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.

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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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Neuraxial Blockade in Patients with Preexisting Spinal Stenosis, Lumbar Disk Disease, or Prior Spine Surgery: Efficacy and Neurologic Complications

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BACKGROUND: Patients with spinal canal pathology, including spinal stenosis and lumbar disk disease, are often not considered candidates for neuraxial blockade because of the risk of exacerbating preexisting neurologic deficits or developing new neurologic dysfunction. In contrast, a history of spine surgery is thought to increase the likelihood of difficult or unsuccessful block. In this retrospective study we investigated the risk of neurologic complications and block efficacy in patients with preexisting spinal canal pathology, with or without a history of spine surgery, after neuraxial anesthesia.

METHODS: During the 15-year study period, all patients with a history of spinal stenosis or lumbar radiculopathy undergoing a neuraxial technique were studied. Patient demographics, preoperative neurologic diagnoses and neurologic findings at the time of surgery/neuraxial block, details of the neuraxial block including technique (spinal vs. epidural, single injection vs. continuous), injectate, technical complications (paresthesia elicitation, bloody needle/catheter placement, inability to advance catheter, accidental dural puncture), and block success were noted. New or progressive neurologic deficits were identified. All patients were followed until resolution or last date of evaluation.

RESULTS: There were 937 patients included, 207 (22%) of whom had undergone spinal surgery. A history of spinal stenosis was present in 187 (20%), lumbar radiculopathy in 570 (61%), and peripheral neuropathy in 210 (22%) patients; 180 patients (19%) had multiple neurologic diagnoses. A majority of patients had active but stable neurologic symptoms at the time of surgery. Overall block success was 97.2%. A history of spine surgery did not affect the success rate or frequency of technical complications. Ten (1.1%; 95% confidence interval [CI] 0.5%–2.0%) patients experienced new deficits or worsening of existing symptoms. Three (1.4%) complications occurred in patients with a history of spinal surgery, and the remaining 7 (1.0%) in patients without prior surgical decompression or stabilization (P = NS). Although an orthopedic procedure was not a risk factor, in 5 of the 6 patients in which the surgery was a unilateral lower extremity procedure, the postoperative deficit involved the operative side. Likewise, in both patients undergoing bilateral orthopedic procedures who developed bilateral deficits, the outcome was worse on the previously affected side. A surgical cause was presumed to be the primary etiology in 4 (40%) of 10 patients. The primary etiology of the remaining 6 (60%) complications was judged to be nonsurgical (including anesthetic-related factors). The presence of a preoperative diagnosis of compressive radiculopathy (P = 0.0495) or multiple neurologic diagnoses (P = 0.005) increased the risk of neurologic complications postoperatively.

CONCLUSIONS: We conclude that patients with preexisting spinal canal pathology have a higher incidence of neurologic complications after neuraxial blockade (1.1%; 95% CI 0.5%–2.0%) than that previously reported for patients without such underlying pathology. However, in the absence of a control group of surgical patients with similar anatomic pathology undergoing general anesthesia, we cannot determine whether the higher incidence of neurologic injury is secondary to the surgical procedure, the anesthetic technique, the natural history of spinal pathology, or a combination of factors and the relative contributions of each. (Anesth Analg 2010;111:1511–9)

Patients with preexisting spinal canal pathology, including spinal stenosis and lumbar disk disease (with or without prior spine surgery), are often not considered candidates for neuraxial blockade. For example, previous case reports have specifically attributed the presence of spinal stenosis or lumbar disk disease (with compressive radiculopathy) to an increased risk of neurologic complications. The mechanisms of injury are presumed to be ischemic,[1–3] mechanical trauma,[4,5] local anesthetic toxicity,[6,7] or a multifactorial etiology. Large series and an increasing number of case reports suggest that undiagnosed spinal stenosis may be a significant contributor to neurologic complications after neuraxial block.[3,5,6,8] Additional surgical factors such as intraoperative positioning, prolonged tourniquet ischemia, or high tourniquet inflation pressure may contribute in a synergistic (rather than simple additive) manner.[9–11] Furthermore, it is unclear whether patients who have undergone prior spine surgery (e.g., laminectomy, diskectomy, spinal fusion) for these conditions are at additional risk of neurologic injury or block failure secondary to anatomic alterations or scarring of the

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central canal. Previous investigations have included small numbers of patients, typically with a history of extensive instrumentation or fusion who underwent epidural catheter placement for labor analgesia.[12–15] However, the applicability of these results to other patient populations is unknown.

Therefore, the primary aim of this investigation was to examine the overall success and neurologic complication rates among patients with spinal stenosis or lumbar disk disease, with or without a history of prior spinal surgery, undergoing neuraxial blockade.

METHODS

After IRB approval and patient consent, the medical records of all patients with a history of spinal stenosis or lumbar disk disease (i.e., compressive radiculopathy) who underwent a subsequent spinal or epidural anesthetic during a 15-year study period were retrospectively reviewed. Patients were identified if "spinal stenosis" or "lumbar disk disease" was entered on their Master Diagnosis List within the Mayo Clinic database. Neurologic diagnoses were limited to abnormalities of the spinal canal and did not include patients with primary central nervous systems disorders such as multiple sclerosis, amyotrophic lateral sclerosis, or postpolio syndrome. All neurologic diagnoses were confirmed by clinical examination and radiographic imaging by a neurologist or neurosurgeon before study inclusion. During the 15-year study period, multiple anesthesiologists (>15 faculty members) participated in the care of all patients.

Demographic data including age, gender, height, and weight were collected. The date of each neurologic diagnosis and details such as the presence of (1) motor deficits, (2) sensory deficits, (3) paresthesias or dysesthesias, or (4) hyperreflexia at the time of their spinal or epidural anesthetic were collected for each patient. Neurologic symptoms at the time of their procedure were further classified as (1) acute (exacerbation of symptoms within the last 30 days), (2) subacute (exacerbation of symptoms within the last 1 to 6 months), or (3) chronic/stable (no change in symptoms within the last 6 months).

Indications for neuraxial anesthesia or analgesia included surgical anesthesia, labor analgesia, or postoperative analgesia only. If a surgical indication occurred, it was classified as (1) orthopedic, (2) urologic, (3) general/abdominal, (4) cesarean delivery, or (5) other. Neuraxial blockade was categorized as (1) epidural, (2) single-injection spinal, (3) continuous spinal, or (4) combined spinal–epidural. Details of each neuraxial technique, including awake placement (yes or no), approach (midline, paramedian, both), number of attempts, and local anesthetic(s) used were collected. The use of epinephrine or other local anesthetic additives was also noted. Technical complications occurring at the time of block placement—such as difficulty identifying the epidural space, difficulty advancing an epidural or subarachnoid catheter, traumatic block placement (evidence of blood), unplanned dural puncture, difficulty obtaining cerebrospinal fluid, paresthesia elicitation, or unintended "total" or "high" spinal—were identified. Block efficacy was categorized as (1) satisfactory (surgery performed without additional intervention), (2) unilateral anesthesia or analgesia, (3) segmental or incomplete anesthesia or analgesia, or (4) no block/block failure.

New or progressive postoperative neurologic deficits (motor or sensory deficits, painful paresthesias, or bowel or bladder dysfunction) were identified in the daily progress notes of the primary surgical service or the anesthesia acute pain service. Complications were also noted during the patient's 2-, 4-, or 6-week surgical follow-up visit. The presence of infectious (neuraxial abscess) or hematologic (neuraxial hematoma) complications was also documented. All complications were followed until complete resolution or until the last documented date of evaluation.

Data are summarized using mean ± SD for continuous variables and percentages for categorical variables. The percentage of failed blocks and technical and neurologic complications was compared in patients with and without a history of spine surgery using the χ^2 test or Fisher's exact test as appropriate. Patients with and without neurologic complications were compared to identify risk factors using Fisher's exact test. $P < 0.05$ was considered statistically significant.

RESULTS

Nine hundred thirty-seven ($n = 937$) patients were identified as having spinal stenosis or lumbar disk disease and undergoing subsequent neuraxial anesthesia or analgesia; 207 of these had a history of spinal surgery before undergoing subsequent spinal or epidural anesthesia (Table 1). Of these, 210 (22%) patients had a coexisting peripheral neuropathy in addition to their spinal canal pathology. At the time of neuraxial blockade, painful paresthesias or dysesthesias were the most common neurologic deficit, followed by nonspecific sensory and motor deficits (Table 1). The majority of patients ($n = 335$; 51%) had active neurologic signs or symptoms (motor or sensory deficits, dysesthesias/paresthesias, or hyperreflexia) at the time of surgery and subsequent neuraxial blockade.

The type of neuraxial blockade included spinal anesthesia in 545 (58%) patients, epidural anesthesia or analgesia in 358 (38%) patients, continuous spinal anesthesia in 24 (3%) patients, and a combined spinal–epidural technique in 10 (1%) patients. Eighty of the patients undergoing epidural anesthesia or analgesia noted above received postoperative epidural analgesia with local anesthetic infusions.

One hundred ninety-three (24%) patients underwent block placement precisely at the level of spinal canal pathology, and >75% of patients had block placement within 2 vertebral levels (Table 2). In addition, the majority of patients with a history of spine surgery ($n = 165$; 83%) underwent subsequent neuraxial anesthesia or analgesia within 2 vertebral levels of their prior spinal surgery.

There was no significant difference in block efficacy when comparing those patients with or without a history of spinal surgery (Table 3). Nine hundred eleven (97.2%) patients had a satisfactory block, 10 (1.1%) patients reported a patchy or segmental block, and 16 (1.7%) patients experienced complete block failure. Overall, there were 107 (11.4%) technical complications. There

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Table 1. Neurologic History of Patients with Spinal Stenosis or Lumbar Disk Disease

Neurologic feature	Number of patients (N = 937)	Percentage (%)[a]
Neurologic diagnosis		
Spinal stenosis	187	20
Compressive radiculopathy	530	57
Disk herniation (without radiculopathy)	40	4
Peripheral neuropathy	210	22
Multiple (>1) diagnoses	180	19
Neurologic history		
Motor deficits	479	51
Sensory deficits	568	61
Pain/dysesthesias	882	94
Hyperreflexia	74	8
History of prior spinal surgery		
s/p Laminectomy	193	21
s/p Diskectomy	9	1
s/p Spinal fusion or other	5	0.5
Disease status at time of block placement		
Acute exacerbation (<30 days)	32	3
Subacute exacerbation (1–6 months)	69	8
Chronic/stable (>6 months)	828	89
Unknown	8	—
Disease progression within last 12 months		
Yes	153	19
No	632	81
Unknown	152	—
Active symptoms at time of neuraxial block		
Yes	335	51
No	323	49
Unknown	279	—

[a] Percentages based upon those patients with available data.
s/p = status post (previous condition).

Table 2. Block Characteristics of Patients with Spinal Stenosis or Lumbar Disk Disease Undergoing Subsequent Neuraxial Anesthesia or Analgesia

Block characteristic	Number of patients (N = 937)	Percentage (%)[a]
Neuraxial technique		
Spinal	545	58
Continuous spinal	24	3
Epidural	358	38
Combined spinal–epidural	10	1
Indication		
Labor analgesia	34	4
Postoperative analgesia only	65	7
Surgical	838	89
Orthopedic	535	64
Urologic	192	23
Intraabdominal	50	6
Cesarean delivery	10	1
Other	51	6
Block placement relative to spinal canal pathology		
Precisely at the level	193	24
Within 1–2 levels	430	52
Within 3–4 levels	172	21
More than 4 levels	26	3
Unknown	116	—
Block placement relative to prior spinal surgery (if applicable)		
Precisely at the level	26	13
Within 1–2 levels	139	70
Within 3–4 levels	28	14
More than 4 levels	7	3
Unknown	7	—
Number of attempts required		
One	712	79
Two	153	17
Three or more	39	4
Unknown	33	—
Local anesthetic use[b]		
Intraoperatively	867	93
Postoperatively	80	9
Epinephrine used		
Yes	422	46
No	505	54
Unknown	10	—

[a] Percentages based upon those patients with available data.
[b] Ten patients received both intra- and postoperative local anesthetics.

was no significant difference in technical complications comparing those patients with and without a history of spinal surgery (Table 3).

There were 10 (1.1%; 95% confidence interval [CI] 0.5%–2.0%) patients who experienced new deficits (3 patients) or worsening of preexisting symptoms (7 patients) postoperatively [Table 4 (cases 1 to 4), and Table 5 (cases 5 to 10)]. Three (1.4%) complications occurred in patients with a history of spinal surgery, and the remaining 7 (1.0%) in patients without prior surgical intervention. Eight of 10 patients with complications underwent an orthopedic surgical procedure; in 5 of the 6 cases in which the surgery was a unilateral lower extremity procedure, the postoperative deficit involved the operative side (cases 3, 4, and 8 to 10). Likewise, in both patients undergoing bilateral orthopedic procedures who developed bilateral deficits, the outcome was worse on the previously affected side (cases 2, 6). From exploratory analyses, the frequency of complications was found to be significantly higher in patients with a preoperative diagnosis of compressive radiculopathy versus other neurologic diagnoses (9/530 vs. 1/407; Fisher exact test $P = 0.049$) and also in patients with multiple versus single preoperative neurologic diagnoses (6/180 vs. 4/757; Fisher exact test $P = 0.005$).

A surgical cause was presumed to be the primary etiology in 4 (40%) of 10 patients (Table 4). Specifically, direct nerve trauma/stretch was the principal mechanism in 3 patients (cases 1, 2, 4), tourniquet ischemia in 2 patients (cases 2, 4), and ischemia from a popliteal cyst in 1 patient (case 3). However, it is possible that neurotoxicity produced by local anesthetic injection at the site of existing root compromise may have contributed in 3 cases (cases 2 to 4), because the postoperative deficits were a worsening of existing deficits. In 3 (75%) of the 4 patients, the neurologic deficits returned to baseline (complete resolution did not occur); in the remaining patient (25%), the symptoms persisted for 3 years (time of last patient follow-up).

The primary etiology of the remaining 6 (60%) complications was judged to be nonsurgical (including anesthetic-related factors) (Table 5). Contributing factors were identified in 3 of these patients and included a previously undiagnosed L2 to L3 ependymoma that became symptomatic after failed epidural anesthesia (likely resulting from needle trauma/bleeding of the neoplasm or alterations in cerebrospinal fluid flow) (case 5), compartment syndrome (with a delay in the diagnosis and

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Table 3. Outcomes of Neuraxial Blockade in Patients with Spinal Stenosis or Lumbar Disk Disease with or without Prior Spinal Surgery

	Patients without prior history of spine surgery			Patients with prior history of spine surgery			
Outcome	*N*	*n*	%	*N*	*n*	%	*P* value[a]
Block efficacy	730			207			0.72
Satisfactory		709	97.1		202	97.6	
Unilateral		0	0.0		1	0.5	
Patchy or segmental		9	1.2		0	0.0	
No block (block failure)		12	1.7		4	1.9	
Technical complications							
Epidural	285			73			0.88
Unable to reach epidural space		5	1.8		0	0.0	
Unable to advance catheter		14	4.9		3	4.1	
Unplanned dural puncture		8	2.8		2	2.7	
Spinal	439			130			1.00
Unable to obtain CSF		1	0.2		0	0.0	
Epidural or spinal	730			207			0.91
Traumatic (blood)		19	2.6		8	3.9	
Unintentional paresthesia		37	5.1		9	4.3	
Unintended "high" spinal		1	0.1		0	0.0	
Neurologic complications	730	7	1.0	207	3	1.4	0.54

CSF = cerebrospinal fluid.

[a] The percentage of patients experiencing one or more of the listed complications within the given category was compared between groups using the Chi-square test or Fisher's exact test as appropriate.

worsening of outcome attributed to epidural infusion) (case 6), and neuropathy of critical illness (case 10). The remaining 3 patients, each with a preoperative diagnosis of spinal stenosis, underwent an uneventful surgery under spinal anesthesia. In these 3 patients (cases 7 to 9), the spinal anesthetic, in the setting of a preexisting neurologic condition, was determined to be the primary etiology, because no positioning or surgical factors were identified. Thus, in 5 of 6 patients with a neurologic complication attributed to a nonsurgical primary etiology, the neuraxial anesthetic was presumed to be the primary mechanism (cases 7 to 9) or a contributing factor in the outcome (cases 5, 6). Decompressive laminectomy was performed in 2 of 6 patients with deficits associated with a nonsurgical etiology (1 for ependymoma; 1 for radiculopathy/spinal stenosis). However, complete resolution was documented in only 2 (33%) of the 6 patients (1 of whom underwent laminectomy; case 5).

DISCUSSION

Patients with preexisting neurologic conditions are typically considered to be at increased risk of neurologic complications after spinal or epidural techniques. Previous studies have investigated the risk associated with multiple sclerosis or polio[16] or diabetic sensorimotor neuropathy[17] and concluded that the risk of severe postoperative neurologic injury in these patients is relatively uncommon but appears to be higher than that reported for the general population.[6,18] Our series is the first to characterize the frequency and severity of neurologic events after neuraxial blockade in patients with previously diagnosed spinal stenosis or lumbar disk disease (with or without prior spinal decompressive surgery). Our major findings suggest that this patient population is at increased risk for worsening of preexisting or the development of new neurologic deficits postoperatively when compared with the general population,[6,18] and that the presence of multiple neurologic diagnoses (radiculopathy, spinal stenosis, peripheral neuropathy) increases the risk.

Our results also demonstrated that a history of spinal surgery did not increase the risk of technical or neurologic complications or affect block success. Previous spinal surgery has often been considered a relative contraindication to the use of neuraxial blockade. Many of these patients experience chronic back pain and are reluctant to undergo epidural or spinal anesthesia, fearing exacerbation of their preexisting back complaints. Several postoperative anatomic changes make needle or catheter placement more difficult and complicated after major spinal surgery. The presence of adhesions or obliteration of the epidural space from scar tissue may increase the incidence of dural puncture or decrease the spread of local anesthetic within the epidural space, producing an incomplete or failed block. Needle placement in an area of the spine that has undergone bone grafting and posterior fusion may not be possible with midline or lateral approaches; needle insertion can be accomplished only at unfused segments.

Previous series investigating the efficacy and safety of neuraxial block after spinal surgery typically involved parturients with a history of Harrington rod instrumentation and fusion. For example, the largest series by Daley et al.[19] included 18 patients with previous Harrington rod instrumentation who underwent 21 attempts at epidural anesthesia for obstetric analgesia. Continuous lumbar epidural anesthesia was successfully established in 20 of 21 attempts, but only 10 procedures were performed easily on the first attempt. The remaining 11 patients required larger amounts of local anesthetics or complained of a patchy block or both. There were no side effects except for low back pain in 2 patients with multiple attempts at catheter placement. Similar results were reported by Crosby and Halpern[12] and Hubbert.[14] Thus, historically, it was concluded that epidural anesthesia may be successfully performed in patients who have had spinal surgery, but successful catheter placement may be possible on the first attempt in only 50% of patients, even by an experienced

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Table 4. Patients with Postoperative Neurologic Complications Associated with a Primary Surgical Etiology

Age (years)/ gender	Preoperative neurologic diagnosis	Neurologic history of symptoms	Current surgery	Symptoms at time of surgery	Neuraxial technique	Postoperative complication	Diagnostic evaluation of deficits	Outcome	Comments
Case 1: 53/F	Lt S1 radiculopathy (mild)	Lt LE pain, paresthesias, and numbness	Rt tibial osteotomy; TTT: 29 minutes	No	Epidural; lidocaine 2% (13 mL) with 1:200,000 epi; Bupiv 0.5% (15 mL); 1 attempt at L3–4 with no paresthesia	Rt tibial neuropathy (pain and numbness) immediately postoperative	3 years postoperative: EMG within normal limits; no evidence of a generalized peripheral neuropathy or right tibial neuropathy	Persistent symptoms after 3 years	Differential diagnoses: intraoperative surgical trauma vs. plantar bone spurs for persistent pain and numbness
Case 2: 77/M	Lt L4–S1 radiculopathy and severe L3–S1 spinal stenosis; s/p Lt partial L4 laminectomy 20 years prior; repeat L3–4 laminectomy 2 years prior	Lt LE pain, numbness, and motor weakness	Bilateral TKAs; Lt TTT: 229 minutes; Rt TTT: 197 minutes	Mild Lt LE pain and numbness; no weakness	Failed L5–S1 epidural; lidocaine 2% (18 mL) with 1:200,000 epi; Bupiv 0.5% (40 mL); multiple attempts with no paresthesias; converted to GA	Lt peroneal neuropathy immediately postoperative	EMG: bilateral peroneal neuropathies at the fibular head (L > R); old Lt L5–S1 radiculopathies	Complete resolution of Lt peroneal neuropathy after 6 years; baseline preoperative deficits remain	Differential diagnoses: preexisting deficits, surgical trauma, and prolonged tourniquet ischemia
Case 3: 76/M	Rt L5 radiculopathy and PSMN	Rt LE pain, numbness, paresthesias, and motor weakness	Rt knee arthroscopy	Rt LE pain, numbness, paresthesia, and weakness	Spinal; Bupiv 0.75% (1.5 mL); 1 attempt at L2–3; no paresthesia	Increased Rt LE pain and progressive paresthesias developed 24 hours postoperatively	No diagnostic evaluation	Resolution to preoperative baseline after 3 months	Chronic polyradiculopathy and hereditary axonal neuropathy diagnosed 8 months prior to knee arthroscopy; developed postoperative septic knee. Differential diagnoses: complication secondary to septic knee, surgical trauma, localized neural ischemia from popliteal cyst
Case 4: 83/M	Rt peroneal neuropathy (compressive fibular cyst); lumbar polyradiculopathy; PSMN	Rt LE pain and motor weakness (foot drop)	Rt TKA and cystic decompression; TTT: 225 minutes	Rt LE pain and motor weakness (foot drop)	Spinal; Bupiv 0.75% (1.5 mL); 2 attempts at L3–4 with Rt-sided paresthesia	Immediate worsening of Rt LE motor deficit postoperative	No diagnostic evaluation	Resolution to preoperative baseline after 6 weeks	Differential diagnoses: exacerbation of preexisting neurologic deficit due to localized surgical procedure and tourniquet ischemia (TTT: 225 minutes)

M = male; F = female; Lt = left; Rt = right; LE = lower extremity; PSMN = peripheral sensorimotor neuropathy; TKA = total knee arthroplasty; Bupiv = bupivacaine; epi = epinephrine; TTT = total tourniquet time; GA = general anesthesia; EMG = electromyography; s/p = status post (previous condition).

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Table 5. Patients with Postoperative Neurologic Complications with a Nonsurgical Primary Etiology

Age (years)/ gender	Preoperative neurologic diagnosis	Neurologic history of symptoms	Current surgery	Symptoms at time of surgery	Neuraxial technique	Postoperative complication	Diagnostic evaluation of deficits	Outcome	Comments
Case 5: 25/F	Lt L4–S1 radiculopathy; s/p L4–5 laminectomy 5 years prior to cesarean delivery with resolution of symptoms	Lt LE pain, numbness, and motor weakness	Cesarean delivery	No	Epidural; Bupiv 0.125% plus fentanyl 2 μg/mL; 1 attempt at L3–4 with no paresthesia; failed (unilateral) epidural for cesarean delivery	Developed Rt LE pain, numbness, and motor weakness 1 week postpartum	MRI: L2–3 ependymoma extending across the midline (R > L)	Complete resolution 8 weeks after ependymoma resection	Postpartum work-up revealed L2–3 ependymoma; underwent ependymoma resection 2 months postpartum
Case 6: 71/M	Chronic Rt L3–5 radiculopathy	Rt LE pain	Bilateral TKAs; Rt TTT: 135 minutes; Lt TTT: 83 minutes	No	Epidural; lidocaine 2% Bupiv 0.5%; 1:200,000 epi; 1 attempt at L2–3; no paresthesia; postoperative epidural infusion: Bupiv 0.1% + fentanyl 7.5 μg/mL for 48 hours	Bilateral LE weakness and numbness after epidural discontinued	Lumbar MRI revealed no hematoma or abscess; EMG: severe bilateral tibial and peroneal neuropathies at the level of the popliteal fossa	Lt: complete resolution after 12 months; Rt: sensorimotor deficits persist 7 years postoperatively	Patient developed bilateral compartment syndrome (unrecognized) during postoperative epidural analgesia; postoperative (B) tibial & peroneal neuropathies; surgical complication with outcome likely worsened because of epidural (delay in diagnosis and intervention)
Case 7: 84/M	Chronic Rt L3–4 and acute Lt L3–4 radiculopathy; moderate L2–L4 spinal stenosis; PSMN	Rt and Lt LE pain	TURP	Lt LE pain	Spinal; procaine 70 mg; 1 attempt at L3–4; no paresthesia	Worsening Lt LE pain and new Lt LE motor deficit immediately postoperatively	EMG: old Rt L3–4 radiculopathy and acute Lt L3–4 radiculopathy and moderate to severe peripheral neuropathy	Partial resolution 4 weeks after laminectomy	New postoperative motor deficits due to exacerbation of Lt LE radiculopathy in setting of spinal stenosis; underwent laminectomy 1 week after TURP
Case 8: 70/F	Mild L2–4 spinal stenosis; Parkinson's disease	Bilateral LE pain	Lt THA	Bilateral LE pain	Spinal; Bupiv 0.75% (2 mL); 1:1,000 epi (0.2 mL); 4 attempts at multiple levels; (+) heme and Rt-sided L2–3 paresthesia	Worsening Rt LE pain and new onset Rt LE weakness 3 weeks postoperatively	EMG: within normal limits; no evidence of a lumbosacral radiculopathy	Partial resolution of pain after 8 weeks	Differential diagnoses: progressive pain secondary to existing condition vs. new onset fibromyalgia or restless leg syndrome
Case 9: 78/M	Bilateral L5–S1 radiculopathy; moderate L4–S1 spinal stenosis; s/p L5 laminectomy 5 years prior	Rt LE pain, numbness, and motor weakness	Rt THA	Partial Rt LE pain and numbness; no weakness	Spinal; tetracaine 1% (1 mL); 1:1,000 epi (0.3 mL); 1 attempt at L3–4 without paresthesia	Immediate worsening of Rt LE numbness postoperative (L5–S1 distribution)	No diagnostic evaluation	Resolution to preoperative baseline after 1 week	Etiology of new postoperative deficits unclear: no obvious surgical trauma to sciatic nerve. Potential contributing factors include existing spinal stenosis with exacerbation by positioning (hyperextension during surgery) and spinal anesthetic

(*Continued*)

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Table 5. (Continued)

Age (years)/ gender	Preoperative neurologic diagnosis	Neurologic history of symptoms	Current surgery	Symptoms at time of surgery	Neuraxial technique	Postoperative complication	Diagnostic evaluation of deficits	Outcome	Comments
Case 10: 71/M	Lumbosacral polyradiculopathy; diabetic PSMN	Lt LE Pain; bilateral LE numbness	Lt TKA; TTT: 109 minutes	Lt LE pain; bilateral LE numbness	Spinal; tetracaine 1% (1 mL); procaine 10% (1 mL); 1:1,000 epi (0.2 mL); 1 attempt at L3–4 with Rt-sided paresthesia	New Lt LE severe motor deficit (foot drop) upon extubation 3 weeks postoperatively	EMG: Lt-sided lumbosacral polyradiculopathy; Lt-sided sciatic neuropathy (site of injury indeterminate); patient refused lumbar MRI	Partial resolution after 4 weeks; lost to follow-up	Complicated postoperative ICU course with prolonged intubation; patient refused full neurologic work-up; differential diagnoses: multifactorial (prolonged immobilization, neuropathy of critical illness, surgical trauma)

M = male; F = female; Lt = left; Rt = right; LE = lower extremity; PSMN = peripheral sensorimotor neuropathy; TURP = transurethral resection of the prostate; TKA = total knee arthroplasty; THA = total hip arthroplasty; Bupiv = bupivacaine; epi = epinephrine; TTT = total tourniquet time; EMG = electromyography; s/p = status post (previous condition); MRI = magnetic resonance imaging; ICU = intensive care unit.

anesthesiologist. Although adequate epidural anesthesia was eventually produced in 40% to 95% of patients, there appeared to be a higher incidence of traumatic needle placement, unintentional dural puncture, and unsuccessful epidural needle or catheter placement, especially if spinal fusion extends to between L-5 and S-1. However, the small numbers of patients and an indication of "labor analgesia" made extrapolation to patients with other surgical indications undergoing a nonepidural technique difficult.

A single study by Berkowitz and Gold[20] evaluated the success rate and complications in 33 patients with prior laminectomy who underwent tetracaine spinal anesthesia. On the basis of their 100% success rate and lack of neurologic complications, including evidence of a prolonged block, the authors concluded that there was "no logical basis for avoiding spinal anesthesia in postlaminectomy patients." Our 97.6% block success rate, trauma frequency of 3.9%, and paresthesia elicitation rate of 4.3% in patients with prior spine surgery support the conclusions of Berkowitz and Gold regarding block efficacy after laminectomy. Furthermore, their frequency of technical complications was similar to those for patients in our series (Table 3) and previous series from our outcomes database.[21] Unfortunately, the frequency of persistent postoperative neurologic deficits in the current retrospective series (1.1%; 95% CI 0.5%–2.0%) is much higher than was expected; prospective epidemiologic investigations have reported frequencies between 1:1000 and 1:10,000.[6,22] Although the neuraxial block was not the primary etiology of all 10 neurologic deficits in our series, it may have been a contributing factor in nearly all the cases because of the "double crush" phenomenon.

The *double crush syndrome*, first proposed by Upton and McComas[11] in 1973, is a general term referring to the coexistence of dual compressive lesions along the course of a nerve, where the presence of a proximal lesion renders the nerve vulnerable to further injury with a more distal compression. Importantly, the 2 minor (perhaps even subclinical) insults synergistically result in a clinical and potentially permanent nerve injury.[23] Although initially described in patients with concomitant cervical radiculopathies and median or ulnar neuropathies, the term has been extended to include injury from noncompressive mechanisms, such as toxic (chemotherapeutic agents, local anesthetics),[24] metabolic (aging, diabetic sensorimotor neuropathy),[17,25,26] ischemic (tourniquet inflation),[27] and traumatic (surgical traction, needle/catheter placement)[28] etiologies. The presence of a preexisting neurologic condition, whether neuraxial or peripheral in nature, would represent an additional mechanism by which minor or subclinical symptoms may interact to result in a new or worsened neurologic deficit.[6,17] Our results suggest an additive effect of multiple preexisting neurologic conditions; we reported that neuraxial block performed in patients with a preoperative diagnosis of a compressive radiculopathy or multiple neurologic diagnoses significantly increased the risk of neurologic complications postoperatively. Previous surgical treatment did not affect the frequency.

The performance of a neuraxial technique in patients with a preexisting neurologic condition may predispose

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the patient to further functional worsening. The mechanism(s) of nerve injury after neuraxial block in patients with spinal stenosis or lumbar disk disease is presumed to be the additive effects of local anesthetic toxicity and neural ischemia (from the volume effects of the injectate/infusate)[1,6,8,29] with epidural techniques and local anesthetic neurotoxicity with spinal blockade. The neurotoxic effects may be enhanced by maldistribution created by degenerative changes within the spinal column.[30] Spinal cord ischemia from microcirculatory derangement has also been proposed.[3,31] Perioperative positioning may also contribute to injury; postoperative cauda equina syndrome and paresis have been reported in previously asymptomatic patients with spinal stenosis after uneventful spinal[6,8] or general anesthesia.[32] Often these cases involve an orthopedic procedure.[8,32]

Patients undergoing orthopedic surgical procedures may be especially prone to the double crush syndrome. Two or more insults along the course of a nerve may readily occur, because the surgical procedure is often associated with a defined postoperative neuropathy (due to surgical neurapraxia), and tourniquet inflation is routinely used to assure a bloodless field. For example, total knee replacement is associated with an overall 2.2% frequency of peroneal nerve palsy (from surgical traction to the peroneal nerve at the fibular head), which increases to 7.7% with prolonged tourniquet inflation (producing ischemia to both the tibial and peroneal components of the sciatic nerve at thigh level).[9,27] A preexisting L5/S1 radiculopathy may render the sciatic components even more sensitive to ischemia and traction, as well as to local anesthetic neurotoxicity. Although an orthopedic procedure was not a risk factor in our series, in 7 of the 8 orthopedic cases with complications, surgery was to the previously affected extremity. Although the relative roles of patient, surgical factors, and anesthetic factors remain undetermined, our results suggest that the overall risk may be high in this patient population.

We conclude that patients with preexisting spinal canal pathology have a higher incidence of neurologic complications after neuraxial blockade than that previously reported for patients without such underlying pathology. However, in the absence of a control group of surgical patients with similar anatomic pathology undergoing general anesthesia, we cannot determine whether the higher incidence of neurologic injury is secondary to the surgical procedure, the anesthetic technique, the natural history of spinal pathology, or a combination of factors and the relative contributions of each.

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FOUNDATION FOR ANESTHESIA EDUCATION AND RESEARCH

Denham S. Ward, MD, PhD, Named Next President of Foundation for Anesthesia Education and Research

For Immediate Release

Rochester, MN, October 28, 2010 – At their annual fall meeting October 16, 2010, the Board of Directors of the Foundation for Anesthesia Education and Research (FAER) named Denham S. Ward, MD, PhD, as successor for long-time President, Alan D. Sessler, MD. Dr. Ward will begin his term as President in 2011.

"Dr. Ward is an accomplished physician scientist with a long history of leadership in anesthesiology," said Dr. Sessler. "Over the months ahead, I will assume a support role in advocating for FAER's mission, confident that my successor will act wisely to create new pathways to propel the specialty's next generation to a new and higher level of patient care, innovation and discovery."

Dr. Ward has been a member of the FAER Board of Directors since 2002 and is now serving as Board Chair. He is currently Professor of Anesthesiology and Biomedical Engineering at the University of Rochester, Rochester, NY, where he is also Chair of the Anesthesiology Department and Associate Dean for Faculty Development – Medical Education.

FAER is a 501(c)(3) organization whose mission is to advance medicine through education and research in anesthesiology. FAER provides research grants and career development opportunities for medical students, residents and anesthesiologists. Since it was founded by the American Society of Anesthesiologists in 1986, the foundation has awarded more than $20 million in research grants, and typically awards more than $1 million in research grants annually.

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