

## SPINE SECTION

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### Original Research Article

# Are Outer Annular Fissures Stimulated During Diskography the Source of Diskogenic Low-Back Pain? An Analysis of Analgesic Diskography Data

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### ABSTRACT

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*Objective.* This study aimed to clarify whether painful annular fissures stimulated during provocation diskography are the likely source of diskogenic pain.

*Design.* A retrospective analysis was conducted of prospectively collected data.

*Setting.* Multidisciplinary, academic spine center.

*Patients.* The study was completed in a cohort of 28 consecutive patients who were enrolled presenting with 6 months duration of axial low-back pain recalcitrant to physical therapy, oral analgesics, and epidural steroid injections and who have diskogenic pain based on history, exam, magnetic resonance imaging, and diskography.

*Interventions.* Subjects underwent provocation diskography and analgesic diskography utilizing a balloon-tipped intradiskal catheter allowing intradiskal injection of anesthetic.

*Outcome Measures.* Visual analog scale, finger-to-floor distance were utilized as outcome measures.

*Results.* 80% of painful intervertebral disks as detected by provocation diskography were sufficiently anesthetized resulting in >50% reduction in low-back pain during analgesic diskography.

*Conclusion.* Diskogenic pain is in varying degrees caused by the sensitized nociceptors within annular tears.

*Key Words.* Analgesic Diskography; Provocation Diskography; Annular Fissure

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### Introduction

The intervertebral disk is the most common source of chronic axial lumbar pain [1–3]. Chemical mediators of inflammation are elevated in both annular and nuclear tissue of painfully degenerated lumbar intervertebral disks [4,5]. Cyclical mechanical loading coupled with inflammatory stimuli increases prostaglandin E2

production by both nuclear and annular cells *in vitro*, with the latter causing a stronger reactivity than the former [6]. Compared with asymptomatic disks, painful disrupted lumbar intervertebral disks have higher concentrations of sensory fibers in their endplates and nucleus [1,7]. This high concentration of sensory fibers, combined with increased levels of proinflammatory mediators such as IL-8 and PGE<sub>2</sub>, provides a substrate for hyperalgesia and presumably pain [4,8]. In this hyperalgesic state, even normal mechanical loading will be painful.

It is difficult to prove that axial back pain is caused by painful annular disruption. The history and physical examination are unreliable and lack validity [4,9,10]. Conventional imaging does not distinguish diskogenic pain from other sources of back pain and does not distinguish symptomatic from asymptomatic subjects [11–16]. The presence of annular tears extending to the outer third of the annulus, however, is the strongest predictor of concordant low-back pain (LBP) during disk stimulation [17–19]. According to Bogduk, “in no other instance in the study of low back pain have such strong correlations been obtained between demonstrable morphology and symptom-reproduction” [20]. The current operational criterion for identifying painful annular tears is reproduction of concordant pain upon injection of nonionic contrast into the disk nucleus flowing into radial annular tears [21].

If LBP is, indeed, caused by sensitized nociceptors that are provoked by disk stimulation, then, anesthetizing the disk should relieve that pain and should protect patients from aggravation of their pain by functional maneuvers for the duration of action of the local anesthetic. The converse, null hypothesis is that anesthetizing the disk should not afford patients any relief of their pain. Allowing for a placebo response rate of 30%, the null hypothesis could be rejected, in the first instance, using a sample of at least 30 subjects if more than 50% of subjects achieved relief. Such an outcome seemed achievable because previous studies of intradiskal injections of local anesthetic have indicated that 75–100% achieve relief [22,23].

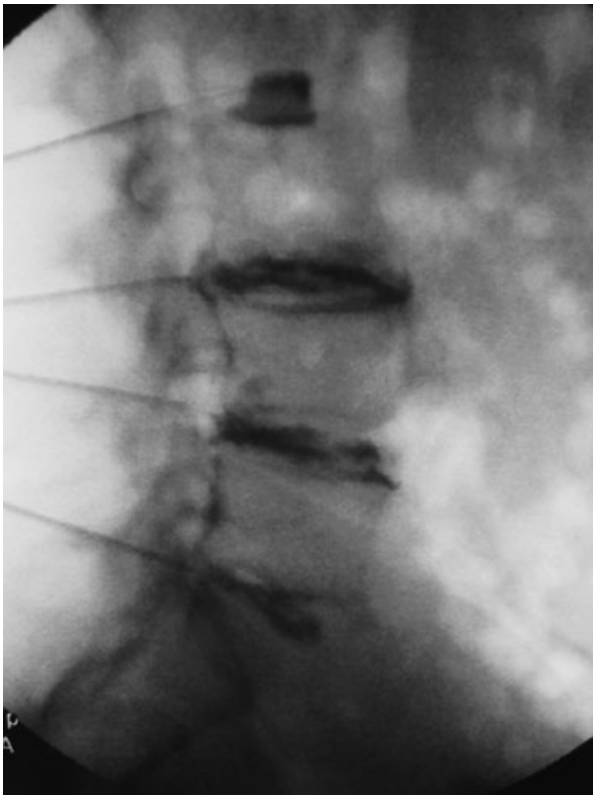
Analgesic diskography can be performed by inserting a balloon-tipped catheter into the symptomatic disk after completion of provocation diskography [24]. The balloon is inflated with contrast for visualization and serves to anchor the catheter within the nucleus during functional maneuvers. Each patient is asked to assume a series of positions that consistently exacerbates his/her

LBP. The symptomatic disk(s) is then anesthetized, and, after a time delay, the patient repeats the painful maneuvers, and a change in pain and function is noted. The intradiskal catheter utilized in our study has U.S. Food and Drug Administration (FDA) approval for intradiskal use for diagnostic purposes and has been used safely in approximately 3,000 patients to date [25].

### Materials and Methods

After obtaining Institutional Review Board approval, prospectively collected data were retrospectively harvested and analyzed. The sample reported in this study was drawn from patients who had experienced functionally limiting chronic axial LBP for at least 6 months despite physical therapy, oral analgesic medications, nonsteroidal anti-inflammatory medications, and targeted transforaminal epidural steroid injections, and who eventually underwent provocation lumbar diskography of the L2-S1 intervertebral disks. The L1-L2 intervertebral disk was additionally stimulated in one patient. In appropriate cases, alternate sources of the LBP were ruled out by controlled, precision, diagnostic blocks of suspected facet or sacroiliac joints. Magnetic resonance imaging revealed at least one morphologically abnormal lumbar spine intervertebral disk.

Lumbar diskography was performed under fluoroscopic control by using a parapedicular, posterolateral approach [21] using an 18-gauge, 5-in. spinal introducer needle allowing placement of a 7- or 8-in. 22 gauge spinal needle within the nucleus of each disk [21]. Approximately 1.5–3.0 mL of omnipaque contrast dye, or gadolinium in the instance of a contrast-dye allergy, was injected into each disk under lateral fluoroscopic view (Figure 1) while assessing for concordant or partial concordant LBP, or no pain at all, and outer annular disruption. A positive diskogram was defined as  $\geq 7/10$  concordant or partial concordant LBP at low pressure occurring simultaneously with extension of contrast dye to the outer annular fibers on direct lateral fluoroscopic visualization with two negative adjacent control disks [21]. A guide wire was threaded through the spinal needle into the painful intervertebral disk nucleus over which the balloon-tipped catheter was placed into the nucleus after removing the spinal needle. Once the radiodense markers on the distal tip of the catheter passed the medial edge of the ipsilateral pedicle, the balloon was inflated with approximately 0.3 mL of contrast dye, confirming nuclear

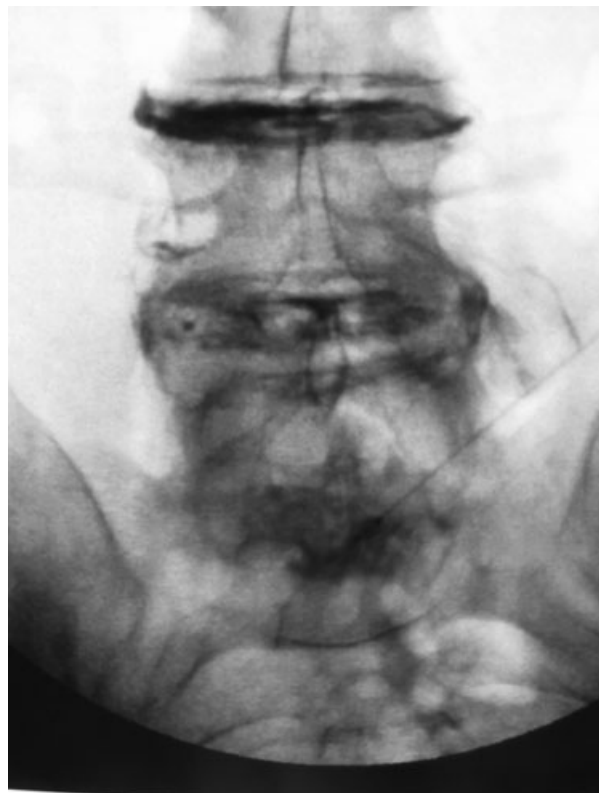


**Figure 1** Lateral fluoroscopic view during diskography depicting outer annular disruption by extension of contrast posteriorly within the outer annular fibers at L3-L4 to L5-S1.

placement fluoroscopically (Figure 2). Each patient then underwent postdiskography computed tomography, allowing confirmation of injection of contrast within the targeted nucleus as well as nuclear placement of the balloon-tipped catheter and grading of the annular disruption [26] (Figure 3). After each patient returned from the computed axial tomography (CT) scanner, baseline LBP scores were collected by using a visual analog score (VAS) from 0 to 10 of his or her LBP during unsupported sitting, forward flexion, and forward flexion while arms were extended with lumbar axial rotation. An amount 0.8 mL of 4% preservative-free xylocaine was injected into the painful disk(s) while recording the patient's report of LBP during this injection. After 5–10 minutes elapsed, 0.8 mL of sterile saline was injected to confirm that the disk was anesthetized by little or no pain provocation. The patient was blinded to the identity of the agent being injected and had been informed that either anesthetic or saline would be injected without revealing which prior to each injection. The painful positions and maneuvers were then repeated, and LBP was reassessed

by using the VAS. After these functional maneuvers were repeated, 0.8 mL of 0.5% marcaine was injected into the disk for long-term anesthetization, the balloon tip was then deflated, and the catheter was removed. A pressure dressing was applied over the catheter site. Duration of anesthetic affect was not routinely evaluated. Patients were deemed to have been relieved of their pain and protected from aggravation, if they reported at least 50% reduction of their pain at rest and during maneuvers. This criterion was adopted in accordance with previous studies of other sources of back pain, in which 50% relief of pain has been deemed as a clinical meaningful reduction of pain [27,28].

Included in the analysis were the data obtained from patients who reported concordant or partial concordant pain ( $\geq 7/10$  on a numerical pain scale), at one or two levels during diskography, with two negative responses at adjacent, control levels; extension of contrast dye to the outer annular fibers at the time of concordant LBP; grade III or greater annular tear on postdiskography computed



**Figure 2** Anteroposterior fluoroscopic view of the balloon-tipped catheter within the L5-S1 disk space with the balloon filled with contrast dye surrounded by a proximal and distal radiodense marker.



**Figure 3** Post-diskography, axial CT scan of a left grade IV annular tear at L4-L5 with the analgesic catheter located within the nucleus.

tomography; reproduction of lumbar pain upon injection of 0.8 mL of 4% xylocaine but only pressure upon subsequent injection of 0.8 mL of sterile saline into the painful disk(s) during the analgesic diskography procedure.

Excluded were patients who had a painful Schmorl’s node, concordant or partial concordant pain without outer annular disruption, or more than two painful disks during diskography.

**Results**

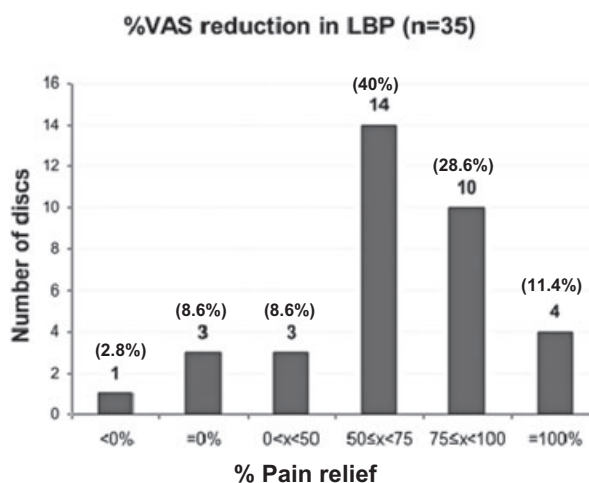
A total of 37 lumbar intervertebral disks in 30 consecutive patients, 17 males, underwent both provocation lumbar diskography and analgesic diskography (AD). Two subjects, two total disks, were excluded due to the absence of outer annular fissures (Table 1). The mean age of the subjects was 41 years (20–61 years). Seventeen L5-S1 disks, 17 L4-L5 disks, and 1 L1-L2 disk were evaluated. Statistical analysis was performed by using SPSS 10.0 software (SPSS, Inc., Chicago, IL). The mean percentage reduction of pain during AD was  $59.77 \pm 30.13$  (Figure 4), which was statistically significant using a Student *t*-test,  $P < 0.001$ . Post-diskography CT scans revealed 13 Grade V, 13 Grade IV, and 9 Grade III annular tears [26] occurring in the concordant or partially concordant lumbar disks. Overall, greater than 50% reduction of LBP was achieved after anesthetiza-

**Table 1** Subject Demographics

Patients characteristics	
Sex (M : F)	16:12
Age (years)	40.6 ± 10.4
Height (in.)	69.1 ± 3.7
Weight (lbs)	214.1 ± 55.1
CT tear (grade 3:4:5)	9:13:13
Disk level	
L1-2	1
L2-3	0
L3-4	0
L4-5	17
L5-S1	17
Concordance	
Partial	12
Full	23
Psychological history	
No history	22
Depression	11
Alcoholism	1
Drugs/Bipolar	1
AD result	
Corroborate	28
None	7
Single level	21 pts
Two levels	7 pts

CT = computed axial tomography; AD = analgesic diskography.

tion of 28 disks (80%) during the functional tasks during AD. There was no statistically significant difference between concordant and partially concordant LBP and pain reduction, between negative psychiatric history and history of depression (Chi-square test  $P = 0.221$ ), or between single-level and two-level painful disks. Eleven patients presented a history of depression that was mild, and one patient had a history of bipolar disorder that was not functionally limiting at the time of presentation.



**Figure 4** Bar graph of the distribution of lumbar intervertebral disks resulting in variable degrees of disk analgesia.

## Discussion

Injecting 0.8 mL of 4% xylocaine into disks diagnosed as the source of chronic LBP by data combined from history, physical examination, imaging studies, and stringent provocation diskography alleviated LBP. Specifically, we found that 80% of painful lumbar intervertebral disks were sufficiently anesthetized, achieving equal to or greater than 50% pain relief (Figure 4). This response is incompatible with the null hypothesis that the disks are not the source of pain and supports our postulate that diskogenic pain is in varying degrees caused by the sensitized nociceptors within annular tears.

Heretofore, the concept of diskogenic pain caused by nociceptive ingrowth into annular fissures has been supported only indirectly by observing a strong correlation between pain provocation and the presence of annular fissure during provocation diskography and subsequent CT imaging. In 1992, Maezawa and Muro reported that injecting contrast into disks with herniations caused pain [29]. Years earlier, Vanharanta et al., in a series of independent articles, studied patients with axial back pain without significant disk herniations. They reported a strong correlation between pain provocation during disk stimulation and age, degree of disk degeneration, and degree of annular disruption [17,26,30,31]. However, their findings were obtained by using univariate statistical calculations and thus were at risk for finding spurious associations [18]. Employing a multivariate statistical technique to review 833 diskograms, Moneta et al. found that the presence of outer annular disruption was the best predictor of pain reproduction during diskography [18]. Indeed, 75% of Grade III [26] annular tears are associated with concordant or similar LBP during diskography [17], and, conversely, 77–95% of concordantly painful lumbar intervertebral disks exhibit  $\geq$  Grade III [26] annular tears [17,19]. Moneta et al. did, however, underscore the fact that their findings provided only circumstantial evidence that the lumbar pain produced by outer annular stimulation during diskography is in fact the source of chronic LBP [18]. A cause-and-effect relationship cannot be confirmed by their observations [18]. Nonetheless, Moneta et al.'s [18] and Vanharanta et al.'s [17,26,30,31] clinical observations are supported by immunohistochemical studies [1,32] demonstrating few nociceptors and rare pain provocation in the inner annulus, whereas there are high densities of

nociceptive nerve fibers in frequently painful outer annular tears.

As depicted in Figure 4, patients reported varying degrees of pain relief during provocative testing. Ideally, all patients should have reported complete relief of their pain. However, several mitigating factors explain the absence of this outcome.

Painful disks exhibit differences in thresholds to mechanical stimulation. Some are sensitive to low pressures of injection; others require moderate pressures [33]. Stress gradients within the disk differ with varying degrees of annular disruption and stages of degeneration [34,35].

A low volume of 0.8 mL of local anesthetic injected into the nucleus may not reach or only reach in insufficient concentration to anesthetize nociceptors responsible for pain caused by these high stress gradients. In addition, it is possible that elevation in intradiskal pH within the injured disk [3] may hinder an effective anesthetic block. Finally, both provocative and analgesic disk testing require a subjective response from the patient. As such, circa 80% of patients reporting at least 50% pain relief may represent the average ceiling where one cannot fully control for confounding factors associated with subjective patient responses.

The findings of this audit support Moneta et al.'s suggestion that annular fissures reaching the outer annular fibers are in fact the source of symptoms in patients suffering from chronic diskogenic LBP. Presumably, painful nociceptors within torn annular fissures or the immediately adjacent end plate or disk outer annulus, are the source of diskogenic pain. Examination of Figures 1 and 3 demonstrates contrast adjacent to the end plate as it extends into the outer annulus. However, pain provocation clearly occurred in each case at the moment when the contrast extended to the outer annular fibers, not as it approximated the end plate. Additionally, concordant pain upon disk stimulation was documented at low pressure (<50 psi). End plate deflection and presumably end plate mediated pain occur at intradiskal pressures >50 psi [36]. Controlled disk analgesia, of these outer annular fissures, can be achieved via an indwelling intradiskal catheter and measured by subjective patient report and physical performance measures. The pointed ramification of these findings is confirmation that 1) outer annular fissures as identified by stringent provocation diskography are the source of pain in patients with chronic diskogenic LBP; and 2) tissue specific therapeutic interventions targeting painful outer

annular fissures are appropriate. Future work is now warranted to verify how certain parameters—Modic endplate changes, intradiskal pH, exact pressure upon pain provocation, disk dessication, intradiskal gas, intradiskal contrast dye, militating factors such as litigation, worker's compensation claims, and psychometric parameters—affect the degree of disk analgesia in single-level and multi-level painful lumbar disks.

### Disclosures

First author is paid consultant to Kyphon/Medtronic and serves on the Clinical Advisory Board for Functional Anesthetic Discography product.

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