Neuraxial Blockade in Patients with Spinal Stenosis: Between a Rock and a Hard Place

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Although severe or disabling neurologic complications after neuraxial block are rare, an epidemiologic series suggests that the frequency of some serious complications is increasing.[1] The presence of new or progressive neurologic deficits necessitates prompt evaluation to detect potentially treatable sources of neurologic injury. In this setting, magnetic resonance imaging (MRI) is the preferred technique to diagnose spinal hematoma, epidural abscess, and mechanical trauma; acute spinal cord ischemia may be undetectable by conventional MRI. However, prior performance of a neuraxial technique may affect interpretation of the images. Radiologists must discern between benign "coincidental" findings, normal procedural-related changes, and those that represent pathologic processes. For example, spinal MRI findings in patients receiving continuous epidural analgesia may mimic those of epidural abscess (e.g., posterior epidural enhanced "lesion" with spinal cord compression) even in the absence of infection.[2] Misinterpretation of MRI findings may lead to unnecessary therapies, including surgery. Despite these implications, MRI after uneventful neuraxial techniques remains largely undefined. Previous investigations have involved single cases or small series.[2,3]

In this issue of *Anesthesia & Analgesia*, Davidson et al.[4] systematically characterized the MRI findings of 30 parturients, 15 of whom had undergone a combined spinal epidural technique, to define normal MRI appearance after uneventful epidural analgesia. MRIs were performed approximately 10 h after delivery. There were no significant fluid collections, hematomas, or compression of the thecal sac noted in any of the MRI studies. However, the presence of an injection track, abnormal soft tissue abnormalities, and/or epidural air allowed the image readers to correctly identify which parturients had undergone a neuraxial technique in 93% of cases. The investigators concluded that the lack of pathologic MRI findings after uncomplicated epidural analgesia suggests that the presence of significant fluid collection or mass effect, in the setting of new neurologic deficits, warrants immediate intervention. This imaging study illuminates our understanding of the anatomic changes, as defined by MRI, induced by neuraxial block. Additional studies are needed to characterize the MRI findings in other patient populations, particularly those with preexisting pathology of the vertebral column, such as spinal stenosis. This knowledge is crucial in understanding the apparent increased risk of neurologic complications associated with neuraxial blockade in these patients.

Pathology of the spine has been proposed as a risk factor for complications after neuraxial techniques. Recent series and case reports support this hypothesis, although the mechanism of injury, ischemia, or neurotoxicity is unclear. An epidemiologic study evaluating severe neurologic complications after neuraxial block conducted in Sweden between 1990 and 1999 revealed some disturbing trends.[1] During the 10-yr study period, approximately 1,260,000 spinal and 450,000 epidural blocks (including 200,000 epidural blocks for labor analgesia) were performed. A total of 127 serious complications were noted, including spinal hematoma,[33] cauda equina syndrome/paraparesis,[36] meningitis,[29] and epidural abscess.[13] The nerve

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damage was permanent in 85 patients. Fourteen of the patients had preexisting spinal stenosis. However, the spinal stenosis was known preoperatively in only 1 case; the remaining 13 cases were diagnosed in the subsequent investigation of the complication. Furthermore, in patients with spinal stenosis, the frequency of cauda equina syndrome and spinal hematoma increased with age. This large series suggests that the incidence of severe anesthesia-related complications is not as low as previously reported (the overall frequency was approximately 1:10,000) and preexisting spinal canal pathology may be a "neglected risk factor."[1] A 1-yr survey in France of nerve root and spinal cord injury after neuraxial block revealed 12 cases of severe and long-lasting complications, including 5 cases of spinal stenosis and 2 spinal arachnoid cysts.[5] A growing number of case reports implicate severe asymptomatic spinal stenosis as a contributing factor in the occurrence or severity of nerve injury after neuraxial block.[6–9] The majority of cauda equina cases involved epidural analgesia, suggesting an ischemic component (from mechanical compression of the cord by the infusate) to the injury.

Spinal stenosis is a narrowing of the spinal canal and neural foramina produced by age-associated changes in the disks and facet joints, including disk degeneration, facet joint capsule hypertrophy, infolding of the ligamentum flavum, and osteophyte formation.[10] The mechanism by which spinal nerve root compression results in the signs and symptoms of spinal stenosis (back/leg radicular pain, usually without sensory or motor deficits, which is exacerbated with extension and alleviated flexion) has not been fully explained. However, both laboratory and clinical models correlate symptomatology to increases in intraspinal pressure.[11] An increase in mechanical pressure as low as 10 mm Hg may produce venous occlusion as well as reduce cerebrospinal fluid and blood flow, resulting in metabolic impairment of the nerve roots and spinal cord.[12] This is notable in that increases in epidural pressure are common in elderly patients undergoing epidural analgesia. In a classic investigation reported in 1967, Usubiaga et al.[13] measured epidural pressures in 405 patients scheduled for elective surgery. In all patients, epidural injection of 10 mL of lidocaine produced an instantaneous increase in epidural pressure; peak pressures ranged from 5 to 65 cm H_2O (4 to 40 mm Hg). The highest pressures occurred in sitting patients. After injection, the pressure "normalized" within 2 min in patients younger than 50 yr. A slow rate of descent, with higher residual pressures, was reported in elderly patients. The pressure changes were transmitted to the intrathecal space at the same level. Finally, the authors correlated high epidural pressures with extent of block, which was interpreted as a "confinement of a larger amount of solution inside the epidural space" due to age-related changes. The effect of epidural injection on cerebrospinal fluid displacement was more recently assessed by Takiguchi et al.[14] who reported that injection of 10 mL of epidural saline 10 min after spinal anesthesia resulted in a spinal level 4 segments higher. In addition, serial epidural injections of 5 mL (to a total of 20 mL) resulted in a reduction in the diameter of the subarachnoid space to approximately 40% after the first injection and to 25% after the second injection. Further decreases were observed with the third and fourth injections. Although these effects may not be clinically significant in many patients, in combination with spinal stenosis, they may result in irreversible neural compromise. Essentially, the prolonged increase of epidural pressure may exacerbate preexisting pathology and increase the risk of nerve root ischemia. The ischemic effects may be further enhanced by the neurotoxicity of local anesthetic solution.

The relative risk of neuraxial blockade in patients with preexisting spinal canal pathology is unknown. In a series of 230 patients undergoing spinal anesthesia, the frequency of paresthesia during needle placement (20% vs 9%) or injection (16% vs 6%) was higher in patients with known lumbar spine pathology compared with those with normal spines.[15] Importantly, although the elicitation of a paresthesia may increase the risk of postoperative persistent paresthesia, no patient developed transient or permanent nerve deficits. Conversely, the cases of cauda equina syndrome/paraparesis often occur after an uneventful neuraxial technique.[1,7,8] A single study, for which only preliminary results are available, examined the overall success and neurologic complication rates among 937 patients with spinal stenosis or lumbar disk disease undergoing neuraxial block between 1988 and 2000.* Of these, 210 patients had a coexisting peripheral neuropathy in addition to their spinal cord pathology. Neurologic diagnoses were present 5 ± 6 yr; half of the patients had active symptoms at the time of the block. In addition, 207 patients had a history of spinal surgery before undergoing neuraxial block, although a large number of the procedures were simple laminectomies or discectomies. Ten patients (1.1%; 95% confidence interval 0.5%–2.0%) experienced new or progressive neurologic deficits when compared with perioperative findings. Although the majority of the deficits were related to surgical trauma or tourniquet ischemia, the neuraxial block was likely the primary etiology in 4 patients.

The preliminary nature of these data warrants care in their interpretation. Even more troubling are spontaneous cases of cauda equina syndrome that have occurred during general anesthesia in the absence of neuraxial block.[16] Additional large series and imaging studies are required to quantify the risk and characterize the mechanism of severe neurologic complications after uneventful neuraxial (primarily epidural)

*Hebl JR, Horlocker TT, Schroeder DR. Neurologic complications after neuraxial anesthesia or analgesia in patients with preexisting spinal stenosis or lumbar disc disease. Reg Anesth Pain Med 2005;29:A89.

blockade and the additive or synergistic contribution made by preexisting spinal stenosis. Until then, we are between a rock and a hard place.

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