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ORIGINAL ARTICLE

Dutch Multidisciplinary Guideline for Invasive Treatment of Pain Syndromes of the Lumbosacral Spine

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■ **Abstract**

Objectives: When conservative therapies such as pain medication or exercise therapy fail, invasive treatment may be indicated for patients with lumbosacral spinal pain. The Dutch Society of Anesthesiologists, in collaboration with the Dutch Orthopedic Association and the Dutch Neurosurgical Society, has taken the initiative to develop the guideline "Spinal low back pain," which describes the evidence regarding diagnostics and invasive treatment of the most common spinal low back pain syndromes, that is, facet joint pain, sacroiliac joint pain, coccygodynia, pain originating from the intervertebral disk, and failed back surgery syndrome.

Methods: The aim of the guideline is to determine which invasive treatment intervention is preferred for each included pain syndrome when conservative treatment has failed. Diagnostic studies were evaluated using the EBRO criteria, and studies on therapies were evaluated with the Grading of Recommendations Assessment, Development and Evaluation system. For the evaluation of invasive treatment options, the guideline committee decided that the outcome measures of pain, function, and quality of life were most important.

Results: The definition, epidemiology, pathophysiological mechanism, diagnostics, and recommendations for invasive therapy for each of the spinal back pain syndromes are reported.

Discussion: The guideline committee concluded that the categorization of low back pain into merely specific or nonspecific gives insufficient insight into the low back pain problem and does not adequately reflect which therapy is effective for the underlying disorder of a pain syndrome. Based on the guideline "Spinal low back pain," facet joint pain, pain of the sacroiliac joint, and disk pain will be part of a planned nationwide cost-effectiveness study. ■

Key Words: low back pain, mechanical, dorsal root, epidural, evidence-based medicine, facet joint, multidisciplinary pain centers, radiofrequency ablation, spinal cord stimulation, guidelines

INTRODUCTION

Low back pain is a widespread problem with major social and economic impact. About 85% to 90% of the patients with low back pain suffer from what is (until now) described as "nonspecific" low back pain; this is

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defined as low back pain not attributable to an identifiable, acknowledged specific pathology, such as an infection, tumor, osteoporosis, or fracture.[1]

Current guidelines on nonspecific low back pain generally assume that spontaneous recovery occurs in the majority of these patients. However, a systematic review (2012) has shown that spontaneous recovery from nonspecific low back pain during the first 3 months after onset occurs in only about one-third of the patients; the majority still experiences pain 1 year after onset.[2] In practice, a proportion of these patients is generally referred to a pain clinic where some are diagnosed with, for example, facet joint pain, sacroiliac joint (SIJ) pain, coccygodynia, discogenic pain, and failed back surgery syndrome (FBSS). If indicated, invasive treatment is applied.

There is no consensus among practitioners and policymakers about the place of this kind of diagnosis. In the current guidelines on nonspecific low back pain, such diagnoses are usually classified as "nonspecific low back pain" and treatment is limited to reassurance, analgesics, and activation/mobilization.[3]

However, pain specialists claim that these diagnoses should not be classified as nonspecific but rather as "specific." It is suggested that better identification of these patients in an earlier phase and, if indicated, the use of invasive treatment would improve the prognosis of those patients. [4]

The Dutch Society of Anesthesiologists felt a strong need to bring clarity to this field. In collaboration with the Dutch Orthopedic Association and the Dutch Neurosurgical Society, they developed a multidisciplinary clinical guideline to deal with this topic. This guideline describes the evidence with regard to the diagnostics and effectiveness of the invasive treatment of 5 spinal low back pain syndromes, that is, (1) facet joint pain, (2) SIJ pain, (3) coccygodynia, (4) discogenic pain, and (5) FBSS. This guideline is available only in Dutch. The choice of topics and the interventions described in this guideline are based on those commonly used in daily clinical practice. The guideline aims to provide answers to clinically relevant problems. The main purpose of the guideline is to determine the evidence of invasive treatment when conservative treatment has failed. Because there is no consensus about the place of the 5 above-mentioned pain syndromes, the guideline pays special attention to the definition, epidemiology, underlying pathophysiology, and validity of the diagnosis, as well as to the effectiveness of invasive treatment of these 5 spinal low back pain syndromes. [5]

The task force proposes to classify spinal low back pain syndromes into (1) "uncomplicated and complicated" degenerative pain syndromes and (2) nondegenerative pain syndromes (Figure 1).

The guideline discussed here focuses on the degenerative uncomplicated spinal low back pain syndromes (Figure 1).

The diagnosis and treatment of the degenerative complicated and nondegenerative spinal low back pain syndromes will be reviewed in separate guidelines, which are currently being developed. The diagnosis and therapy of the lumbosacral radicular pain syndrome has been reviewed in a guideline developed earlier.[6]

To our knowledge, this is the first guideline on spinal low back pain which makes use of the Grading of Recommendations Assessment, Development and Evaluation (GRADE) method. This new method of assessment is gaining popularity in guideline development. An important difference compared with earlier assessment methods is that, instead of focusing on the study design, the GRADE method focuses on assessment of the strength of evidence for prior defined, relevant outcome measures. This brings the GRADE method more in line with actual clinical practice.

The aim of this article was to provide an English summary of the main findings of the Dutch guideline for invasive treatment of degenerative uncomplicated pain syndromes of the lumbosacral spine (http:/www.anesthesiologie.nl/richtlijnen: in Dutch).

METHODS

Task Force

A multidisciplinary task force was set up in 2009 to develop the guideline. The task force comprised representatives of specialties related to the diagnostics and clinical decision-making process of spinal low back pain syndromes amenable for invasive treatment, that is, anesthesiology (pain medicine), orthopedics, and neurosurgery. All members of the task force are acknowledged experts and key players in the clinical and scientific field of low back pain; no member of the task force had anything to disclose in relation to the development of this guideline. A focus group of 8 patients was also involved in the development of the guideline. Methodological support was provided by epidemiologists from the Quality of Healthcare Center of the Dutch Association of Medical Specialists.

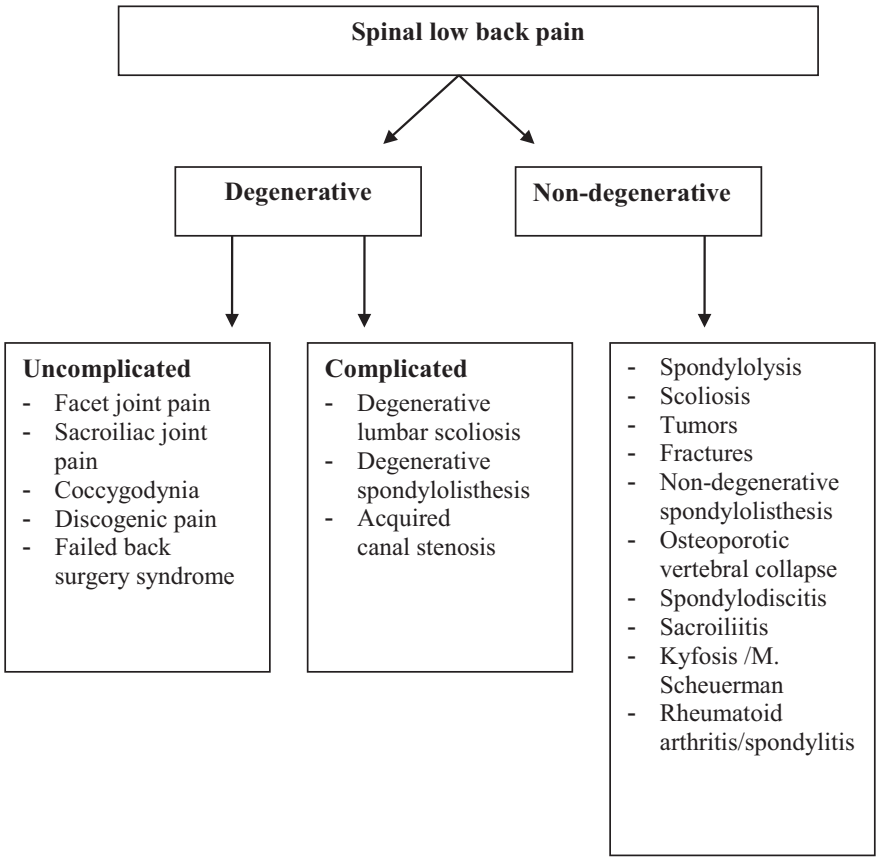

Figure 1. Proposal for a new classification system for "Spinal low back pain."

Primary Clinical Question

The primary clinical question in the guideline is as follows: *Which invasive treatment intervention is preferred if conservative treatment has failed?*

Outcome Measures

During the preparation phase, the relevant outcomes were inventoried and arranged according to the sequence of importance for the patient. For evaluation of the invasive treatment options, the task force decided that the outcome measures of "pain," "functionality," and "quality of life" were the most important. For clinically relevant differences in pain and functionality, use was made of the values as proposed by Ostelo et al. [7] (Table 1).

Table 1. Threshold Values for Clinically Relevant Differences in Pain and Functionality for Patients with Low Back Pain

Questionnaire* (Range)	Absolute Threshold	Relative Threshold with Regard to Baseline Value, %
VAS (0 to 100)	15	30
NRS (0 to 10)	2	30
RDQ (0 to 24)	5	30
ODI (0 to 100)	10	30
QBPDQ (0 to 100)	20	30

*The different classification system for spinal low back pain.
VAS, visual analog scale; NRS, Numerical Rating Scale; RDQ, Roland Morris Disability Questionnaire; ODI, Oswestry Disability Index; QBPDQ, Quebec Back Pain Disability Scale.

Systematic Literature Review

Specific English and Dutch search terms for each diagnosis included in this guideline were used to identify relevant studies (published between 1990 and June 2011) in MEDLINE (OVID) and in EMBASE (Embase.com). In addition, a manual research was made in the reference lists of the identified papers. Initially, the search strategy aimed to identify (systematic reviews or meta-analyses of) double-blind randomized sham-controlled trials (RCTs). If absent, an additional search was made for prospective controlled studies, comparative studies, and prospective noncomparative studies. For the search strategies and evidence tables, see the Guideline literature site of the Erasmus Medical Center (www.erasmusmc.nl/pijn/guidelineliterature).

Primary assessment of the identified literature was performed by at least 2 members of the task force, whereas the results were discussed by the entire task force. For 3 pain syndromes (SIJ pain, coccygodynia, and FBSS), no articles remained after applying our inclusion and exclusion selection procedure [8] www.anesthesiologie.nl/richtlijnen).

Selection criteria were as follows: (1) studies describing a patient population with complaints of low back pain persisting for more than 3 months for which conservative therapy (TENS, activation mobilization) was not effective; (2) a relevant minimal follow-up of at least 3 months; (3) a minimal study population of 15 patients (RCT 2 × 15 patients); (4) no prior surgery; (5) patient selection based on a test treatment with at least 50% reduction in complaints; and (6) in RCTs, the control group is sham or placebo.

Evaluation of Therapeutic Intervention Studies

Intervention studies were evaluated using the GRADE method (http://www.gradeworkinggroup.org/).

A combination of the evaluated studies is used to determine the level of the burden of proof for each outcome measure. This evaluation determines the evidence level as represented in the classification shown in Table 2.

The GRADE method has 4 evidence levels: high, moderate, low, and very low. The study design determines the starting level of the strength of evidence, that is, systematic literature analyses of RCTs start high and systematic literature analyses of observational studies start low. Five factors (limitations of study design, inconsistency, indirectness, imprecision, and publication bias) can downgrade the strength of evidence by 1 or 2 levels; the guideline committee decided on the relative importance of each of these respective factors.

In addition, 3 factors can upgrade the burden of proof of a systematic literature analysis of an observational study, that is, large effect, dose–response relationship, and confounding that underestimates the actual effect or overestimates an actual nonexisting effect.

For each investigated diagnosis (ie, facet joint pain, SIJ pain, coccygodynia, discogenic pain, and FBSS), the guideline gives a description of the definition, epidemiology, pathophysiology, validity of the diagnosis, and the evidence for invasive therapy when conservative treatment has failed. Based on the validity of the diagnosis and the evidence of the therapy, the task force describes considerations to be taken into account to answer the primary clinical question and arrives at a recommendation for clinical practice.[9] If there is no, and/or conflicting, or not enough evidence to give a clear recommendation, the task force recommends to perform the treatment in a study-related way. When no literature is available, or case reports are available but insufficient to indicate the effectiveness or safety to give a clear recommendation for practice, the task force recommends that this treatment should be considered and *preferably be administered in a study-related way*. When there is not enough and/or conflicting evidence, and benefits are clearly balanced with risks and burdens, to give a clear recommendation for practice, the task force recommends this treatment should be used *only study-related* (Table 3).

Table 2. The Grading of Recommendations Assessment, Development and Evaluation Categorization of the Quality of Studies for Each Outcome Measure

Quality	Study Design	Quality Downgrade	Quality Upgrade
High (4)	RCT	*1. Study limitations*	*1. Large effect*
Moderate (3)		−1 severe	+1 large
Low (2)	Observational comparative study	−2 very severe	+2 very large
	(eg, patient control study, cohort study)	*2. Inconsistency*	*2. Dose–response relationship*
Very low (1)	Nonsystematic clinical observation	−1 severe	+1 evidence for relationship
	(eg, case series or case reports)	−2 very severe	*3. Plausible confounding*
		3. Indirectness	+1 would underestimate the effect
		−1 severe	+1 would overestimate the effect when
		−2 very severe	no effect was shown
		4. Imprecision	
		−1 severe	
		−2 very severe	
		5. Publication bias	
		−1 probable	
		−2 very probable	

NB: randomized controlled trials (RCTs) start "high" (4), observational studies start "low" (2).
For RCTs: for example, total 1-point downgrade: then from high (4) to moderate (3); for RCTs: for example, 2-point downgrade: then from high (4) to low (2); for RCTs: in total ≥ 3-point downgrade: then from high (4) to very low (1).
For observational studies: for example, 1-point upgrade: then from low (2) to moderate (3).

This recommendation (ie, study-related) implies that there is always systematic registration of the patient's characteristics, diagnostic process, treatment (including details of the technique involved), evaluation of the results, and registration of any adverse effects and/or complications.

Facet Joint Pain

Definition. Facet joint pain is defined as pain that originates from every structure that comprises the facet joints, including the fibrous capsule, synovial membrane, hyaline articular cartilage, and bone.[10]

Epidemiology. From the studies in which facet joint pain was carefully selected by means of controlled diagnostic blocks, the prevalence of facet joint pain in a group of patients with lumbar back pain referred to a pain specialist was estimated at 10% to 30%.[11–14]

Pathophysiology. Degenerative processes in the spinal column are the main cause of facet joint pain.[15,16] Degenerative inflammation fills the facet joint with fluid causing the joint to swell until it reaches the joint capsule, thereby causing pain. Degenerative disk disease and degenerative spondylolisthesis are predisposing factors.[17]

Diagnosis. Unilateral or bilateral back pain is the most prevalent symptom.[18] Pain originating from the upper lumbar facet joints radiates toward the flanks, hips, and lateral upper legs. Pain from the lower lumbar facet joints radiates toward the posterior upper legs. Pain distal from the knee is rarely an indication for facet joint pathology.[18] The pain can be triggered or aggravated by unilateral pressure on the facet joint or the transverse process. Extension, lateroflexion, or rotation toward the ipsilateral side causes pain. There is unilateral muscular hypertonia in the area of the affected facet joint. There is a limitation of local unilateral movement or increased stiffness on the side of the facet pain. Flexion relieves the pain.

When causes such as fracture, tumor, or infection have been excluded and imaging technology is inconclusive, then arthritis, gout, arthrosis, and a differential diagnosis of synovitis should also be considered in case of facet pathology.[19] However, there are no signs or symptoms which are pathognomonic for the diagnosis. Using the Delphi technique, a test nerve block was seen as the most decisive step to confirm the diagnosis.[20] Diagnostic or prognostic nerve blocks can be performed by administering a small volume of local anesthetic at the medial branch of the dorsal ramus or intra-articular. The injections are performed under X-ray guidance.[21–23] Cohen et al.[24] reported on the value of the facet test blockade. The number needed to treat (NNT) without a test blockade is 3, with 1 test blockade 2.3, and with 2 test blockades 1.28. Other studies report a NNT of 1.9 without a test blockade, 1.6 with 1 test blockade,[12] and 1.1 with 2 test blockades.[25]

Clearly, 2 test blockades result in a greater specificity and a lower NNT; however, from a pragmatic

Table 3. Summary of Evidence Scores and Implications for Clinical Practice

Score	Description	Implication
I	Effectiveness demonstrated in various RCTs. The benefits clearly outweigh risk and burdens. One RCT or more RCTs with methodological weakness demonstrate effectiveness. The benefits clearly outweigh risk and burdens. One RCT or more RCTs with methodological weakness demonstrate effectiveness. The benefits are clearly balanced with risks and burdens.	Positive implication for practice
II	Multiple RCTs with methodological weakness yield contradictory results better or worse than the control treatment. The benefits are clearly balanced with risk and burdens, or uncertainty in the estimates of benefits and risks and burdens. Effectiveness only demonstrated in observational studies. Given that there is no conclusive evidence of the effect, the benefits are closely balanced with risk and burdens.	Considered, preferably study-related
III	No literature is available, or case reports are insufficient to indicate the effectiveness and/or safety. These treatments should only be applied in relation to research.	Only study-related
IV	Observational studies indicate no or too short-lived effectiveness. Given there is no positive clinical effect, the risks and burdens outweigh the benefits. One or more RCTs with methodological weakness, or large observational studies that do not indicate any superiority to the control treatment. Given there is no positive clinical effect, the risks and burdens outweigh the benefits. RCT of a good quality which does not exhibit any clinical effect. Given there is no positive clinical effect, the risks and burdens outweigh the benefits.	Negative implication for practice

RCT: randomized controlled trial (Van Boxem et al.[157]).

point of view, the task force advises to use 1 test blockade. Dreyfus et al.[26] studied the most ideal position of the needle tip during a test blockade; they compared an end position of the needle at the upper edge of the processes articularis and the ligamentum mammiloaccesorius. This latter end position gives the lowest spread of local anesthetic to the segmental nerves if 0.5 mL local anesthetic is used; the task force advises the use of this end position. Based on the expert opinion, the task force also advises to continuously evaluate the test blockade for 30 minutes. The use of MRI or CT is of no additional value in this process [15,27–36] (Table 4).

Invasive Treatment: Implications for Practice

Of the several invasive pain treatments available for facet joint pain, we investigated (1) radiofrequency (RF) lesion, (2) intra-articular corticosteroid injection, (3) pulsed radiofrequency lesion, and (4) surgery.

In the search for the effectiveness of invasive treatment in facet joint pain, we identified 34 papers. After the selection procedure, 9 papers fulfilled the inclusion criteria and were used to formulate the scientific conclusion. Details on the search strategy used are described in the Guideline literature site of the Erasmus Medical Center (www.erasmusmc.nl/pijn/guidelineliterature).

Radiofrequency lesion

After the selection procedure, 4 RCTs fulfilled the selection criteria [12,37–39] to formulate a scientific conclusion:

- It is plausible that RF treatment of the ramus dorsalis in patients with facet joint pain has a favorable effect on pain for 3 to 12 months.[12,37–39]
- There is evidence that RF lesion of the ramus dorsalis in patients with facet pain has a beneficial effect on functionality for 3 to 6 months.[12,39]

The results of these 4 studies cannot be pooled due to differences in the methods of reporting. All show a significant reduction in pain in the treatment group compared with the sham group. The methodological quality of the RCTs showed the following limitations: In all 4 studies, the power is low, the evidence for functionality is low, and none of the studies validated the outcome "quality of life."

Based on these considerations, the task force developed the following *positive implication for practice* (because effectiveness is demonstrated in various RCTs and the benefits clearly outweigh the risks and burdens):

- In facet joint pain, if conservative therapy has failed, radiofrequency lesion of the innervating medial branches of the rami dorsalis of the affected segmental nerves can be performed.[12,37–39]

Intra-articular Corticosteroid Injection

After the selection procedure, 3 [40–42] studies fulfilled the inclusion criteria to formulate a scientific conclusion:

- It is concluded that there is conflicting evidence regarding the efficacy of intra-articular injections with corticosteroids on pain and there is no beneficial effect on functionality.[40,42]
- The evidence that intra-articular injections are beneficial for the outcome parameter pain is low.

The RCTs of Manchikanti et al. [40,41] show no significant difference between the intervention and control group. Carette et al. [42] reported a significant difference in pain between the group receiving an intra-articular injection of prednisolone and a group receiving physiological salt. However, the power of the studies is low and the evidence for the outcome parameter "functionality" is moderate. Only Manchikanti et al.[40] studied functionality and found no significant difference between the studied groups. In severe facet arthrosis, it is generally not possible to place a needle intra-articularly.

Based on the scientific conclusion and these additional considerations, the task force developed the following *negative implication for practice* (because there is no positive clinical effect or risk and the burdens outweigh the benefits).

- Patients with facet pain who have insufficient result from conservative therapy should not be treated with intra-articular corticosteroid injections.

Pulsed Radiofrequency Lesion

After the selection procedure, 1 paper fulfilled the inclusion criteria to formulate a scientific conclusion. There is an increasing use of pulsed radiofrequency

Table 4. Evidence Score for Grading Quality of Intervention Studies for the Diagnoses Facet Pain and Discogenic Pain

Diagnosis	Interventions	Outcome Measure	Number of Studies	Design	Limitations	Inconsistent	Indirect	Imprecise	*n* = Intervention	*n* = Control group	Final Quality
Facet Pain	Corticosteroid Injections	Pain	3	RCT	No serious side effects	Not serious inconsistent	Not serious indirect	Serious imprecise	172/334	162/334	Low
		Function	3	RCT	No serious side effects	Not serious inconsistent	Not serious indirect	Serious imprecise	131/261	130/261	Moderate
	Radio Freq. Lesions	Pain	4	RCT	No serious side effects	Not serious inconsistent	Not serious indirect	Serious imprecise	73/141	68/141	Moderate
		Function	3	RCT	No serious side effects	Not serious inconsistent	Serious indirect	Serious imprecise	55/91	56/91	Low
	Pulsed Radio Freq. Lesions	Pain	2	RCT	No serious side effects	Not serious inconsistent	Not serious indirect	Serious imprecise	33/66	33/66	Moderate
		Function	2	RCT	No serious side effects	Not serious inconsistent	Not serious indirect	Serious imprecise	13/26	13/26	Moderate
Discogenic Pain	Corticosteroid and Meth. Blue Injections	Pain	3	RCT	No serious side effects	Serious inconsistent	Not serious indirect	Serious imprecise	96/194	98/194	Low
		Function	2	RCT	No serious side effects	Serious inconsistent	Not serious indirect	Serious imprecise	82/169	87/169	Low
	Intradiscal electrothermal therapy	Pain	2	RCT/comparative study	No serious side effects	Serious inconsistent	Not serious indirect	Serious imprecise	88/207	119/207	Very low
		Function	2	RCT/comparative study	No serious side effects	Serious inconsistent	Not serious indirect	Serious imprecise	81/145	64/145	Very low
		Quality of life	2	RCT	No serious side effects	Serious inconsistent	Not serious indirect	Serious imprecise	70/113	43/113	Very low

The corresponding references are: *Facet injections:* Carette et al.[42], Manchikanti et al.[41], Manchikanti et al. [40]. *Radio Frequency and Facet pain:* Gallagher et al.[37], Nath et al.[38], Van Kleef et al.[12], Tekin et al.[39]. *Pulsed RF and Facet pain:* Tekin et al.[39], Kroll et al.[43]. *Discogenic pain and injections:* Khot et al.[134], Oh and Shim[136], Peng et al. [128]. *Discogenic pain and IDET:* Derby et al.[133], Pauza et al.[122], Freeman et al.[137]
Key components of quality of evidence: *Limitations* of the study are those characteristics of design or methodology that impacted or influenced the application or interpretation of the results of your study. *Consistency* refers to the degree of similarity in the effect sizes of different studies within an evidence base. If effect sizes indicate the same direction of effect and if the range of effect sizes is narrow, an evidence base can be judged to be consistent. *Directness* concerns whether the evidence being assessed reflects a single, direct link between the interventions of interest and the ultimate health outcome under consideration (whether a benefit or harm). *Precision* is the degree of certainty surrounding an estimate of effect with respect to a specific outcome.

lesion (PRF) in the treatment of the dorsal root ganglia (cervical) and several peripheral nerves. Two RCTs compared RF lesion with PRF lesion.[39,43] One RCT [43] is only included in the "other considerations without a scientific conclusion" to enable the task force to formulate the implications for clinical practice; the other RCT [39] is included with a "scientific conclusion" and in the "other considerations" in order to formulate the implications for clinical practice. Both RCTs show that patients with facet joint pain derive more benefit from RF lesion of the ramus dorsalis than from PRF lesion.[39,43]

Based on the scientific conclusion and these other considerations, the task force developed the following *negative implication for practice* (because there is no positive clinical effect, or risks and burdens outweigh the benefits)

- PRF has no place in the treatment of lumbar facet pain.

Surgery

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. Also, in the remaining considerations, the task force found no additional evidence for surgical interventions in facet joint pain.[44]

Based on the lack of a scientific conclusion and these other considerations, the task force developed the following *negative implication for practice* (because there is no positive clinical effect, or risks and burdens outweigh the benefits).

- Surgical interventions are not indicated in the treatment of facet joint pain.

Pain in the Sacroiliac Joint

Definition. Pain in the SIJ is defined as a pain localized in the area of the SIJ that can be provoked/elicited by stress tests and by provocation tests of the SIJ and that is completely relieved after infiltration of the SIJ with a local anesthetic.[45] The SIJ is a diarthrodial synovial joint and is primarily innervated by the sacral dorsal rami S1-3.[46,47]

Epidemiology. Together with the facet joint, the SIJ is one of the most important sources of mechanical low back pain. The prevalence of SIJ pain is reported to range from 16% to 30%.[48–50] In about 35% of the cases, there is no known cause for the pain.

Pathophysiology. There are intra-articular and extra-articular causes of SIJ pain. When the etiology is intra-articular, there may be an infection, inflammation, or malignancy. When the cause is extra-articular, the pain is probably related to anatomical structures: enthesopathy, fractures, ligament injury, or myogenic injury. The risk factors for this are trauma (abrupt rotation and axial strain, status after lumbosacral arthrodesis [≥ 30%]), postural defect [51] due to, for example, a discrepancy in leg length,[52] scoliosis,[53] pregnancy,[54] strenuous work,[55] and inflammation due to rheumatologic diseases.

Diagnosis. Diagnostics comprise the usual procedures: medical history, physical examination, and a diagnostic nerve block. Apart from ruling out red flags (tumor, inflammation, infection, fracture, anatomical anomalies), diagnostic examinations and imaging (such as MRI) have little added value for SIJ pain.[56] There is no correlation between findings on radiographs, CT, and bone scan with a positive outcome of a block.[57]

MRI has no added value in imaging normal anatomy, but can reveal early spondyloarthropathies and cartilage inflammation of the SIJ.[58,59]

Most patients report SIJ pain to be localized in the area of the buttock (94%). The pain can refer to the lower lumbar region/spine (72%), groin (14%), higher lumbar region (6%), and abdomen (2%). Referred pain toward the leg appears in 28% of the patients, of which 12% state they have pain radiating to the foot.[60] Standing up from a sitting position can provoke the pain.[61] On physical examination, a positive Fortin finger test is found, if the patient indicates with 1 finger that the pain is right in the middle and inferior to the posterior superior iliac crest. There are 6 provocation tests with, individually, low sensitivity and specificity, that is, the approximation test, gapping test, FABER test, pelvic torsion test, femoral shear test, and the Gillet's test.[62] With several positive stress tests (at least 3 of 5/6 provocation tests), the specificity and sensitivity are reported to be 80% and 85% to 94%, respectively.[61,63–66]

Young et al.[61] found a positive correlation between SIJ pain and the worsening of symptoms when getting up from sitting position, unilateral pain, and 3 positive provocation tests. As one of its diagnostic criteria, the International Association for the Study of Pain states that

SIJ pain has to disappear on intra-articular infiltration with local anesthesia. For this purpose, the use of single or double blockades, with a long- or short-acting local anesthetic, has been described.[41,46,48,50,60,63–65,67–69]

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion about the diagnostic value of a test block for SIJ. In the remaining considerations, a study was identified which describes a diagnostic nerve block under fluoroscopy to make the diagnosis more specific when there is a clinical suspicion of SIJ pain.[56] Based on these considerations, the task force developed the following *positive implication for practice* (because effectiveness is demonstrated in 1 or more RCTs and the benefits clearly outweigh risk and burdens).

- When there is a clinical suspicion of SIJ pain, it can be useful to perform a diagnostic nerve block under fluoroscopy to enable a more specific diagnosis.

Invasive Treatment: Implications for Practice

Of the several invasive pain treatments available for SIJ pain, we investigated (1) intra-articular corticosteroid injection, (2) (cooled) radiofrequency lesion, and (3) surgery.

In the search for the effectiveness of invasive treatment in SIJ pain, 21 papers were identified. After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. More details on the search strategy used are described in the Guideline literature site of the Erasmus Medical Center. (www.erasmusmc.nl/pijn/guidelineliterature). The task force developed its implications for practice based on the remaining considerations only.

Intra-articular Corticosteroid Injection

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. In the remaining considerations, 2 RCTs were identified. Both trials showed significant pain reduction after intra-articular corticosteroid injections, and both showed a low risk-benefit ratio. Based on the lack of a scientific conclusion and these other considerations, the task force developed the following implication for practice:

- Intra-articular corticosteroid injection can be applied for patients with SIJ pain who have insufficient or no effect from conservative therapy.

However, because larger studies are required to arrive at a more definite scientific conclusion, the implication for practice is to carry out this procedure *only study-related* (because no literature is available, or case reports are insufficient to indicate effectiveness or safety to give a clear recommendation for practice).[70,71]

(Cooled) Radiofrequency Lesion

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. In the remaining considerations, 1 RCT on radiofrequency treatment of SIJ pain was identified but failed to meet the inclusion criteria because the study group was too small.[72] In this latter trial, significant pain reduction was shown after RF lesion.

Based on the lack of a scientific conclusion and these other considerations, the task force developed the following implication for practice:

- If an intra-articular corticosteroid injection provides insufficient effect, treatment using cooled RF lesion or RF lesion can be considered.

However, because larger and longer-term studies are required to arrive at a more definite scientific conclusion, the implication for practice is to carry out those procedures *only study-related* (because no literature is available, or case reports are insufficient to indicate effectiveness or safety to give a clear recommendation for practice).[70–75]

Surgery

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. In the remaining considerations, small studies were identified which describe successful minimal invasive treatment for pain and function after treatment of a carefully selected group of patients for whom surgery was a last resort treatment.[76] In contrast, several studies were found with disappointing results.[77–80]

Based on the lack of a scientific conclusion and these other considerations, the task force developed the following *negative implication for practice* (because there is no positive clinical effect, or risks and burdens outweigh the benefits).

- Most surgical treatments are generally not advised for patients with SIJ pain.

Surgical treatment (if applicable) should be performed only after a comprehensive noninvasive and minimally invasive treatment and careful consideration. Preference should be given to minimally invasive surgical techniques.

Coccygodynia

Definition. Coccygodynia is pain in the area of the os coccygis.

Epidemiology. The female to male ratio is 5:1.[81] An increased body mass index of > 27.4 in women and > 29.4 in men is a risk factor for coccygodynia.[82]

Pathophysiology. The acute form of coccygodynia mainly appears after a trauma caused by a fall in the sitting position[82,83]; however, childbirth can also cause such a trauma.[30] Similarly, repetitive microtraumata caused by maintaining an unaligned sitting position or through sports (eg, bicycling or motorcycle sports) can also cause coccygodynia.[84,85] The coccygeal joints are involved in 70% of the traumatic cases. There is anterior luxation, hypermobility, coccygeal spicules, subluxation, or luxation.[83,84] The sacrum and os coccygis lie more posterior in women. Also, the os coccygis is longer in women than in men, placing women at increased risk of developing coccygodynia.[82,86–88]

Diagnosis. Patients usually report pain in the location of the coccyx, generally provoked by sitting. Activities such as bicycling are painful because of the direct pressure placed on the coccyx.[88] During physical examination, mobilization of the os coccygis can differentiate between nociceptive pain of the os coccygis with ligamentary and muscular structures, and referred pain by pathology in the lower pelvic area.[88] In coccygodynia, the Valsalva maneuver is positive in diseases based on neural structures and negative with primary involvement of the os coccygis itself.[88] For diagnostic imaging plain, lateral radiographs of the os coccygis are the first choice.[50,82,83] In relation to obesity, determination of the BMI is mandatory.[82] A test nerve block is not indicated. Differential diagnostics are the levator ani syndrome, osteomyelitis, arthritis, intra-ossal lipomata or chondromata, and vascular necrosis of the os coccygis.[89,90]

Invasive Treatment: Implications for Practice

In coccygodynia, the following recommendations are made with regard to invasive therapy when conservative therapy has failed: (1) corticosteroid injection, (2) RF lesion of the ganglion Impar, and (3) surgery.

In the search for the effectiveness of invasive treatment in coccygodynia pain, we identified 18 papers. After the selection procedure, 2 papers fulfilled the inclusion criteria to formulate a scientific conclusion. More details on the search strategy used are described in the Guideline literature site of the Erasmus Medical Center. (www.erasmusmc.nl/pijn/guidelineliterature). The task force developed their recommendation mainly upon the remaining considerations:

Corticosteroid Injection

After the selection procedure, only 1 of 7 papers fulfilled the inclusion criteria to formulate a scientific conclusion.[91] This article shows that the risks of infiltration are low, the procedure is a little stressful, and the effect on pain is relatively positive.

Based on the scientific conclusion and the other considerations, the task force developed the following implication for practice:

- In patients with coccygodynia for whom conservative therapy has provided insufficient results, a corticosteroid injection can be a treatment option.

This treatment should *preferably be carried out study-related* (because there is not enough and/or conflicting evidence and benefits clearly balanced with risk and burdens to give a clear recommendation for practice).[91]

Radiofrequency Lesion of the Ganglion Impar

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. In the remaining considerations, 1 prospective study was included. Reig et al.[92] show that a RF lesion of the ganglion Impar has a positive effect on pain. However, this trial is underpowered with too few patients and shows a relative risk of serious side effects. Based on the

lack of a scientific conclusion and these other considerations, the taskforce developed the following *negative implication for practice* (because there is no positive clinical effect, or risks and burdens outweigh the benefits):

Patients with coccygodynia who do not have sufficient effect of conservative therapy should not be treated with a RF lesion of the ganglion Impar.

Surgery

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. In the remaining considerations, 4 nonrandomized studies showed a moderate positive effect of surgical interventions on coccygodynia in patients with a clear anatomical anomaly.[93–96]

Based on the lack of a scientific conclusion and these other considerations, the task force developed the following implication for practice:

- When there is an evident anatomical anomaly of the os coccygis, surgical intervention can be a treatment option for patients with coccygodynia for whom conservative therapy and corticosteroid injection have provided insufficient results or have had no effect.

This treatment should *only* be carried out *study-related* (because no literature is available, or case reports are insufficient to indicate effectiveness or safety to give a clear recommendation for practice).[97]

Discogenic Pain

Definition. Discogenic low back pain is defined as pain that originates from any structure that comprises the discus intervertebralis, that is, the nucleus pulposus, the annulus fibrosus, the vertebral end plate, and the accompanying innervation.

Epidemiology. The prevalence of chronic discogenic low back pain in a population with back pain is reported to be 28% to 43%.[98–100] Discogenic back pain seems to be related to a certain degree of disk degeneration.[101] The prevalence of disk degeneration appears to cover the entire population; however, in some persons it develops at a markedly younger age and/or to a more severe degree. Genetic factors play an essential role and mechanical influence is relatively slight.[102–105] The effect of smoking as an accompanying harmful factor is well established.[106,107]

Pathophysiology. The intervertebral disk consists of a nucleus pulposus which is surrounded by a fiber-like structure called the annulus fibrosus. The healthy disk is avascular and for feeding dependent on diffusion through the annulus fibrosus and the vertebral endplates. The nerves of the disk comprise 2 extensive nerve plexuses, the anterior plexus and the posterior plexus. The sinuvertebral nerve is the largest branch of the posterior plexus; this plexus is a diffuse network where somatic and autonomic branches converge. The anterior plexus receives branches of the rami communicantes. In a healthy disk, the surrounding innervation is limited to the external third of the annulus.[108]

Fissures develop in the annulus fibrosus during disk degeneration. Disk degeneration is accompanied by an inflammatory response partially induced through the nucleus pulposus material.[109–112] If the fissures reach the outer third part of the annulus, neo-neurovascular ingrowth can develop from the nerve plexus described above. Nociceptive stimulation then develops through a combination of this neo-neurovascular ingrowth and the inflammatory chemical changes. Degeneration of the intervertebral disk is characterized by signs of inflammation and nerve growth and is seen as an important cause of discogenic low back pain.

Diagnosis. A period of acute low back pain is often an expression of discogenic pain. This period can coincide with the initial development of a tear in the annulus fibrosus. The pain is often located medially and centrally. Axial strain, such as with sitting or standing, usually exacerbates the pain, while lying down alleviates the pain. Strolling is very poorly tolerated, whereas normal walking is better tolerated. Discogenic pain emanating from the level of L3/L4 usually radiates to the anterior side of the upper leg. From the level L4/L5, pain radiates toward the lateral side of the upper leg and sometimes toward the posterior side of the upper leg. Pain emanating from the level L5/S1 usually causes pain on the anterior side of the upper leg along with localized pain located medially on the back. Pain in the groin is also often reported; this may be because the L2 nerve root not only innervates the groin, but is also the most important source of afferent pathways to the lumbosacral disk.[113–116]

The main clinical features of discogenic pain during a physical examination are the biphasic rise from flexion

(the corkscrew phenomenon) and pressure pain on the processus spinosus; however, these are not specific and are therefore only indicative. In 1994, Vanharanta described pain originating from the disk provoked by a vibrating tuning fork pressed on the processus spinosus of the affected segment.[117]

Diagnostic techniques, such as imaging of the lumbar spine (particularly with CT and MRI), can reveal anatomical defects and disk degeneration.[118,119] A limitation of these diagnostic techniques is that degenerative findings on imaging can also be observed in the asymptomatic population [120] and therefore cannot be proven to be the source of the patient's pain syndrome. Provocative discography is a physiological test that is considered to be the most specific diagnostic for discogenic back pain, as well as for determining the degree of disk degeneration.[100,121,122] Although controversial, the test is assumed to be able to identify the disk as pain source.

There are indications that a diagnostic blockade of the disk can play a role in the diagnosis of discogenic pain [123] (Table 5).

Provocative discography is recommended to substantiate the discogenic low back pain diagnosis after clinical suspicion of severe discogenic problems has been confirmed by diagnostic techniques (preferably MRI).[124–127]

Table 5. ISIS/IASP Criteria for Provocative Discography: From Bogduk et al.[163]

1. Absolute discogenic pain
Stimulation of target discus 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 2 adjacent disks is not painful
2. Highly probable discogenic pain
Stimulation of target discus 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 disks is not painful
3. Discogenic pain
Stimulation of target discus 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 2 adjacent disks is not painful
4. Possible discogenic pain
Stimulation of target discus 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 disks is not painful, and stimulation of another discus is painful at a pressure greater than 50 psi above the opening pressure, and the pain is discordant

Invasive Treatment: Implications for Practice

Of the several invasive treatments available for discogenic pain, we investigated (1) intradiscal injection of methylene blue, restorative solution, and corticosteroids; (2) intradiscal RF lesion, ramus communicans RF lesion, and intradiscal electrothermal therapy; and (3) surgery.

In the search for the effectiveness of invasive treatment in discogenic pain, we identified 23 papers. After the selection procedure, 10 studies fulfilled our inclusion criteria and were used to formulate the scientific conclusions. Details on the search strategy used are described in the Guideline literature site of the Erasmus Medical Center (www.erasmusmc.nl/pijn/guidelineliterature).

Intradiscal Injection of Methylene Blue

After the selection procedure, 1 of 4 papers fulfilled the inclusion criteria to formulate a scientific conclusion[128]: An intradiscal injection with methylene blue seems to have a positive effect on reduced pain and restorative function in patients with discogenic back pain. In a high-quality RCT, Peng et al. showed an extremely positive effect after intradiscal injection with methylene blue on pain and function in patients with discogenic back pain. Risk of complications is low but, if they do occur, they are serious (eg, discitis). The exact degeneration degree of the disk, in which a methylene blue injection is indicated, is not entirely clear. There is a risk of potential neurotoxicity of methylene blue.

Based on the scientific conclusion and other considerations,[129–131] the task force developed the following implication for practice:

- Patients with discogenic low back pain for whom conservative therapy has provided insufficient or no effect should be treated with intradiscal injection of methylene blue *only study-related* (because there no literature is available, or case reports are insufficient to indicate effectiveness or safety to give a clear recommendation for practice).

Intradiscal Injection of Restorative Solution

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion for intradiscal injections with a restorative solution. Some studies describe a relatively positive effect of intradiscal restorative solutions.[132,133]

Intradiscal injection therapy is a relatively inexpensive and low-impact surgical procedures. The risk of complications is low. However, complications can be serious (discitis). It is obvious to give no injections at grade 5 disk degeneration. Long-term effects of intradiscal punctures are unknown.

Based on the lack of a scientific conclusion and these other considerations, the task force developed the following implication for practice:

- Patients with discogenic low back pain for whom conservative therapy has provided insufficient or no effect can be treated with intradiscal injection of restorative solution *only study-related* (because no literature is available, or case reports are insufficient to indicate effectiveness or safety to give a clear recommendation for practice).

Intradiscal Injection of Corticosteroids

After the selection procedure, 1 of 5 papers fulfilled the inclusion criteria to formulate a scientific conclusion for intradiscal corticosteroid injections[134]: Patients with discogenic low back pain appeared to derive no benefit from treatment with intradiscal injections with corticosteroids.

There was no difference between intradiscal corticosteroid injection and a placebo injection.

Based on the scientific conclusion and other considerations, the task force developed this *negative implication for practice* (because there is no positive clinical effect, or risks and burdens outweigh the benefits):

- Patients with discogenic low back complaints should not be treated with an intradiscal injection of corticosteroids.

Intradiscal Radiofrequency Lesion

After the selection procedure, 1 of 2 papers fulfilled the inclusion criteria to formulate a scientific conclusion.[135] An intradiscal RF lesion seems to have no effect on pain reduction and function in patients with discogenic back pain. The absence of any treatment effect of an RF lesion of the discus is well explained by the large distance from the RF electrode to the nerve fibers for destruction.

Based on the scientific conclusion and these other considerations, the task force developed the following *negative implication for practice* (because there is no positive clinical effect, or risks and burdens outweigh the benefits):

- Patients with discogenic low back pain with insufficient effect of conservative treatment should not be treated with a RF lesion of the discus.

Ramus Communicans Radiofrequency Lesion

After the selection procedure, 1 paper fulfilled the inclusion criteria to formulate a scientific conclusion for treatment of intradiscal pain with a ramus communicans blockade.[136] A RF lesion of the ramus communicans seems to have a positive effect on pain reduction and restorative function. The ramus communicans is a minimally invasive procedure with a low risk of complications. The side effects are minimal and the costs are low.

Based on the scientific conclusion and these other considerations, the task force developed the following *positive implication for practice* (because effectiveness is demonstrated in various RCTs and the benefits clearly outweigh the risks and burdens):

- In patients with discogenic low back pain without a positive effect from conservative treatments, a RF lesion of the ramus may be considered.

Intradiscal Electrothermal Therapy

After the selection procedure, 2 of 7 papers fulfilled the inclusion criteria to formulate a scientific conclusion for intradiscal electrothermal therapy (IDET): IDET seems to have a cautiously positive effect on pain, function, and quality of life in patients with discogenic low back pain.[122,137] In other studies of lower quality, the benefit from IDET over a RF treatment of the disk or the disk surgery was demonstrated.

Based on the scientific conclusion and the other considerations, the task force developed the following implication for practice:

- For patients with discogenic low back pain for whom conservative therapy has provided insufficient effect or no effect, IDET may be considered.

This treatment should be used *only study-related* (because no literature is available, or case reports are insufficient to indicate effectiveness or safety to give a clear recommendation for practice).

Surgery

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion

for surgical interventions in discogenic pain. The assumption that movement of the affected segment worsens the pain (mechanical back pain) has led to the concept that immobilization or stabilization of a painful degenerative motion segment will reduce the pain. The immobilization is realized by means of posterior, anterior, or circumferential lumbar fusion.

Based on the literature, no overall judgment can be made regarding the efficacy of fusion vs. a conservative policy for discogenic low back pain.[138]

An etiological hypothesis would be to eliminate the source of pain (ie, the disk) while preserving motion, by means of placing a lumbar disk prosthesis (total disk replacement, TDR). Comparing TDR (with preserved motion) and lumbar fusion (stabilized interlumbar motion), no clear differences were found in clinical outcome and safety between TDR and lumbar fusion.[139,140]

Based on the lack of a scientific conclusion and these other considerations, the task force developed the following implication for practice:

- In patients with discogenic pain for whom conservative therapy has provided insufficient or no effect, a lumbar fusion or TDR can be a treatment option.

This treatment should *preferably be administered study-related* (because there is not enough and/or conflicting evidence, and benefits are clearly balanced with risks and burdens, to give a clear recommendation for practice) (see Table 4).

Failed Back Surgery Syndrome

Definition. Failed back surgery syndrome is a broadly defined diagnosis usually attributed to persistent or renewed pain after previous back surgery. The "failed" in the FBSS diagnosis does *not* refer to any potential failure of the surgeon, such as through an incorrect surgical indication, technique, or level. It refers to not achieving the intended objective of the spinal surgery, that is pain reduction, possibly due to the absence of a surgically treatable cause. Therefore, the term postsurgical pain syndrome would be more appropriate.[141] Usually, there has to be some form of nerve compression caused by degenerative defects, such as spinal disk herniation, benign stenosis, ligamentum flavum hypertrophy, or spondylolisthesis.

In this guideline, the term "FBSS" applies to patients who have undergone 1 or more lumbosacral operations, or who have found no reduction of pain or a comparable level of pain has returned within, for example, 1 year.

As a rule, the patient has no underlying problem that could successfully be treated surgically and which would further reduce the chance for success with repeated surgery.[141]

Epidemiology. Reported estimates of the incidence/prevalence vary drastically, ranging from anywhere up to 40% of the number of surgical patients.[142–145]

Pathophysiology. There are definite indications that certain pathophysiological factors play a role. The indications are categorized as such: residual or relapse HNP or recurrent disk herniation, nerve damage, spinal canal stenosis, postoperative infection, epidural fibrosis, and/or adhesive arachnoiditis. Also, there is convincing evidence that certain psychological and environmental factors, such as smoking, play a role in the risk for developing FBSS.[146–149]

Diagnosis. The patient's medical history and physical examination, including appropriate imaging (X-ray, CT, MRI), are the main cornerstones for diagnostics. FBSS is a diagnosis of exclusion, which is different from the other diagnoses that fall under the category of uncomplicated degenerative spinal pain. Consultation with a spinal surgeon is needed to rule out surgically treatable causes.

Invasive Treatment Recommendations

Of the several invasive pain treatments available for FBSS, we investigated (1) epiduroscopy and (2) spinal cord stimulation.

In the search for the effectiveness of invasive treatment in FBSS, we identified 22 papers. After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. More details on the search strategy are described in the Guideline literature site of the Erasmus Medical Center (www.erasmusmc.nl/pijn/guidelineliterature). The task force developed recommendations based on the remaining considerations.

Epiduroscopy

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. One RCT [150] of spinal endoscopic adhesiolysis in

chronic refractory low back and lower extremity pain showed significant pain relief and functional recovery remaining after some months. Serious complications are described due to pressure increase in the epidural space during the epiduroscopy.

Based on the lack of a scientific conclusion and on these other considerations, the task force developed the following recommendation:

- Epiduroscopy can be a treatment option for patients with FBSS for whom conservative therapy has provided insufficient or no effect.

This treatment option is *preferably administered study-related* (because there is not enough and/or conflicting evidence, and benefits are clearly balanced with risk and burdens, to give a clear recommendation for practice).

Spinal Cord Stimulation

After the selection procedure, no papers fulfilled the inclusion criteria to formulate a scientific conclusion. No placebo-controlled studies can be performed with the recently available stimulation paradigms. Two prospective randomized comparative trials clearly show a positive effect of spinal cord stimulation on leg pain in FBSS.[143,151]

Based on the lack of a scientific conclusion and these other considerations, the task force developed the following *positive recommendation for practice* (because effectiveness is demonstrated in various RCTs, and the benefits clearly outweigh the risks and burdens):

- Neuromodulation is recommended for patients with FBSS who have pronounced leg pain and for whom conservative therapy has provided insufficient or no effect.

DISCUSSION

In the development of this guideline, the task force concluded that categorization into "specific and nonspecific" low back pain provides insufficient insight into low back pain and that this categorization fails to effectively indicate which available therapy is effective for which underlying disorder of a back pain syndrome. Therefore, the task force proposed to categorize spinal low back pain into "degenerative and nondegenerative" disorders. The degenerative disorders are then further subdivided into "uncomplicated and complicated" disorders (Figure 1). It is acknowledged that such a categorization is a provisional solution that may need to be adjusted as new data and insights emerge.

The current guideline addresses the invasive treatment of uncomplicated degenerative disorders. Later on, this guideline will be expanded to include complicated degenerative as well as nondegenerative disorders of the spine. Conservative treatments for the various disorders will also be inventoried, evaluated, and embedded in future guidelines.

This is not the first guideline on this topic. Among others, in 2008, a collaborative task force of the Dutch and Belgian Anesthesiology Pain societies succeeded in reviewing the diagnosis and treatment of 25 pain diagnoses. The majority of these reviews were translated into English (published in Pain Practice) and later collected in a textbook entitled "Evidence-based interventional pain medicine according to clinical diagnoses." Some of these diagnoses cover the diagnoses described in the underlying guideline on spinal low back pain.[9,19,152–157]

In the Dutch–Belgian collaboration, the search, selection, and evaluation procedure was based on the system described by Guyatt et al.[158] These authors use an Evidence-Based Guideline Development (EBGD) method and combine this with a risk-benefit consideration; especially in invasive pain medicine, this latter item is of considerable value. In this new underlying guideline, instead of using an EBGD method,[9] the GRADE method is used. An important difference is that assessment based on an EBGD method [158] focuses on the study design; that is, *the systematic review* is seen as the highest level of evidence, followed by *RCTs and observational studies*, and the lowest level of evidence is *expert opinion*. In the GRADE system, the main focus lies in assessment of the strength of evidence for prior defined, relevant outcome measures. This brings the GRADE method more in alignment with actual clinical practice.[159] One of the characteristics of the GRADE method is that inclusion criteria for the studies have to be described in advance; however, such criteria can be disputed. For example, in the new guideline discussed here, the inclusion criterion states a minimal study population of 15 patients (RCT = 2 × 15 patients). However, this criterion was an "educated guess" and could be considered a limitation of this guideline. Nevertheless, if necessary, smaller studies could be (and were) included in the "remaining considerations." This means that information and data from smaller studies were taken into account when developing the

recommendations and advice. A consequence of using the GRADE method is that, in many of the treatments for spinal low back pain syndromes, there is no evidence that a specific treatment actually helps. Application of the EBGD (or Guyatt) method would probably have led to a more positive image. On the other hand, the GRADE method not only examines the evidence regarding whether a therapy *in fact* helps, but also acknowledges that there has to be evidence that a specific treatment *does not* help.

An important limitation of the new guideline is that it describes the state of science only up to June 2011. Unfortunately, this is a limitation of most guidelines and ours is no exception to this rule. At the moment of publication, guidelines have generally already been overtaken by later reports. For example, new evidence has emerged regarding the treatment of SIJ pain, that is, positive reports from RCTs using cooled RF treatment.[160,161] These data will change the conclusions and recommendations about the place of invasive treatment in SIJ pain. For this reason, guidelines need to be updated regularly.

The GRADE method clearly shows the gaps in knowledge and provides a strategy for future research, not only regarding the themes but also concerning the way in which this research should be performed.

In the Netherlands, the decision as to whether (or not) a therapy is reimbursed is regulated by law (Health Council of the Netherlands). During the development of this guideline, a negative advice regarding the reimbursement of invasive treatment of spinal low back pain was issued by the Council. However, the Council based its decision mainly on systematic reviews commissioned by the Council itself.[3,4]

Based on our new guideline on spinal lumbar pain syndromes, the Council reconsidered the situation and has decided to support new research. A cost-effective study is in progress that aims to analyze minimally invasive treatment procedures in combination with a multidisciplinary rehabilitation program vs. a multidisciplinary rehabilitation program alone[6] (http://www.mintstudie.nl).

The results of this ongoing study will serve as a guide to future developments.[162]

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REFERENCES

1. Waddell G. The clinical course of low back pain. In: Waddell G, ed. *The Back Pain Revolution*. Ch 7. Edinburgh: Churchill Livingstone; 1998:115–37.

2. Itz CJ, Geurts JW, van Kleef M, Nelemans P. Clinical course of non-specific low back pain: a systematic review of prospective cohort studies set in primary care. *Eur J Pain*. 2012;17:5–15.

3. 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;26:2504–2513; discussion 13–4.

4. Willems P, de Bie R, Oner C, Castelein R, de Kleuver M. Clinical decision making in spinal fusion for chronic low back pain. Results of a nationwide survey among spine surgeons. *BMJ Open*. 2011;1:e000391.

5. Maas ET, Juch JN, Groeneweg JG, et al. Cost-effectiveness of minimal interventional procedures for chronic mechanical low back pain: design of four randomised controlled trials with an economic evaluation. *BMC Musculoskelet Disord*. 2012;13:260–269.

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

7. Ostelo RW, Deyo RA, Stratford P, et al. Interpreting change scores for pain and functional status in low back pain: towards international consensus regarding minimal important change. *Spine*. 2008;33:90–94.

8. Dickenson D, Vineis P. Evidence-based medicine and quality of care. *Health Care Anal*. 2002;10:243–259.

9. Van Zundert J, Hartrick C, Patijn J, Huygen F, Mekhail N, van Kleef M. Evidence-based interventional pain medicine according to clinical diagnoses. *Pain Pract*. 2011;11:423–429.

10. Cohen SP, Raja SN. Pathogenesis, diagnosis, and treatment of lumbar zygapophysial (facet) joint pain. *Anesthesiology*. 2007;106:591–614.

11. Dreyfuss P, Halbrook B, Pauza K, Joshi A, McLarty J, Bogduk N. Efficacy and validity of radiofrequency neurotomy

for chronic lumbar zygapophysial joint pain. *Spine*. 2000;25:1270–1277.

12. 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;24:1937–1942.

13. Hicks GE, Morone N, Weiner DK. Degenerative lumbar disc and facet disease in older adults: prevalence and clinical correlates. *Spine*. 2009;34:1301–1306.

14. Eubanks JD, Lee MJ, Cassinelli E, Ahn NU. Prevalence of lumbar facet arthrosis and its relationship to age, sex, and race: an anatomic study of cadaveric specimens. *Spine*. 2007;32:2058–2062.

15. Lewinnek GE, Warfield CA. Facet joint degeneration as a cause of low back pain. *Clin Orthop Relat Res*. 1986;213:216–222.

16. Butler D, Trafimow JH, Andersson GB, McNeill TW, Huckman MS. Discs degenerate before facets. *Spine*. 1990;15:111–113.

17. Yang KH, King AI. Mechanism of facet load transmission as a hypothesis for low-back pain. *Spine*. 1984;9:557–565.

18. McCall IW, Park WM, O'Brien JP. Induced pain referral from posterior lumbar elements in normal subjects. *Spine*. 1979;4:441–446.

19. van Kleef M, Vanelderen P, Cohen SP, Lataster A, Van Zundert J, Mekhail N. 12. Pain originating from the lumbar facet joints. *Pain Pract*. 2010;10:459–469.

20. Wilde VE, Ford JJ, McMeeken JM. Indicators of lumbar zygapophyseal joint pain: survey of an expert panel with the Delphi technique. *Phys Ther*. 2007;87:1348–1361.

21. Marks RC, Houston T, Thulbourne T. Facet joint injection and facet nerve block: a randomised comparison in 86 patients with chronic low back pain. *Pain*. 1992;49:325–328.

22. Dreyfuss PH, Dreyer SJ. Lumbar zygapophysial (facet) joint injections. *Spine J*. 2003;3:50S–59S.

23. Sowa G. Facet-mediated pain. *Dis Mon*. 2005;51:18–33.

24. Cohen SP, Strassels SA, Kurihara C, et al. Randomized study assessing the accuracy of cervical facet joint nerve (medial branch) blocks using different injectate volumes. *Anesthesiology*. 2010;112:144–152.

25. 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;26:1411–1416; discussion 7.

26. Dreyfuss P, Schwarzer AC, Lau P, Bogduk N. Specificity of lumbar medial branch and L5 dorsal ramus blocks. A computed tomography study. *Spine*. 1997;22:895–902.

27. Carrera GF. Lumbar facet joint injection in low back pain and sciatica: preliminary results. *Radiology*. 1980;137:665–667.

28. Helbig T, Lee CK. The lumbar facet syndrome. *Spine*. 1988;13:61–64.

29. Carrera GF, Williams AL. Current concepts in evaluation of the lumbar facet joints. *Crit Rev Diagn Imaging*. 1984;21:85–104.

30. Dolan AL, Ryan PJ, Arden NK, et al. The value of SPECT scans in identifying back pain likely to benefit from facet joint injection. *Br J Rheumatol*. 1996;35:1269–1273.

31. Murtagh FR. Computed tomography and fluoroscopy guided anesthesia and steroid injection in facet syndrome. *Spine*. 1988;13:686–689.

32. Raymond J, Dumas JM. Intraarticular facet block: diagnostic test or therapeutic procedure? *Radiology*. 1984;151:333–336.

33. Revel ME, Listrat VM, Chevalier XJ, et al. Facet joint block for low back pain: identifying predictors of a good response. *Arch Phys Med Rehabil*. 1992;73:824–828.

34. Fairbank JC, Park WM, McCall IW, O'Brien JP. Apophyseal injection of local anesthetic as a diagnostic aid in primary low-back pain syndromes. *Spine*. 1981;6:598–605.

35. Jackson RP, Jacobs RR, Montesano PX. 1988 Volvo award in clinical sciences. Facet joint injection in low-back pain. A prospective statistical study. *Spine*. 1988;13:966–971.

36. Schwarzer AC, Wang SC, O'Driscoll D, Harrington T, Bogduk N, Laurent R. The ability of computed tomography to identify a painful zygapophysial joint in patients with chronic low back pain. *Spine*. 1995;20:907–912.

37. Gallagher J, Vadi PLP, Wesley JR. Radiofrequency facet joint denervation in the treatment of low back pain-a prospective controlled double-blind study in assess to efficacy. *Pain Clinic*. 1994;7:193–198.

38. Nath S, Nath CA, Pettersson K. Percutaneous lumbar zygapophysial (Facet) joint neurotomy using radiofrequency current, in the management of chronic low back pain: a randomized double-blind trial. *Spine*. 2008;33:1291–1297; discussion 1298.

39. Tekin I, Mirzai H, Ok G, Erbuyun K, Vatansever D. A comparison of conventional and pulsed radiofrequency denervation in the treatment of chronic facet joint pain. *Clin J Pain*. 2007;23:524–529.

40. Manchikanti L, Singh V, Falco FJ, Cash KA, Pampati V. Lumbar facet joint nerve blocks in managing chronic facet joint pain: one-year follow-up of a randomized, double-blind controlled trial: Clinical Trial NCT00355914. *Pain Physician*. 2008;11:121–132.

41. Manchikanti L, Pampati V, Bakhit CE, et al. Effectiveness of lumbar facet joint nerve blocks in chronic low back pain: a randomized clinical trial. *Pain Physician*. 2001;4:101–117.

42. 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;325:1002–1007.

43. Kroll HR, Kim D, Danic MJ, Sankey SS, Gariwala M, Brown M. A randomized, double-blind, prospective study comparing the efficacy of continuous versus pulsed radiofrequency in the treatment of lumbar facet syndrome. *J Clin Anesth*. 2008;20:534–537.

44. Esses SI, Moro JK. The value of facet joint blocks in patient selection for lumbar fusion. *Spine*. 1993;18:185–190.

45. Merskey H. *Classification of Chronic Pain, International Association for the Study of Pain; Task Force on Taxonomy*. Canada: IASP Press; 1994.

46. Fortin JD, Kissling RO, O'Connor BL, Vilensky JA. Sacroiliac joint innervation and pain. *Am J Orthop (Belle Mead NJ)*. 1999;28:687–690.

47. Szadek KM, Hoogland PV, Zuurmond WW, de Lange JJ, Perez RS. Nociceptive nerve fibers in the sacroiliac joint in humans. *Reg Anesth Pain Med*. 2008;33:36–43.

48. Bernard TN Jr, Kirkaldy-Willis WH. Recognizing specific characteristics of nonspecific low back pain. *Clin Orthop Relat Res*. 1987;217:266–280.

49. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. *Spine*. 1995;20:31–37.

50. Maigne JY, Aivaliklis A, Pfefer F. Results of sacroiliac joint double block and value of sacroiliac pain provocation tests in 54 patients with low back pain. *Spine*. 1996;21:1889–1892.

51. Herzog W, Conway PJ. Gait analysis of sacroiliac joint patients. *J Manipulative Physiol Ther*. 1994;17:124–127.

52. Schuit D, McPoil TG, Mulesa P. Incidence of sacroiliac joint malalignment in leg length discrepancies. *J Am Podiatr Med Assoc*. 1989;79:380–383.

53. Schoenberger M, Hellmich K. Sacroiliac dislocation and scoliosis. *Hippokrates*. 1964;35:476–479.

54. Albert H, Godskesen M, Westergaard J. Prognosis in four syndromes of pregnancy-related pelvic pain. *Acta Obstet Gynecol Scand*. 2001;80:505–510.

55. Marymont JV, Lynch MA, Henning CE. Exercise-related stress reaction of the sacroiliac joint. An unusual cause of low back pain in athletes. *Am J Sports Med*. 1986;14:320–323.

56. Hansen HC, McKenzie-Brown AM, Cohen SP, Swicegood JR, Colson JD, Manchikanti L. Sacroiliac joint interventions: a systematic review. *Pain Physician*. 2007;10:165–184.

57. Song IH, Carrasco-Fernandez J, Rudwaleit M, Sieper J. The diagnostic value of scintigraphy in assessing sacroiliitis in ankylosing spondylitis: a systematic literature research. *Ann Rheum Dis*. 2008;67:1535–1540.

58. Puhakka KB, Melsen F, Jurik AG, Boel LW, Vesterby A, Egund N. MR imaging of the normal sacroiliac joint with correlation to histology. *Skeletal Radiol*. 2004;33:15–28.

59. Puhakka KB, Jurik AG, Schiottz-Christensen B, et al. MRI abnormalities of sacroiliac joints in early spondylarthropathy: a 1-year follow-up study. *Scand J Rheumatol*. 2004;33:332–338.

60. Slipman CW, Jackson HB, Lipetz JS, Chan KT, Lenrow D, Vresilovic EJ. Sacroiliac joint pain referral zones. *Arch Phys Med Rehabil*. 2000;81:334–338.

61. Young S, Aprill C, Laslett M. Correlation of clinical examination characteristics with three sources of chronic low back pain. *Spine J*. 2003;3:460–465.

62. Cattley P, Winyard J, Trevaskis J, Eaton S. Validity and reliability of clinical tests for the sacroiliac joint. A review of literature. *Australas Chiropr Osteopathy*. 2002;10:73–80.

63. Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests. *Man Ther*. 2005;10:207–218.

64. van der Wurff P, Buijs EJ, Groen GJ. A multitest regimen of pain provocation tests as an aid to reduce unnecessary minimally invasive sacroiliac joint procedures. *Arch Phys Med Rehabil*. 2006;87:10–14.

65. van der Wurff P, Buijs EJ, Groen GJ. Intensity mapping of pain referral areas in sacroiliac joint pain patients. *J Manipulative Physiol Ther*. 2006;29:190–195.

66. Szadek KM, van der Wurff P, van Tulder MW, Zuurmond WW, Perez RS. Diagnostic validity of criteria for sacroiliac joint pain: a systematic review. *Pain*. 2009;10:354–368.

67. Dreyfuss MD. Practice Guidelines and protocols for Sacroiliac Joint blocks. In: Society ISI ed. 9th Annual Scientific Meeting; 1989:35–49.

68. Laslett M, Young SB, Aprill CN, McDonald B. Diagnosing painful sacroiliac joints: a validity study of a McKenzie evaluation and sacroiliac provocation tests. *Aust J Physiother*. 2003;49:89–97.

69. Maigne JY, Boulahdour H, Chatellier G. Value of quantitative radionuclide bone scanning in the diagnosis of sacroiliac joint syndrome in 32 patients with low back pain. *Eur Spine J*. 1998;7:328–331.

70. Maugars Y, Mathis C, Berthelot JM, Charlier C, Prost A. Assessment of the efficacy of sacroiliac corticosteroid injections in spondylarthropathies: a double-blind study. *Br J Rheumatol*. 1996;35:767–770.

71. Luukkainen RK, Wennerstrand PV, Kautiainen HH, Sanila MT, Asikainen EL. Efficacy of periarticular corticosteroid treatment of the sacroiliac joint in non-spondylarthropathic patients with chronic low back pain in the region of the sacroiliac joint. *Clin Exp Rheumatol*. 2002;20:52–54.

72. Cohen SP, Hurley RW, Buckenmaier CC 3rd, Kurihara C, Morlando B, Dragovich A. Randomized placebo-controlled study evaluating lateral branch radiofrequency denervation for sacroiliac joint pain. *Anesthesiology*. 2008;109:279–288.

73. Buijs EJ, van Wijk RM, Geurts JW, Weeseman RR, Stolker RJ, Groen GG. Radiofrequency lumbar facet denervation: a comparative study of the reproducibility of lesion size after 2 current radiofrequency techniques. *Reg Anesth Pain Med*. 2004;29:400–407.

74. Vallejo R, Benyamin RM, Kramer J, Stanton G, Joseph NJ. Pulsed radiofrequency denervation for the treatment of sacroiliac joint syndrome. *Pain Med*. 2006;7:429–434.

75. Ferrante FM, King LF, Roche EA, et al. Radiofrequency sacroiliac joint denervation for sacroiliac syndrome. *Reg Anesth Pain Med*. 2001;26:137–142.

76. Wise CL, Dall BE. Minimally invasive sacroiliac arthrodesis: outcomes of a new technique. *J Spinal Disord Tech*. 2008;21:579–584.

77. Ziran BH, Heckman D, Smith WR. CT-guided stabilization for chronic sacroiliac pain: a preliminary report. *J Trauma*. 2007;63:90–96.

78. Khurana A, Guha AR, Mohanty K, Ahuja S. Percutaneous fusion of the sacroiliac joint with hollow modular anchorage screws: clinical and radiological outcome. *J Bone Joint Surg Br*. 2009;91:627–631.

79. Buchowski JM, Kebaish KM, Sinkov V, Cohen DB, Sieber AN, Kostuik JP. Functional and radiographic outcome of sacroiliac arthrodesis for the disorders of the sacroiliac joint. *Spine J*. 2005;5:520–528; discussion 529.

80. Schutz U, Grob D. Poor outcome following bilateral sacroiliac joint fusion for degenerative sacroiliac joint syndrome. *Acta Orthop Belg*. 2006;72:296–308.

81. Peyton FW. Coccygodynia in women. *Indiana Med*. 1988;81:697–698.

82. Maigne JY, Doursounian L, Chatellier G. Causes and mechanisms of common coccydynia: role of body mass index and coccygeal trauma. *Spine*. 2000;25:3072–3079.

83. Maigne JY, Guedj S, Straus C. Idiopathic coccygodynia. Lateral roentgenograms in the sitting position and coccygeal discography. *Spine*. 1994;19:930–934.

84. Frazier LM. Coccydynia: a tail of woe. *N C Med J*. 1985;46:209–212.

85. van Kleef M, Barendse G, Wilmink JT. Percutaneous intradiscal radio-frequency. *Pain Clinic*. 1996;9: 259–268.

86. Moore K, Delley A. *Clinically Oriented Anatomy*. Philadelphia: Lippincott Williams & Wilkins; 1999.

87. Ng CL. Levator ani syndrome – a case study and literature review. *Aust Fam Physician*. 2007;36:449–452.

88. De Andres J, Chaves S. Coccygodynia: a proposal for an algorithm for treatment. *J Pain*. 2003;4:257–266.

89. Lourie J, Young S. Avascular necrosis of the coccyx: a cause of coccydynia? Case report and histological findings in 16 patients. *Br J Clin Pract*. 1985;39:247–248.

90. Maigne JY, Chatellier G. Comparison of three manual coccydynia treatments: a pilot study. *Spine*. 2001;26:E479–E483; discussion E84.

91. Wray CC, Easom S, Hoskinson J. Coccydynia. Aetiology and treatment. *J Bone Joint Surg Br*. 1991;73:335–338.

92. Reig E, Abejon D, del Pozo C, Insausti J, Contreras R. Thermocoagulation of the ganglion impar or ganglion of Walther: description of a modified approach. Preliminary results in chronic, nononcological pain. *Pain Pract*. 2005;5:103–110.

93. Sehirlioglu A, Ozturk C, Oguz E, Emre T, Bek D, Altinmakas M. Coccygectomy in the surgical treatment of traumatic coccygodynia. *Injury*. 2007;38:182–187.

94. Kerr EE, Benson D, Schrot RJ. Coccygectomy for chronic refractory coccygodynia: clinical case series and literature review. *J Neurosurg Spine*. 2011;14:654–663.

95. Trollegaard AM, Aarby NS, Hellberg S. Coccygectomy: an effective treatment option for chronic coccydynia: retrospective results in 41 consecutive patients. *J Bone Joint Surg Br*. 2010;92:242–245.

96. Balain B, Eisenstein SM, Alo GO, et al. Coccygectomy for coccydynia: case series and review of literature. *Spine*. 2006;31:E414–E420.

97. Karadimas EJ, Trypsiannis G, Giannoudis PV. Surgical treatment of coccygodynia: an analytic review of the literature. *Eur Spine J*. 2011;20:698–705.

98. 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.

99. 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.

100. 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.

101. Hadjipavlou AG, Simmons JW, Pope MH, Necessary JT, Goel VK. Pathomechanics and clinical relevance of disc degeneration and annular tear: a point-of-view review. *Am J Orthop*. 1999;28:561–571.

102. Sambrook PN, MacGregor AJ, Spector TD. Genetic influences on cervical and lumbar disc degeneration: a magnetic resonance imaging study in twins. *Arthritis Rheum*. 1999;42:366–372.

103. Hancock MJ, Battie MC, Videman T, Gibbons L. The role of back injury or trauma in lumbar disc degeneration: an exposure-discordant twin study. *Spine*. 2010;35:1925–1929.

104. Battie MC, Videman T, Kaprio J, et al. The Twin Spine Study: contributions to a changing view of disc degeneration. *Spine J*. 2009;9:47–59.

105. Videman T, Gibbons LE, Kaprio J, Battie MC. Challenging the cumulative injury model: positive effects of greater body mass on disc degeneration. *Spine J*. 2010;10:26–31.

106. Battie MC, Videman T, Gill K, et al. 1991 Volvo Award in clinical sciences. Smoking and lumbar intervertebral disc degeneration: an MRI study of identical twins. *Spine*. 1991;16:1015–1021.

107. Fogelholm RR, Alho AV. Smoking and intervertebral disc degeneration. *Med Hypotheses*. 2001;56:537–539.

108. Groen GJ, Baljet B, Drukker J. Nerves and nerve plexuses of the human vertebral column. *Am J Anat*. 1990;188:282–296.

109. Hasegawa T, An HS, Inufusa A, Mikawa Y, Watanabe R. The effect of age on inflammatory responses and nerve root injuries after lumbar disc herniation: an experimental study in a canine model. *Spine*. 2000;25:937–940.

110. Nerlich AG, Schleicher ED, Boos N. 1997 Volvo Award winner in basic science studies. Immunohistologic markers for age-related changes of human lumbar intervertebral discs. *Spine*. 1997;22:2781–2795.

111. O'Donnell JL, O'Donnell AL. Prostaglandin E2 content in herniated lumbar disc disease. *Spine*. 1996;21:1653–1655; discussion 1655-1656.

112. Oegema TR Jr, Johnson SL, Aguiar DJ, Ogilvie JW. Fibronectin and its fragments increase with degeneration in the human intervertebral disc. *Spine*. 2000;25:2742–2747.

113. 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.

114. Ohnmeiss DD, Vanharanta H, Ekholm J. Relationship of pain drawings to invasive tests assessing intervertebral disc pathology. *Eur Spine J* 1999;8:126–131.

115. 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.

116. Takahashi Y, Sato A, Nakamura SI, Suseki K, Takahashi K. Regional correspondence between the ventral portion of the lumbar intervertebral disc and the groin mediated by a spinal reflex. A possible basis of discogenic referred pain. *Spine*. 1998;23:1853–1858; discussion 1859.

117. Vanharanta H. The intervertebral disc: a biologically active tissue challenging therapy. *Ann Med*. 1994;26:395–399.

118. 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 1259-1260.

119. 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.

120. Jensen PD, Jensen FT, Christensen T, Ellegaard J. Non-invasive assessment of tissue iron overload in the liver by magnetic resonance imaging. *Br J Haematol*. 1994;87:171–184.

121. Carragee EJ, Barcohana B, Alamin T, van den Haak E. Prospective controlled study of the development of lower back pain in previously asymptomatic subjects undergoing experimental discography. *Spine*. 2004;29:1112–1117.

122. Pauza KJ, Howell S, Dreyfuss P, Peloza 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.

123. Ohtori S, Yamashita M, Inoue G, et al. L2 spinal nerve-block effects on acute low back pain from osteoporotic vertebral fracture. *J Pain*. 2009;10:870–875.

124. 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.

125. Buenaventura RM, Shah RV, Patel V, Benyamin R, Singh V. Systematic review of discography as a diagnostic test for spinal pain: an update. *Pain Physician*. 2007;10:147–164.

126. Manchikanti L, Glaser SE, Wolfer L, Derby R, Cohen SP. Systematic review of lumbar discography as a diagnostic test for chronic low back pain. *Pain Physician*. 2009;12:541–559.

127. Shah RV, Everett CR, McKenzie-Brown AM, Sehgal N. Discography as a diagnostic test for spinal pain: a systematic and narrative review. *Pain Physician*. 2005;8:187–209.

128. Peng B, Pang X, Wu Y, Zhao C, Song X. A randomized placebo-controlled trial of intradiscal methylene blue injection for the treatment of chronic discogenic low back pain. *Pain*. 2010;149:124–129.

129. Peng B, Hao J, Hou S, et al. Possible pathogenesis of painful intervertebral disc degeneration. *Spine*. 2006;31:560–566.

130. Peng B, Hou S, Wu W, Zhang C, Yang Y. The pathogenesis and clinical significance of a high-intensity zone (HIZ) of lumbar intervertebral disc on MR imaging in the patient with discogenic low back pain. *Eur Spine J*. 2006;15:583–587.

131. 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.

132. 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.

133. 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.

134. 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 837.

135. 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.

136. 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.

137. Freeman BJ, Fraser RD, Cain CM, Hall DJ, Chapple DC. A randomized, double-blind, controlled trial: intradiscal electrothermal therapy versus placebo for the treatment of chronic discogenic low back pain. *Spine*. 2005;30:2369–2377; discussion 2378.

138. Ohtori S, Koshi T, Yamashita M, et al. Surgical versus nonsurgical treatment of selected patients with discogenic low back pain: a small-sized randomized trial. *Spine*. 2011;36:347–354.

139. Blumenthal S, McAfee PC, Guyer RD, et al. A prospective, randomized, multicenter Food and Drug Administration investigational device exemptions study of lumbar total disc replacement with the CHARITE artificial disc versus lumbar fusion: part I: evaluation of clinical outcomes. *Spine*. 2005;30:1565–1575; discussion E387-91.

140. Zigler J, Delamarter R, Spivak JM, et al. Results of the prospective, randomized, multicenter Food and Drug Administration investigational device exemption study of the ProDisc-L total disc replacement versus circumferential fusion

for the treatment of 1-level degenerative disc disease. *Spine*. 2007;32:1155–1162; discussion 63.

141. Wilkinson HA. Spinal cord stimulation versus reoperation for failed back surgery syndrome: a cost effectiveness and cost utility analysis based on a randomized, controlled trial. *Neurosurgery*. 2008;63:E376; author reply E376.

142. Stojanovic MP. Stimulation methods for neuropathic pain control. *Curr Pain Headache Rep*. 2001;5:130–137.

143. North RB, Kidd D, Shipley J, Taylor RS. Spinal cord stimulation versus reoperation for failed back surgery syndrome: a cost effectiveness and cost utility analysis based on a randomized, controlled trial. *Neurosurgery*. 2007;61:361–368; discussion 368-369.

144. Weir BK, Jacobs GA. Reoperation rate following lumbar discectomy. An analysis of 662 lumbar discectomies. *Spine*. 1980;5:366–370.

145. Law JD, Lehman RA, Kirsch WM. Reoperation after lumbar intervertebral disc surgery. *J Neurosurg*. 1978;48:259–263.

146. Waddell G, Kummel EG, Lotto WN, Graham JD, Hall H, McCulloch JA. Failed lumbar disc surgery and repeat surgery following industrial injuries. *J Bone Joint Surg Am*. 1979;61:201–207.

147. Spengler DM, Freeman C, Westbrook R, Miller JW. Low-back pain following multiple lumbar spine procedures. Failure of initial selection? *Spine*. 1980;5:356–360.

148. Wiltse LL, Rocchio PD. Preoperative psychological tests as predictors of success of chemonucleolysis in the treatment of the low-back syndrome. *J Bone Joint Surg Am*. 1975;57:478–483.

149. An HS, Silveri CP, Simpson JM, et al. Comparison of smoking habits between patients with surgically confirmed herniated lumbar and cervical disc disease and controls. *J Spinal Disord*. 1994;7:369–373.

150. Manchikanti L, Boswell MV, Rivera JJ, et al. [ISRCTN 16558617] A randomized, controlled trial of spinal endoscopic adhesiolysis in chronic refractory low back and lower extremity pain. *BMC Anesthesiol*. 2005;5:10.

151. Kumar K, Taylor RS, Jacques L, et al. The effects of spinal cord stimulation in neuropathic pain are sustained: a 24-month follow-up of the prospective randomized controlled multicenter trial of the effectiveness of spinal cord stimulation. *Neurosurgery*. 2008;63:762–770; discussion 70.

152. Van Zundert J, Hartrick C, Patijn J, Huygen F, Mekhail N, van Kleef M. Evidence-based Interventional Pain Medicine According to Clinical Diagnoses. *Pain Pract*. 2011;11:423–429.

153. Vanelderen P, Szadek K, Cohen SP, et al. 13. Sacroiliac joint pain. *Pain Pract*. 2010;10:470–478.

154. Patijn J, Janssen M, Hayek S, Mekhail N, Van Zundert J, van Kleef M. 14. Coccygodynia. *Pain Practice*. 2010;10:554–559.

155. Kallewaard JW, Terheggen MA, Groen GJ, et al. . 15. Discogenic low back pain. *Pain Pract*. 2010;10:560–579.

156. De Andres J, Van Buyten JP. Neural modulation by stimulation. *Pain Pract*. 2006;6:39–45.

157. Van Boxem K, Cheng J, Patijn J, et al. 11. Lumbosacral radicular pain. *Pain Pract*. 2010;10:339–358.

158. 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.

159. Boluyt N, Rottier BL. Transparent reporting of all GRADE criteria in clinical practice guidelines. *Am J Respir Crit Care Med*. 2012;185:1129; author reply 1129–1130.

160. Karaman H, Kavak GO, Tufek A, et al. Cooled radiofrequency application for treatment of sacroiliac joint pain. *Acta Neurochir*. 2011;153:1461–1468.

161. Patel N, Gross A, Brown L, Gekht G. A randomized, placebo-controlled study to assess the efficacy of lateral branch neurotomy for chronic sacroiliac joint pain. *Pain Med*. 2012;13:383–398.

162. Enthoven AC, van de Ven WP. Going Dutch–managed-competition health insurance in the Netherlands. *N Engl J Med*. 2007;357:2421–2423.

163. Bogduk N. Lumbar disc stimulation. In: Bogduk N, ed. *Practice Guidelines for Spinal Diagnostic and Treatment Procedures*. San Francisco, CA: International Spine Intervention Society; 2004:20–46.