Techniques in Regional Anesthesia and Pain Management (2009) 13, 85-92

ELSEVIER

Techniques in
Regional Anesthesia
& Pain Management

Lumbar discography: Diagnostic role in discogenic pain

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KEYWORDS:
Discogenic pain;
Discography;
Lumbar;
Back pain;
Disk degeneration;
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Although modern imaging techniques such as magnetic resonance imaging and computed tomography are useful in detecting anatomic abnormalities within the intervertebral disc, they cannot provide any information regarding the presence or absence of pain arising from anatomic abnormalities. Discography is currently the only diagnostic method that is available to determine whether an anatomically abnormal intervertebral disc is the cause of a given patient's ongoing pain. However, discography is a subjective test, relying entirely on the patient's pain experience during the conduct of this brief procedure, which is without effective controls. This article discusses the current scientific evidence regarding the use of discography at lumbar spinal levels, describes the technique and associated complications, and explores the controversies surrounding the usefulness of this diagnostic test.

Lindblom first reported the nucleographic patterns of 13 patients in 1948, the first description of what would come to be termed discography, and controversy has surrounded the use of this approach ever since.[1] The term "discography" refers to the injection of radiographic contrast material into the nucleus pulposus of the intervertebral disc coupled with radiographs to determine the pattern of contrast spread. The methods to conduct this study and its perceived usefulness have changed markedly with time. In the 1950s and early 1960s, discography became the imaging study of choice for delineating anatomic abnormalities within the intervertebral disc, particularly herniation of the nucleus pulposus.[1-3] In recent decades, this application of discography has been replaced by newer and less invasive imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), which enable clinicians to assess structural changes within the discs. Although imaging of the disc has improved, there remains a lack of correlation between clinical symptoms and anatomic abnormalities identified on imaging studies.[4-6] Indeed, studies from four decades past had already shown little correlation between morphologic changes within the intervertebral disc and the presence of pain.[7,8]

Cervical and thoracic discs may well differ from the lumbar discs in the pathologic process leading to pain,[9] and our understanding of the role of discography at these spinal levels is even less clear, thus we will limit our discussion to the lumbar discography.

Discogenic pain and disc degeneration

Our understanding of the pathophysiology of disc degeneration, and pain originating from such a disc, is evolving. The term "discogenic pain" is used to describe pain emanating from an intervertebral disc and not the surrounding neural structures. Classically, discogenic pain is described

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1084-208X/$ -see front matter
doi:10.1053/j.trap.2009.05.004

as axial low back pain, exacerbated by activities that increase intradiscal pressure, such as coughing, straining, or rotating. Studies that have looked at pain referral patterns of patients undergoing discography have reported that, in addition to axial low back pain, they may present pain in one or both lower extremities that mimics radicular pain.[10,11] Although conflicting evidence exists on the prevalence of discogenic pain, studies suggest that it is an important source of chronic low back pain in approximately one-third of patients.[12-14]

The normal lumbar intervertebral disc receives sensory innervation that extends into the outer third of the annulus.[15] The nerves supplying the dorsal aspect of the disc appear to differ from those that innervate the anterior and lateral portions of the discs. Posteriorly, the disc is innervated by the sinuvertebral nerve, which is formed by a recurrent branch of the ventral ramus and a branch of the gray ramus communicates.[16] Each lumbar sinuvertebral nerve sends branches superiorly and inferiorly, supplying the disc above and below, in addition to the disc at the level of origin of the nerve. The ventral surface of the disc receives its innervation from a plexus formed by the sympathetic trunk, its rami communicantes, and perivascular nerves. Branches from the gray rami communicantes also supply the lateral surface of the annulus.[17] In degenerated discs, the innervation appears to be widespread and deeper, such that nerve fibers can reach and penetrate the nucleus pulposus.[17,18]

Disc degeneration can result from multiple insults, including aging. Normally, discs receive their nutrients by diffusion through the vertebral end plate. The traditional concept is that age-related changes are secondary to a decrease in blood flow to the endplates, resulting in diminishing nutrient supply to the intervertebral disc.[19-21] In a cadaveric study, Boos and coworkers[21] showed diminishing blood supply to the endplate as early as 10 years of age. The obliteration of blood vessels paralleled an increase in cartilage disorganization, endplate density, and microfractures.[21] On a microscopic level, the concentration of proteoglycans in the nucleus pulposus declines with age, as well as the proteins that link them.[22] The type of collagen within the nucleus transforms from type II to type I, which makes the nucleus more fibrous.[22] With increasing collagen–proteoglycan binding, fewer polar groups are available to bind water, progressively dehydrating the nucleus.

Some of the features of disc degeneration have been associated with other factors. Diminished disc height, a common surrogate for degenerative disc disease, is far more common in the lower lumbar levels than in the upper lumbar spine, suggesting a mechanical factor.[23] Similarly, the presence, severity, and frequency of radial tears within the annulus fibrosus do not seem to correlate with age.[24] Environmental factors, such as smoking, also result in changes consistent with disc degeneration. In a study by Akmal and coworkers,[25] nicotine inhibited proliferation and synthesis of extracellular matrix in cells from bovine nucleus pulposus. Other factors, including vibration,[26] torsion[27,28] and compression,[29] as well as genetics,[30,31] toxic,[25] and metabolic factors,[32] have all been implicated in the pathogenesis of degenerative disc disease.

Discography as a pain challenge

Discography is currently used most widely to determine whether a given intervertebral disc is the source of back pain. It is thought to stimulate nociceptive nerve endings within the disc via mechanical distortion induced through raising the pressure within the central portion of each disc. Whereas proponents of discography insist that it is the only diagnostic modality that correlates disc pathology and symptoms, opponents question the validity of provoked symptoms. In the 1960s, two studies by Holt dismissed the usefulness of discography because of high false-positive rates in cervical (100%) and lumbar (37%) discs in a population of prison inmates.[33,34] Holt's data have received widespread criticism because of methodology flaws, including selection of test subjects, the high technical failure rate, neurotoxicity of the injectate, and the technique used in the studies. More recent controlled prospective studies and a recent meta-analysis have reported low false-positive rates ranging from 0% to 10%.[35-38]

Efforts to reduce the false-positives rates have focused on proposing more restrictive criteria for interpreting a positive result. One such restriction is to limit the pressure exerted within the disc during discography through use of pressure monitoring. It is clear that many normal volunteers without back pain will report pain during provocative discography if high pressure is applied within the disc. Derby and coworkers[36] recruited 13 volunteers without low back pain and performed discography in 43 discs, looking at intensity of response and pressure of injection. They found that 56% of the discs were not painful regardless of the pressure generated during injection. When asymptomatic discs became painful, the pain was usually mild, with subjects rating an average pain score of only 2 or 3 on a 10-point scale. Another study agreed with these findings, showing that the group with a concordant response (pain similar to the usual back pain) reported higher pain scores at lower intradiscal pressure than the group with discordant pain (pain unlike their usual back pain).[39]

However, not all studies agree. Carragee and coworkers[40] tested the hypothesis that adding "pain produced with low injection pressure" to the established diagnostic criteria would reduce the rate of false positives. Although this was a retrospective review of the data from three previously published studies, the results did not support this hypothesis. Later, in a prospective cohort study, Carragee and coworkers compared a group of patients with pain undergoing spinal fusion after a positive, single level discogram to a control group of patients undergoing spinal fusion for an unstable spondylolisthesis.[41] The "discogenic pain" group consisted of 30 patients with axial low back pain who had failed conservative therapy and had a positive, single-level,