than the annulus fibrosus and thus permits the nucleus to perform as a hydrostatic load-bearing structure, contained by the less elastic annulus fibrosus.

The intervertebral disc is made up of collagen, proteoglycans, connective tissue and water. The molecular structure of collagen leads to its enormous tensile strength, which provides the secure attachment to the vertebral bodies and assists in the ability to withstand daily shearing forces.

Pathophysiology

Disc degeneration begins in early childhood as vessels within the disc space slowly regress. The etiology of this vascular regression is thought to be secondary to the change from quadrupedal to bipedal locomotion in childhood, thus loading the disc in the upright posture, increasing intradiscal pressure, and causing involution of the blood supply. By 4 years of age, chondrocytes are dependent on diffusion alone for metabolic supply. The porous surface in the lamina cribrosa decreases with age. With increasing age the metabolic strain to the intervertebral disc increases because of decreasing blood supply and decreasing diffusion. These metabolic changes lead to alterations in overall charge in the disc space, decreasing the net inward flow of fluid and decreasing water content from 90% to 70%; this change ultimately leads to loss of disc height and expandability. Once the degenerative cascade begins, spinal mechanics are altered, and abnormal loading on adjacent levels may result in ligamentous strain causing pain in liked structures (facet joints, sacroiliac joints and adjacent disc levels).

An MRI study examined 50 patients with chronic low back pain under axial loading and found that 20% of patients showed no changes, whereas 80% had accentuation of their lumbar disease (stenosis, disc protrusion or listhesis). The authors concluded that axial loading may not cause the initial disease, but a

prior injury may lead to a degenerative cascade allowing axial loading that negatively affects the intervertebral disc.

The DDD process also has an inflammatory component that can often be seen on the anterior disc space on MRI scans or intraoperatively during anterior lumbar spine surgical exposures. This inflammatory component may explain why intradiscal corticosteroid injections and/or oral anti-inflammatory medications have occasionally been reported as beneficial in some patients. A comparative study on inflammatory mediators in disc tissue that were removed from patients undergoing routine discectomy for a herniated disc compared with disc tissue from patients undergoing total discectomy and fusion for discogenic pain, found significantly higher levels of proinflammatory mediators (interleukin-6 and interleukin-8) in the group with discogenic pain. These findings suggest an inflammatory role within the nucleus pulposus as one of the possible components of the degenerative cascade.

Clinical Presentation

The patient history and physical examination are extremely important in determining the etiology of the patient's back pain. Several historic and physical examination findings can help direct the physician to the appropriate diagnostic test or treatment option. The patient with DDD typically presents with daily pain that is worse with weight bearing (standing or sitting) for prolonged periods. Patients may occasionally report subjective radicular irritability, but it is not the primary reported condition. Back pain is generally daily and persistent, whereas radicular symptoms are usually transient.

Physical Examination

Results of the physical examination may be nearly normal; therefore, a diagnosis by exclusion will be required. Patients will typically have a normal neurologic examination with intact motor, reflex and sensory findings. Patients generally deny radiculopathy, and although the gait may be guarded or antalgic, it is generally normal. A small percentage of patients may have transient radicular symptoms if their DDD has caused a loss of disc height and subsequent foraminal stenosis with weight bearing. A common physical finding is mild transverse low back tenderness that may not be associated with true paraspinous muscle spasm. The most typical subjective symptoms is a deep aching pain that is relieved with bed rest and aggravated by weight bearing. The classic question to ask the patient is: "Can you sit through a movie without fidgeting?" It is a consistent finding that patients with true discogenic pain cannot sit with comfort for even short periods. Examination findings regarding straight-leg raising and other radicular test are normal. The range of motion in the lumbar spine may be limited in flexion and extension because of aggravation caused by loading of the intervertebral discs.

The surgeon should always question the patient about common red flags such as pain at night, unexplained systemic symptoms, fever and weight loss. The patient should have a complete history and physical examination because back pain may be associated with other disease processes.

Psychological evaluation is required for patients with any history of mental illness, including depression, or in those who have a long history of narcotic use.

Diagnostic Evaluation

Plain radiography is the best initial study. The normal degenerative processes in the foramen and intervertebral space such as disc space narrowing with osteophyte formation or sclerosis at the endplates should be evaluated. With severe narrowing, there may be the characteristic "vacuum phenomenon" or gas-formation within the disc space. Other possible sources for back pain should be excluded by

analyzing the plain radiograph for any suspicious areas, absent pedicles, compression fractures and other possible degenerative, oncologic or infectious changes.

MRI has become one of the most commonly used tools in the diagnostic workup of a patient with back pain. Sagittal T2-weighted MRI yields the best initial view of the desiccated disc space. In the typical degenerated disc segment, the T2-weighted MRI scan will show decreased signal intensity (dark) within the affected disc space and increased signal intensity in the normally hydrated disc spaces. The appearance of degeneration of the vertebral bodies adjacent to the affected disc level on MRI scans has been described by Modic as three different signal intensity patterns. A type I pattern is defined as decreased signal intensity on T1-weighted MRI scans and increased signal on T2-weighted MRI scans. Type II changes have increased signal intensity on T1-weighted MRI scans and are isointense on T2-weighted MRI scans. Type II changes have been examined pathologically to reveal endplate disruption with increased lipid content in the marrow, which are believed to represent an inflammatory response to the painful disc. Type III changes have decreased signal intensity on T1- and T2-weighted MRI scans, which corresponds pathologically to significant loss of marrow and endplate sclerosis. The clinical significance of these findings of DDD on T2-weighted MRI scans and Modic changes at the endplate is yet to be elucidated. One study found no significant correlation between an MRI scan that is positive for vertebral endplate changes and concordant pain with provocative discography. The results of additional biomechanical studies have led to the conclusion that there is no direct correlation between degenerative changes to the disc and the adjacent vertebral bodies. Thus, Modic changes may demonstrate degenerative changes within the body and/or disc but have failed to clinically correlate with findings on provocative discography.

Discography is strictly a confirmatory examination and not a stand-alone test. The test itself is a provocative examination requiring a cooperative effort from the radiologist and the patient. The test is beneficial to confirm the definitive DDD level, but should always include at least one normal level. The rationale for including the normal level is to exclude the adjacent levels as a cause for pain and to further confirm that the disc with abnormal internal architecture is the concordantly painful disc.

Nonsurgical Treatment

Most patients with low back pain will recover without surgery. All patients (excluding those with a neurologic emergency) should be managed initially with nonsurgical treatment options that are supported by the literature. Exercise programs are strongly encouraged for a variety of reasons. Strengthening the abdominal wall muscle groups and lumbar musculature (core muscle groups) has demonstrated a definitive benefit in relieving low back pain of discogenic origin. The physiologic basis of strengthening the muscles to unload the joints is well-supported in the literature. Patients who improve their strength through a trial of physical therapy but still require surgery tend to better tolerate surgery and recover faster. Several studies have analyzed the use of bracing for patients with discogenic pain and found limited benefit, with some studies showing aggravation of the pain. Bracing has typically been used for the diagnosis and treatment of mechanical back pain and has limited use in treating discogenic pain. Special orthosis that unweight the lumbar spine by pneumatic pistons may have diagnostic as well as therapeutic use.

Chiropractic manipulation has been shown to be more beneficial in the treatment of acute low back pain than placebo and has been recommended for acute treatment of low back pain in the neurologically intact patient. Data suggest, however, that chiropractic treatment of chronic back pain (pain lasting > 12 weeks) is of no greater benefit than the use of a placebo.

Traction is often used in the cervical spine for temporary relief of DDD and mild cervical disc herniations. It is more difficult to use traction in the lumbar spine, and the literature supporting its use for patients with discogenic pain is limited.

Several pharmacologic studies have demonstrated the benefits of nonsteroidal anti-inflammatory medications in the treatment of discogenic back pain, this adding support to the hypothesis of an inflammatory factor in the disease process. Each patient undergoing treatment of acute or chronic low back pain should be initially prescribed a trial of nonsteroidal anti-inflammatory medications along with other nonsurgical treatments such as physical therapy or chiropractic manipulation that have evidence-based benefits.

Surgical Treatment

In the past decade, the most common surgical treatment for discogenic lower back pain has been disc excision with interbody fusion performed either anteriorly, posteriorly or circumferentially, depending on the surgeon's experience and the clinical findings. The main goal in the treatment of discogenic pain is removal of the pain source (for example, discectomy and fusion of the diseased segment, thus prohibiting motion). The success rate of posterolateral fusion surgery for relieving discogenic pain without removal of the disc has been fairly poor. Several studies on posterolateral fusion for DDD have shown high fusion rates (upward of 90%), but with only a 60% clinically beneficial result. Posterolateral fusions have a definite role in the elimination of mechanical back pain because of their ability to reduce motion; however, laboratory investigations have demonstrated that posterolateral fusions can reduce motion within a spinal segment by 40%, whereas anterior lumbar interbody fusions reduce motion in the segment by 80% and eliminate all motion within the disc space. One study reported on five patients who had persistent discogenic pain after posterolateral fusions and subsequently had positive discography leading to an anterior interbody fusion, which provided pain relief in all patients. An essential factor in the treatment of discogenic pain is the complete removal of the pain generator. Anterior and posterior interbody fusions have reports of fusion rates of more than 90%, but posterolateral interbody fusions show a lower success rate in discogenic pain relief (ranging from 60% to 90%). Anterior interbody fusions have been found to be consistently superior for pain relief.

An additional negative factor associated with the posterior approach to the lumbar spine is violation of the posterior paraspinal musculature. Muscle damage by dissection and retraction can be a significant source for early post operative pain. If significant muscle ischemia occurs during surgery, muscles may scar post operatively and lead to post fusion syndrome. In addition to the muscles, the posterior approach exposes the dura and neural elements to possible damage and risks for post operative perineural fibrosis.

The anterior approach for interbody fusion avoids the risk of posterior paraspinal muscle damage, reduces risks for direct injury or scarring to the neural elements and permits a total discectomy with restoration of disc space height. Achieving a total discectomy and implanting an appropriate interbody device allows restoration of the disc space height and indirectly increases the volume of neural foramen. When placing interbody devices for fusion, the anterior approach allows for higher fusion rates by obeying the essential rules of successful fusion: allowing a better graft-bone contact area than can be achieved using the posterior approach; easier compression of the graft-bone interfaces, which are more evenly distributed; and obtaining segmental stabilization, which can be achieved by numerous routes, either through posterior percutaneous pedicle screws, an anterior plate, or in certain instances, by stand-alone anterior lumbar interbody fusion with the addition of recombinant human bone morphogenic protein (rhBMP).

Traditional stand-alone anterior lumbar interbody fusions with autogenous iliac crest grafts have been reported to have a high rate of pseudoarthrosis, graft extrusions and subsidence. Subsidence is governed by the cross-sectional area of contact and the relationship of the strut to the margin of the endplate. Three essential factors for the prevention of subsidence should be addressed intraoperatively. (1) It is necessary

to appropriately size the bone graft to the vertebral body. A good fit will permit Wolff's law (bone placed under appropriate stress is remodeled, whereas bone not under stress is resorbed) to operate. (2) The contact surface area between the graft and the vertebral body should be maximized. The extent of subsidence is inversely proportional to the surface area of contact between the graft and the vertebral body. (3) The character and quality of contact surfaces should be optimized. The cartilaginous endplate must be removed to expose the cortical bone surface and allow access to bleeding subchondral bone. Increased fusion rates without increased subsidence have been shown by burring off the endplate to allow the cortical vertebral body to fuse. With the introduction of rhBMP, fusion rates have increased substantially. A comparative study analyzing rhBMP with allograft versus iliac crest autograft found similar clinical outcomes: however, the rhBMP group had significantly higher fusion rates (95% versus 89%). In addition, surgical time and blood loss were both lower in the rhBMP group.

Circumferential surgery has gained acceptance as a means of addressing graft subsidence noted in stand-alone anterior lumbar interbody fusion procedures. Circumferential spinal fusions have resulted in fusion rates of 90% to 100%, with more than 80% of patients having clinical improvement. Patient selection and the need for circumferential fusions should be clearly identified before surgery. In a recent Swedish study that examined three surgical techniques for patients with chronic low back pain and analyzed both fusion rates and long-term outcome scales, patients were grouped into those treated with posterolateral fusion alone, posterolateral fusion with pedicle screws and posterolateral fusion with pedicle screws and interbody graft. The fusion rates were highest in the circumferential fusion group (91%), followed by the group with posterolateral fusion with pedicle screws (87%). The lowest fusion rates were in the group treated with posterior lateral fusion alone (72%). In terms of surgical time, post operative hospital days, blood loss and complications, all were highest in the circumferential group and lowest in the onlay fusion group without instrumentation. The patient outcomes did not differ among the three groups, leading the authors to conclude that, in this study population, there was no disadvantage to using the least surgically demanding and lowest cost procedure (uninstrumented onlay posterolateral fusion) despite the low fusion rate.

Circumferential surgery can and should be used in appropriate clinical situations for either an additional posterior tension band or to prevent graft subsidence in a high-risk patient. When circumferential surgery is necessary, the anterior lumbar interbody fusion can be performed first, followed by a posterior Wiltse muscle-splitting approach with placement of percutaneous pedicle screws unilaterally or bilaterally. The tissue-sparing retroperitoneal anterior approach to the lumbar spine and the minimally invasive percutaneous pedicle screw insertion posterolaterally allow mobilization of the patient on the afternoon of surgery, and result in relatively short hospital stays of 1 to 3 days.

Artificial disc replacement is now approved for use in the treatment of discogenic pain. Arthroplasty may become more commonly recommended than fusion for the surgical treatment of discogenic pain for several reasons. Arthroplasty involves less intraoperative blood loss, shorter surgical times and shorter hospitalization than fusion surgery. Patients who receive disc replacement are rehabilitated faster and return to work sooner than patients treated with fusion. It is believed that motion preservation may decrease the incidence of adjacent level disease (transition syndrome) that occurs with fusion surgery. Large prospectively randomized cohorts of patients have been identified for the US Food and Drug Administration Investigational Device Exemption studies of several lumbar artificial discs. These cohorts will be followed over time to more scientifically evaluate the long-term outcomes of arthroplasty versus fusion.

Summary

Discogenic back pain has a multi-factorial pathogenesis, with substantial published research showing a degenerative cascade occurring after an initial insult, probably beginning at the molecular level. Discogenic back pain, often referred to as internal disc derangement, DDD, mechanical back pain or

segmental instability all require the appropriate diagnostic algorithm and eventual treatment based on that algorithm. The importance of following the diagnostic algorithm to identify the patients with true discogenic pain as opposed to an internal pain generator cannot be overemphasized. With appropriate patient selection, the treatment of discogenic back pain can be successful.

Annotated Bibliography

General

Saal JS: General principles of diagnostic testing as related to painful lumbar spine disorders: A critical appraisal of current diagnostic techniques. *Spine* 2002;27:2538-2545.

The available information and data on invasive diagnostic tests to evaluate chronic low back pain were reviewed. Inherent limitations in the accuracy of the diagnostic tests were found.

Incidence

Devereaux MW: Neck and low back pain. *Med Clin North Am* 2003;87:643-662.

This article discusses cases that represent a sample of conditions that affect the spine and paraspinous structures.

Frymoyer JW, Wiesel SW: *The Adult and Pediatric Spine*, ed3, Philadelphia, PA, Lippincott Williams & Wilkins 2004, PP 899-905.

A discussion of low back pain and other conditions that affect the spine is presented.

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.

The efficacy of intradiscal electrothermal therapy is compared with that of placebo treatment.

Pawl RP: Pain treatment and spine surgery. *Surg Neurol* 2004;61:320-322.

This article discussed methods of pain treatment during spine surgery.

Etiologies

Ala-kokko L: Genetic risk factors for lumbar disc disease. *Ann Med* 2002;34:42-47.

The hypothesis that genetic factors play a role in lumbar disc disease is strengthened by the identification of two collagen IX alleles associated with sciatica and lumbar disc herniation.

Battie MC, Videman T, Parent E: Lumbar disc degeneration: Epidemiology and genetic influences. *Spine* 2004;29:2679-2690.

A review of the scientific literature discussing the prevalence of lumbar disc degeneration and genetic influences is presented.

Chung SA, Khan SN, Diwan AD: The molecular basis of intervertebral disc degeneration. *Orthop Clin North Am* 2003;34:209-219.

Molecular evidence involved in intervertebral disc degeneration is still being investigated. The involvement of cytokines and other inflammatory mediators in this process is controversial.

Elfering A, Semmer N, Birkhofer D, Zanetti M, Hodler J, Boos N: Risk factors for lumbar disc degeneration: A 5-year prospective MRI study in asymptomatic individuals. *Spine* 2002;27:125-134.

Risk factors involved in the development of lumbar disc degeneration are studied.

Hartvigsen J, Christensen K, Frederiksen H, Pedersen HC: Genetic and environmental contributions to back pain in old age: A study 2108 Danish twins aged 70 and older. *Spine* 2004;29:897-901.

A discussion of the relative contribution of genetic and environmental factors to back pain in old age is presented.

Iwashashi M, Matsuzaki H, Tokuhashi Y, et al: Mechanism of intervertebral disc degeneration caused by nicotine in rabbits to explicate intervertebral disc disorders caused by smoking. *Spine* 2002;27:1396-1401.

The effects of nicotine on the vascular beds of rabbits were studied to determine the mechanism of nicotine-induced vertebral disc degeneration.

Manenti G, Liccardo G, Sergiancomi G, et al: Axial loading MRI of the lumbar spine. *In Vivo* 2003;17:413-420.

According to this study, axial loading MRI provides important information for specific nonsurgical treatment of low back pain.

Sobajima S, Kim JS, Gilbertson LG, Kang JD: Gene therapy for degenerative disc disease. *Gene Ther* 2004;11:390-401.

The ability of gene therapy to affect biologic processes in the degenerated intervertebral disc is reviewed.

Anatomy and Physiology

Aoki Y, Takahashi Y, Ohtori S, Moriya H, Takahashi K: Distribution and immunocytochemical characterization of dorsal root ganglion neurons innervating the lumbar intervertebral disc in rats: A review. *Life Sci* 2004;74:2627-2642.

This study suggests that nerve growth factor is involved in the generation of discogenic low back pain.

Burke JG, Watson RW, Conhyea D, et al: Human nucleus pulposus can respond to a pro-inflammatory stimulus. *Spine* 2003;28:2685-2693.

This study set out to confirm that human intervertebral disc is responsive to a pro-inflammatory stimulus and to identify the specific mediators involved.

Butterman GR: The effect of spinal steroid injections for degenerative disc disease. *Spine J* 2004;4:495-505.

Spinal steroid injections are beneficial to some patients with advanced degenerative disc disease and chronic low back pain.

Norcross JP, Lester FE, Weinhold P, Dahnert LE: An in vivo model of degenerative disc disease. *J Orthop Res* 2003;21:183-188.

An animal model of degenerative disc disease using the intervertebral discs in the tails of rats was studied in order to determine possible treatments.

Neural Innervation

Davis TT, Delamarter RD, Sra P, Goldstein TB: The IDET procedure for chronic discogenic low back pain. *Spine* 2004;29:752-756.

The purpose of this study was to assess the functional status, symptoms and treatments of patients treated with intradiscal electrothermal therapy.

Fagan A, Moore R, Vernon R, et al: The innervations of the intervertebral disc: A quantitative analysis. *Spine* 2003;28:2570-2576.

This article presents the first quantitative analysis of the innervations of the lumbar intervertebral disc.

Khot A, Bowditch M, Powell J, et al: The use of intradiscal steroid therapy for lumbar spinal discogenic pain: A randomized controlled trial. *Spine* 2004;29:833-836.

This study determined whether intradiscal steroid injection influenced clinical outcome after 1 year in patients with chronic discogenic low back pain.

Oh WS, Shim JC: A randomized controlled trial of radiofrequency denervation of the ramus communicans nerve for chronic discogenic low back pain. *Clin J Pain* 2004;20(1):55-60.

The efficacy of percutaneous radiofrequency thermocoagulation of the ramus communicans nerve in patients with chronic discogenic low back pain was studied.

Diagnostic Evaluation

Anderson MW: Lumbar discography: An update. *Semin Roentgenol* 2004;39:52-67.

Discography remains the only test that can provide physiologic information about the role an intervertebral disc plays in a patient's symptom complex. Assessment of the patient's pain response is the most important component of the procedure.

Carragee EJ, Alamin TF: Discography: A review. *Spine J* 2001;1:364-372.

Current uses of discography, technique involved and recent studies on its validity are discussed.

Ferguson SJ, Steffen T: Biomechanics of the aging spine. *Eur Spine J* 2003;12(suppl 2):S97-S103.

Advancing age is not the only factor in spine degeneration. Additional study is needed for full understanding of the aging spine's unique biomechanical function.

Willems PC, Jacobs W, Duinkerke ES, De Kleuver M: Lumbar discography: Should we use prophylactic antibiotics?: A study of 435 consecutive discograms and a systematic review of the literature. *J Spinal Disord Tech* 2004;17:243-247.

According to this study, not enough evidence was found that prophylactic antibiotics can prevent discitis.

Nonsurgical Treatment

Borman P, Keskin D, Bodur H: The efficacy of lumbar traction in the management of patients with low back pain. *Rheumatol Int* 2003;23:82-86.

The efficacy of lumbar traction in the treatment of patients with low back pain was studied. No specific effect of traction on standard physical therapy was observed.

Jellema P, van Tulder MW, van Poppel MN, Nachemson AL, Bouter LM: Lumbar supports for prevention and treatment of low back pain: A systematic review within the framework of the Cochrane Back Review Group. *Spine* 2001;26:377-386.

The effects of lumbar supports for the prevention and treatment of low back pain were assessed.

Surgical Treatment

Burkus JK, Gornet MF, Dickman CA, et al: Anterior lumbar interbody fusion using rhBMP-2 with tapered interbody cages. *J Spinal Disord Tech* 2002;15(5):337-349.

Lumbar fusion with rhBMP-2 and tapered interbody fusion cages can lead to solid union and eliminate the need to harvest iliac crest bone graft.

Fritzell P, Hagg O, Wessberg P, et al: Chronic low back pain and fusion: A comparison of three surgical techniques: A prospective multi-center randomized study from the Swedish lumbar spine study group. *Spine* 2002;27:1131-1141.

Three commonly used surgical techniques to achieve lumbar fusion were compared according to their ability to reduce pain and decreased disability in patients with chronic low back pain.

McAfee PC, Fedder IL, Saiedy S, Shucosky EM, Cunningham BW: SB Charité disc replacement: Report of 60 prospective randomized cases in a US center. *J Spinal Disord Tech* 2003;16:424-433.

This study is the first to show improvement of functional outcome measures in a prospective randomized design, with disc arthroplasty treating mechanical back pain and achieving comparable results to lumbar fusion-interbody fusion cage and bone morphogenic protein or interbody autograft and pedicle screw instrumentation.

Park P, Garton HJ, Gala VC, et al: Adjacent segment disease after lumbar or lumbosacral function: Review of the literature. *Spine* 2004;29:1938-1944.

The etiology, incidence and risk factors associated with adjacent segment disease are discussed.

Zigler JE: Lumbar spine arthroplasty using the Prodisc II. *Spine J* 2004;4:260S-267S.

Results from this study suggest that total disc arthroplasty may be an option to lumbar fusion for surgical treatment of disabling mechanical low back pain secondary to lumbar disc disease.

Classic Bibliography

Annunen S, Paassilta P, Lohiniva J, et al: An allele of COL9A2 associated with intervertebral disc disease. *Science* 1999;285:409-412.

Blumenthal SL, Baker J, Dossett A, et al: The role of anterior lumbar fusion for internal disc disruption. *Spine* 1988;13:566-569.

Boden SD, Davis DO, Dina TS, et al: Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects: A prospective investigation. *J Bone Joint Surg Am* 1990;72(3):403-408.

Brown WC, Orme TJ, Richardson HD: The rate of pseudoarthrosis in patients who are smokers and patients who are nonsmokers: A comparison study. *Spine* 1986;11:942-943.

Burdorf A: Exposure assessment of risk factors for disorders of the back in occupational epidemiology. *Scand J Work Environ Health* 1992;18(1):1-9.

Carragee EJ, Paragioudakis SJ, Khurana S: Lumbar high-intensity zone and discography in subjects without low back pain. *Spine* 2000;25:2987-2992.

Carragee EJ, Tanner CM, Khurana S, et al: The rates of false-positive lumbar discography in select patients without low back symptoms. *Spine* 2000;25:1373-1380.

Cloward RB: Posterior lumbar interbody fusion updated. *Clin Orthop Relat Res* 1985;193:16-19.

Cloward RB: Lesions of the intervertebral discs and their treatment by interbody fusion methods. *Clin Orthop Relat Res* 1963;27:51-77.

Cypress BK: Characteristics of physician visits for back symptoms: A national perspective. *Am J Public Health* 1983;73:389-395.

de Vernejoul MC, Bielakoff J, Herve M, et al: Evidence for defective osteoblastic function: A role for alcohol and tobacco consumption in osteoporosis in middle-aged men. *Clin Orthop Relat Res* 1983;179:107-115.

Dennis S, Watkins R, Landaker S, et al: Comparison of disc space heights after anterior lumbar interbody fusion. *Spine* 1989;14(8):876-878.

Deyo RA, Bass JE: Lifestyle and low back pain: The influence of smoking and obesity. *Spine* 1989;14:501-506.

Dickerman RD, Pertusi R, Smith GH: The upper range of lumbar spine bone mineral density? *Int J Sports Med* 2000;21:469-470.

Emery SE, Bolesta MJ, Banks MA, et al: Robinson anterior cervical fusion: Comparison of the standard and modified techniques. *Spine* 1994;19:660-664.

Frymoyer JW, Pope MH, Clements JH, Wilder DG, Macpherson B, Ashikaga T: Risk factors in low back pain: An epidemiological survey. *J Bone Joint Surg Am* 1983;65:213-218.

Glassman SD, Anagnost SC, Parker A, et al: The effect of cigarette smoking and smoking cessation on spinal fusion. *Spine* 2000;25:2608-2615.

Guiot BH, Fessler RG: Molecular biology of degeneration disc disease. *Neurosurgery* 2000;47:1034-1040.

Hanley EN Jr, Shapiro DE: The development of low back pain after excision of lumbar disc. *J Bone Joint Surg Am* 1989;71(5):719-721.

Hollo I, Gergely I, Boross M: Smoking results in calcitonin resistance. *JAMA* 1977;237(23):2470.

Hopper JL, Seeman E: The bone density of female twins discordant for tobacco use. *N Engl J Med* 1994;330:387-392.

Horton WC, Daftari TK: Which disc as visualized by magnetic resonance imaging is actually a source of pain? *Spine* 1992;17:S164-S171.

Hutton WC, Ganey TM, Elmer WA, et al: Does long-term compressive loading on the intervertebral disc cause degeneration? *Spine* 2000;25:2993-3004.

Ito M, Incorvaia KM, Yu SF, Fredrickson BE, Yuan HA, Rosenbaum AE: Predictive signs of discogenic lumbar pain on magnetic resonance imaging with discography correlation. *Spine* 1998;23(11):1252-1258.

Jackson RK, Boston DA, Edge AJ: Lateral mass fusion: A prospective study of a consecutive series with long-term follow-up. *Spine* 1985;10:828-832.