
EVIDENCE-BASED MEDICINE

Evidence-Based Interventional Pain Medicine
according to Clinical Diagnoses

15. Discogenic Low Back Pain

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■ **Abstract:** An estimated 40% of chronic lumbosacral spinal pain is attributed to the discus intervertebralis. Degenerative changes following loss of hydration of the nucleus pulposus lead to circumferential or radial tears within the annulus fibrosus. Annular tears within the outer annulus stimulate the ingrowth of blood vessels and accompanying nociceptors into the outer and occasionally inner annulus. Sensitization of these nociceptors by various inflammatory repair mechanisms may lead to chronic discogenic pain.

The current criterion standard for diagnosing discogenic pain is pressure-controlled provocative discography using strict criteria and at least one negative control level. The strictness of criteria and the adherence to technical detail will allow an acceptable low false positive response rate. The most important determinants are the standardization of pressure stimulus by using a validated pressure

monitoring device and avoiding overly high dynamic pressures by the slow injection rate of 0.05 mL/s. A positive discogram requires the reproduction of the patient's typical pain at an intensity of > 6/10 at a pressure of < 15 psi above opening pressure and at a volume less than 3.0 mL. Perhaps the most important and defensible response is the failure to confirm the discus is symptomatic by not meeting this strict criteria. Various interventional treatment strategies for chronic discogenic low back pain unresponsive to conservative care include reduction of inflammation, ablation of intradiscal nociceptors, lowering intranuclear pressure, removal of herniated nucleus, and radiofrequency ablation of the nociceptors. Unfortunately, most of these strategies do not meet the minimal criteria for a positive treatment advice. In particular, single-needle radiofrequency thermo-coagulation of the discus is not recommended for patients with discogenic pain (2 B-). Interestingly, a little used procedure, radiofrequency ablation of the ramus communicans, does meet the (2 B+) level for endorsement. There is currently insufficient proof to recommend intradiscal electrothermal therapy (2 B±) and intradiscal biacuplasty (0). It is advised that ozone discolysis, nucleoplasty, and targeted disc decompression should only be performed as part of a study protocol. Future studies should include more strict inclusion criteria. ■

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INTRODUCTION

This review on Discogenic Low Back Pain is part of the series “Evidence-based Interventional Pain Medicine according to clinical diagnoses”. Recommendations formulated in this chapter are based on “Grading strength of recommendations and quality of evidence in clinical guidelines” described by Guyatt et al.¹ and adapted by van Kleef et al.² in the editorial accompanying the first article of this series (Table 1). The latest literature update was performed in October 2009.

Each year, many people become disabled as a result of back complaints. Back pain is a multifactorial ailment. In approximately 45% of the cases, low back pain appears to be of discogenic in origin.^{3,4} The sacroiliac joint or the facet joints are indicated as the cause of the pain in 13% and 15% to 40% of the cases, respectively.⁴ Furthermore, in clinical practice, often, more than one cause can be found simultaneously that might be held responsible for the patients’ pain. Discogenic pain shares clinical signs with lumbosacral radicular pain characterized by radiating pain in one or more lumbar or sacral dermatomes with or without neurological deficits. Discus herniation in patients under the age of 50 and spine degeneration in older patients are often associated with chronic low back pain. The development of interventional techniques to treat discogenic

pain has stimulated the refinement of diagnostic procedures with a high specificity and sensitivity, to confirm or refute the hypothesis that the patients’ pain is primarily due to a painful internally disrupted discus.

ANATOMY OF THE DISCUS INTERVERTEBRALIS

The discus intervertebralis is composed of the nucleus pulposus (NP), the annulus fibrosus (AF), and the vertebral end-plates (VE). The corpora vertebrae lie above and below the discus. On the posterior side, the discus is supported by two facet joints. Together, the weight-bearing joints provide support and stability, especially by limiting movement of the spine in all directions.⁵ The healthy discus is avascular, and its nutrition depends on diffusion via the AF and the VE. The nucleus itself has no blood supply.

Nerve Supply

The nerve supply of the discus intervertebralis is complex. The sensory innervation of the discus intervertebralis occurs via branches of the truncus sympathicus.⁶ The dorsal circumference of the discus annulus is innervated via branches of the nervi sinuvertebrales (or recurrentes meningei) (Figure 1), which stem from rami communicantes. The nervus sinuvertebralis runs ventral to the nerve root, back to the canalis spinalis, where the nerve splits into finer branches, which form nerve networks—one in the ligamentum longitudinale

Table 1. Summary of Evidence Scores and Implications for Recommendation

Score	Description	Implication
1 A +	Effectiveness demonstrated in various RCTs of good quality. The benefits clearly outweigh risk and burdens	Positive recommendation
1 B +	One RCT or more RCTs with methodologic weaknesses, demonstrate effectiveness. The benefits clearly outweigh risk and burdens	
2 B +	One or more RCTs with methodologic weaknesses, demonstrate effectiveness. Benefits closely balanced with risk and burdens	
2 B ±	Multiple RCTs, with methodologic weaknesses, yield contradictory results better or worse than the control treatment. Benefits closely balanced with risk and burdens, or uncertainty in the estimates of benefits, risk and burdens.	Considered, preferably study-related
2 C +	Effectiveness only demonstrated in observational studies. Given that there is no conclusive evidence of the effect, benefits closely balanced with risk and burdens	
0	There is no literature or there are case reports available, but these are insufficient to suggest effectiveness and/or safety. These treatments should only be applied in relation to studies.	Only study-related
2 C –	Observational studies indicate no or too short-lived effectiveness. Given that there is no positive clinical effect, risk and burdens outweigh the benefit	Negative recommendation
2 B –	One or more RCTs with methodologic weaknesses, or large observational studies that do not indicate any superiority to the control treatment. Given that there is no positive clinical effect, risk and burdens outweigh the benefit	
2 A –	RCT of a good quality which does not exhibit any clinical effect. Given that there is no positive clinical effect, risk and burdens outweigh the benefit	

RCT, randomized controlled trial.

posterius (LLP) and a network in the ventral dura.⁶ The nerve plexus is characterized by many left-right connections and many cranio-caudal connections. Ultimately, the posterior discus and corpus vertebrae are innervated via this nerve network in the LLP. The same accounts for the ventral dura. The ligamentum longitudinale anterius (LLA) also contains a network of nerves with many left-right and high-low connections of branching nerves. It is formed by branches from the trunci sympathici from both sides. The ventral and lateral sides of the discus intervertebralis are supplied by branches of the rami communicantes, direct branches of the truncus sympathicus, and by the LLA nerve plexus⁶ (Figure 1).

Because many of the afferent fibers from the discus travel along with nervi sympathici, some investigators have sought to prove the discus has a sympathetic innervation and that both nerve networks consist of interconnected nerves with somatic and autonomic branches from various lumbar spinal nerves.⁶ This assumption has been endorsed by Suseki et al.⁷ and indirectly supported

by a recent RCT showing pain relief following radiofrequency (RF) lesioning of the rami communicantes.⁸

Significance of This Innervation Pattern

The observation of left-right and cranio-caudal connections in these nerve plexuses further suggest that lateralized disorders, in which nociceptive stimuli reach the spinal cord via nervi sinuvertebrales from the other side, can cause pain at a side that is contralateral to its origin. This could explain why patients complain about pain on the left side at one time and another time about pain on the right. Another implication is that the majority of spinal structures, including the discs, are innervated multisegmentally.⁶ Via the mechanism of deep somatic referred pain, this innervations pattern leads to an overlap in distribution of referred pain areas from adjacent structures. As a result, the pain projections are not always reliable for determining the source of the pain.

Finally, if the human disci receive significant afferent fibers via sympathetic pathways, their cell bodies may be

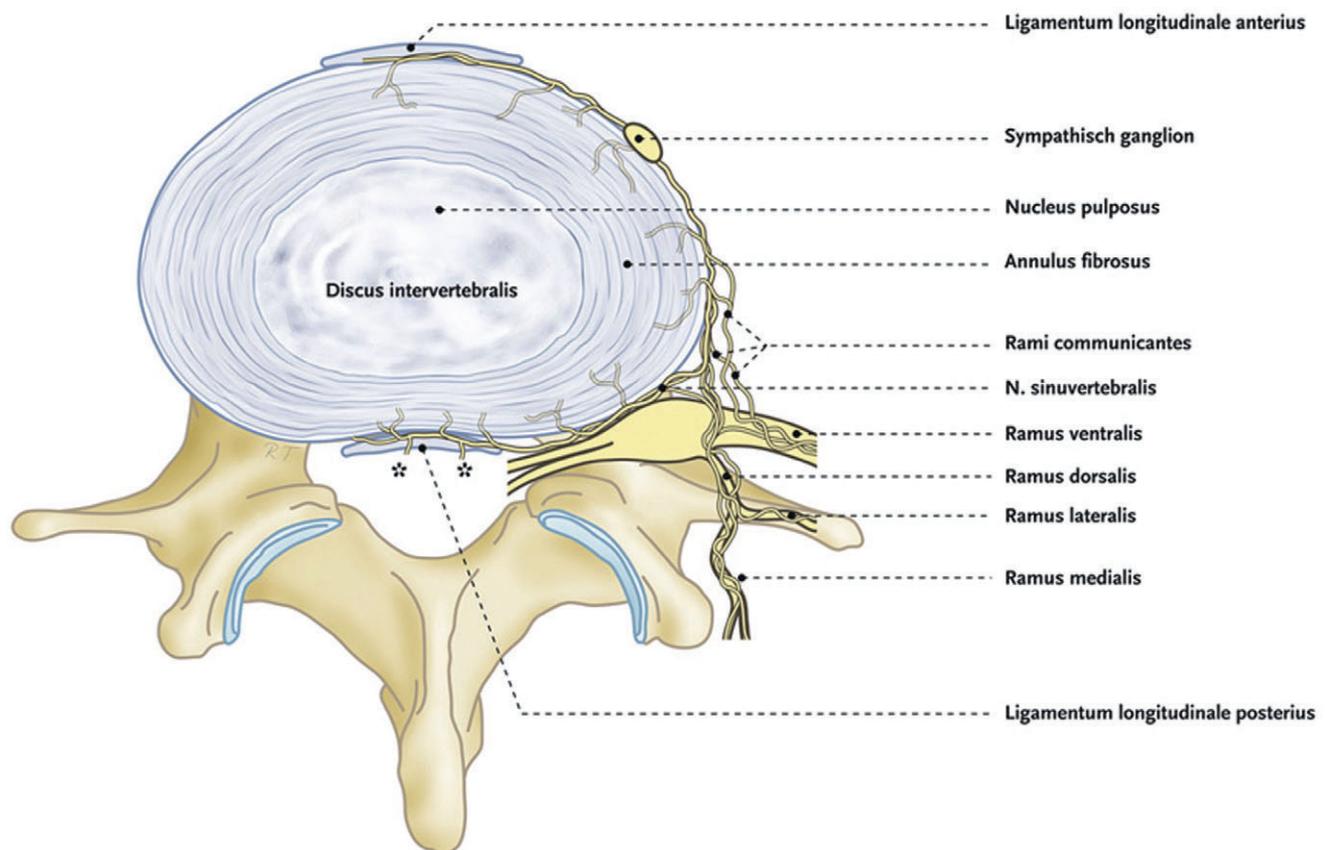


Figure 1. Schematic drawing of the lumbosacral innervation.⁶ *Connections to the dural nerve plexus. Illustration: Rogier Trompert Medical Art. <http://www.medical-art.nl>.

primarily located in the ganglia spinalia (dorsal root ganglia, DRGs) of C8-L2 nerves, ie, the levels at which the sympathetic nerve fibers leave the spinal cord.⁶ Although it has yet to be proven true, some researchers have utilized this hypothesis to obtain a specific block of the nervus spinalis L2 for low lumbar discogenic pain.⁹

I. DIAGNOSIS

I.A HISTORY

There are no specific characteristics in the patients' history that confirm or disprove the diagnosis of discogenic low back pain.¹⁰ More typical features include persistent, nociceptive low back, groin and/or leg pain that worsens with axial loading and improves with recumbence. Patients may have experienced a prior episode of acute, intense pain caused by an acute tear in the innermost part of the AF (although no scientific proof of this exists).

Discogenic low back pain is often localized medially in the back, and more detailed referral patterns were reported by Ohnmeiss et al. during provocative discography.^{11,12} Discogenic pain originating from the L3/L4 level typically radiates to the front (anterior) side of the thigh, L4/L5 to the outside (lateral) of the thigh, and sometimes to the back (posterior) of the thigh, and L5/S1 usually causes pain on the back of the thigh.

I.B PHYSICAL EXAMINATION

There are no typical characteristics of discogenic pain in the physical examination. Biphasic straightening from flexion is considered by some to be an indication of a discus complaint. Pain as a result of pressure on the processus spinosus is considered characteristic of discogenic low back pain ("Federung"). Vanharanta¹³ has described pain radiating from the discus due to provocation with a tuning fork pressed on the processus spinosus of the affected segment. Although suggestive, these physical examination characteristics have not been validated, and the current criterion standard for confirming a clinical diagnosis of discogenic pain is a positive discogram and the demonstration of a Grade 3 annular tear.^{14,15}

I.C ADDITIONAL TESTS

Imaging techniques such as CT and MRI are highly effective means of demonstrating detailed anatomical abnormalities in the vertebral column.^{16,17} These imaging techniques are limited in that only an indication can be given for the cause of the pain. Recently, the

presence of a high-intensity zone (HIZ) has been correlated with the presence of discogenic pain at that level. The HIZ may be an indication of an annulus tear that extends to the outer third of the annulus. The HIZ may be caused by the presence of inflammatory cytokines. Conflicting studies can also be found in the literature concerning this subject. On the one hand, a study done by Wolfer and Derby showed an 80% correlation between the HIZ and discogenic pain. Carragee, on the other hand, claims that this HIZ regularly occurs in asymptomatic control patients as well.^{14,18} In spite of the regular appearance of conflicting literature, especially between Carragee's and Derby's groups, provocative discography remains the *gold standard* for the diagnosis of discogenic pain. Although MRI images are helpful in visualizing such pathology as discus degeneration and desiccation, HIZs, and loss of disk height, the results commonly correlate poorly with clinical findings, leaving open the critical question of causality. To date, provocation discography is the only available method of linking the morphologic abnormalities seen on MRI with clinically observed pain, and its predictive value has been repeatedly questioned, mainly as a result of reported false positive rates.

Pathophysiology of Discogenic Pain and Discography

In the normal discus intervertebralis, sensory nerves innervate the outermost third of the annulus. In the degenerated discus, this innervation is deeper and more widespread; some fibers even penetrate the NP.¹⁹⁻²⁶ By now, it is also an accepted fact that the discus can be a frequent and significant source of low back pain. Every discus has a nucleus that is surrounded by a fibrous structure, the AF. As a result of aging, an anomalous posture of the back, or injury, the discus intervertebralis can become weaker, and fissures and tears can arise in the annulus (Figure 2). These tears can cause chronic pain if the tear in the annulus extends to its outermost third.

Based on CT-discography studies, the annular tear is becoming more frequently implicated as the basis for discogenic pain. The emphasis lies more on the extent and the dimensions of the annular tear than on disc degeneration. Sachs et al.²⁷ developed the "Dallas Discogram Scale," a 4-point scale that specifies the degree of discus degeneration. Grade 0 indicates a discus in which the contrast agent remains entirely in the NP. Grades 1 through 3 indicate tears in which the contrast agent extends to the innermost, middle, and outermost sections, respectively, of the AF. Later, Grade 4 was

added; the Grade 4 fissure has expanded into an arch-shaped tear outside of or in the innermost ring of the annulus (Figure 3).

Subsequently, Vanharanta²⁸ demonstrated the relationship between the expansion of the tear in the annulus and pain reproduction during discography.

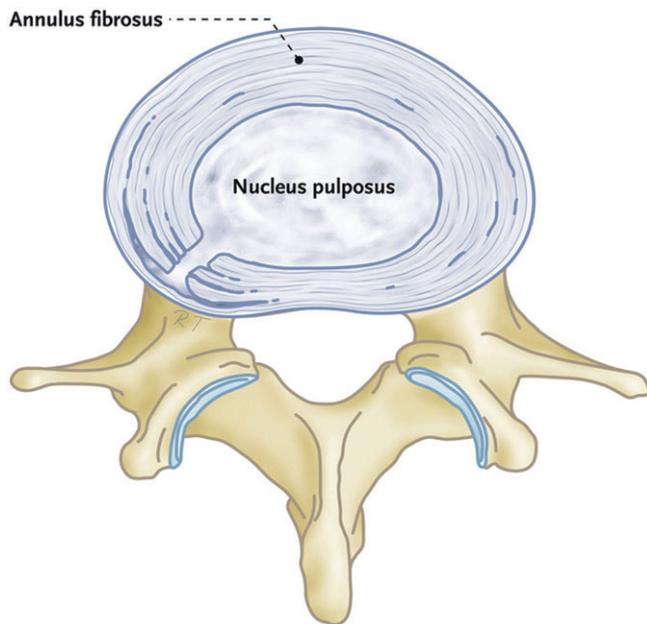


Figure 2. Discus intervertebralis with tears and fissures in the annulus fibrosus. Illustration: Rogier Trompert Medical Art. <http://www.medical-art.nl>.

Grades 0 and 1 are almost never painful. In Grade 3 annulus ruptures, more than 75% of the discographies are accompanied with exact reproduction of concordant pain. On the other hand, it has been shown that in pain reproduction during discography, 77% of the discs intervertebrales have an internal morphology with a Grade 3 rupture. This concordant pain is also present very intermittently in Grade 2 ruptures.

Chemical changes. There are two types of chemical changes that occur in the degenerative discus. First, a fracture in the vertebral endplate can lead to the introduction of inflammatory cytokines in the NP. This inflammation response changes the delicate nutrient balance in the nucleus, resulting in diminished oxygen diffusion, increase in local lactate concentration, and decrease in pH inside the discus.

In some cases, the cytokines themselves can be the source of pain, and outer annular rupture may facilitate the “leakage” of these inflammatory mediators to the adjacent epidural structures such as the ligamentum longitudinale posterius, dura, and ganglion spinale (dorsal root ganglion, DRG). The ingrowth of nociceptors into the deeper layers of the discus may sensitize the discus to normal mechanical loads. In addition, irritation of the nerve endings in the VE can produce pain. All or some of these mechanisms may cause a “chemically or mechanically” sensitized discus.²⁸

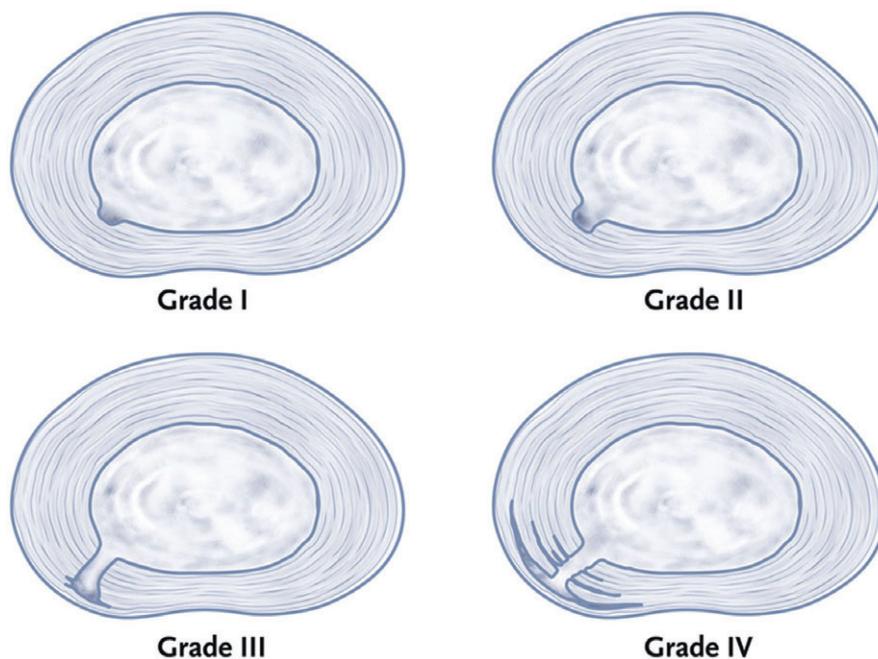


Figure 3. Gradation of the radial fissures visible on CT discography. Illustration: Rogier Trompert Medical Art. <http://www.medical-art.nl>.

Lumbar Discography

Definitions. Stimulation of a discus intervertebralis is a procedure that was developed for the purpose of confirming or refuting a clinical hypothesis of discogenic low back pain. The procedure is performed by inserting a needle in the NP of the target discus and injecting contrast agent (or another suitable medium) in order to test the sensitivity of the discus to gradually increasing distending pressures.

Discus stimulation is the more accurate name for a procedure that until now has often been described as (provocative) discography.

Discography is a procedure in which a contrast agent is introduced into the nucleus of a discus with the goal of describing the morphology of that discus.

Discography thus differs from disc stimulation—a procedure in which attention is focused on the reaction of the patient. Discus stimulation is usually followed by discography in order to verify the correct needle position or to elucidate the internal morphology of the discus. A combination of these definitions could be called provocative discography.

Patient selection. Suitable patients for this procedure are those with chronic low back pain, with or without pseudo-radicular referral, which lasts for longer than 3 months and which does not react to medication, transcutaneous electric nerve stimulation (TENS) and other conservative measures, and for which minimal invasive treatments of the facet joints and the sacroiliac joints do not prove to be effective or are not sufficiently effective. The implementation of the discography procedure is only advisable as a preparation for a possible interventional treatment aimed at reducing discogenic pain. An X-ray and an MRI of the lumbar spinal column must be performed not earlier than 6 months prior to the procedure.

Contraindications

Absolute

- absence of informed consent for discography (or other interventional treatments);
- local infection;
- pregnancy;
- local infection at injection site; and
- systemic infection

Relative

- allergy to contrast agent, local anesthetics, or antibiotics;

- known increased tendency to hemorrhage; and
- use of anticoagulants.

Procedure. Provocative discography is performed in the operating room under strict sterile conditions. Thirty minutes before the intervention, the patient is administered intravenous antibiotics (2 g cephazolin, i.v.). Many interventionalists also mix antibiotics within the intradiscally injected contrast at a concentration between 1 and 10 mg/mL (eg, 3 mg/mL cephazolin). The administration of antibiotics for the prevention of a discitis is disputed.²⁹ In their review, Willems et al. indicate that the side effects of antibiotics (allergic reactions) are even greater than the potential benefits and advise against administering antibiotics.²⁹ Yet currently, international consensus exists to administer periprocedural antibiotics as part of the discography procedure. The most important condition for the prevention of a discitis is observance of strict sterile technique.

Position. In the operation room, the patient lies in the prone position on an X-ray permeable table.

Sterility. The skin of the low back and the gluteal region is thoroughly disinfected. The operator and the assistant must wash their hands according to the local protocol of the hospital, and must wear protective clothing (surgical caps, surgical jackets and sterile gloves). After the injection point has been marked, the patient is covered with a sterile drape. The same must be done with the C-arm. Due to the limited rotation of the C-arm, it must be located on the side of the patient where the needle will be inserted.

Level determination. The levels to be examined are chosen based on a combination of patient history, physical examination, and additional examinations. The symptomatic level and the two adjacent levels are examined. Heretofore, the one or two adjacent disci intervertebrales serve as control levels, although recent evidence³⁰ showing a ~20% increase in long-term degenerative changes on the side of needle puncture may preclude needle puncture of MRI normal-appearing discs for the sole purpose of a control level. Typically, the least degenerated or more likely asymptomatic levels are studied first. The patient should be blinded to the discus level and should not be aware of the start of the discus stimulation. The patient should preferably be only be lightly sedated during the procedure, but those on copious narcotics should be given a judicious dose

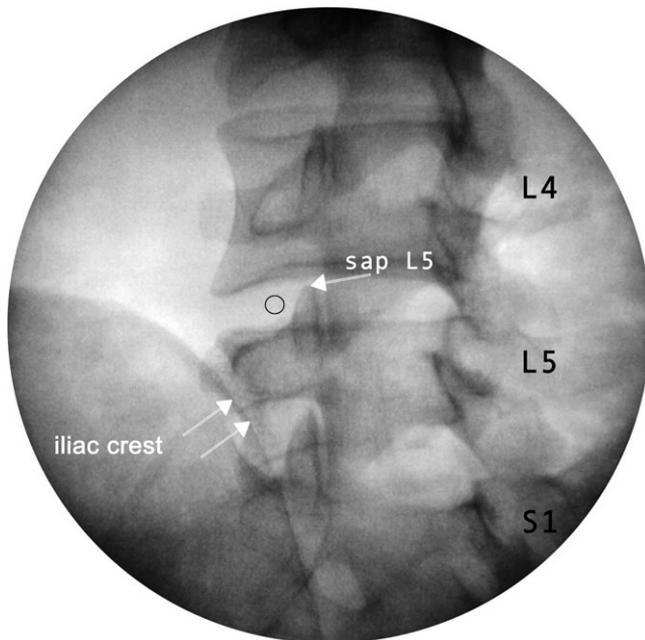


Figure 4. Starting point of the needle, assuming a maximal disc height, is such that the C-arm is rotated so that the facet column is between 1/3 and 1/2 of the corpus vertebrae. The injection point is then directly lateral to the processus articularis superior (superior articular process, sap).

so that there pain sensitivity is not exaggerated. The patient must be awake and able to reliably report during the disc stimulation.

The C-arm is first positioned with the direction of the radiation beam parallel to the subchondral plate of the lower vertebral plate of the discus. In the discs above L5-S1, the C-arm is then rotated ipsilaterally until the lateral aspect of the processus articularis overlies the axial middle of the discus to be punctured (Figure 4), and the disc height is at its maximum. In this projection, the needle can be inserted parallel to the direction of the radiation beam and brought into position (tunnel view). The target for the puncturing of the AF is the lateral-middle side of the discus, just lateral to the lateral edge of the processus articularis superior (Figure 5).

At the L5-S1 level, the crista iliaca does not allow access to the discus using a down-the-beam approach. The fluoroscopy tube is rotated until the lateral edge of processus articularis superior of S1 is positioned approximately 25% over the posterior to anterior distance of the corpus vertebrae.

Needle positioning. A new needle is used for each discus to be examined. After anesthetizing the skin

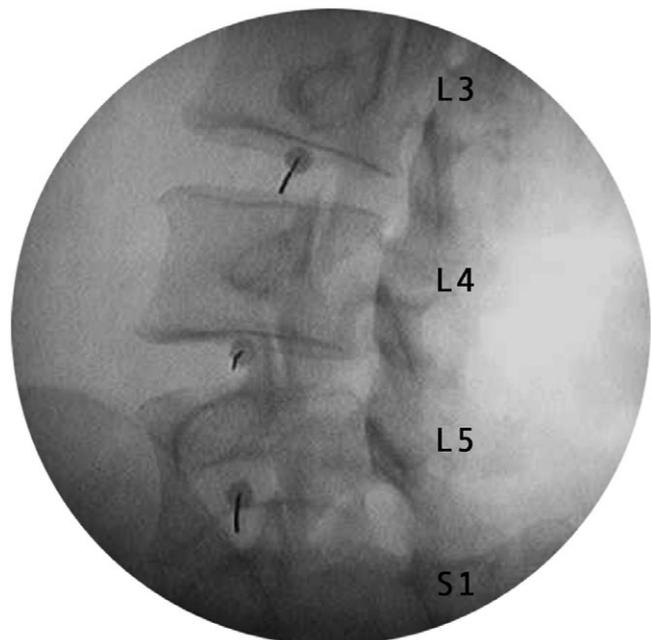


Figure 5. Needle position for an ideal discogram at the L3-L4, L4-L5, and L5-S1 levels.

and the underlying tissue, a one-needle or a two-needle technique can be used to approach the discus. In a two-needle technique, a 20-G needle is advanced over the lateral edge of the processus articularis superior. A 25-G hollow needle is then inserted through this needle and into the AF until it reaches the middle of the nucleus. The two-needle technique may help reduce the incidence of discitis and allow entering the discus with needles of a small diameter (eg, 27 G) which might help prevent the incidence of iatrogenic disc degeneration.³⁰

The needle is carefully advanced to the needle-point end position. Beyond the processus articularis superior, the needle passes through the foramen intervertebrale in the vicinity of the ramus ventralis. In case of paresthesia, the needle must be repositioned. A strong resistance is felt as the needle passes through the annulus. The needle is pushed through the annulus to the center of the discus. The needle's progress is followed in various projections, first in AP and then in lateral projection (Figure 6). Ideally, after placement, the needle is situated in the middle of the disc's nucleus, as seen in the AP as well as in the lateral projection. Other examples are given in Figures 7 and 8.

Discus stimulation. After verification of the correct needle position, the stylet is removed from the needle

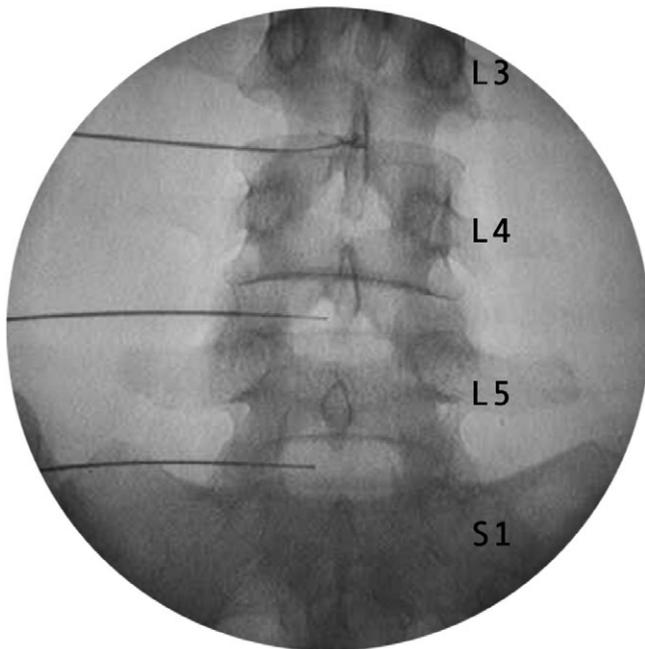


Figure 6. AP-position of the needles at discography in which the needles have been positioned in the middle of the nucleus pulposus.

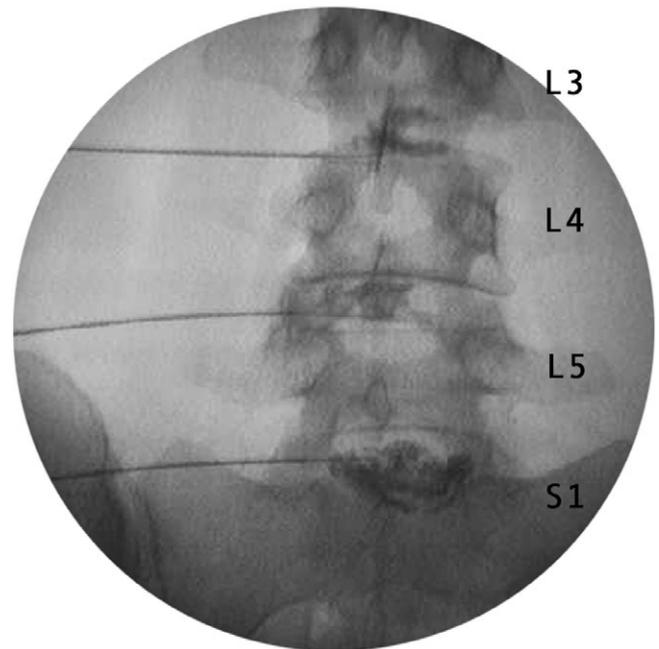


Figure 8. Discography at 3 levels: L3-L4, L4-L5 and L5-S1, all in anteroposterior view

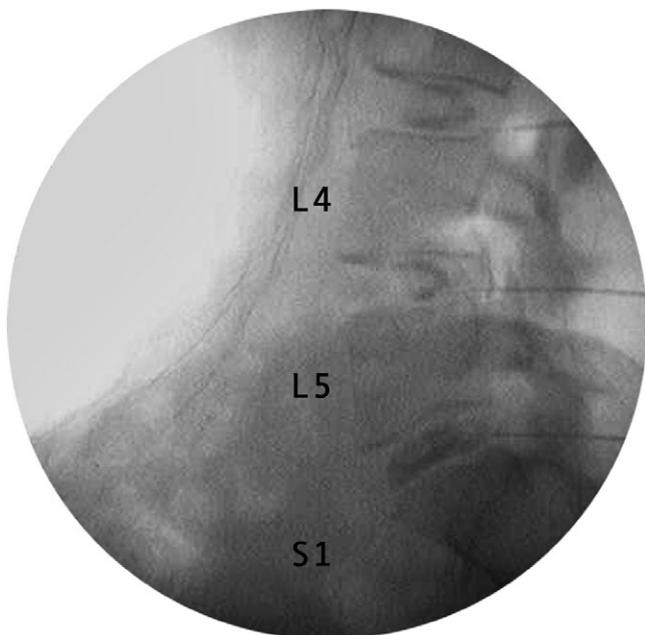


Figure 7. Discography at 3 levels where Grades 1 to 2 discs are visible at L3-L4 and L4-L5, and a disc with Grades 3 to 4 rupture is visible at level L5-S1.

and the needle is connected to a contrast agent delivery system which can measure the intradiscal pressure (manometry). The rate of infusion of the contrast agent should not exceed 0.05 mL/s.³¹⁻³³ This

rate reflects a static flow that corresponds to the distension pressure in the discus intervertebralis. If a higher flow is used, false positive discographies can occur due to the resultant pressure peaks. Pain is often provoked by these pressure peaks due to vertebral end-plate compression and distention of the adjacent facet joint. It is important that the discus expected to be most painful is stimulated last; the patient must not be able to see which discus is being stimulated. If the painful discus is stimulated first, it is possible that the echo of that pain lasts long enough to make adequate stimulation at other levels no longer possible. If these conditions have been met, the stimulation can be started.

The following parameters must be carefully monitored during the injection of the contrast solution: the opening pressure (OP), the pressure at which contrast is first visible in the discus; the provocation pressure, the pressure greater than the opening pressure at which complaints of pain arise; and the peak pressure or the final pressure at the end of the procedure. Ideally, pressure, volume, and provocation details are recorded at 0.5 mL increments, with additional notation made for the aforementioned events.

The procedure, per level, is continued until the following events:

- Concordant pain is reproduced at a level of 7 or greater (on a 0 to 10 numeric rating scale; NRS), and subsequent injected volume confirms the response.
- The volume infused reaches the 3.0 mL. (Up to 4 mL may be injected into a very degenerated disc when pressures remain less than 15 psi.)
- The pressure rises to 50 psi above opening pressure in discs with a Grade 3 annular tear.
- If contrast leaks through the outer annulus or through the endplates, one may not be able to pressurize the disc to a pressure sufficient to test the disc sensitivity. In these cases, the rapid manual injection may be acceptable, but must be noted and a negative response is a more defensible response.

Assessment criteria. The guidelines of the IASP (International Association for the Study of Pain), as well as those of the ISIS (International Spine Intervention Society), state that two levels must always be tested as controls when performing provocative discography (except if the target disc is that of L5-S1).³⁴⁻³⁶ A disc is only considered to be provocative (positive) if concordant pain can be induced at the target level, and if the control levels were negative for provocation of pain.

Manometry: Overestimation of discogenic pain due to a false positive response to provocative discography is also possible. Asymptomatic discs, with overpressurization, may become painful because normally quiescent nociceptors and mechanoreceptors in the endplates and ligamenta longitudinalia posteriora, and perhaps capsules of the facet joints, are stimulated. The diagnosis of discogenic pain can only be made if there is reproduction of concordant pain resulting from a pressure that does not produce pain in a normal disc or in an asymptomatic patient.

The concept and definition of a chemically sensitive disc was first described by Derby et al.³⁷ In 2004, O'Neill further described subgroups: discs with a pain threshold of 0 psi—these discs are described as chemically sensitive discs and³¹ discs with a pain threshold of 1 psi or higher—these discs are considered to be pressure sensitive. Pain thresholds ≥ 50 psi above the opening pressure correlated with a 100% chance of a false positive discography, whereas pain thresholds between 25 and 50 psi above the opening pressure still lead to 50% false positive results. This chance of a false positive disc decreases to 14% in a pain-sensitive disc at 15 psi above opening pressure. The true pressure-

sensitive disc probably has a pain threshold of 1–9 psi above opening pressure, or is considered a chemically sensitive disc (0 psi). The latter (chemically sensitive) disc intervertebralis is usually already extremely painful at the time of puncture. The classification of discs based on the pressure at which pain arises is illustrated in Table 2.

Morphologically, these discs are Grades 2 to 3 based on the Dallas Discogram Scale (Table 3). The international (IASP and ISIS) guidelines are based on these operational criteria:

1. Absolute discogenic pain:
 - Stimulation of target disc reproduces concordant pain.
 - The intensity of this pain has a Numeric Rating Scale (NRS) score of at least 7 on an 11-point scale.
 - The pain is reproduced by a pressure of less than 15 psi above the opening pressure.
 - Stimulation of the two adjacent discs is not painful.
2. Highly probable discogenic pain:
 - Stimulation of target disc reproduces concordant pain.
 - The intensity of this pain has a NRS score of at least 7 on an 11-point scale.
 - The pain is reproduced by a pressure of less than 15 psi above the opening pressure.
 - Stimulation of *one* of the adjacent discs is not painful.

Table 2. Classification of Discs on the Basis of the Pressure at which Pain Arises

- Discs that are painful at a pressure lower than 15 psi above opening pressure
- Discs that are painful between 15 and 50 psi above opening pressure
- Discs that are painful at greater than 50 psi above opening pressure
- Discs that are not painful in spite of the fact that the pressure is higher than 50 psi above opening pressure

Table 3. Assessment of the Morphology of the Discus Intervertebralis Using Discography

Dallas Discogram Scale:

Grade 0: The contrast remains entirely in the nucleus pulposus.

Grades 1 through 3: Indicate tears in which the contrast agent extends to the innermost, middle, and outermost sections, respectively, of the annulus fibrosus.

Grade 4: Here the Grade 3 fissure has expanded into an arc-shaped tear, outside of or in the innermost ring of the annulus fibrosus.

3. Discogenic pain:
 - Stimulation of target disc reproduces concordant pain.
 - The intensity of this pain has a NRS score of at least 7 on an 11-point numerical scale.
 - The pain is reproduced by a pressure of less than 50 psi above the opening pressure.
 - Stimulation of the two adjacent discs is not painful.
4. Possible discogenic pain:
 - Stimulation of target disc reproduces concordant pain.
 - The intensity of this pain has a NRS score of at least 7 on an 11-point numerical scale.
 - The pain is reproduced by a pressure of less than 50 psi above the opening pressure.
 - Stimulation of *one* of the adjacent discs is not painful, and stimulation of another disc is painful at a pressure greater than 50 psi above the opening pressure, and the pain is discordant.

Given that a strict selection process will improve the outcome of minimally invasive and surgical treatments, the goal must be to strive toward criteria 1 and 2 for the purpose of concluding that 1 and/or 2 discs are actually positive.

During discography the distribution of the contrast agent is monitored via lateral and AP radiographic examination.

Postoperative care. After the discography, the patient goes to the ward or to the recovery room. The patient may be discharged if the pain is under control and there are no signs of loss of neurological function. The patient may experience worsening of the pain symptoms in the first postoperative days and should be prescribed pain-relieving medication. The patient should be instructed to contact the doctor immediately if she/he experiences an increase in symptoms, loss of neurological function, and/or fever.

I.D DIFFERENTIAL DIAGNOSIS

The differential diagnosis is first and foremost directed at ruling out *red flags*, such as trauma and fractures, infection, tumors, and neurological complications. Thereafter, one strives to rule out visceral pain. Before making a decision about the interventional treatment plan, it is important to demonstrate that the disc intervertebralis is the cause of the (pseudo-) radicular pain.

II. TREATMENT OPTIONS

II.A CONSERVATIVE MANAGEMENT

There are no known studies that have demonstrated that long-term antinociceptive medication has any significant positive effect in patients with discogenic low back pain. Generally, medication such as NSAIDs and weak opioids are recommended for a limited time (maximum of three months).³⁸ A systematic review found no evidence for the added value of active exercise therapy in relation to inactive treatment (bed rest) and other conservative treatments such as traction, manipulation, hot packs, or corsets.^{39,40}

II.B INTERVENTIONAL MANAGEMENT

In the last few years, various minimally invasive treatments have been advanced to treat discogenic pain, such as intradiscal injections, IDET (intradiscal electrothermal therapy), discrode, biacuplasty, intradiscal radiofrequency (RF) thermocoagulation, and RF treatment of the ramus communicans. Several small-scale prospective and anatomical studies have been published recently concerning the possible role of nucleoplasty in chronic discogenic low back pain. In spite of the fact that these minimally invasive treatments may be an effective alternative to surgical treatments, they remain experimental. The definitive value of these treatments must be determined in the coming years with randomized, controlled studies.

Intradiscal Corticosteroid Injections

The goal of intradiscal corticosteroid injections is the suppression of the inflammation that is considered to be responsible for discogenic pain. The literature on this topic is limited to case reports that only yield positive results. However, positive and negative results are been found in prospective studies. Butterman published in 2004 a prospective study comparing patients with degenerative disc disease (DDD) and end-plate inflammatory changes on MRI (Modic Type 1) with a patient group having DDD and no end-plate inflammatory changes. The group with Modic Type-1 changes had significantly better results after intradiscal steroid injection compared with the group without Modic Type-1 changes.⁴¹

In 1992, Simmons published a study in which 25 patients received 80 mg methylprednisolone intradiscally versus a control group to whom 1.5 mL bupivacaine (0.5%) was administered.⁴² No significant

difference was found between the two groups. Khot et al. published a comparable study of 12 patients in which, after positive discography, the patients were randomly divided into two groups.⁴³ In one group, intradiscal corticosteroids were administered, and in the control group, physiological saline solution was administered. The authors concluded that intradiscal corticosteroids do not improve clinical outcomes in patients with discogenic low back pain relative to placebo.⁴⁴ Intradiscal injections with other chemical substances are being investigated. Klein et al. published a pilot study in which a glucosamine and chondroitin sulfate solution combined with hypertonic dextrose and dimethyl sulfoxide (DMSO) were injected intradiscally.⁴⁵ It has been suggested that the injection of these substances synergistically promotes the hypermetabolic response of chondrocytes and retards the enzymatic degradation of cartilage. The authors reported positive results in the VAS score and in the “disability score”. Derby et al. performed a comparable study in which he described effects analogous to those of IDET.⁴⁶ Given that this was only a pilot study, we must wait for RCTs to be able to make a judgment about the effect of these injections.

Intradiscal Electrothermal Therapy (IDET)

Saal and Saal published the first use of IDET for discogenic pain. The procedure consists of percutaneous insertion of a thermocoil into the disc under radiographic examination.⁴⁷ The catheter must be placed along the internal aspect of the posterior annulus. The distal portion of the catheter (5 cm) is heated for 16 min to 90°C. Experimental veterinary studies have demonstrated that this will result in temperatures exceeding 60°C in the posterior annulus and to a possible local denervation.

The first results were promising, with 50–70% of the patients experiencing significant pain reduction. Recent controlled studies are fueling much discussion about the actual effectiveness of this treatment.^{48,49} Concerning this, it must be said that it is unclear whether the inclusion criteria of the patients was selective enough, and whether the discography was considered the most important method of selection in conformity with what has already been described in this chapter.

Pauza et al. performed a randomized, placebo-controlled prospective study of the effectiveness of IDET in the treatment of chronic discogenic low back pain.⁵⁰ His group screened 1,360 patients with low back pain; 64 of these patients were selected for study after positive discography results. Thirty-seven patients were ran-

domized to the IDET group, and 27 patients to the sham group; the IDET catheter was inserted into the sham group, but without application of the RF current. Patients in both groups indicated improvement. In the IDET group, the average improvement in pain score, disability, and depression scale was significantly higher. Approximately 40% of the IDET group patients had an improvement of more than 50% in their pain scores. The NNT (number needed to treat) to reach more than 75% pain reduction was 5. These results suggest that the results of the IDET treatment cannot be completely ascribed to the placebo effect. These results also correspond with the results of various small-scale prospective study populations, which allow one to conclude that IDET can be effective in chronic, discogenic low back pain in a population selected with strict criteria. Pauza used the following inclusion criteria: age between 18 and 65 years, back pain more severe than leg pain, duration of pain symptoms at least 6 months, no improvement after a minimum of 6 weeks of conservative treatment (including medication, physical therapy, rehabilitation), back pain worsens with sitting and standing and is lessened by lying down, a score lower than 20 on the Beck Depression Inventory, no surgical interventions in the last 3 months, and less than 20% loss of disc height in the lumbar spine. In discography, the symptomatic level is indicated by way of negative control levels. A relative contraindication was obesity.

In 2006, Appelby et al. published a systematic review of the literature, and concluded that there was sufficient evidence for the effectiveness and safety of the IDET procedure.⁵¹ Contrary to Appelby's report is that of Freeman et al.⁵¹ This group took a very critical look at the existing literature, and came to the conclusion that the evidence for the effectiveness of the IDET procedure was weak and had a scientifically insufficient foundation. To date, a positive RCT, a negative RCT, various positive prospective studies, and two negative studies have been published. Notably, the fact that no more than two discs are degenerative is important. The outcomes in the cases with more extensive disc degeneration have been shown to be significantly worse. A serious limitation among the available IDET studies is that the selection criteria do not concur: a critical factor for achieving useful results. New studies with internationally defined inclusion criteria are needed in order to arrive at definitive judgments about the clinical effectiveness of the IDET procedure.

The mechanism by which IDET might act is not yet known. Two hypotheses have been proposed. The first

hypothesis assumes that electrothermal therapy of the annulus produces local pain reduction by way of denervation of the nociceptors. The second mechanism proposed states that changes occur in the structure of the collagen fibers in the annulus due to heating; these changes improve the stability of the annulus. As of yet, there is little histological proof to support this hypothesis.

The following are described as complications: catheter breakage, nerve injury (cauda equina lesion), post-IDET spinal disc herniation, discitis, local infection, epidural abscess.

Biacuplasty

Intradiscal biacuplasty is the latest in a series of minimally invasive posterior annulus heating techniques. This technology works specifically by concentrating RF current between the ends of two straight probes. Relatively even heating over the larger area of the posterior annulus is achieved by internally cooling the electrodes.^{52,53}

The procedure is completed under fluoroscopy, with the patient lying in the prone position. Two TransDiscal 18 G electrodes via introducers are placed bilaterally in the posterior annulus of the discus intervertebralis. The generator controls the delivery of RF energy by monitoring the temperature measured by a thermocouple at the tip of the probe. The temperature increases gradually over a period of 7–8 min to 50°C, with final heating at 50°C for another 7 min. It should be noted that although the temperature is set to 50°C on the RF generator, tissue temperature reaches 65°C due to ionic heating. During this time, the patient should be awake and able to communicate with the physician.

First, two pilot studies involving 8 and 15 patients demonstrated significant pain relief following the discus biacuplasty procedure at 3, 6, and 12 months.⁵⁴ In the European case series involving 8 patients, there was an average of about 50% pain reduction at 3 months, with overall good patient satisfaction. In the prospective pilot study involving 15 patients, Kapural et al. reported patient improvements in several pain assessment measures after undergoing discus biacuplasty procedure for discogenic pain.⁵⁴ Results from these pain assessment measures included a reduction in the median VAS pain score from 7 to 4 at 1 month, which remained at a level of 3 at 6 and 12 months follow-up, improvement in Oswestry index from 23.3 to 16.5 points at 1 month, which remained similarly improved after 12 months, and an increase in the SF-36 Bodily Pain score from 38

to 54 points.⁵⁴ Pilot studies and case series, even when designed as prospective trials, tend to exaggerate the positive outcomes. Therefore, we await results of sham controlled, prospective randomized studies before accepting or refuting this approach to the treatment of discogenic pain. Still, intradiscal biacuplasty may hold several advantages over previous techniques. There is minimal disruption to the native tissue architecture, and thus the biomechanics of the spine are likely unchanged. Additionally, the relative ease of electrode placement eliminates the need to thread a long-heating catheter (eg, compared with IDET).

Intradiscal Radiofrequency (RF) Thermocoagulation

Intradiscal RF thermocoagulation is used for the treatment of discogenic pain. Barendse et al. performed a double-blind, randomized prospective study on 28 patients.⁵⁵ The discogenic pain diagnosis was made on the basis of the injection of a mixture of 2 mL lidocaine (2%) with contrast agent. Patients who indicated more than a 50% reduction in pain within 30 min were included and randomized into 2 groups. Patients in the RF group ($n = 13$) received an RF treatment of the discus intervertebralis lasting 70 s at 90°C in which the needle was placed in the center of the discus. Patients in the control group underwent the same procedure, except that no RF current was administered. Eight weeks after the treatment, there was no difference between the VAS scores of the two groups for pain and global perceived effect, or in the Oswestry Disability Index. The conclusion was that RF is ineffective for the treatment of discogenic pain. Two important remarks can be made about this study. First of all, the discography was not performed using a method that is currently accepted. It has subsequently become clear that discogenic pain is caused by nociceptors that are found in the outermost layer of the annulus. Heating the center of the nucleus will not necessarily lead to the destruction of nociceptors in the annulus.

Ercelen et al. performed another randomized prospective study with RF for discogenic pain using an improved selection and treatment method.⁵⁶ Ercelen's group selected 39 patients on the basis of a provocative discography. These patients were randomized into 2 groups. In the first group, the discus was heated for 360 s to 80°C; in the other group, for 120 s to 80°C. In this study, there were also no significant differences in pain reduction and functionality.

Recently a new intradiscal RF method has been introduced—discTRODE™ (Valleylab, Boulder, CO,

U.S.A.). The DiscTrode is positioned along the posterior interface between the nucleus and the annulus. In an open trial, Erdine et al.⁵⁷ found improvement of symptoms as measured by the SF-36 and the VAS score in 10 of 15 patients (66.6%). Finch et al. reported a case-control study of 46 patients with mono-discopathy with an annular tear confirmed by means of a provocative discography.⁵⁸ Thirty-one patients underwent the disc treatment with heat via the DiscTrode, and 15 patients functioned as control group. In the control group, conservative treatment was continued. The VAS score was significantly reduced in the RF group, and this reduction persisted for 12 months. In the control group, the VAS score did not change. The authors concluded that heating the annulus, particularly at the level of the annular tear, can potentially be a good alternative for the treatment of discogenic pain. More recently, Kvarstein et al.⁵⁹ published a randomized controlled trial comparing intra-annular RF to sham treatment. The authors concluded that there was no beneficial effect of DiscTrode compared with the sham group. Another conclusion was the advice not to use the DiscTrode because of the high number of patients with increased pain in the treatment group.⁵⁹ However, the study of Kvarstein et al. was criticized for its lack of power and the fact that the study was terminated early.⁶⁰ This technology proved to be ineffective in improving functional capacity and VAS scores when compared with IDET during the study where strict patient selection criteria were employed.⁶¹

Ramus Communicans Block

Discogenic low back pain could be considered to be deep somatic pain, if viewed from its neural origin. However, the innervation of the discus shows a multisegmental origin. As described above, the sensory nerve fibers reach the spinal cord via adjacent and more distant rami communicantes and ganglia spinalia (dorsal root ganglia, DRGs) (Figure 1). Based on the work of Groen et al., Ohtori's group recently demonstrated that in rats the low lumbar intervertebral discs are chiefly innervated by L1-L2 ganglia spinalia (DRGs) via the truncus sympathicus and the ramus communicans.^{6,62} Fibers from the L3-L6 ganglia spinalia (DRGs) directly innervate the LLP via the nervi sinuvertebrales. Nakamura et al. looked at the afferent pathways that could be responsible for the discogenic low back pain by selectively blocking the L2 root in 33 patients.⁹ On the basis of these findings, the authors concluded that the L2 segmental nerve could possibly be the most impor-

tant afferent pathway for discogenic pain of the low lumbar discs, mainly by way of sympathetic afferent fibers of the nervi sinuvertebrales. Infiltration of the L2 root can then also be useful as a diagnostic procedure and as a therapy.

A block and destruction of the ramus communicans is also described as a treatment for discogenic low back pain or for pain in the vertebra itself.⁶³ Chandler et al. described the ramus communicans block as being an effective treatment for pain originating from a vertebral compression fracture.⁶⁴ Oh and Shim investigated the effectiveness of RF thermocoagulation of the ramus communicans in 49 patients.⁷ These patients had chronic discogenic low back pain at 1 level, and had previously received no effect from an IDET treatment. Patients were randomized into an RF group and a control group. The control group received a lidocaine injection near the ramus communicans without RF. After 4 months, there was significant improvement in VAS scores and improvements in the Short Form (36) Health Survey (SF-36) in the RF group relative to the control group. The authors concluded that the RF thermocoagulation of the ramus communicans could be considered as one of the treatments for discogenic low back pain.

In spite of the promising initial results, further randomized studies of the effects of the ramus communicans block on discogenic pain are also needed in this case. A number of questions must still be answered. What is the definitive role of L1-L2 in discogenic low back pain; what is the role of the ramus communicans in this? Which patients react best to a ramus communicans block, and how long is this treatment effective?

Other interventional techniques. Although this overview is not complete, the following techniques have been used frequently in the past. In chemonucleolysis, the enzyme chymopapain is injected into the discus intervertebralis; as a result, the NP is dissolved. This therapy has been almost completely abandoned due to problems related to dosage reliability, difficulties with the supply of chymopapain, and a number of serious complications. Otherwise, the treatment appears to be effective as demonstrated by various RCTs.⁶⁵

Automated percutaneous lumbar nucleotomy (APLD) is a technique in which a section of the nucleus is mechanically removed percutaneously in order to effect decompression of the nucleus. However, the technique has been proven to be less effective in comparison

with other treatments, and is therefore not advised.⁶⁵ A more modern variant of percutaneous nucleotomy using the Dekompressor™ (Stryker Corp., Kalamazoo, MI, U.S.A.) is still being used; it has a smaller diameter than the original APLD apparatus. There is no evidence present in the literature for this technique, and until otherwise shown, it can be considered to be the same as the classic APLD. Percutaneous laser disc-decompression (PLDD) is a treatment method that has been utilized on a large-scale world-wide since the beginning of the 1990s. Laser heat is used to bring about the evaporation of nuclear material. Unfortunately, until now, only case series have been reported.⁶⁵ Currently, the following techniques are applied most often world-wide: Nucleoplasty® (Arthrocare, Stockholm, Sweden), Ozone Discolysis, Targeted DISC Decompression, and the aforementioned Dekompressor™.

Percutaneous Intradiscal Treatments for Disc Herniation

As previously mentioned in the introduction, there is a clear overlap of the clinical signs of discogenic lumbago and the symptoms of spinal discus herniation. Discus herniation usually leads to a combination of discogenic lumbago and radicular leg pain. There seems to be evidence of a complex interaction between biochemical factors originating from the NP of the discus intervertebralis and mechanical factors (nerve root compression), which together cause the pain. Also, see the practice guideline on radicular pain.⁶⁶

The goal of epidural injection of steroids in cases of herniated discus is primarily anti-inflammatory and therefore pain lessening. The goal of this treatment is rapid reduction in pain symptoms compared with a conservative treatment. The treatment must be considered conservative during the natural course of the acute lumbosacral radicular syndrome, which is the result of a discus herniation. In the long term, there are no differences in outcome in comparison with conservative treatment without epidural injection of steroids.

The differences between conservative treatment and operative discectomy are also not demonstrated in the long term. Operative discectomy is nonetheless utilized on a large scale. The reason for this is that the intervention can often lead to a more rapid reduction in symptom complaints when compared with a conservative treatment policy.⁶⁵ The disadvantages are the operative and anesthesiological risks and the risk of epidural adhesions, which are associated with the so-called postlaminectomy syndrome, or the failed back-surgery

syndrome. Otherwise, the indications for operative discectomy are larger discus protrusions and extrusions that show signs of nerve root compression on MRI. Smaller, focal protrusions without nerve root compression appear to be less apt to spontaneously resorb, and have a less favorable natural course; in other words, these small hernias often produce long-term pain symptoms with a slow spontaneous recovery.⁶⁷

Over the years, the aforementioned considerations have led to various percutaneous, minimally invasive intradiscal techniques directed at the mechanical factor of discus herniation with the underlying idea of capitalizing on the advantages of operative therapy with as few of the disadvantages as possible. Most of these techniques—in contrast to the surgical discectomy—have the common goal of decompressing the nucleus so that there is a change in volume and an accompanying reduction in the pressure on the nerve and/or a lessening of the inflammatory reaction as a result. For these purposes, these techniques are usually only possible in the case of a so-called “contained” hernia.

Nucleoplasty. The decompression method utilizes “coblation,” in which a high-energy plasma field is generated with the help of a bipolar RF probe. This plasma field breaks molecular bonds. For this reason, the technique is also called plasma disc decompression (PDD). Tissue can be evaporated in this way at relatively low temperatures (40 to 70°C). However, the plasma field can only arise in conductive surroundings. In practice, this means that the treatment is not effective in a dehydrated discus (“black disc” on MRI). After a 16-G needle has been positioned in the nucleus, the probe is moved back and forth and rotated intradiscally. In this way, 6 or more tunnels are made in the nucleus, and the intradiscal pressure drops. Meanwhile, the treatment has been utilized on a large scale, and the complication level appears to be low and acceptable.^{68–70}

Percutaneous Disc Decompression Using Dekompressor™

The percutaneous disc decompression (Dekompressor™) technology extracts nuclear discus material by an auger within a cannula that ends inside the nucleus. A significant change in intradiscal pressure should follow the reduction of nuclear volume within the closed hydraulic space. It is imperative that the annular wall should be intact in order to retract the bulging section. Therefore provocative discography may occasionally be needed to confirm the affected level and to rule out any

annular disruption. In their case series, Alo and colleagues reported an 80% success rate with this technique.⁷¹ Although there are no controlled studies published on Dekompressor efficacy, a European study reported also pain score improvements in the majority of patients treated with Dekompressor. This seems to suggest that patients with posterolateral foraminal disc protrusions can typically expect more pain relief than those with posteromedian ones.⁷² There are no randomized, sham studies on percutaneous discectomy using Dekompressor device.

Ozone discolysis. Ozone Discolysis consists of the injection of a mixture of O₃ and O₂, usually both intradiscally, as well as epidurally. As a result, an oxidative dehydration takes place in the nucleus; this is comparable with chemonucleolysis by means of chymopapain. In addition, upregulation of the intracellular antioxidant scavenger system occurs due to oxidative stress; this results in an increase in the endogenous anti-inflammatory response.⁷³ In addition to various large case series with remarkably good results, two comparative studies have been published.^{74,75} In Gallucci's study, intradiscal and transforaminal epidural corticosteroid injection is compared with intradiscal transforaminal epidural steroid injection with the addition of an O₃/O₂ mixture.⁷⁶ Bonnetti et al. had already published a comparative study examining transforaminal epidural injection of an O₃/O₂ mixture versus transforaminal epidural steroid injection.⁷⁷ In both studies, ozone resulted in a significantly better effect than corticosteroids. There are no significant complications of the technique described. Ozone Discolysis can be utilized for "contained," as well as for "noncontained" spinal disc herniation. The extent to which the degree of disc degeneration has an influence on the clinical result is not yet clear. Although the technique is primarily meant for spinal disc herniation with prominent radicular pain, it is also utilized for discogenic lumbago associated with spinal disc herniation.

Targeted Disc Decompression (TDD). This technique stems from the IDET technique for discogenic lumbago. In connection with the IDET technique, there have been some reports of unintentional shrinking of the size of disc protrusions as an effect of the technique. TDD makes use of just this property. The catheter used has approximately the same configuration as an IDET catheter; however, the active zone, where coagulation of disc tissue occurs, is markedly shorter. The goal is to

position the active zone on the annulus-nucleus boundary at the point of the "contained" protrusion. Given that this technique is a thermocoagulation, the degree of hydration of the nucleus is, in principle, not important. Although the technique is increasingly utilized and appears to provide good results, no literature has as yet been published about TDD.

Evidence for New Developments

The techniques described in new developments above are currently being investigated for effectiveness and complications. At this time, it does not appear to be possible to formulate an evidence rating and recommendations.

II.C COMPLICATIONS OF INTERVENTIONAL MANAGEMENT

Although all these procedures are associated with minimal tissue damage, a short recovery time, and low infection risk, various rare complications have been reported such as catheter breakage, nerve root injuries, post-IDET disc herniation, discitis, radicular pain, severe headache, cauda equina syndrome, and vertebral body osteonecrosis.⁵⁹ The most important complication of minimally invasive intradiscal procedures is discitis. The incidence is very low at 0.25% to 0.7%.^{29,78} Any patient who complains about increased pain within 1 week after the procedure must be carefully examined. At the very minimum, this examination must include patient history, physical examination, and laboratory examination (infection parameters). If the infection parameters are elevated or abnormal, or in case of doubt, an MRI must be performed in order to rule out discitis.

Staphylococcus aureus is the major cause of discitis. The chance of discitis can be reduced by the routine prophylactic use of intravenous or intradiscal antibiotics. Sharma et al. reviewed the literature and described that the chance of discitis is reduced from 2.7% to 0.7% with the use of the "through the needle technique"; in this technique, the needle is advanced through the skin until the annulus is reached, and another thin needle (25 G) is then advanced through the first needle into the discus.⁷⁸ Willems published a 0.25% incidence of discitis in a series of 4,981 patients on which the "through the needle" technique was used and to whom no prophylactic antibiotic were administered.²⁹ They also concluded that the routine use of antibiotics is not necessary for this procedure. However, the international

guidelines currently prescribe routine use of periprocedural prophylactic antibiotics.

II.D EVIDENCE FOR INTERVENTIONAL MANAGEMENT

A summary of the available evidence is given in Table 4.

III. RECOMMENDATIONS

Intradiscal corticosteroid injections and RF treatment of the discus are not advised for patients with discogenic low back pain. The current body of evidence does not provide sufficient proof to recommend intradiscal treatments, such as IDET and biacuplasty for chronic, non-specific low back complaints originating from the discus intervertebralis. We are also of the opinion that at this time the only place for intradiscal treatments for chronic low back pain is in a research setting. RF treatment of the ramus communicans is recommended.

III.A CLINICAL PRACTICE ALGORITHM

Figure 9 illustrates the practice algorithm for the management of low back pain of discogenic origin.

III.B TECHNIQUES

IDET

The procedure takes place under sterile OR conditions on a patient lying in the prone position with the aid of radiographic examination. While administering prophylactic antibiotics, a 17 G needle is inserted posterolaterally into the discus, generally on the side with the least complaints. Thereafter, a 30 cm-long catheter with a flexible tip, 5 cm of which can be heated, is advanced through the needle. This tip is advanced circumferentially through the NP until it covers the entire posterior section of the annulus. After placement of the tip has been checked radiographically, the tip of the catheter is heated for 18 min to 90°C according to a standard protocol. This temperature is reached after 14 min and is then maintained for 4 min at this level. Then the

Table 4. Evidence of Interventional Pain Management of Discogenic Pain

Technique	Assessment
Intradiscal corticosteroid administration	2 B-
Radiofrequency (RF) treatment of the discus intervertebralis	2 B-
IDET (intradiscal electrothermal therapy)	2 B±
Biacuplasty	0
Distrode	0
Radiofrequency (RF) of the ramus communicans	2 B+

needle and the catheter are removed, and the patient can be discharged after the recovery period. If during the procedure, the patient complains of leg pain, it is possible that a spinal nerve is being irritated. In this case, the heating process should be immediately terminated. After the procedure, the patient must follow a strict 12-week long rehabilitation protocol. In patients with a large tear in the annulus, it may appear to be impossible to maneuver the catheter into the correct position.

Ramus Communicans

Diagnostic block. The C-arm is positioned in such a way that the direction of the radiation beam in the transverse plane is approximately 20° oblique such that the facet joints are projected away and the vertebral column is clearly visible. For the angle in the sagittal plane, the C-arm is rotated on its axis. As a result, the processus transversus changes location relative to the corpus vertebrae. The direction of the radiation beam must be such that the axis of the processus transversus lies slightly above the middle of the corpus vertebrae. Usually, an SMK-C15 cannula (Radionics, Burlington,

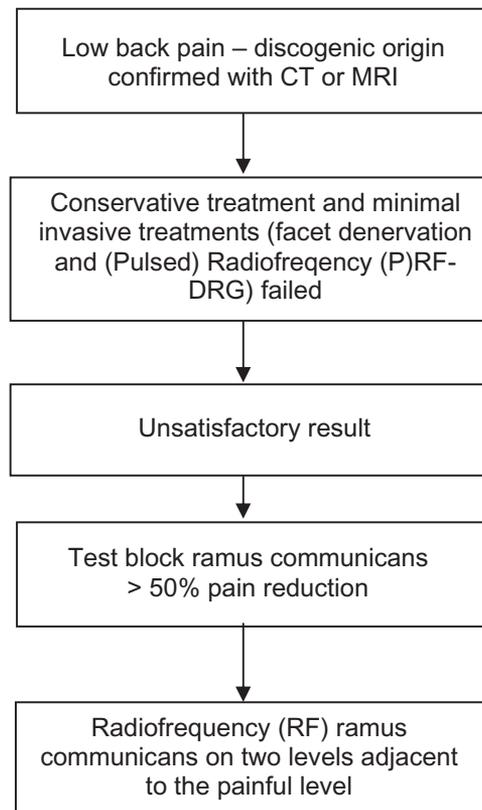


Figure 9. Practice algorithm for the interventional treatment of discogenic pain.

MA, U.S.A.) is used for this procedure. An injection point is marked just caudally to the processus transverses, and somewhat medially to the lateral edge of the corpus vertebrae. After local anesthetization of the skin, the needle is advanced using a tunnel view, for which the general rules of this technique must be observed; in other words, corrections to the direction of the needle must be made while the needle is in the superficial layers, and the depth of the needle must be checked regularly on the lateral projection. Do not try to make contact with the processus transversus. The needle is advanced until contact is made with the corpus vertebrae. On the lateral projection, the point of the needle lies somewhat ventral to the posterior side of the corpus vertebrae. Contrast agent (0.5 mL) is then injected. On the anteroposterior projection, this usually results in a very compact shadow; on the lateral projection, the contrast agent spreads anteriorly over the corpus vertebrae. In case of intravascular dispersal, a minimal change in position is usually sufficient. Finally, 1 mL lidocaine (2%) is injected.

RF treatment. An SMK-C15 cannula with a 2 mm active point is used. Fluoroscopy and the insertion of the needle conform completely with the technique described for the diagnostic block. When the needle has been correctly positioned, stimulation at 50 Hz causes sensations in the back at a voltage of < 1.5 V. Thereafter, 2 Hz stimulation is applied. Contractions of the leg muscles may not be allowed to occur at below twice the value of the sensory threshold. If these conditions are not met, then the needle is moved slightly laterally and anteriorly until a safe position has been achieved. A RF treatment is made for 60 s at 80°C.

The L5 level. This level deserves special mention since the anatomical relationships can require an adapted technique. This can be the result of a high crista iliaca or of a broad processus transversus. In these cases, the L5 segmental nerve exits the foramen intervertebrale more horizontally than the other lumbar nerves do. While adjusting the C-arm axially, it is best to project the processus transversus as high as possible. By doing so, a safe needle position can often be found for this level. Nevertheless, the intervention at this level is not possible in all cases.

IV. SUMMARY

Lumbar discography, provocative discography, and disc manometry are all examinations whose goal is to deter-

mine whether a discus intervertebralis is the cause of patients' pain symptoms. In spite of the unceasing stream of contradictory literature, provocative discography remains the gold standard for the determination of the diagnosis of discogenic pain. For the purpose of improving the results of minimally invasive intradiscal treatments, it is important to use a strict selection process to select discography patients' and to perform discography with manometry. It must be noted that the studies performed up to now have not included patients selected in the correct manner generally, and in particular by not adequately performing discography. This has certainly not had a positive influence on the results. For the treatment of discogenic pain, a RF treatment of the ramus communicans can be recommended.

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REFERENCES

1. Guyatt G, Gutterman D, Baumann MH, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an american college of chest physicians task force. *Chest*. 2006;129:174-181.
2. van Kleef M, Mekhail N, van Zundert J. Evidence-based guidelines for interventional pain medicine according to clinical diagnoses. *Pain Pract*. 2009;9:247-251.
3. Kuslich SD, Ulstrom CL, Michael CJ. The tissue origin of low back pain and sciatica: a report of pain response to tissue stimulation during operations on the lumbar spine using local anesthesia. *Orthop Clin North Am*. 1991;22:181-187.
4. Schwarzer AC, Aprill CN, Derby R, Fortin J, Kine G, Bogduk N. The relative contributions of the disc and zygapophyseal joint in chronic low back pain. *Spine*. 1994;19:801-806.
5. Cohen SP, Larkin TM, Barna SA, Palmer WE, Hecht AC, Stojanovic MP. Lumbar discography: a comprehensive review of outcome studies, diagnostic accuracy, and principles. *Reg Anesth Pain Med*. 2005;30:163-183.

6. Groen GJ, Baljet B, Drukker J. Nerves and nerve plexuses of the human vertebral column. *Am J Anat*. 1990;188:282–296.
7. Suseki K, Takahashi Y, Takahashi K, Chiba T, Yamagata M, Moriya H. Sensory nerve fibres from lumbar intervertebral discs pass through rami communicantes. A possible pathway for discogenic low back pain. *J Bone Joint Surg Br*. 1998;80:737–742.
8. 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;20:55–60.
9. Nakamura SI, Takahashi K, Takahashi Y, Yamagata M, Moriya H. The afferent pathways of discogenic low-back pain. Evaluation of L2 spinal nerve infiltration. *J Bone Joint Surg Br*. 1996;78:606–612.
10. Schwarzer AC, Aprill CN, Derby R, Fortin J, Kine G, Bogduk N. The prevalence and clinical features of internal disc disruption in patients with chronic low back pain. *Spine*. 1995;20:1878–1883.
11. Ohnmeiss DD, Vanharanta H, Ekholm J. Relation between pain location and disc pathology: a study of pain drawings and CT/discography. *Clin J Pain*. 1999;15:210–217.
12. Ohnmeiss DD, Vanharanta H, Ekholm J. Relationship of pain drawings to invasive tests assessing intervertebral disc pathology. *Eur Spine J*. 1999;8:126–131.
13. Vanharanta H. The intervertebral disc: a biologically active tissue challenging therapy. *Ann Med*. 1994;26:395–399.
14. Carragee EJ, Hannibal M. Diagnostic evaluation of low back pain. *Orthop Clin North Am*. 2004;35:7–16.
15. Zhou Y, Abdi S. Diagnosis and minimally invasive treatment of lumbar discogenic pain—a review of the literature. *Clin J Pain*. 2006;22:468–481.
16. 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;23:1252–1258; discussion 9–60.
17. Gilbert FJ, Grant AM, Gillan MG, et al. Low back pain: influence of early MR imaging or CT on treatment and outcome—multicenter randomized trial. *Radiology*. 2004;231:343–351.
18. Wolfer LR, Derby R, Lee JE, Lee SH. Systematic review of lumbar provocation discography in asymptomatic subjects with a meta-analysis of false-positive rates. *Pain Physician*. 2008;11:513–538.
19. Coppes MH, Marani E, Thomeer RT, Groen GJ. Innervation of “painful” lumbar discs. *Spine*. 1997;22:2342–2349; discussion 9–50.
20. Freemont AJ, Peacock TE, Goupille P, Hoyland JA, O’Brien J, Jayson MI. Nerve ingrowth into diseased intervertebral disc in chronic back pain. *Lancet*. 1997;350:178–181.
21. Hurri H, Karppinen J. Discogenic pain. *Pain*. 2004;112:225–228.
22. Peng B, Hao J, Hou S, et al. Possible pathogenesis of painful intervertebral disc degeneration. *Spine (Phila Pa 1976)*. 2006;31:560–566.
23. Peng B, Chen J, Kuang Z, Li D, Pang X, Zhang X. Expression and role of connective tissue growth factor in painful disc fibrosis and degeneration. *Spine (Phila Pa 1976)*. 2009;34:E178–E182.
24. Peng B, Wu W, Hou S, Li P, Zhang C, Yang Y. The pathogenesis of discogenic low back pain. *J Bone Joint Surg Br*. 2005;87:62–67.
25. Freemont AJ, Watkins A, Le Maitre C, et al. Nerve growth factor expression and innervation of the painful intervertebral disc. *J Pathol*. 2002;197:286–292.
26. Freemont AJ. The cellular pathobiology of the degenerate intervertebral disc and discogenic back pain. *Rheumatology (Oxford)*. 2009;48:5–10.
27. Sachs BL, Vanharanta H, Spivey MA, et al. Dallas discogram description. A new classification of CT/discography in low-back disorders. *Spine*. 1987;12:287–294.
28. Vanharanta H, Sachs BL, Spivey MA, et al. The relationship of pain provocation to lumbar disc deterioration as seen by CT/discography. *Spine*. 1987;12:295–298.
29. 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.
30. Carragee EJ, Don AS, Hurwitz EL, Cuellar JM, Carrino J, Herzog R. Does discography cause accelerated progression of degeneration changes in the lumbar disc: a ten-year matched cohort study. *Spine (Phila Pa 1976)*. 2009;34:2338–2345.
31. O’Neill C, Kurgansky M. Subgroups of positive discs on discography. *Spine*. 2004;29:2134–2139.
32. Seo KS, Derby R, Date ES, Lee SH, Kim BJ, Lee CH. In vitro measurement of pressure differences using manometry at various injection speeds during discography. *Spine J*. 2007;7:68–73.
33. Shin DA, Kim SH, Han IB, Rhim SC, Kim HI. Factors influencing manometric pressure during pressure-controlled discography. *Spine (Phila Pa 1976)*. 2009;34:E790–E793.
34. Bogduk N. Provocation discography: lumbar disc stimulation. In: ISIS, ed. *Practice Guidelines for Spinal Diagnostic and Treatment Procedures*. Seattle, WA: International Spine Intervention Society; 2004:20–46.
35. Merskey H, Bogduk N. *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms*. 2nd ed. Seattle, WA: IASP Press; 1994.
36. Saboeiro GR. Lumbar discography. *Radiol Clin North Am*. 2009;47:421–433.
37. Derby R, Howard MW, Grant JM, Lettice JJ, Van Peteghem PK, Ryan DP. The ability of pressure-controlled

discography to predict surgical and nonsurgical outcomes. *Spine*. 1999;24:364–371; discussion 71–72.

38. Koes BW, van Tulder MW, Peul WC. Diagnosis and treatment of sciatica. *BMJ*. 2007;334:1313–1317.

39. Luijsterburg PA, Verhagen AP, Ostelo RW, et al. Physical therapy plus general practitioners' care versus general practitioners' care alone for sciatica: a randomised clinical trial with a 12-month follow-up. *Eur Spine J*. 2008;17:509–517.

40. Luijsterburg PA, Verhagen AP, Ostelo RW, van Os TA, Peul WC, Koes BW. Effectiveness of conservative treatments for the lumbosacral radicular syndrome: a systematic review. *Eur Spine J*. 2007;16:881–899.

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

42. Simmons JW, McMillin JN, Emery SF, Kimmich SJ. Intradiscal steroids. A prospective double-blind clinical trial. *Spine*. 1992;17:S172–S175.

43. Khot A, Bowditch M, Powell J, Sharp D. The use of intradiscal steroid therapy for lumbar spinal discogenic pain: a randomized controlled trial. *Spine*. 2004;29:833–836; discussion 7.

44. Muzin S, Isaac Z, Walker J 3rd. The role of intradiscal steroids in the treatment of discogenic low back pain. *Curr Rev Musculoskelet Med*. 2008;1:103–107.

45. Klein RG, Eek BC, O'Neill CW, Elin C, Mooney V, Derby RR. Biochemical injection treatment for discogenic low back pain: a pilot study. *Spine J*. 2003;3:220–226.

46. Derby R, Eek B, Lee SH, Seo KS, Kim BJ. Comparison of intradiscal restorative injections and intradiscal electrothermal treatment (IDET) in the treatment of low back pain. *Pain Physician*. 2004;7:63–66.

47. Saal JA, Saal JS. Intradiscal electrothermal treatment for chronic discogenic low back pain: a prospective outcome study with minimum 1-year follow-up. *Spine*. 2000;25:2622–2627.

48. Freeman BJ. IDET: a critical appraisal of the evidence. *Eur Spine J*. 2006;15(suppl 3):S448–S457.

49. Urrutia G, Kovacs F, Nishishinya MB, Olabe J. Percutaneous thermocoagulation intradiscal techniques for discogenic low back pain. *Spine*. 2007;32:1146–1154.

50. Pauza KJ, Howell S, Dreyfuss P, Pelozo JH, Dawson K, Bogduk N. A randomized, placebo-controlled trial of intradiscal electrothermal therapy for the treatment of discogenic low back pain. *Spine J*. 2004;4:27–35.

51. Appleby D, Andersson G, Totta M. Meta-analysis of the efficacy and safety of intradiscal electrothermal therapy (IDET). *Pain Med*. 2006;7:308–316.

52. Kapural L, Mekhail N, Hicks D, et al. Histological changes and temperature distribution studies of a novel bipolar radiofrequency heating system in degenerated and nondegenerated human cadaver lumbar discs. *Pain Med*. 2008;9:68–75.

53. Pauza K. Cadaveric intervertebral disc temperature mapping during disc biacuplasty. *Pain Physician*. 2008;11:669–676.

54. Kapural L, Nageeb F, Kapural M, Cata JP, Narouze S, Mekhail N. Cooled radiofrequency system for the treatment of chronic pain from sacroiliitis: the first case-series. *Pain Pract*. 2008;8:348–354.

55. Barendse GA, van Den Berg SG, Kessels AH, Weber WE, van Kleef M. Randomized controlled trial of percutaneous intradiscal radiofrequency thermocoagulation for chronic discogenic back pain: lack of effect from a 90-second 70 C lesion. *Spine*. 2001;26:287–292.

56. Ercelen O, Bulutcu E, Oktenoglu T, et al. Radiofrequency lesioning using two different time modalities for the treatment of lumbar discogenic pain: a randomized trial. *Spine*. 2003;28:1922–1927.

57. Erdine S, Yucel A, Celik M. Percutaneous annuloplasty in the treatment of discogenic pain: retrospective evaluation of one year follow-up. *Agriculture* 2004;16:41–47.

58. Finch PM, Price LM, Drummond PD. Radiofrequency heating of painful annular disruptions: one-year outcomes. *J Spinal Disord Tech*. 2005;18:6–13.

59. Kvarstein G, Mawe L, Indahl A, et al. A randomized double-blind controlled trial of intra-annular radiofrequency thermal disc therapy—a 12-month follow-up. *Pain*. 2009;145:279–286.

60. van Kleef M, Kessels AG. Underpowered clinical trials: time for a change. *Pain*. 2009;145:265–266.

61. Kapural L, Hayek S, Malak O, Arrigain S, Mekhail N. Intradiscal thermal annuloplasty versus intradiscal radiofrequency ablation for the treatment of discogenic pain: a prospective matched control trial. *Pain Med*. 2005;6:425–431.

62. Ohtori S, Takahashi K, Chiba T, Yamagata M, Sameda H, Moriya H. Sensory innervation of the dorsal portion of the lumbar intervertebral discs in rats. *Spine*. 2001;26:946–950.

63. Sluijter ME. Radiofrequency lesions of the communicating ramus in the treatment of low back pain. In: Raj PP, ed. *Current Management of Pain*. Philadelphia: Kluwer Academic publishers; 1989:145–159.

64. Chandler G, Dalley G, Hemmer J, Jr, Seely T. Gray ramus communicans nerve block: novel treatment approach for painful osteoporotic vertebral compression fracture. *South Med J*. 2001;94:387–393.

65. Gibson JN, Waddell G. Surgical interventions for lumbar disc prolapse: updated Cochrane Review. *Spine*. 2007;32:1735–1747.

66. Van Boxem K, Cheng J, Patijn J, van Kleef M, Lataster A, Mekhail N, Van Zundert J. 11. Lumbosacral radicular pain. *Pain Pract*. 2010;10:339–358.

67. Jensen TS, Albert HB, Soerensen JS, Manniche C, Leboeuf-Yde C. Natural course of disc morphology in patients

with sciatica: an MRI study using a standardized qualitative classification system. *Spine*. 2006;31:1605–1612; discussion 13.

68. Boswell MV, Trescot AM, Datta S, et al. Interventional techniques: evidence-based practice guidelines in the management of chronic spinal pain. *Pain Physician*. 2007;10:7–111.

69. Al-Zain F, Lemcke J, Killeen T, Meier U, Eisen-schenk A. Minimally invasive spinal surgery using nucleoplasty: a 1-year follow-up study. *Acta Neurochir (Wien)*. 2008;150:1257–1262.

70. Manchikanti L, Derby R, Benyamin RM, Helm S, Hirsch JA. A systematic review of mechanical lumbar disc decompression with nucleoplasty. *Pain Physician*. 2009;12:561–572.

71. Alo KM, Wright RE, Sutcliffe J, Brandt SA. Percutaneous lumbar discectomy: clinical response in an initial cohort of fifty consecutive patients with chronic radicular pain. *Pain Pract*. 2004;4:19–29.

72. Amoretti N, David P, Grimaud A, et al. Clinical follow-up of 50 patients treated by percutaneous lumbar discectomy. *Clin Imaging*. 2006;30:242–244.

73. Bocci V. Ozone as a bioregulator. Pharmacology and toxicology of ozonotherapy today. *J Biol Regul Homeost Agents*. 1996;10:31–53.

74. Buric J, Molino Lova R. Ozone chemonucleolysis in non-contained lumbar disc herniations: a pilot study with 12 months follow-up. *Acta Neurochir Suppl*. 2005;92:93–97.

75. Muto M, Andreula C, Leonardi M. Treatment of herniated lumbar disc by intradiscal and intraforaminal oxygen-ozone (O₂-O₃) injection. *J Neuroradiol*. 2004;31:183–189.

76. Gallucci M, Limbucci N, Zugaro L, et al. Sciatica: treatment with intradiscal and intraforaminal injections of steroid and oxygen-ozone versus steroid only. *Radiology*. 2007;242:907–913.

77. Bonetti M, Fontana A, Cotticelli B, Volta GD, Guindani M, Leonardi M. Intraforaminal O(2)-O(3) versus periradicular steroidal infiltrations in lower back pain: randomized controlled study. *AJNR Am J Neuroradiol*. 2005;26:996–1000.

78. Sharma SK, Jones JO, Zeballos PP, Irwin SA, Martin TW. The prevention of discitis during discography. *Spine J*. 2009;9:936–943.