Neurologic Complications After Chlorhexidine Antisepsis for Spinal Anesthesia

Hans P. Sviggum, MD, Adam K. Jacob, MD,* Katherine W. Arendt, MD,* Michelle L. Mauermann, MD,† Terese T. Horlocker, MD,* and James R. Hebl, MD**

Background and Objectives: Recent reports of infectious complications after neuraxial procedures highlight the importance of scrupulous aseptic technique. Although chlorhexidine gluconate (CHG) has several advantages over other antiseptic agents; including a more rapid onset of action, an extended duration of effect, and rare bacterial resistance, it is not approved by the US Food and Drug Administration for use before lumbar puncture because of absence of clinical safety evidence. The objective of this retrospective cohort study was to test the hypothesis that the incidence of neurologic complications associated with spinal anesthesia after CHG skin antisepsis is not different than the known incidence of neurologic complications associated with spinal anesthesia.

Methods: All patients 18 years or older who underwent spinal anesthesia at Mayo Clinic Rochester from 2006 to 2010 were identified. The primary outcome variable was the presence of any new or progressive neurologic deficit documented within 7 days of spinal anesthesia. The etiology of a patient's neurologic complication was independently categorized as possibly or unlikely related to the spinal anesthetic by 3 investigators. Consensus among all reviewers was required for final category assignment.

Results: A total of 11,095 patients received 12,465 spinal anesthetics during the study period. Overall, 57 cases (0.46%; 95% confidence interval, 0.34%–0.58%) met criteria for neurologic complication. Spinal anesthesia was felt to be the possible etiology of 5 neurologic complications (0.04%; 95% confidence interval, 0.00%–0.08%); all completely resolved within 30 days.

Discussion: The incidence of neurologic complications possibly associated with spinal anesthesia (0.04%) after CHG skin antisepsis is consistent with previous reports of neurologic complications after spinal anesthesia. These results support the hypothesis that CHG can be used for skin antisepsis before spinal placement without increasing the risk of neurologic complications attributed to the spinal anesthetic.

(*Reg Anesth Pain Med* 2012;37: 139–144)

Infection within the spinal canal or around the spinal cord is one of the most concerning complications following neuraxial anesthetic techniques. The frequency of these serious infections has historically been considered extremely low.[1–3] However, recent European studies and anecdotal case reports from the United States suggest that the frequency of infectious complications associated with neuraxial anesthesia and analgesia may be increasing.[4–6] Specifically, Moen and colleagues[4] and the Centers for Disease Control and Prevention[5] have reported several cases of post–spinal anesthesia meningitis likely due to breaches in aseptic technique. Furthermore, it is believed that the appropriate selection and application of skin antiseptic before neuraxial blockade may have a significant role in preventing many of these infectious complications.[7,8]

Chlorhexidine gluconate (CHG) is a potent broad-spectrum germicide that is effective against most nosocomial yeasts, gram-positive, and gram-negative bacteria.[9–11] The addition of isopropyl alcohol to CHG further accelerates its bactericidal effects. Chlorhexidine gluconate has a number of advantages over other commonly used antiseptic solutions, including a more rapid onset of action, an extended duration of effect, fewer and less severe skin reactions, retained effectiveness in the presence of blood and other organic compounds, and rare bacterial resistance.[8] Because of these advantages, the American Society of Regional Anesthesia and Pain Medicine, the American Society of Anesthesiologists (ASA), and the Royal College of Anaesthetists have recommended CHG as the antiseptic of choice before all regional techniques.[8,12,13]

Despite these evidence-based recommendations, many clinicians remain concerned about the use of CHG during neuraxial anesthesia and analgesia because of US Food and Drug Administration (FDA) product labeling that warns against the use of CHG for "lumbar puncture or in contact with the meninges" (CareFusion, San Diego, California; available at: http://www.chloraprep.com/pdf/Directions_of_Use/10_5_Orange_label_10.pdf; accessed October 25, 2011). This warning may be based, in part, on a paucity of clinical data and early laboratory studies that suggested an association between CHG and neurotoxicity when applied directly to neural tissue or the meninges.[14,15] Despite these early animal studies, there are currently no published reports within the literature describing CHG neurotoxicity in humans; nor are there clinical studies evaluating the potential neurotoxic risk or neurologic complications associated with CHG skin antisepsis before neuraxial techniques. Therefore, the goal of the current investigation was to test the hypothesis that the incidence of neurologic complications associated with spinal anesthesia after CHG skin antisepsis is not different than the known incidence of neurologic complications associated with spinal anesthesia.

METHODS

After Mayo Clinic Institution Review Board approval and written informed consent, all patients 18 years or older undergoing spinal anesthesia from January 1, 2006, to November 1, 2010, were retrospectively identified using the Mayo Clinic Department of Anesthesiology Quality Database and the electronic medical record (EMR). In July 2005, CHG in isopropyl

From the Departments of *Anesthesiology and †Neurology, Mayo Clinic, Rochester, MN.

Accepted for publication November 15, 2011.

Address correspondence to: James R. Hebl, MD, Department of Anesthesiology, Mayo Clinic, 200 First St SW, Rochester, MN 55905 (e-mail: hebl.james@mayo.edu).

This work represents a collaborative effort of Mayo Clinic's Departments of Anesthesiology and Neurology.

Support for this study was provided by the Department of Anesthesiology and College of Medicine, Mayo Clinic, Rochester, MN.

Material from this manuscript was presented at the Midwest Anesthesia Residents Conference Meeting (April 1–3, 2011, Chicago, IL) and the American Society of Regional Anesthesia and Pain Medicine 36th Annual Meeting (May 5–8, 2011, Las Vegas, NV).

The authors declare no conflict of interest.

ISSN: 1098-7339
DOI: 10.1097/AAP.0b013e318244179a

alcohol became the exclusive antiseptic used at our institution before all regional techniques. Patient demographics, including age, sex, preexisting neurologic disease, ASA physical status, and the surgical indication for spinal anesthesia, were recorded. Preexisting neurologic disease was defined as the presence of lumbar spinal stenosis, multiple sclerosis, postpolio syndrome, diabetic peripheral neuropathy, or idiopathic or hereditary peripheral neuropathy documented in the master diagnosis list as *International Classification of Diseases, Ninth Revision* codes 138, 249.6, 250.6, 336.9, 340, 356, 357, or 724.0. Anesthetic characteristics, including the type and size of the spinal needle and the level of spinal placement, were also collected from the Anesthesia Quality Database.

The primary outcome variable was the presence of new or progressive sensory or motor deficits after spinal anesthesia. Postoperative sensorimotor deficits were identified using a free-text electronic query of the EMR. All clinical notes within the EMR were searched for the following terms: tingling, neurapraxia, nerve apraxia, motor deficit, weakness, arachnoiditis, meningitis, nerve dysfunction, numbness, hypoesthesia, neuropathic pain, neuropathy, nerve palsy, and nerve injury. Identification of false-positive cases was minimized by excluding those cases identified as "peripheral neuropathy," "diabetic neuropathy," "no tingling," "no weakness," and "no numbness." The medical records of patients identified as having 1 or more key words were then manually reviewed. The onset, description, and clinical course of neurologic symptoms, physical examination findings, and data from electrodiagnostic studies or radiographic imaging were collected. Patients with preexisting neurologic deficits that were unchanged after spinal anesthesia were excluded.

Neurologic complications were defined as new or progressive numbness, paresthesias, hypesthesias, dysesthesias, or weakness identified within 1 week of the spinal anesthetic. New or progressive neurologic deficits were classified as either sensory or sensorimotor based on the subjective description or objective findings documented within the medical record. Neurologic deficits were identified in the daily progress notes of the primary surgical service or the anesthesia acute pain service or within consultation notes from the Department of Neurology. Complications were also identified during the patient's 2-, 4-, or 6-week surgical follow-up visit.

The medical records of all patients with a neurologic complication were independently reviewed by 2 board-certified anesthesiologists (A.K.J., K.W.A.) and a board-certified neurologist (M.L.M.). Each reviewer categorized the spinal anesthetic as a "possible" or "unlikely" primary etiology of the neurologic complication. Cases were considered "unlikely" if neurologic deficits were identified within a peripheral nerve distribution in a patient who had undergone an ipsilateral peripheral nerve block within a similar anatomic distribution, or if they were consistent with a recognized complication of surgical positioning. Cases were also considered "unlikely" if neurologic findings were most consistent with a muscular cause (eg, rhabdomyolysis) or had a delayed onset. Cases were considered "possibly" related to the neuraxial technique if neurologic signs were present immediately after surgery and the distribution of neurologic signs and symptoms was anatomically localized to the lumbar or sacral nerve roots, or if they did not meet criteria for "unlikely." A consensus among all 3 reviewers was required for final categorization.

The clinical course of each complication, including the presence or absence of a neurologic deficit at the time of hospital discharge, the diagnostic evaluation by a board- certified neurologist, electrodiagnostic studies, date of last follow-up, time to maximal neurologic recovery (≤1 week, 1 week to 1 month, 1–3 months, 3–6 months, 6–12 months, or ≥12 months), and degree of neurologic recovery (complete, partial, none), was recorded. Complete neurologic recovery was defined as a return to baseline neurologic status. Partial neurologic recovery was defined as an improvement of neurologic symptoms, but a persistent deficit was documented at the time of last follow-up. Patients with no improvement in neurologic symptoms at the time of last follow-up were defined as having no recovery.

Data are summarized as mean (SD) for continuous parametric variables and median (interquartile range) for continuous nonparametric variables. Categorical variables are reported as frequency percentages. The frequency of neurologic complications was summarized using point estimates with 95% confidence intervals (CIs).

RESULTS

A total of 11,095 patients underwent 12,465 spinal anesthetics with CHG antisepsis during the study period. Patient and procedure details for all spinal anesthetics are summarized in Table 1. The majority of spinal anesthetics were performed for orthopedic surgical procedures. Peripheral nerve blockade was performed in conjunction with the spinal anesthetic in 5317 cases (42.7%), with 1469 patients (11.8%) having more than 1 peripheral nerve block. A preexisting neurologic condition was present in 732 patients (6.6%), with 177 patients (1.6%) having more than 1 neurologic diagnosis.

A total of 1188 potential neurologic complications were identified using the EMR free-text query of key terms. However, after manual chart review, 57 cases met inclusion criteria for a new or progressive neurologic deficit resulting in an overall incidence of 0.46% (95% CI, 0.34%–0.58%). Of these, 52 cases were categorized as unlikely related to the spinal anesthetic, whereas 5 cases were categorized as possibly related for an incidence of 0.04% (95% CI, 0.00%–0.08%).

The clinical characteristics and natural course of all 57 neurologic complications are summarized in Table 2. The median (25th, 75th) length of follow-up was 0.9 (0.3–2.0) years. The majority of patients (n = 46) had a complete neurologic recovery. Of these, 40 patients (87%) experienced complete recovery within 6 months of surgery, 4 (8.7%) experienced complete recovery within 12 months of surgery, and 2 (4.3%) completely recovered more than 12 months after spinal placement. Of the 10 patients who experienced partial neurologic recovery during the follow-up period, 5 patients (50%) achieved maximal improvement within 6 months of surgery, 2 (20%) improved within 12 months of surgery, and 3 (30%) required more than 12 months to achieve maximal neurologic recovery. One patient, an 88-year-old man who had a peroneal nerve palsy following total hip arthroplasty, had no improvement in his neurologic symptoms at a follow-up of 24 months.

Twenty patients experienced an isolated sensory deficit after their spinal technique. Of these, 19 patients (95%) experienced a complete neurologic recovery. In contrast, only 27 (73%) of the 37 patients with a combined sensorimotor deficit experienced a complete neurologic recovery. All 19 patients (100%) with an isolated sensory deficit who experienced complete neurologic recovery did so within 6 months of surgery, whereas only 21 (77.8%) of 27 patients with a combined sensorimotor deficit experienced a complete neurologic recovery over the same period.

Of the 57 cases of new or worsened neurologic deficit, 5 cases (8.8%) were categorized as possibly related to the spinal anesthetic (Table 3). All 5 cases were lumbar spinal anesthetics with small-gauge Whitacre needles. Four spinal procedures were

TABLE 1. Patient and Procedural Characteristics

Characteristic	Overall (n = 12,465), n (%)	Neurologic Complication (n = 57), n (%)
Age, y	56.2 ± 18.2	56.4 ± 18.3
Sex		
Male	5506 (44.2)	24 (42.1)
Female	6959 (55.8)	33 (57.9)
ASA physical status		
I	1046 (8.4)	2 (3.5)
II	8504 (68.2)	43 (75.4)
II	2809 (22.5)	12 (21.1)
IV	106 (0.9)	0
Type of procedure		
Orthopedic	6913 (55.5)	24 (42.1)
Urologic	1849 (14.8)	16 (28.1)
Obstetric	1777 (14.3)	9 (15.8)
Gynecologic	808 (6.5)	3 (5.3)
General	786 (6.3)	5 (8.8)
Cardiovascular and thoracic	151 (1.2)	0
Radiation oncology	71 (0.6)	0
Neurologic	65 (0.5)	0
Other	45 (0.4)	0
Needle size		
18-gauge	150 (1.2)	0
22-gauge	4862 (39.0)	23 (40.4)
24-gauge	550 (4.4)	5 (8.8)
25-gauge	5922 (47.5)	27 (47.4)
27-gauge	455 (3.7)	1 (1.8)
Other/not available	526 (4.2)	1 (1.8)
Needle type		
Whitacre	11,144 (89.4)	50 (87.7)
Quincke	377 (3.0)	1 (1.8)
Sprotte	569 (4.6)	5 (8.8)
Tuohy	103 (0.8)	0
Other/not available	242 (1.9)	1 (1.8)
Level of placement		
L2-3	1190 (9.5)	5 (8.8)
L3-4	7840 (62.9)	38 (66.7)
L4-5	3157 (25.3)	13 (22.7)
L5-S1	84 (0.7)	1 (1.8)
Unknown	194 (1.6)	0
Local anesthetic used in spinal	11,183 (89.7)	54 (94.7)
Peripheral nerve block performed	5317 (42.7)	22 (38.6)
Preexisting neurologic condition*		
Spinal stenosis	315 (2.8)	3 (5.3)
Multiple sclerosis	13 (0.1)	0
Postpolio syndrome	16 (0.1)	0
Diabetic neuropathy	160 (1.4)	0
Idiopathic or hereditary peripheral neuropathy	413 (3.7)	0

*Based on N = 11,095 patients; *International Classification of Diseases, Ninth Revision* diagnosis code documented before first spinal anesthetic.

TABLE 2. Evaluation and Follow-Up of Neurologic Complications After Spinal Anesthesia

Characteristic	n (%) (n = 57)
Type of nerve injury	
Sensory	20 (35.1)
Sensorimotor	37 (64.9)
Nerve injury documented before hospital discharge	
Yes	53 (93.0)
No	4 (7.0)
Neurology consultation obtained	
Yes	13 (22.7)
No	44 (77.3)
Electromyogram or nerve conduction study obtained	
Yes	7 (12.3)
No	50 (87.7)
Degree of neurologic recovery	
None	1 (1.8)
Partial	10 (17.5)
Complete	46 (80.7)
Time to maximal neurologic recovery or last follow-up	
<1 wk	21 (36.8)
1 wk to 1 month	11 (19.3)
1–3 mo	7 (12.3)
3–6 mo	6 (10.5)
6–12 mo	6 (10.5)
>12 mo	6 (10.5)

technically uncomplicated. One patient described a transient right-sided paresthesia during needle placement and subsequently experienced right-sided thigh weakness. A second patient underwent multiple attempts at a left-sided psoas compartment nerve block before a technically uncomplicated spinal anesthetic and subsequently complained of left-sided radicular leg pain. In each of the 5 cases, all deficits completely resolved within 1 month of the injury first being reported. There were no reported cases with clinical signs or symptoms consistent with neurotoxicity, including arachnoiditis, aseptic meningitis, or diffuse lumbar plexopathy.

In addition to the 5 neurologic deficits possibly related to spinal anesthesia after CHG antisepsis, we identified 1 patient with rheumatoid arthritis and long-standing immune suppression who developed bacterial meningitis 1 day after spinal anesthesia for irrigation and debridement of an infected total knee arthroplasty. The patient was admitted with community-acquired *Staphylococcus aureus* bacteremia and sepsis with acute septic arthritis and pneumonitis. Approximately 8 hrs after admission, the patient underwent irrigation and debridement of her left knee under spinal anesthesia. The patient received organism-sensitive, triple-antibiotic coverage with levofloxacin, cefepime, and vancomycin within 12 hrs of spinal placement. The organism cultured from the cerebrospinal fluid (CSF) was identical to that cultured from the left knee joint and bloodstream before surgery. The patient was dismissed after 4 months with a normal neurologic examination.

DISCUSSION

Despite recent advancements in regional anesthesia techniques and improved perioperative care, patients continue to experience significant morbidity—and even death—from infectious

TABLE 3. Neurologic Complications Categorized as "Possibly" Related to Spinal Anesthesia

Patient, Sex, Age (y)	Procedure	Level, Needle Type and Size	Medication	Perioperative Observations	Injury Description	Outcome
Female, 21	Cesarean delivery after trial of labor	L3-4 Whitacre 25-gauge	Bupivacaine 12 mg, fentanyl 25 μg, morphine 0.1 mg	Transient right-sided paresthesia during spinal placement	Right thigh weakness	Full recovery within 10 d
Female, 72	Left total hip arthroplasty	L3-4 Whitacre 22-gauge	Bupivacaine 15 mg	Failed psoas block after multiple attempts	Radicular pain left leg, no focal neurologic deficits	Full recovery within 7 d
Female, 43	Vaginal hysterectomy	L2-3 Whitacre 25-gauge	Bupivacaine 13 mg, hydromorphone 60 μg	None	Numbness around medial aspect of knees bilaterally	Full recovery within 30 d
Male, 51	Radical retropubic prostatectomy	L4-5 Whitacre 25-gauge	Bupivacaine 15 mg, hydromorphone 40 μg, clonidine 50 μg	None	Bilateral leg weakness	Full recovery within 2 d
Female, 36	Cesarean delivery after trial of labor	L3-4 Whitacre 25-gauge	Bupivacaine 12.5 mg, fentanyl 25 μg	None	Right leg paresthesias/pain in L4, L5 distribution	Full recovery within 2 d

complications after neuraxial anesthesia and analgesia. Breaches in aseptic technique and the inappropriate selection or application of antiseptic solutions are thought to be major contributors to these adverse events.[4,5,7,8,16] Previous investigations have described the enhanced efficacy and clinical advantages of CHG in regional anesthesia,[17,18] whereas a limited number of animal studies have suggested concern regarding neurotoxicity.[14,15,19,20] This single-center, retrospective investigation in which all patients received skin antisepsis with CHG in isopropyl alcohol before spinal placement found an incidence of neurologic complications possibly related to the anesthetic technique (0.04%; 95% CI, 0.00%–0.08%) to be similar to previous large-scale estimates of neurologic complications after spinal anesthesia (0.03%–0.06%).[21,22] Furthermore, none of the identified neurologic complications had a clinical course consistent with CHG neurotoxicity, namely, arachnoiditis, aseptic meningitis, or diffuse lumbar plexopathy.

In 2006, the American Society of Regional Anesthesia Practice Advisory on Infectious Complications Associated With Regional Anesthesia and Pain Medicine recommended that CHG in an alcohol base be considered the antiseptic of choice before all regional techniques.[8] As expected, this recommendation led to discordance between evidence-based practice and expert clinical opinion and CHG product labeling. Following this recommendation, the ASA Task Force on Infectious Complications Associated With Neuraxial Anesthesia and Analgesia conducted a survey of expert consultants and a random sampling of ASA members regarding skin antiseptic preferences and reported discrepant results.[12] Although expert consultants indicated a preference for CHG with alcohol for skin preparation before neuraxial techniques, at-large ASA members had a slight preference for povidone-iodine. The reason for this discordance is unclear but may be due to US Food and Drug Administration–recommended product labeling that warns against the use of CHG for lumbar puncture or in contact with the meninges (CareFusion). This warning may be based, in part, on the absence of clinical testing on the use of CHG for skin antisepsis before lumbar puncture. Animal studies have demonstrated profound histologic and clinical evidence of neurotoxicity when high concentrations of CHG compounds are applied to the middle ear, anterior chamber of the eye, or directly into the neuraxis.[14,15,19,20] Although isolated anecdotal reports of CHG-related neurotoxicity are present in the lay press, there are no clinical reports to support this association in humans.

The descriptions of neurologic injuries possibly attributed to spinal anesthesia in this study are consistent with classic descriptions of nerve injury following neuraxial blockade.[23,24] As a result, the mechanism(s) of injury (eg, mechanical trauma) are likely to be similar. In contrast, CHG neurotoxicity would likely present as arachnoiditis, aseptic meningitis, or a diffuse plexopathy.[15,25–27] None of the complications possibly attributed to spinal anesthesia in this study fit patterns thought to be consistent with CHG neurotoxicity. Furthermore, all neurologic deficits possibly related to spinal anesthesia were completely resolved within 30 days of the neuraxial technique.

The current study determined that the overall incidence of perioperative neurologic complications, including both neuraxial and peripheral nerve injuries, was 0.46%. A similar incidence of perioperative nerve injury (0.79%) was demonstrated in a recent study of patients undergoing total knee arthroplasty using similar methods of outcome definition and case ascertainment.[28] The present study included a large proportion of patients (56%) undergoing lower-extremity orthopedic procedures in conjunction with peripheral nerve blockade. This clinical patient population may partially account for the higher incidence of

overall perioperative neurologic complications when compared with other large-scale epidemiologic studies.[29,30]

Finally, a unique clinical scenario was identified in the current report that included a case of bacterial meningitis in an immunocompromised patient approximately 1 day after undergoing irrigation and debridement of an infected knee under spinal anesthesia. The organism cultured from the CSF and the associated antibiotic sensitivity was identical to that identified in both the knee joint and blood cultures before surgery. Of note, the organism (*S. aureus*) cultured in all locations (CSF, blood, and joint) was sensitive to the antibiotics administered during the preoperative period. As a result, this infectious complication was likely not related to the spinal anesthetic, and it was certainly separate from other reported cases of spinal meningitis occurring in noninfected patients in which breaches in aseptic technique were believed to be a major contributor.[4,5]

There are some important limitations to our study. First, the retrospective collection of information introduces the possibility of missing transient, yet clinically significant events that may not be documented in the medical record. This includes subtle signs and symptoms of chemical (aseptic) meningitis, such as headache, neck pain, or fever that may not have been identified during the free-text search query in the absence of neurologic deficits. Nevertheless, use of the search terms "meningitis" and "arachnoiditis" may have captured patients with these subtle findings if either of these diagnoses was being considered. Second, demonstrating a true cause-effect relationship between the neuraxial technique and the postoperative neurologic deficit is challenging, as there may be multiple contributing factors. However, in the current study, 2 anesthesiologists and a neurologist who specializes in neuromuscular disorders independently reviewed all cases of neurologic injury and collectively determined the relatedness of the neurologic deficit(s) to the spinal anesthetic. Even in those nerve injuries possibly related to the spinal technique, the retrospective study design did not allow us to identify the precise mechanism of injury.

In summary, this study reports an incidence of neurologic complications possibly related to spinal anesthesia after antisepsis with CHG plus isopropyl alcohol of 0.04%. This incidence is similar to previously reported rates of neurologic complications after spinal anesthesia without attention to the skin antiseptic used.[21,22] Furthermore, the description of identified injuries in the current study is similar to typical injuries reported after spinal anesthesia, with no clinical evidence of CHG neurotoxicity. All neurologic complications possibly related to the spinal anesthetic were resolved within 30 days. Although the results of this study are reassuring regarding the association of perioperative nerve injury and CHG antisepsis before spinal anesthesia, we are unable to determine the relative safety of CHG for skin disinfection. Further large-scale studies are necessary to more formally establish its safety in the practice of neuraxial regional anesthesia. At this time, however, given the increase in infectious complications following neuraxial block and the superiority of CHG as a skin disinfectant, the benefits of using CHG for skin antisepsis appear to outweigh the risks.

REFERENCES

1. Ready LB, Helfer D. Bacterial meningitis in parturients after epidural anesthesia. *Anesthesiology.* 1989;71:988–990.
2. Baker AS, Ojemann RG, Swarts MN, Richardson EP. Spinal epidural abscess. *N Engl J Med*. 1975;293:463–468.
3. Ericsson M, Algers G, Schliamser SE. Spinal epidrual abscesses in adults: review and report of iatrogenic cases. *Scand J Infect Dis*. 1990;22:249–257.
4. Moen V, Dahlgren N, Irested L. Severe neurological complications after central neuraxial blockades in Sweden 1990-1999. *Anesthesiology.* 2004;101:950–959.
5. Centers for Disease Control and Prevention. Bacterial meningitis after intrapartum spinal anesthesia—New York and Ohio, 2008–2009. *MMWR*. 2010;59:65–66.
6. Wang LP, Hauerberg J, Schmidt JF. Incidence of spinal epidural abscess after epidural analgesia: a national 1-year survey. *Anesthesiology.* 1999;91:1928–1936.
7. Horlocker TT, Wedel DJ. Infectious complications of regional anesthesia. *Best Pract Res Clin Anaesthesiol*. 2008;22:451–475.
8. Hebl JR. The importance and implications of aseptic techniques during regional anesthesia. *Reg Anesth Pain Med*. 2006;31:311–323.
9. Maki DG, Ringer M, Alvarado CJ. Prospective randomised trial of povidone-iodine, alcohol, and chlorhexidine for prevention of infection associated with central venous and arterial catheters. *Lancet*. 1991;338:339–343.
10. Mimoz O, Karim A, Mercat A, et al. Chlorhexidine compared with povidone-iodine as skin preparation before blood culture. A randomized, controlled trial. *Ann Intern Med*. 1999;131:834–837.
11. Mimoz O, Pieroni L, Lawrence C, et al. Prospective, randomized trial of two antiseptic solutions for prevention of central venous or arterial catheter colonization and infection in intensive care unit patients. *Crit Care Med*. 1996;24:1818–1823.
12. American Society of Anesthesiologists Task Force on infectious complications associated with neuraxial techniques. Practice Advisory for the Prevention, Diagnosis, and Management of Infectious Complications Associated With Neuraxial Techniques: a report by the American Society of Anesthesiologists Task Force on infectious complications associated with neuraxial techniques. *Anesthesiology.* 2010;112:530–545.
13. Cook T. Major complications of central neuraxial block in the United Kingdom: the 3rd National Audit Project of the Royal College of Anaesthetists. 2009. Available at: http://www.rcoa.ac.uk/docs/NAP3_web-large.pdf. Accessed October 25, 2011.
14. Henschen A, Olson L. Chlorhexidine-induced degeneration of adrenergic nerves. *Acta Neuropathol*. 1984;63:18–23.
15. Hurst EW. Adhesive arachnoiditis and vascular blockage caused by detergents and other chemical irritants; an experimental study. *J Med Liban*. 1955;8:310–317.
16. Hebl JR, Horlocker TT. Dirty rotten scoundrels! *Reg Anesth Pain Med*. 2010;35:485–487.
17. Kinirons B, Mimoz O, Lafendi L, Naas T, Meunier J, Nordmann P. Chlorhexidine versus povidone iodine in preventing colonization of continuous epidural catheters in children: a randomized, controlled trial. *Anesthesiology.* 2001;94:239–244.
18. Shapiro JM, Bond EL, Garman JK. Use of a chlorhexidine dressing to reduce microbial colonization of epidural catheters. *Anesthesiology.* 1990;73:625–631.
19. Aursnes J. Cochlear damage from chlorhexidine in guinea pigs. *Acta Otolaryngol*. 1981;92:259–271.
20. Aursnes J. Vestibular damage from chlorhexidine in guinea pigs. *Acta Otolaryngol*. 1981;92:89–100.
21. Auroy Y, Benhamou D, Bargues L, et al. Major complications of regional anesthesia in France: the SOS Regional Anesthesia Hotline Service. *Anesthesiology.* 2002;97:1274–1280.
22. Auroy Y, Narchi P, Messiah A, Litt L, Rouvier B, Sammi K. Serious complications related to regional anesthesia: results of a prospective survey in France. *Anesthesiology.* 1997;87:479–486.
23. Sorenson EJ. Neurological injuries associcated with regional anesthesia. *Reg Anesth Pain Med*. 2008;33:442–448.

24. Wong CA. Nerve injuries after neuraxial anaesthesia and their medicolegal implications. *Best Pract Res Clin Obstet Gynaecol*. 2010;24:367–381.

25. Aldrete JA. Neurologic deficits and arachnoiditis following neuroaxial anesthesia. *Acta Anaesthesiol Scand*. 2003;47:3–12.

26. Nishimura C, Tsubokawa K, Kasama S, Otagiri T. Two cases of chemical meningitis following spinal anesthesia. *J Anesth*. 2001;15:111–113.

27. Gibbons RB. Chemical meningits following spinal anesthesia. *JAMA*. 1969;210:900–902.

28. Jacob AK, Mantilla CB, Sviggum HP, Schroeder DR, Pagnano MW, Hebl JR. Perioperative nerve injury after total knee arthroplasty: regional anesthesia risk during a 20-year cohort study. *Anesthesiology.* 2011;114:311–317.

29. Warner MA, Martin JT, Schroeder DR, Offord KP, Chute CG. Lower-extremity motor neuropathy associated with surgery performed on patients in a lithotomy position. *Anesthesiology.* 1994;81:6–12.

30. Welch MB, Brummett CM, Welch TD, et al. Perioperative peripheral nerve injuries: a retrospective study of 380,680 cases during a 10-year period at a single institution. *Anesthesiology.* 2009;111:490–497.