Intradiscal Pressure Monitoring in the Cervical Spine
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INTRODUCTION: Discography has been widely used in the lumbar and cervical spine as a diagnostic tool to identify sources of discogenic pain that may be amenable to surgical treatment. In the lumbar spine, intradiscal pressure monitoring as an adjunct to traditional provocative discography has aided the discographer in determining clinically significant internal disc disruption. The addition of pressure monitoring has increased the interobserver reliability and reproducibility, as well as the objectivity of the study. A pressure gradient of up to 80-90 psi is used to determine the high end of physiological pressure in the lumbar spine. Intradiscal pressure also provides an endpoint at which the disc may be graded normal in the absence of concordant pain. Minimum lumbar intradiscal rupture pressure has been determined to be 110-212 psi, and this has provided a safety parameter for lumbar discography. Additional in-vivo investigation has shown that there is no transmission of pressure to adjacent discs in the lumbar spine when a normal appearing disc with a concordant pain response is infused at 100 psi.

Discography in the cervical spine is currently performed without the benefit of pressure monitoring, and corresponding pressure parameters have not been determined. The purpose of this study was to develop the framework for intradiscal pressure monitoring in the cervical spine and the basis for a pressure curve that will reflect clinically significant cervical internal disc disruption. We also sought to determine whether there is any pressure increase in adjacent discs during cervical discography that might result in false-positive diagnosis during in-vivo discography. An additional goal was to establish safe upper parameters for infusion volume and intradiscal pressure in the cervical spine.

METHODS: We investigated 26 discs in 5 fresh-frozen cadaveric cervical spines aged 45 to 68 with no prior history of cervical spine disease. T2 MRI was performed on each specimen and radiographically abnormal discs were noted. Pressure-controlled, fluoroscopically guided discography was performed on each level using a right lateral approach. A 25G spinal needle was placed into each disc and Omnipaque 300 (Nycomed, Princeton, NJ) contrast dye was infused utilizing a pressure-controlled inflation syringe with digital pressure monitor (Merit Universal Syringe, MeritMedical, South Jordan, UT). The operating pressure range of the monitor was 0 - 367 psi (+/- 2.5%). Opening
pressure, rupture pressure, volume infused, and location of rupture were recorded. Pressures were simultaneously recorded at each adjacent disc level using additional pressure monitors and identical needle placement. Immediately following discography, CT was performed on each specimen according to the discography protocol. A musculoskeletal radiologist who was blinded to the pressure and volume recordings read all radiographic studies.

RESULTS: 26 discs C2-3 to C7-T1 were grossly intact for evaluation. The median opening pressure was 30 psi (range 14 – 101 psi). Two discs did not rupture and were pressurized to 367 psi. In 24 discs, the median intradiscal rupture pressure was 40 psi (range 14 – 171 psi). The median volume infused at rupture was 0.5 ml (range 0.25 – 1.0 ml). When grouped, the median intradiscal rupture pressure in the C2-3, C3-4, and C7-T1 discs was 53 psi (range 16 - 171 psi) compared to 36.5 psi (range 14 – 150 psi) in the C4-5, C5-6, and C6-7 discs. There was no measurable pressure change in any of the 30 adjacent disc levels evaluated.

CONCLUSIONS: Investigation of intradiscal rupture pressure exhibited a bimodal distribution with higher intradiscal rupture pressures observed in the C2-3, C3-4, and C7-T1 discs compared with those of the C4-5, C5-6, and C6-7 discs. Despite maximum pressurization, there was no transmission of pressure to adjacent cervical discs. During in-vivo provocative discography, it is possible to observe a disc with normal appearance and dye distribution, but with a positive concordant pain response. Similar to findings in the lumbar spine, this finding suggests that pressure transmission to an adjacent symptomatic level is not the pathophysiology with such an observation. The minimum pressure for disc rupture was 14 psi with infusion of only 0.25 ml. Thus, cervical discs may have appreciably lower pressure parameters than those of lumbar spine, and the potential for iatrogenic disc injury may exist with low pressures and volumes. The cadaveric testing in this study was limited by sample size, but begins to establish parameters for intradiscal pressure measurement as a component of provocative cervical discography. Further cadaveric testing is necessary to develop robust parameters for intradiscal pressure monitoring in the cervical spine.