

CLINICAL PRACTICE

Persistent Low Back Pain

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This Journal feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.

A 49-year-old maintenance worker with a history of depression and previous reports of minor back pain is seen after four months of continuing low back pain. He has remained out of work for fear of worsening the injury. Magnetic resonance imaging (MRI) two weeks after the onset of pain showed only mild degenerative changes in the lumbar region without spinal stenosis or disk collapse or extrusion. How should this patient be evaluated and treated?

THE CLINICAL PROBLEM

Low back pain without sciatica, stenosis, or severe spinal deformity is common, with a reported point prevalence as high as 33 percent¹ and a one-year prevalence as high as 73 percent.² In physically active adults not seeking medical attention, the annual incidence of clinically significant low back pain (pain level, 4 or more on a 10-point scale) with functional impairment is approximately 10 to 15 percent.³ Acute low back pain (lasting three to six weeks) usually resolves in several weeks, although recurrences are common and low-grade symptoms are often present years after an initial episode. Serious or persistent disability is uncommon even among those with low back pain lasting more than three months.² Risk factors for the development of disabling chronic or persistent low back pain (variously defined as lasting more than three months or more than six months) include preexisting psychological distress, disputed compensation issues, other types of chronic pain, and job dissatisfaction.⁴⁻⁷ However, even among patients with one or more of these factors, only 6 percent were out of work for more than one week during a five-year period.⁷

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STRATEGIES AND EVIDENCE

EVALUATION

The history and physical examination are helpful mainly in identifying risk factors for delayed recovery that may have a psychosocial basis or identifying signs of serious underlying diseases (such as fracture, tumor, infection, or deformity) that require specific treatment. Back pain associated with predominant sciatica (manifested by more radicular pain in the legs than back pain) or neurogenic claudication requires a different therapeutic approach and must be distinguished from low back pain alone. This article focuses on disabling and persistent low back pain without prominent sciatica.

IMAGING

Imaging studies in the great majority of persons with low back pain reveal nonspecific findings but no serious pathology. Case series of patients referred with chronic disabling low back pain have shown that disk degeneration,⁸ annular disruption,⁸⁻¹⁰ and end-plate changes¹¹ have been associated with the severity of pain (Fig. 1). However, these findings are also common in cross-sectional studies of asymptomatic sub-

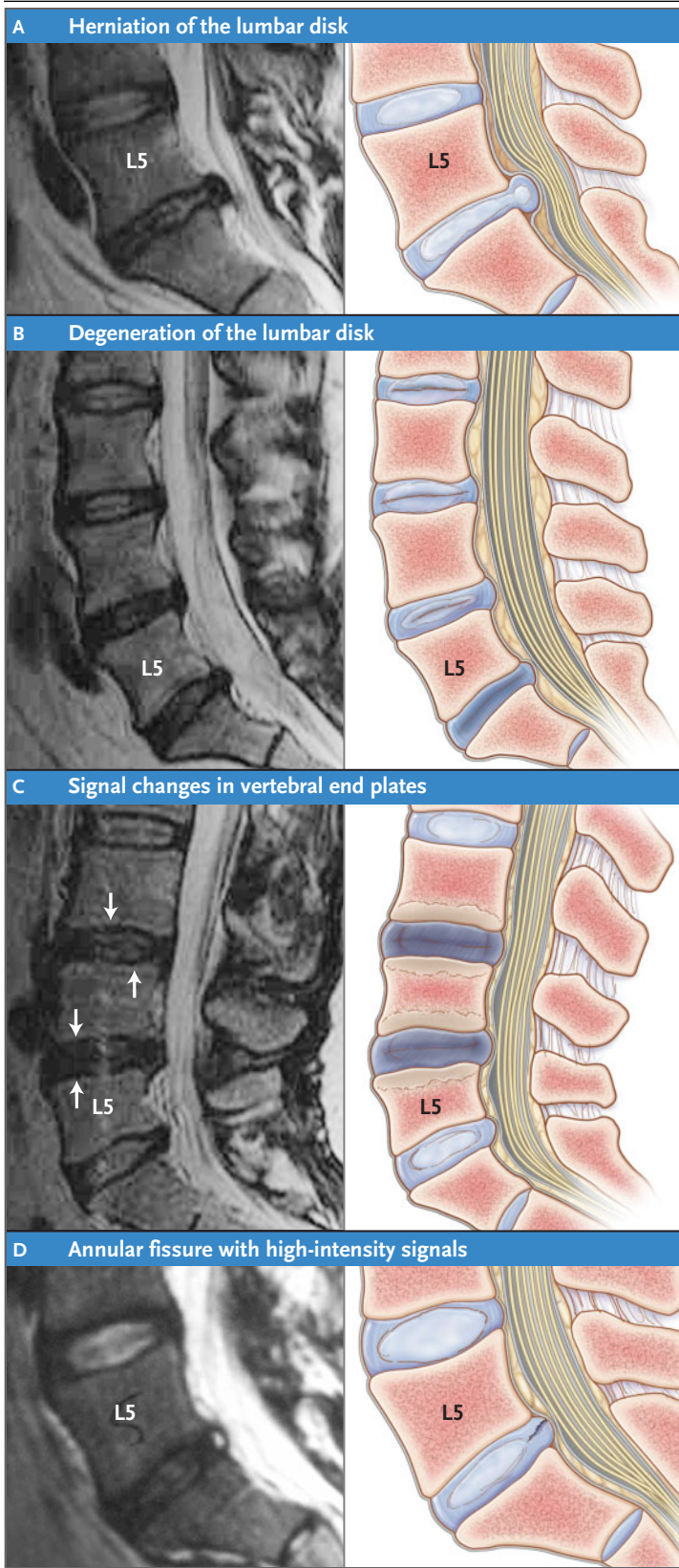


Figure 1. Images of the Spine from Normal Volunteers.

Herniation of the lumbar disk, as shown in Panel A, is found in 25 to 50 percent of asymptomatic subjects; extrusion of the disk material is found in 1 to 18 percent. Degeneration of the lumbar disk, shown in Panel B, increases with age and is found in 25 to 70 percent of asymptomatic subjects. Signal changes in the vertebral end plates (Panel C, arrows) are found in 10 percent of asymptomatic subjects; severe changes are found less frequently. Panel D shows a disk with a bright signal in the annular fissure. This represents degenerative changes that are found in 14 to 33 percent of asymptomatic subjects. Despite the high prevalence in healthy persons, these findings are often described as causing serious low back pain and are treated with spinal fusion.

jects.^{10,12,13} Furthermore, in prospective studies of subjects with no or trivial low back pain who underwent MRI, neither baseline MRI findings nor changes over time were useful predictors of the subsequent development of low back pain.^{6,7,14,15}

MRI or radiography early in the course of an episode of low back pain do not improve clinical outcomes or reduce costs of care.¹⁶ MRI is best used to rule out the possibility of impending neurologic injury, infection, or tumors. Appropriate candidates for MRI include patients with low back pain who have associated neurologic symptoms or signs; associated systemic symptoms; risk factors for cancer, infection, or occult fractures; or persistent pain in the absence of neurologic signs or symptoms after four to eight weeks. Patients should understand that the reason for imaging is to rule out these serious conditions, and that common degenerative findings are expected. Ill-considered attempts to make a diagnosis on the basis of imaging studies may reinforce the suspicion of serious disease, magnify the importance of nonspecific findings, and label patients with spurious diagnoses.

OTHER DIAGNOSTIC TECHNIQUES

Among patients with persistent disabling low back pain, there are no characteristic findings on physical examination or standard imaging. Therefore, attempts have been made to use provocative injections and anesthetic blockade to identify a hypothetical primary symptomatic structure (“pain generator”). One test used by some clinicians to direct invasive therapy is provocative diskography, which involves injecting dye into an intervertebral disk. Proponents of the test suggest that if injection into a disk reproduces a patient’s usual low back pain,

then that disk must be the cause of the patient's pain. However, injection into a disk can simulate the quality and location of pain known not to originate from that disk.¹⁷ Furthermore, disk injections are painful 30 to 80 percent of the time for patients who do not have symptomatic disk disease but who have had previous disk surgery or who have psychological distress, remote chronic pain, or disputed compensation claims.^{18,19} A controlled study comparing outcomes of spinal fusion when diskography was or was not used in the preoperative evaluation showed no differences between groups.²⁰

PSYCHOSOCIAL FACTORS

Psychosocial factors strongly predict future disability and the use of health care services for low back pain. Chronic disabling low back pain develops more frequently in patients who, at the initial evaluation for low back pain, have a high level of "fear avoidance" (an exaggerated fear of pain leading to avoidance of beneficial activities), psychological distress, disputed compensation claims, involvement in a tort-compensation system, or job dissatisfaction.^{5-7,21,22} These psychosocial factors are particularly prevalent in persons with low back pain for whom imaging shows only degenerative changes; 70 to 80 percent of such patients demonstrate psychological distress on psychometric testing or have disputed compensation issues, compared with 20 to 30 percent of patients whose imaging studies reveal definite pathologic or destructive processes.^{23,24} These psychosocial factors should be routinely assessed in patients with low back pain and taken into account in decisions regarding treatment.

TREATMENT

There is little consensus in practice about how to manage persistent disabling low back pain for which the only structural findings are nonspecific. Some clinicians have focused on the identification and treatment of an occult local "pain generator," assuming there is specific pathology in the spine that accounts for the magnitude of symptoms. However, since the same findings on imaging studies in severely symptomatic patients are commonly seen in minimally symptomatic persons, it has been suggested that psychosocial factors and factors affecting pain tolerance influence the degree of illness in patients with persistent disabling low back pain. In this approach, treatment and prevention are directed at restoring function and supporting adaptive

techniques, as opposed to medically or surgically treating the common spinal changes.

PHARMACOLOGIC THERAPY

Pharmacologic treatment of chronic low back pain usually includes analgesics, antiinflammatory drugs, and muscle relaxants, but the evidence for their efficacy is not compelling. In randomized trials, the differences in pain after a patient has taken nonsteroidal antiinflammatory agents as compared with placebo have generally been in the minimally detectable range.²⁵ For example, in a four-week trial involving patients with a flare of chronic back pain,^{25,26} pain scores (on a 100-point scale) decreased from 75 to 35 with valdecoxib, and to 45 with placebo. These marginal improvements do not warrant the long-term use of cyclooxygenase-2 inhibitors for patients with chronic back pain, particularly given the new data about increased cardiovascular risk associated with their use.²⁷⁻²⁹ Another short-term trial, with 30 patients, showed that diflunisal (Dolobid, 500 mg twice daily) was more effective in reducing chronic back pain than was acetaminophen (1000 mg four times daily), but interpretation is limited by the small sample and the recognition that there is often spontaneous variation in levels of back pain.²⁵

Muscle relaxants may also alleviate pain only moderately. In a pooled analysis of two randomized trials involving 222 subjects, treatment with tetrazepam (50 mg three times daily for 14 days) resulted in a statistically significant but clinically marginal reduction in pain intensity as compared with placebo.³⁰ Similar results are reported with other classes of muscle relaxants (such as cyclobenzaprine), and no particular class has proved superior. Long-term treatment with narcotics or sedatives is generally discouraged, given the associated risks of tolerance and side effects.^{25,30}

Antidepressant drugs, specifically tricyclic and tetracyclic drugs, have demonstrated small but consistent benefits in pain reduction in randomized trials in patients with chronic low back pain without clinical depression (a 20 to 40 percent greater reduction in pain than with placebo, during a period of four to eight weeks).³¹ However, there were no consistent or substantial functional improvements, and side effects occurred in more than 20 percent of subjects (Table 1).⁴⁷ Selective serotonin-reuptake inhibitors and trazodone have not been more effective than placebo in patients with chronic low back pain.³¹

Table 1. Common Therapies for Patients with Chronic Low Back Pain.

Treatment	Outcome
Tricyclic and tetracyclic antidepressants: nortriptyline (25 to 100 mg), amitriptyline (50 to 150 mg), maprotiline (50 to 150 mg), and others*	Good evidence of decreased levels of pain and decreased use of analgesics ³¹
Massage: one or two treatments a week for 5 to 10 weeks	Moderately decreased levels of pain and improvement in function as compared with sham treatment Decreased pain but no improvement in function as compared with exercise Some improvement for up to 1 year after a full 10-week course of massage ³²
Exercise: strengthening, stretching, passive end-range motion treatments, and others	Conflicting evidence, but possible improvement in ability to perform daily activities and in work tolerance as compared with usual care by general practitioners ³³
Manipulation	Moderate improvement in pain (10 points on a 100-point pain scale) as compared with sham treatment No clear superiority over physical therapy, medications, or care by general practitioners ^{34,35} When combined with trunk stabilization and isometric exercises for the torso, marginal improvements as compared with physician consultation in reducing pain, increasing function, and decreasing symptoms of depression
Combined physical training and cognitive behavioral approach	Strong evidence of a decreased amount of sick leave and an improvement in function over 12 months in patients with chronic disabling low back pain Questionable effect on pain reduction per se ^{36,37}
Multidisciplinary programs combining medical, psychological, and rehabilitative components	Strong evidence of improved function and moderate evidence of pain improvement with intensive and comprehensive programs ³⁸ No positive effect with less intensive programs (less than 100 hours)
Corticosteroid injections into or neuroablation of the facet joints of the spine	Uncertain efficacy of corticosteroid injections Moderate evidence to support the efficacy of precise denervation of the facet joints in a very small subgroup of patients with a clear placebo-controlled response to anesthetic injections ³⁹⁻⁴¹
Spinal-fusion surgery	Possible efficacy in patients with isolated one- or two-level spondylosis and few or no coexisting factors for chronic pain (e.g., disputed compensation issues, psychological distress, or other types of chronic pain) ^{42,43} No better results in patients with multiple coexisting factors than aggressive nonoperative management Outcomes that rarely meet the expectations of the patients when coexisting factors are present ^{6,19,43-46}

* Side effects of these antidepressants at dosages of 50 to 150 mg per day include blurred vision, dizziness, dry mouth, tremor, and urinary retention. Trazodone and selective serotonin-reuptake inhibitors have not been effective in trials of patients with chronic low back pain.

NONPHARMACOLOGIC THERAPY

Exercise seems to increase the rate of return to normal activities in patients with persistent low back pain. A Cochrane review of randomized trials of various exercises for persistent low back pain, including strengthening, general stretching, the McKenzie method of passive end-range stretching exercises, and conventional physical therapy (consisting of hot packs, massage, and stretching, flexibility, and coordination exercises), showed that these strategies appeared equivalent and seemed to be more effective than the usual care by a general practitioner.³³

In general, exercise programs, such as two or

three one-hour sessions a week until normal activities are resumed, in four to six weeks, have moderate effects — 10 to 15 points on a 100-point pain scale, or a 5 to 10 percent improvement on scales that assess functional disability, as compared with placebo or usual care.^{33,36} Similarly, randomized trials and systematic reviews have not shown a clear advantage of any particular treatment method over another, including physical therapy, exercise, massage, manipulation by chiropractors or other practitioners of manual medicine, low-impact aerobics, reconditioning on training machines, or back school (classroom-style educational programs for patients with back pain) (Table 1).^{32,34,48,49}

Available data suggest that a combination of medical care with either physical therapy⁴⁹ or manipulation³⁵ may be moderately more effective in reducing pain and self-rated disability than is a single method of treatment. The difference may reflect the patient's confidence in the treatment prescribed. In a trial comparing chiropractic care with medical care with and without physical therapy, the patient's initial confidence in the assigned treatment correlated directly with outcome, whereas the treatment assignment per se did not.^{49,50} At the other extreme, patients with persistent pain should avoid rest or confinement to bed.⁵¹

Given the marginal effect on functional outcomes of most of these interventions when used alone, and given the evidence that psychosocial factors may be important obstacles to recovery, more comprehensive approaches have been developed. Functional restoration programs incorporate physical therapy and medical treatment strategies with a cognitive behavioral approach that focuses on achieving specific functional goals (e.g., certain walking distances and speeds or certain weights lifted and numbers of repetitions). These programs, as compared with usual care by a general practitioner, also seem to decrease the amount of sick leave taken.^{37,38,44,45,52}

However, none of these methods of rehabilitation have consistently been shown to have generalized applicability (partly because of compliance issues in distressed patients), and it is unknown if effects are sustained for the long term.²⁸ Neither less intensive rehabilitation programs, especially those not accompanied by a strong component of behavioral therapy, nor pain-management programs relying on spinal injections and analgesic drugs seem to offer clear advantages over usual care for improving functional outcomes.³⁸

INJECTIONS AND NEUROABLATION PROCEDURES

Invasive treatments and surgery account for a high proportion of expenditures among patients with chronic low back pain, although there are enormous variations in usage according to geographic region. Despite their widespread use, these techniques have not been shown to be particularly effective among patients with chronic low back pain.

In randomized trials, injections of glucocorticoids or anesthetic agents into the epidural space, lumbar disks, lumbar facets, and trigger points have not improved outcomes in patients who have chronic low back pain without radiculopathy,⁵³⁻⁵⁵ nor

have injections of sclerosing agents into the lumbar fascia.^{56,57} Radiofrequency ablation of the small nerves to the facet joints was ineffective in one randomized trial³⁹ and showed a moderate effect (6 percent improvement in disability scores), which lasted only four weeks, in another.⁴⁰ Although data are insufficient, some authors have suggested a possible benefit of neuroablation of the facet joint in the extremely small subgroup of patients with chronic low back pain who respond to placebo-controlled anesthetic blocks.^{41,58}

Percutaneous treatments directed at altering the internal mechanics or innervation of the disk by heat (intradiskal electrothermal treatment) or radiofrequency energy have been used, but data supporting their use are lacking. Recent randomized trials have shown either no effect^{59,60} or a benefit in only a small proportion of highly selected subjects.⁶¹

SURGERY

The role for the surgical treatment of persistent disabling low back pain remains controversial.⁶² Laminectomy is generally not performed in the absence of radiculopathy or cauda equina syndrome. Excision of a prolapsed disk is sometimes performed, but there are no controlled studies to support the use of this technique.

The most common surgical treatment for persistent low back pain with degenerative changes is spinal fusion. A small randomized trial (64 patients) compared spinal-fusion surgery with an aggressive rehabilitation program. The rehabilitation program used a cognitive behavioral approach among patients with chronic back pain and degenerative changes on imaging. The study showed no differences between groups at one year in back pain, function, use of medication, work status, or general satisfaction. After one year, 22 percent of patients in the fusion group returned to work, as compared with 33 percent of those in the rehabilitation group.⁴⁵ A larger trial (294 patients)⁴² showed a greater decrease in the level of back pain and a greater improvement in function after two years among patients who had spinal-fusion surgery as compared with those assigned to an unstructured physical therapy program (excellent outcomes in 16 percent vs. 2 percent, respectively). The study showed no clear benefit of fusion five years after surgery.

The likelihood that spinal-fusion surgery will be beneficial for common degenerative changes may be improved by selecting patients without coexisting psychosocial disorders (including serious psy-

chological distress or disputed compensation issues) or other chronic pain, and those with severe degenerative changes (for example, severe disk collapse).^{10,17,42} However, even for these patients, the results of spinal fusion (excellent results in 30 to 50 percent) are inferior to results of the same surgery for definitive pathologic conditions (such as unstable spondylolisthesis).⁴³

The goals and expectations of the patient should be addressed when making decisions about treatment.⁶³ Educating patients regarding the limitations and risks of various treatment options is particularly important for more invasive treatments, for which expectations are often unrealistic. In a study of patients awaiting spinal fusion for presumed diskogenic pain, more than 90 percent indicated that an acceptable outcome would include, at a minimum, a return to some gainful employment, discontinuation of narcotic medications, and a high level of physical functioning.⁴⁶ Such results are uncommon after surgery in patients with persistent disabling back pain.^{42-45,60,61}

AREAS OF UNCERTAINTY

Attempts to arrest or reverse disk degeneration with the use of biologic factors and gene-transfer technology are under investigation and have unknown efficacy. Similarly, the efficacy of injection or surgical implantation of materials to augment disk function or mechanical stability is unproven, although such techniques are being performed. An unblinded study of disk replacement as compared with spinal fusion showed similar outcomes at 6 months of follow-up, as well as at 12 months.⁶⁴ Longer follow-up is needed to assess the failure rates of these techniques over time, and only a small minority of patients may be appropriate candidates.⁶⁵ An Australian study indicated that a television campaign advising people with back pain to stay active and keep working reduced work-injury claims and medical expenses.⁶⁶ This suggests that public health initiatives may help prevent episodes of low back pain from becoming chronic and disabling; more research is warranted.

GUIDELINES

International guidelines for the treatment of chronic low back pain consistently encourage patients to

become active early and gradually; they also encourage consideration of psychosocial factors as risk factors for chronicity.⁶⁷ These guidelines, and the North American Spine Society guidelines for unremitting low back pain,⁶⁸ do not recommend any one pharmacologic or nonpharmacologic method of treatment over another. The guidelines of the North American Spine Society suggest that surgery be considered only after a two-to-four-month trial of nonoperative measures and only when there are objective findings of structural defects, although the guidelines do not specify whether common degenerative findings constitute sufficient defects. Guidelines of the Washington State Medical Association, which are frequently used, recommend consideration of fusion only for demonstrable instability, deformity, or neurologic injury.

CONCLUSIONS AND RECOMMENDATIONS

The patient described in the vignette should be reassured that no serious disease was detected on MRI and that he is not in danger of a serious neurologic injury. He should be guided to pharmacologic treatment that has limited side effects and begin an aggressive, three-to-six-week rehabilitation program with primary functional and behavioral goals. He should understand that maintaining fitness through exercise is important and should be ongoing after resolution of this episode.

On the basis of trials supporting the use of tricyclic antidepressants, I would prescribe an agent such as amitriptyline, starting at 25 to 50 mg at bedtime, and increasing the dosage as needed. If the patient has a strong preference for certain additional short-term treatments, such as spinal manipulation or massage, it is reasonable to accommodate him, especially since evidence supports better outcomes for patients who feel confident about the treatment prescribed. The patient should understand that the primary goal of treatment is to maximize function, and that some ongoing or recurrent back pain is likely but not dangerous. Accommodations should be made to resume work as soon as possible, even at a low level. In the absence of severe spinal disease or radiculopathy, surgery should generally be avoided.

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REFERENCES

1. Skovron ML, Szpalski M, Nordin M, Melot C, Cukier D. Sociocultural factors and back pain: a population-based study in Belgian adults. *Spine* 1994;19:129-37.
2. Cassidy JD, Carroll LJ, Cote P. The Saskatchewan Health and Back Pain Survey: the prevalence of low back pain and related disability in Saskatchewan adults. *Spine* 1998;23:1860-66.
3. Carragee E, Cohen S. Reliability of LBP history in asymptomatic subjects? The prevalence and incidence of reported back pain correlates with surveillance frequency. In: *Proceedings of the 19th Annual Meeting of the North American Spine Society*, Chicago, October 26-30, 2004:216. abstract.
4. Bigos SJ, Battie MC, Spengler DM, et al. A prospective study of work perceptions and psychosocial factors affecting the report of back injury. *Spine* 1991;16:1-6. [Erratum, *Spine* 1991;16:688.]
5. Burton AK, Tillotson KM, Main CJ, Hollis S. Psychosocial predictors of outcome in acute and subacute low back trouble. *Spine* 1995;20:722-8.
6. Carragee EJ, Alamin TF, Miller JL, Carragee JM. Discographic, MRI and psychosocial determinants of low back pain disability and remission: a prospective study in subjects with benign persistent back pain. *Spine* 2005;5:24-35.
7. Boos N, Semmer N, Elfering A, et al. Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging: predictors of low back pain-related medical consultation and work incapacity. *Spine* 2000;25:1484-92.
8. 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;23:1252-8.
9. Aprill C, Bogduk N. High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. *Br J Radiol* 1992;65:361-9.
10. Carragee EJ, Paragioudakis SJ, Khurana S. 2000 Volvo Award winner in clinical studies: lumbar high-intensity zone and discography in subjects without low back problems. *Spine* 2000;25:2987-92.
11. Weishaupt D, Zanetti M, Hodler J, et al. Painful lumbar disk derangement: relevance of endplate abnormalities at MR imaging. *Radiology* 2001;218:420-7.
12. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects: a prospective investigation. *J Bone Joint Surg Am* 1990;72:403-8.
13. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med* 1994;331:69-73.
14. Borenstein DG, O'Mara JW Jr, Boden SD, et al. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects: a seven-year follow-up study. *J Bone Joint Surg Am* 2001;83:1306-11.
15. Jarvik JJM, Hollingworth W, Heagerty P, Haynor DR, Deyo RA. The Longitudinal Assessment of Imaging and Disability of the Back (LAIDBack) Study: baseline data. *Spine* 2001;26:1158-66.
16. 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;231:343-51.
17. Carragee EJ, Tanner CM, Yang B, Brito JL, Truong T. False-positive findings on lumbar discography: reliability of subjective concordance assessment during provocative disc injection. *Spine* 1999;24:2542-7.
18. Carragee EJ, Chen Y, Tanner CM, Truong T, Lau E, Brito JL. Provocative discography in patients after limited lumbar discectomy: a controlled, randomized study of pain response in symptomatic and asymptomatic subjects. *Spine* 2000;25:3065-71.
19. Carragee EJ, Tanner CM, Khurana S, et al. The rates of false-positive lumbar discography in select patients without low back symptoms. *Spine* 2000;25:1373-80.
20. Madan S, Gundanna M, Harley JM, Boeree NR, Sampson M. Does provocative discography screening of discogenic back pain improve surgical outcome? *J Spinal Disord Tech* 2002;15:245-51.
21. Cassidy JD, Carroll L, Cote P, Berglund A, Nygren A. Low back pain after traffic collisions: a population-based cohort study. *Spine* 2003;28:1002-9.
22. Hurwitz EL, Morgenstern H, Yu F. Cross-sectional and longitudinal associations of low-back pain and related disability with psychological distress among patients enrolled in the UCLA Low-Back Pain Study. *J Clin Epidemiol* 2003;56:463-71.
23. Cairns MC, Foster NE, Wright CC, Pennington D. Level of distress in a recurrent low back pain population referred for physical therapy. *Spine* 2003;28:953-9.
24. Carragee EJ. Psychological and functional profiles in select subjects with low back pain. *Spine* 2001;26:198-204.
25. van Tulder MW, Scholten RJ, Koes BW, Deyo RA. Nonsteroidal anti-inflammatory drugs for low back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine* 2000;25:2501-13.
26. Coats TL, Borenstein DG, Nangia NK, Brown MT. Effects of valdecoxib in the treatment of chronic low back pain: results of a randomized, placebo-controlled trial. *Clin Ther* 2004;26:1249-60.
27. Nussmeier NA, Whelton AA, Brown MT, et al. Complications of the COX-2 inhibitors parecoxib and valdecoxib after cardiac surgery. *N Engl J Med* 2005;352:1081-91.
28. Ostelo R, Tulder M, Vlaeyen J, Linton S, Morley S, Assendelft W. Behavioural treatment for chronic low-back pain. *Cochrane Database Syst Rev* 2005;1:CD002014.
29. Solomon SD, McMurray JVV, Pfeffer MA, et al. Cardiovascular risk associated with celecoxib in a clinical trial for colorectal adenoma prevention. *N Engl J Med* 2005;352:1071-80.
30. van Tulder MW, Touray T, Furlan AD, Solway S, Bouter LM. Muscle relaxants for non-specific low back pain. *Cochrane Database Syst Rev* 2003;2:CD004252.
31. Staiger TO, Gaster B, Sullivan MD, Deyo RA. Systematic review of antidepressants in the treatment of chronic low back pain. *Spine* 2003;28:2540-5.
32. Cherkin DC, Sherman KJ, Deyo RA, Shekelle PG. A review of the evidence for the effectiveness, safety, and cost of acupuncture, massage therapy, and spinal manipulation for back pain. *Ann Intern Med* 2003;138:898-906.
33. van Tulder MW, Malmivaara A, Esmail R, Koes BW. Exercise therapy for low back pain. *Cochrane Database Syst Rev* 2000;2:CD000335.
34. Assendelft WJ, Morton SC, Yu EI, Suttortp MJ, Shekelle PG. Spinal manipulative therapy for low back pain: a meta-analysis of effectiveness relative to other therapies. *Ann Intern Med* 2003;138:871-81.
35. Niemisto L, Lahtinen-Suopanki T, Rissanen P, Lindgren KA, Sarna S, Hurri H. A randomized trial of combined manipulation, stabilizing exercises, and physician consultation compared to physician consultation alone for chronic low back pain. *Spine* 2003;28:2185-91.
36. Staal JB, Hlobil H, Twisk JW, Smid T, Koke AJ, van Mechelen W. Graded activity for low back pain in occupational health care: a randomized, controlled trial. *Ann Intern Med* 2004;140:77-84.
37. Schonstein E, Kenny DT, Keating J, Koes BW. Work conditioning, work hardening and functional restoration for workers with back and neck pain. *Cochrane Database Syst Rev* 2003;1:CD001822.
38. Guzman J, Esmail R, Karjalainen K, Malmivaara A, Irvin E, Bombardier C. Multidisciplinary bio-psycho-social rehabilitation for chronic low back pain. *Cochrane Database Syst Rev* 2002;1:CD000963.
39. Leclaire R, Fortin L, Lambert R, Bergeron YM, Rossignol M. Radiofrequency facet joint denervation in the treatment of low back pain: a placebo-controlled clinical trial to assess efficacy. *Spine* 2001;26:1411-6.
40. van Kleef M, Barendse GA, Kessels A, Voets HM, Weber WE, de Lange S. Randomized trial of radiofrequency lumbar facet denervation for chronic low back pain. *Spine* 1999;24:1937-42.
41. Dreyfuss P, Halbrot B, Pauz K, Joshi A, McLarty J, Bogduk N. Efficacy and validity of radiofrequency neurotomy for chronic

- lumbar zygapophysial joint pain. *Spine* 2000;25:1270-7.
42. Fritzell P, Hagg O, Wessberg P, Nordwall A. 2001 Volvo Award winner in clinical studies: lumbar fusion versus nonsurgical treatment for chronic low back pain: a multicenter randomized controlled trial from the Swedish Lumbar Spine Study Group. *Spine* 2001;26:2521-32.
43. Carragee E, Lincoln T, Parmar V, Alamin T, Carragee J. A gold standard evaluation of the "discogenic pain" diagnosis as determined by provocative discography. *Spine* (in press).
44. Fairbank J, Frost H, MacDonald J, Rivero-Arias O, Campbell H, Gray A. The MRC Spine Stabilization Trial: a randomized controlled trial comparing surgical stabilization of the lumbar spine with intensive rehabilitation in patients with chronic low back pain. In: Proceedings of the 31st Annual Meeting of the International Society for the Study of the Lumbar Spine, Porto, Portugal, May 31-June 5, 2004:38. abstract.
45. Ivar Brox J, Sorensen R, Friis A, et al. Randomized clinical trial of lumbar instrumented fusion and cognitive intervention and exercises in patients with chronic low back pain and disc degeneration. *Spine* 2003;28:1913-21.
46. Carragee E, Alamin T. A prospective assessment of patient expectations and satisfaction in spinal fusion surgery. In: Proceedings of the 30th Annual Meeting of the International Society for the Study of the Lumbar Spine, Vancouver, B.C., Canada, May 13-17, 2003. abstract.
47. Salerno SM, Browning R, Jackson JL. The effect of antidepressant treatment on chronic back pain: a meta-analysis. *Arch Intern Med* 2002;162:19-24.
48. Mannion AF, Muntener M, Taimela S, Dvorak J. A randomized clinical trial of three active therapies for chronic low back pain. *Spine* 1999;24:2435-48.
49. Hurwitz EL, Morgenstern H, Harber P, et al. A randomized trial of medical care with and without physical therapy and chiropractic care with and without physical modalities for patients with low back pain: 6-month follow-up outcomes from the UCLA low back pain study. *Spine* 2002;27:2193-204.
50. Goldstein MS, Morgenstern H, Hurwitz EL, Yu F. The impact of treatment confidence on pain and related disability among patients with low-back pain: results from the University of California, Los Angeles, low-back pain study. *Spine J* 2002;2:391-9.
51. Deyo RA, Diehl AK, Rosenthal M. How many days of bed rest for acute low back pain? A randomized clinical trial. *N Engl J Med* 1986;315:1064-70.
52. van Tulder MW, Ostelo R, Vlaeyen JW, Linton SJ, Morley SJ, Assendelft WJ. Behavioral treatment for chronic low back pain: a systematic review within the framework of the Cochrane Back Review Group. *Spine* 2001;26:270-81.
53. Carette S, Marcoux S, Truchon R, et al. A controlled trial of corticosteroid injections into facet joints for chronic low back pain. *N Engl J Med* 1991;325:1002-7.
54. Nelemans PJ, deBie RA, deVet HC, Sturmans F. Injection therapy for subacute and chronic benign low back pain. *Spine* 2001;26:501-15.
55. 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;29:833-6.
56. Dechow E, Davies RK, Carr AJ, Thompson PW. A randomized, double-blind, placebo-controlled trial of sclerosing injections in patients with chronic low back pain. *Rheumatology (Oxford)* 1999;38:1255-9.
57. Yelland MJ, Glasziou PP, Bogduk N, Schluter PJ, McKernon M. Prolotherapy injections, saline injections, and exercises for chronic low-back pain: a randomized trial. *Spine* 2004;29:9-16.
58. van Kleef M, Weber WE, Kessels A, Dreyfuss P, Pauza K, Bogduk N. Re: Efficacy and validity of radiofrequency neurotomy for chronic lumbar zygapophysial joint pain (*Spine* 2000;25:1270-7). *Spine* 2001;26:E163-4.
59. 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;26:287-92.
60. Freeman BJ, Fraser RD, Cain CM, Hall D. A randomized double-blind controlled efficacy study: Intradiscal Electrothermal Therapy (IDET) versus placebo. *J Bone Joint Surg Br Proc* 2004;86:484-5.
61. 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.
62. Deyo RA, Nachemson A, Mirza SK. Spinal-fusion surgery — the case for restraint. *N Engl J Med* 2004;350:722-6.
63. Weinstein JN. The missing piece: embracing shared decision making to reform health care. *Spine* 2000;25:1-4.
64. Zigler JE. Clinical results with ProDisc: European experience and U.S. investigation device exemption study. *Spine* 2003;28:S163-S166.
65. Huang RC, Lim MR, Girardi FP, Cammisa FP Jr. The prevalence of contraindications to total disc replacement in a cohort of lumbar surgical patients. *Spine* 2004;29:2538-41.
66. Buchbinder R, Jolley D. Population based intervention to change back pain beliefs: three year follow up population survey. *BMJ* 2004;328:321.
67. Koes BW, van Tulder MW, Ostelo R, Kim Burton A, Waddell G. Clinical guidelines for the management of low back pain in primary care: an international comparison. *Spine* 2001;26:2504-13.
68. Wong DA, Mayer T, Watters W, et al. Unremitting low back pain: North American Spine Society Phase III Clinical Guidelines for multidisciplinary spine care specialists. La Grange, Ill.: North American Spine Society, 2000:96.

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