Discogenic back pain is a syndrome of nonradicular pain in the absence of spinal deformity, instability, and neural tension signs.¹ Pain generators from discogenic pain are believed to be produced from the nociceptive fibers through the sinuvertebral nerve, which is stimulated by fissures or tears in the posterior longitudinal ligament and annulus fibrosus.²⁻⁴ More specifically, the outer posterolateral part of the annulus has a rich sensory innervation.⁵ It has been documented that the outer annulus is the tissue of origin in most cases of low back pain.⁶ It is conceivable, then, that increased discal pressure can cause stretching of the annulus and firing of these nerve endings. It is also believed that provocative discograms will reproduce this pain. Discograms had previously been used exclusively in determining the anatomic segment thought to be responsible for the patient’s pain. This use has been controversial, and many published studies have supported⁷⁻⁹ or refuted it.¹⁰⁻¹²

Studies that accepted discography as a valid diagnostic tool treated patients with positive discograms. Surgical treatment outcomes for percutaneous discotomy, discectomy, interbody fusion, posterior lumbar interbody fusion, and global fusion revealed success rates from 30 to 96%.¹³ Donelson et al. reported that the McKenzie assessment reliably differentiated discogenic
pain from nondiscogenic pain as well as competent from an incompetent annulus in symptomatic discs and was superior to magnetic resonance imaging (MRI) in distinguishing painful from nonpainful discs.14

In 1986, Choy and Ascher first employed laser technology to decompress discs.15 This procedure is based on the concept that the intervertebral disc, when contained by the annulus and the dorsal longitudinal ligaments, is a closed hydraulic space, subject to pressure and volume relationships. Choy and others have demonstrated that a small decrease in intradiscal volume results in a substantial reduction on intradiscal pressure.16,17 Despite refinements in technique and the recognition that discogenic pain can cause low back pain, percutaneous laser disc decompression (PLDD) has been recommended only for cases of lumbar radiculopathy and not for low back pain alone. However, over the course of time and aided by the work of Botsford, with discography, low back pain caused by central herniation or disc degeneration (discogenic pain) seemed to be treatable with PLDD. It can be theorized that the decompressive affects of PLDD reduce the activity of the sensory fibers of the outer annulus, decreasing pain sensation.

The objective of this retrospective study is, to evaluate patients (see Table 1.2), using the McNab criteria who were treated with PLDD for discogenic pain. Until now, no long-term follow-up on this patient population has been performed.

A total of 37 patients were selected. The majority of the patients (32) were selected from the Laser Spine Center (D.S.J. Choy) based solely on their clinical symptoms of low back pain with absence of radicular symptoms on initial evaluation. The other five, from Northeastern Neurological Associates (W.A. Black), were selected based on clinical findings as well as a concordant provocative discogram. Patients who underwent PLDD from December 14, 1993 to June 19, 2001, were selected for attempted interview via telephone to evaluate their response to PLDD as outlined by the McNab criteria. One patient was deceased, and three could not be contacted. This left 32 patients participating in the survey.

**Case Studies**

**Case JB**

This patient was first seen in November 1993 with a 3-year history of low back pain, which was progressive. JB had been treated with reduction of activity (quit playing tennis) and chiropractic manipulations. Low back pain occurred with sitting, bending, lifting, or standing in one position. Only changes in activity gave relief. He became intolerant.
Discography performed on July 18, 1994, showed central disc herniation of L2-3 and L3-4 with reproduction of low back pain. The L3-4 pain was more pronounced than L2-3; L1-2 showed no herniation and no reproduction of back pain. JB underwent PLDD on July 20, 1994, at L2-3 and L3-4. He experienced a postoperative facet syndrome, which was treated with exercise and methylprednisolone (Medrol) dose pack. During an interview in December 2001 the patient stated, "The surgery changed my life." He reported that he is able to play golf and engage in other activities that had not been possible before. At times JB still experiences low back pain, which is relieved with massage, but he acknowledged being out of shape and having dropped his exercise program.

Case TG

This patient was a 29-year-old auto mechanic who twisted while crouching and lifting. He had transient pain in the calf. He was treated with physical therapy for 3 months while working in a light-duty position and did well. However, he experienced chronic, recurrent back pain with bending and lifting and could not return to his profession. His MRI scans of June 1992 and 1993 demonstrated Schmorl’s node in the superior end plate of L3. He was seen in June 1994 because of 8 months of inability to work secondary to back pain. A discogram of L2-3 reproduced his back pain, and the contrast medium entered the Schmorl’s node. PLDD, performed on November 19, 1994, resulted in relief of back pain and TG’s return to employment as an auto mechanic. During an interview on December 12, 2001, the patient reported that he continues to work as an auto mechanic. His work includes bending over into engines. He does note occasional back pain, which can be relieved by ibuprofen (Motrin).

Case Summaries

More specifically, 23 male and 9 female patients were contacted. PLDD was performed on 59 separate disc levels: one each at T8-9, T12-13, and L1-2; 4 at L2-3, 11 at L3-4; 20 at L4-5; and 21 at L5-S1 (Table 16.1).

Survey results revealed that 14 patients (44%) reported a good response, 14 (44%) reported a fair response, and 4 (12.5%) reported a poor response. The 28 patients (88%) who reported good or fair responses were considered to represent successful cases. Those 4 patients (12.5%) who reported a poor response were considered to be treatment failures. Of these 4 failures, 2 reported reinjury as the cause of pain in the treated discs. The remaining 2 patients reported having relief postprocedure for a few days before their preprocedure pain levels returned.
Conclusions

These results indicate that discogenic back pain, whether diagnosed clinically or via discogram, is amenable to PLDD. In the future, more comprehensive studies must be performed to support these findings. Based on this small study population, it can be concluded that PLDD may be an effective treatment for discogenic back pain with minimal natural recurrence.

References

7. Birney TJ, et al. Comparison of MRI and discography in the diag-