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A critical evaluation of discography in patients with lumbar intervertebral disc disease

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Abstract

STUDY DESIGN: The study is a prospective observational study of 48 continuous patients with symptomatic lumbar degenerative disk disease. Each patient underwent discography, MRI, and a biochemical analysis of disk lavage fluid.

OBJECTIVES: The purpose of this study was to correlate concordant pain on discography with MRI grade and biochemical markers of inflammation in a clinical setting.

SUMMARY OF BACKGROUND DATA: The pathophysiology of degenerative disk disease is complex. Discography is used to differentiate symptomatic from asymptomatic levels. MRI is used to image changes in disk water content. Biochemical assays have identified molecular markers of inflammation. To date, no study has correlated concordant pain on discography with MRI findings and biochemical markers.

METHODS: Forty-eight (48) continuous patients with symptomatic lumbar degenerative disk disease gave informed consent for study entry. Patient sex, age, insurance, work status and visual analog score (VAS) were recorded. MRI was obtained and Pfirrmann grading was performed by a single spine surgeon. Discography with disc lavage was performed by a single anesthesiologist. Lavage samples were tested for inflammatory markers with high resolution multi-plex bead immunoassays and ELISA with >5 pg/ml resolution.

RESULTS: None of demographic variables was significantly related to concordant pain on discogram by chi-squared tests and Mann-Whitney U-test. The Pfirrmann score was significantly different for patients with and without concordant pain at L3-L4 ($p<0.001$), but was insignificant at other levels after multitest correction. Pfirrmann scores were significantly different at any level in patients with and without concordant pain. VAS scores were not significantly correlated with opening pressures at any level. Despite the presence of serum proteins in the disk lavage fluid, none of the tested inflammatory mediators was identified by multi-plex bead immunoassays and ELISA.

CONCLUSIONS: There are only weak correlations between demographic, discogram, and radiographic variables. Response to discogram cannot be predicted by non-invasive means. The disk lavage method was unable to identify the presence of specific inflammatory peptides with multiplex immunoassays and ELISA.

Keywords: Intervertebral disc disease; Cytokines; Chemical pain mediators; Lumbar disc; Prostaglandins; Neuropeptides

FDA device/drug status: not applicable.

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Introduction

The surgical management and pathophysiology of degenerative disc disease is complex. Discography is an important diagnostic procedure in which disc pressure controlled by needle injection is correlated with degree of lumbar pain reported by the patient. Although imaging studies, such as magnetic resonance imaging (MRI), can be used to document pathologic changes in the disc, some studies have shown a poor correlation between MRI findings and clinical symptomatology [1], because disc degeneration and herniation increase with normal aging. The high incidence of asymptomatic findings on MRI makes interpretation of patient studies more difficult, because each imaging finding must be closely correlated to symptoms which can be relatively nonspecific in many patients. MRI alone is not able to differentiate symptomatic from asymptomatic degenerative disc changes.

In addition to these clinical issues, laboratory studies examining biopsy specimens during clinical removal of disc material in the treatment of lumbar herniated nucleus pulposus refractory to conservative measures have shown that inflammatory mechanisms are most likely involved in the majority of sciatic-like symptoms secondary to lumbar disc herniation [2–6]. It has been suggested that morphologically different types of disc herniation may demonstrate distinct inflammatory properties [7]. Inflammatory cytokines have been identified in cerebrospinal fluid of patients with symptomatic degenerative disc disease, although the source and concentration of inflammatory mediators at the level of the symptomatic nerve root is unknown [3,8]. Despite the apparent role of inflammatory mediators in the pathophysiology of degenerative disc disease, relatively little is known concerning the molecular aspects of pain and the specific biochemical factors involved.

To date, no study has correlated concordant pain on discography with MRI grading and with biochemical analysis of the disc space [7,8]. We hypothesized that we could correlate clinical findings on discography with radiographic MRI changes and specific inflammatory mediators in the disc space using lavage during discography followed by a highly sensitive protein assay.

Methods

After obtaining human investigational review board approval, 48 patients ranging in age from 21 to 75 with axial back pain of at least 6 months duration were enrolled in this study. The patients were identified among 171 consecutive patients offered study enrollment after being referred to an anesthesiologist who specializes in pain management. Patients with a history of corticosteroid medication within a 3-month period before the recommended discography, those with chronic medical conditions (insulin-dependent diabetes mellitus, coronary artery disease requiring surgery or interventional cardiology) or systemic inflammatory disease were excluded from the study.

Demographic information was obtained including gender, age, insurance, work status, and visual analog scores before the procedure. Additionally, extent/grading of disc disease, concordance on discography, and opening pressure on discography were recorded. A board-certified orthopaedic spinal surgeon independently evaluated MRI scans of all patients. Each disc level was classified according to the scale devised by Pfirrmann.

All study participants underwent lumbar discography by a single physician (RD) to evaluate their chronic axial pain. All patients had a discogram performed at a normal level for a control. The patient was placed prone on an operative, radiolucent table. A sterile preparation and draping was then undertaken. Intravenous antibiotics were given before the commencement of discography. After infiltrating the subcutaneous tissue with 1% Xylocaine with epinephrine, a standard double-needle technique was used. Initially, a 20-gauge introducer needle was utilized, then a 25-gauge curved Chiba spinal needle was used to enter the annulus. Placement was then confirmed fluoroscopically. Opening pressure was noted, and then saline solution 1–3 mL was injected and then removed. The discography procedure was continued with a nucleogram with a mixture of Omnipaque and Kefzol. Pain provocation, visual analog scores, and opening and static disc pressures were documented. The lavage fluid was then placed in a sterile container and the rest of the injection procedure was completed. The specimens were placed on ice and then frozen at -20° C and only thawed at the time of biochemical analysis.

The presence of proteins within the aspirated fluid was evaluated by polyacrylamide gel electrophoresis. Using a 4–10% gradient polyacrylamide gel (sodium dodecyl sulfate–polyacrylamide gel electrophoresis, BioRad, Hercules, CA), approximately 40μL of sample was loaded into the gel lanes along with a 4-μL calibration marker and a 4-μL solution of diluted serum protein. Gel staining was performed using Coomassie blue (Sigma Chemical Co., St. Louis, MO) which provides a detection limit of approximately 0.1μg/band. The presence and concentration of specific inflammatory mediators was then evaluated using high-resolution multi-plex bead immunoassays (Cytokine Panel III, Biosource, Camarillo, CA) using the manufacturer protocols. The specific cytokines that were able to be detected with this assay included interleukin (IL)-1β, IL-1ra, IL-2, IL-2R, IL-4, IL-5, IL-6, IL-7, IL-8, IL-10, IL-12p40, IL-13, IL-15, IL-17, tumor necrosis factor (TNF)-α, interferon (IFN)-α, IFN-γ, granulocyte-macrophage colony-stimulating factor (GM-CSF), MIP-1α, MIP-1β, IP-10, MIG, eotaxin, RANTES, and MCP-1. This assay was performed using a 96-well plate format and was analyzed using a Luminex 100 (Luminex Inc., Austin, TX) instrument which monitors the spectral properties of the capture beads while simultaneously measuring the quantity of associated fluorophore.

Standard curves were generated in this assay system extending over several orders of magnitudes of concentrations. The sensitivity and quantization abilities of the assays system are comparable to traditional enzyme-linked immunosorbent assays (ELISA). All 45 lavage samples were also tested using sandwich ELISA in 96-well microtitration plates, following the manufacturer's protocols, and ELISA kits (R&D Systems, Minneapolis, MN) for the cytokines IL-1β, IL-6 and TNF-α. The standards used for each of these cytokines ranged from IL-1β (2–250 pg/mL), IL-6 (4–600 pg/mL), and TNF-α (7–1000 pg/mL). (Vmax microplate reader, Molecular Devices, Sunnyvale, CA). The optical density of the plates was measured at 450 nm. (reference filter 650 nm). All samples were tested in duplicate. The cytokines that were available for testing included IL-1β, IL-1ra, IL-2, IL-2R, IL-4, IL-5, IL-6, IL-7, IL-8, IL-10, IL-12p40, IL-13, IL-15, IL-17, TNF-α, IFN-α, IFN-γ, GM-CSF, MIP-1α, MIP-1β, IP-10, MIG, eotaxin, RANTES, and MCP-1, and neuropeptides.

Multivariate statistical analysis was performed on demographic, radiographic, discographic, and biochemical data with SPSS 12.0 (SPSS, Inc., Chicago, IL). Data were analyzed for probability distribution to guide choice of parametric and nonparametric statistics. An alpha value of 0.05 was considered significant after multitest correction of the Bonferroni type.

Results

Polyacrylamide gel electrophoresis verified the presence of peptides in the lavage solutions (Fig. 1). However, high-resolution multi-plex bead immunoassays and ELISA failed to detect any of the specific peptides tested for in any of the 48 lavage samples down to the detection limits of the assays.

Demographic variables are presented in Table 1. Patient age was normally distributed by the Kolmogorov-Smirnov test (p=.26) but was not normally distributed after multitest correction (p=.016). Radiographic variables are presented in Table 2. The Pfirrmann scores were not normally distributed at any spinal level (p<.05 for all levels). Lumbar discogram variables are presented in Table 3. The visual analog scores were not normally distributed at any spinal level by Kolmogorov-Smirnov test (p<.05 for all levels). The opening pressure scores were not normally distributed by Kolmogorov-Smirnov test after multitest correction at any level (p<.05 at all levels).

None of the demographic variables was significantly related to concordant pain on discogram in a univariate analysis. Gender was not significantly related to concordant pain by chi-square analysis at L5–S1 (p=.65), L4–L5 (p=.65), L3–L4 (p=.33), L2–L3 (p=.98), or L1–L2 (p=.45). Insurance status was not significantly related to concordant pain by chi-square analysis at L5–S1 (p=.26), L4–L5 (p=.26), L3–L4 (p=.56), L2–L3 (p=.66), or L1–L2 (p=.95). Work status was not significantly related to concordant pain by chi-square analysis at L5–S1 (p=.95), L4–L5 (p=.95), L3–L4 (p=.39), L2–L3 (p=.33), or L1–L2 (p=.35). Age was not significantly different between patients with concordant pain by *t* tests at L5–S1 (p=.055), L4–L5 (p=.055), L3–L4 (p=.057), L2–L3 (p=.18), or L1–L2 (p=.70). Age also did not differ by Mann-Whitney *U* test at any level (p>.05 at all levels).

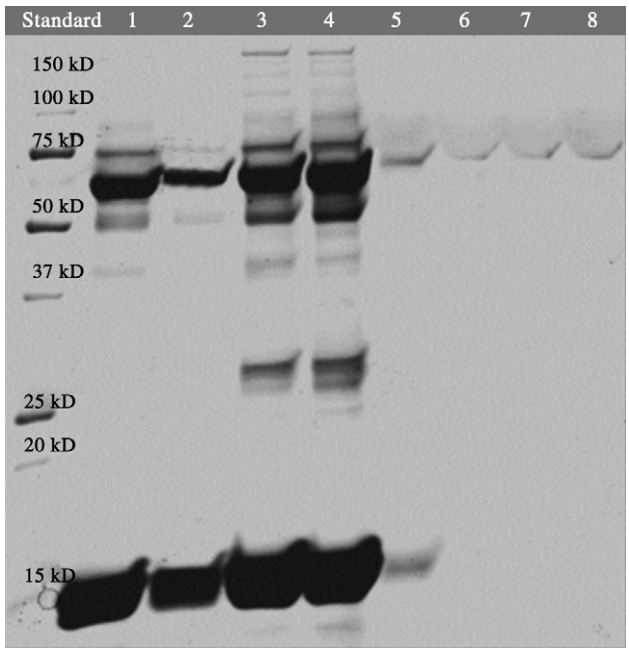

Fig. 1. Polyacrylamide gel electrophoresis of eight disc lavage samples. Lanes 1–4 had evidence of blood within the samples. All lanes showed evidence of protein within the microgram/mL range.

The radiographic Pfirrmann data were not predictive of discogram result variables. Though the Pfirrmann score was significantly different for patients with and without concordant pain by Mann-Whitney *U* test at L5–S1 (p=.028) and L3–L4 (p<.001), the effect was not observed at L4–L5 (p=.50), L2–L3 (p=.89), or L1–L2 (p=.46). Only the significance at L3–L4 was sustained after multitest

Table 1
Demographic variables

Gender	
Female	16 (33%)
Male	32 (67%)
Insurance	
Private	10 (21%)
Workers compensation	38 (79%)
Work status	
Full duty	13 (27%)
Light duty	2 (4.2%)
Out of work	30 (63%)
Unreported	3 (6.3%)
Age (years)	42 (10)

Categorical variables are presented as frequency (percentage). Age is presented as mean (standard deviation).

Table 2
Radiographic variables; frequencies of Pfirrmann classification for all five lumbar levels regardless of symptoms

Level	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5
L5–S1	2	11	25	8	1
L4–L5	1	19	18	10	0
L3–L4	20	13	13	1	0
L2–L3	36	6	5	1	0
L1–L2	40	2	6	0	0

correction (corrected alpha value=0.01). Optimal cutoff values were computed at L5–S1 and L3–L4. A Pfirrmann score greater than 3 was not predictive of concordant pain by chi-square analysis at L5–S1 (p=.088). A Pfirrmann score greater than 2 was predictive of concordant pain by chi-square analysis at L3–L4 (p=.002) even after multitest correction (corrected alpha value=0.007), but the sensitivity was only 41% at a specificity of 85% by receiver-operating characteristic analysis. Similarly, the Pfirrmann radiographic scores were not significantly correlated with visual analog scores by Spearman's rho after multitest correction (corrected alpha=0.01) at L5–S1 (p=.032, rho=0.32), L4–L5 (p=.86, rho=0.028), L2–L3 (p=.89, rho=0.03), or L1–L2 (p=.80, rho=–0.14). Only at L3–L4 (p<.001, rho=0.58) was the Pfirrmann score correlated with visual analog score.

Among the discogram variables, concordant pain was correlated with visual analog scores at all levels by definition; however, opening pressures were not significantly different in patients with and without concordant pain by the Mann-Whitney *U* test at L5–S1 (p=.45), L4–L5 (p=.53), L3–L4 (p=.027), L2–L3 (p=.44), or L1–L2 (p=.49) after multitest correction (corrected alpha=0.01). Visual analog scores were not significantly correlated with opening pressures by the Spearman's rho test after Bonferroni multitest correction (corrected alpha=0.01) at L5–S1 (p=.01, rho=–0.38), L4–L5 (p=.01, rho=–0.38), L3–L4 (p=.025, rho=0.34), L2–L3 (p=.21, rho=0.26), or L1–L2 (p=.95, rho=0.03).

Discussion

Our understanding of the pathophysiology of axial back pain is evolving [9]. Pressure-controlled discography is a tool that may assist physicians in identifying a source of back pain [10]. It would be desirable to have a clinical assay of biochemical markers such as disc lavage to complement routine clinical discography. The purpose of this study was to try to identify inflammatory mediators in the disc space of patients with chronic axial pain that might correlate with symptoms. A similar assay experiment was performed in the epidural space in patients with radiculopathy [11].

Table 3
Discogram variables

Level	Corcordant pain	Visual analog	Opening pressure	n (48 total)
L5–S1	35 (78%)	6.8 (3.4)	20.9 (5.6)	45
L4–L5	35 (78%)	6.8 (3.4)	21.2 (5.3)	45
L3–L4	17 (39%)	3.3 (4.3)	18.8 (2.5)	44
L2–L3	3 (12%)	1.0 (2.6)	18.0 (2.1)	26
L1–L2	3 (50%)	3.5 (4.0)	18.7 (1.0)	6

Concordant pain is presented as frequency (percentage) of positive concordnat pain. The visual analog and opening pressure measurements are presented as mean (standard deviation).

Although many inflammatory cytokines have been identified in the intervertebral disc as it undergoes degeneration, it is not known if any of these specific inflammatory chemicals plays a significant role in radiculopathy or low back pain [12–15]. Identification of matrix fragments, nonenzymatic glycation products, and neuropeptides are representative of the many ongoing products and processes that occur with aging. Much work still needs to be done to determine what process, if any, links anatomical structural degenerative changes of the spine with symptom expression [16]. The identification of specific biomarkers that are associated with symptoms of discomfort will greatly facilitate our understanding of symptomatic degenerative disc disease. Future research is necessary to better understand the mechanisms and genetics that mediate intervertebral disc cell responses to the process of aging and micromechanical and inflammatory stimuli.

Other cytokines have been identified which may play a role in disc-related pain syndromes [4,6,7,17–21]. Several studies have identified TNF-α and IL-8 in surgical disc specimens removed for the treatment of pain [4,17]. Kang et al. identified matrix metalloproteinase, nitric oxide, prostaglandin E2, and interleukin-6 in cultures of discs retrieved in patients with a lumbar disc herniation and radiculopathy [22]. Similarly, Takahashi identified interleukin-1, beta interleukin-6, and TNF-α in prepared tissue specimens from retrieved human herniated disc tissue [23]. Burke et al. identified high levels of both IL-6 and IL-8 in patients with symptomatic degenerative disc disease who underwent spinal fusion [24]. It has been postulated that even a small amount of these factors may be sufficient to initiate an inflammatory process after rupture of the nucleus pulposus due to their ability to recruit other cytokine-producing cells and stimulate the up-regulation of genes coding for proinflammatory mediators [25].

In vitro studies have led to suggestions that certain specific neuropeptides are implicated in the pathophysiology of radiculopathy [26,27]. However, to date this has not been confirmed in vivo.

Several authors have suggested a possible role for pain-related neuropeptides in symptomatic intervertebral disc disease [28]. Substance P (SPLI) and calcitonin gene-related peptide (CGRP-L1) have been evaluated in the cerebrospinal fluid and dorsal root ganglion of humans and animal models respectively and have been shown to be elevated by local disc inflammation [26,27,29,30]. Unfortunately, the site of production of these molecules and the specific role they play in patient symptoms remains to be determined.

The statistical dependencies among demographic, radiographic, and discogram variables are weak. As expected, there is no way of reliably predicting the results of the discogram procedure from demographic and radiographic variables at any lumbar level. In this study, the analysis is confounded by the fact that lumber levels for discogram were chosen a priori based on history, physical examination, and imaging. This effect is probably large at upper lumber levels. At the L5–S1 and L4–L5 levels, 45 of 48 patients received discogram, so the effect is minimal. This confirms the value of discogram as providing additional clinical information.

In this study, we were unable to detect the presence of specific inflammatory peptides in the lavage fluid of patients with axial lumbar pain. There are several possible explanations for these results. First, it is possible that none of the cytokines were present in the disc space in concentrations adequate to be detected by the present detection technique. Alternatively, it may be that although one or more of the cytokines are present in the disc space, they are bound to tissues preventing their removal with the lavage technique. Also, they may only be present in the annulus and therefore undetectable. We only left the lavage fluid in the space for approximately 10 seconds, which may not be enough time for cytokine uptake. Another possible explanation is that the assay technique was not able to detect the peptides because they were not able to be removed in adequate concentrations or because they were degraded during the storage and analytic process. We were only able to remove a small portion (<50%) of lavage fluid in severely degenerated discs. This led us to sometimes use up to 3 cc. Out target was to remove 0.5 to 1 cc of lavage fluid. The machine was calibrated on each of the days sample were run, and positive controls were confirmed. We believe that these proteins are highly metabolically active and degraded during the storage process. We have added protease inhibitors to our most recent efforts and have been able to identify tiny amounts of cytokines. Early data are encouraging but premature at this time. This has validated the technique in the authors' opinion. Our study is being repeated with these modifications.

References

[1] Boden S, Davis DO, Dina TS, et al. Abnormal magnetic resonance scans of the spine in asymptomatic patients. J Bone Joint Surg 1990;72A:403–8.

[2] Olmarker K. Radicular pain: recent pathophysiologic concepts and therapeutic implications. Schmerz 2001;15:425–9.

[3] Brisby H, Olmarker K, Larsson K, et al. Proinflammatory cytokines in cerebrospinal fluid and serum in patients with disc herniation and sciatica. Eur Spine J 2002;11:62–6.

[4] Igarashi T, Kikuchi S, Shubayev V, et al. Exogenous tumor necrosis factor-alpha mimics nucleus pulposus-induced neuropathology. Molecular, histologic, and behavioral comparisons in rats. Spine 2000;25:2975–80.

[5] Cuellar JM, Montesano PX, Carstens E. Role of TNF-alpha in sensitization of nociceptive dorsal horn neurons induced by application of nucleus pulposus to L5 dorsal root ganglion in rats. Pain 2004;110: 578–87.

[6] Saal JS, Franson RC, Dobrow R, et al. High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. Spine 1990;15:674–8.

[7] Nygaard OP, Mellgren SI, Osterud B. The inflammatory properties of contained and noncontained lumbar disc herniation. Spine 1997;22: 2484–8.

[8] Hyyppa MT, Alaranta H, Lahtela K, et al. Neuropeptide converting enzyme activities in CSF of low back pain patients. Pain 1990;43: 163–8.

[9] Borenstein DG. Epidemiology, etiology, diagnostic evaluation, and treatment of low back pain. Curr Opin Rheumatol 2001;13:128–34.

[10] Derby R. The relationship between intradiskal pressure and pain provocation during discography. J Bone Joint Surg [Br] 1995;19: 59–60.

[11] Scuderi GJ, Brusovanik GV, Anderson DG, et al. Cytokine assay of epidural space lavage in patients with lumbar intervertebral disk herniation and radiculopathy. J Spinal Dis Tech (in press).

[12] Takahashi H, Suguro T, Okazima Y, et al. Inflammatory cytokines in the herniated disc of the lumbar spine. Spine 1996;21:218–24.

[13] Carlton SM, Hargett GL. Stereological analysis of Ca2+/calmodulin-dependent protein kinase II alpha–containing dorsal root ganglion neurons in the rat. J Compar Neurol 2002;448:102–10.

[14] Goupille P, Jayson MI, Valot JP, et al. The role of inflammation in disk herniation-associated radiculopathy. Semin Arthritis Rheum 1998;28:60–71.

[15] Gronblad M, Virri J, Tolonen J, et al. A controlled immunohistochemical study of inflammatory cells in disc herniation tissue. Spine 1994;19:2744–51.

[16] Kang JD, Stefanovic-Racie M, Mcintyre LA, et al. Toward a biochemical understanding of human intervertebral disc degeneration. Spine 1997;22:1065–73.

[17] Olmarker K, Rydevik B. Selective inhibition of tumor necrosis factor-alpha prevents nucleus pulposus-induced thrombus formation, intraneural edema, and reduction of nerve conduction velocity: possible implications for future pharmacologic treatment strategies of sciatica. Spine 2001;26(20):863–9.

[18] Cannon JG, St. Pierre BA. Cytokines in exertion-induced skeletal muscle injury. Mol Cell Biochem 1998;179:159–67.

[19] Imasato H, Nagata K, Hashimoto S, Komori H, Inoue A. Objective evaluation of pain in various spinal diseases: neuropeptide immunoreactivity in the cerebrospinal fluid. Spinal Cord 1997;35:757–62.

[20] Lindh C, Thornwall M, Hansen AC, et al. Neuropeptide-converting enzymes in cerebrospinal fluid: activities increased in pain from herniated lumbar disc, but not from coxarthrosis. Acta Orthop Scand 1996;67:189–92.

[21] Ahn S-H, Cho Y-W, Ahn M-W, et al. MRNA expression of cytokines in herniated lumbar intervertebral discs. Spine 2002;27:911–7.

[22] Kang JD, Georgescu HI, Larkin L, et al. Herniated lumbar intervertebral discs spontaneously produce matrix metalloproteinases, nitric oxide, interleukin-6, and PGE2. Spine 1996;21:271–7.

[23] Takahashi H, Suguro T, Okazima Y, Motegi M, Okada Y, Kakiuchi T. Inflammatory cytokines in the herniated disc of the lumbar spine. Spine 1996;21:218–24.

[24] Burke JG, Watson RW, McCormack D, et al. Intervertebral discs, which cause low back pain, secrete high levels of proinflammatory mediators. J Bone Joint Surg [Br] 2002;84:196–201.

[25] Koes BW, Sholten RGPM, Mens JMA, et al. Epidural steroid injection for low back pain and sciatica: an updated systematic review of randomized clinical trials. Pain Digest 1999;9:241–7.

[26] Ohtori S, Takahashi K, Chiba T, et al. Substance P and calcitonin gene-related peptide immunoreactive sensory DRG neurons innervating the lumbar facet joints in rats. Autonom Neurosci Basic Clin 2000;86(1–2):13–7.

[27] Ohtori S, Takahashi K, Chiba T, et al. Substance P and calcitonin gene-related peptide immunoreactive sensory DRG neurons innervating the lumbar intervertebral discs in rats. Ann Anat 2002;184: 235–40.
[28] McCarron RF, Wimpee MW, Hudkins PG, et al. The inflammatory effect of nucleus pulposus: a possible element in the pathogenesis of low back pain. Spine 1987;12:760–4.
[29] Lindh C, Liu Z, Welin M, Ordeberg G, et al. Low calcitonin gene-related, peptide-like immunoreactivity in cerebrospinal fluid from chronic pain patients. Neuropeptides 1999;33:517–21.
[30] Lindh C, Liu Z, Lyrenas S, Ordeberg G, et al. Elevated cerebrospinal fluid substance P-like immunoreactivity in patients with painful osteoarthritis, but not in patients with rhizopathic pain from a herniated lumbar disc. Scand J Rheumatol 1997;26:468–72.

25 Years Ago in Spine

The Roland-Morris Disability Questionnaire

Many disability scales have been developed for patients with low back pain. Although there is no ''gold standard'' to evaluate disability in low back pain, the Roland-Morris Disability Questionnaire, developed 25 years ago, remains one of the most widely used questionnaires internationally for measuring self-rated disability because of back pain [1,2].

The Disability Questionnaire was developed as part of a study designed to describe the natural history of back pain in patients aged 16 to 64 years, who presented to a group practice with low back pain during one calendar year. The authors chose statements from the Sickness Impact Profile that covered a range of aspects of daily living, and the phrase ''because of my back'' was added to each statement to clarify the cause of the patient's disability. Patients can complete the Questionnaire without difficulty and without assistance in about 5 minutes.

The Roland-Morris Disability Questionnaire, is a simple, sensitive, and reliable outcome measure, increasing the chance that a true treatment effect will be detected. The Questionnaire demonstrates content and constructs validity, short-term repeatability, feasibility, and linguistic adaptation. An individual patient's score can vary from 0 (no disability) to 24 (severe disability), and the scores recorded by patients presenting with back pain in primary care suggested that the questionnaire could also be used with a more severely disabled population. Responses to the Questionnaire are not affected by age, sex, or social class, and the authors noted that the flexibility of the scoring system indicated it could be a useful outcome measure in a wide variety of clinical situations.

References

[1] Roland M, Morris R. A study of the natural history of back pain. Part I: Development of a reliable and sensitive measure of disability in low-back pain. Spine 1983;8:141–4.
[2] Roland M, Morris R. A study of the natural history of low-back pain. Part II: Development of guidelines for trials of treatment in primary care. Spine 1983;8:145–50.

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