Regional Anesthesia in the Febrile or Infected Patient

Denise J. Wedel, M.D., and Terese T. Horlocker, M.D.

Infectious complications may occur after any regional anesthetic technique but are of greatest concern if the infection occurs near or within the central neuraxis. Possible risk factors include underlying sepsis, diabetes, depressed immune status, steroid therapy, localized bacterial colonization or infection, and chronic catheter maintenance. Bacterial infection of the central neuraxis may present as meningitis or cord compression secondary to abscess formation. The infectious source may be exogenous (e.g., contaminated equipment or medication) or endogenous (a bacterial source in the patient seeding to the needle or catheter site). Microorganisms can also be transmitted via a break in aseptic technique, and indwelling catheters may be colonized from a superficial site (skin) and subsequently serve as a wick for spread of infection from the skin to the epidural or intrathecal space.

Although individual cases have been reported in the literature, serious central neuraxial infections such as arachnoiditis, meningitis, and abscess after spinal or epidural anesthesia are extremely rare. In a combined series of more than 65,000 spinal anesthetics, there were only 3 cases of meningitis.[1] A similar review of approximately 50,000 epidural anesthetics failed to disclose a single epidural or intrathecal infection.[1] A more recent multicenter, prospective study including 40,640 spinal and 30,413 epidural anesthetics reported no infectious complications.[2] Limited data suggest that spinal or epidural anesthesia during bacteremia is a risk factor for infection of the central neuraxis. Although the authors of the large cited studies did not report how many patients were febrile during administration of the spinal or epidural anesthetic, a significant number of the patients included in these studies underwent obstetric or urologic procedures, and it is likely that some patients were bacteremic after (and perhaps during) needle or catheter placement. In a recent retrospective review by Horlocker et al.[3] of 4,767 consecutive spinal anesthetics, there were 2 infectious complications noted. One patient, who developed a disc-space infection after spinal anesthesia, was noted to have had a recent untreated episode of urosepsis. The second patient developed a paraspinal abscess 11 days after spinal anesthesia, performed after unsuccessful attempts at caudal block for suspected rectal fistula. Despite the apparent low risk of central nervous system infection after regional anesthesia, anesthesiologists have long considered sepsis to be a relative contraindication to the administration of spinal or epidural anesthesia. This impression is based largely on anecdotal reports and conflicting laboratory and clinical investigations.

The clinical presentation of infections of the central nervous system, the laboratory and clinical studies evaluating the association between meningitis and dural puncture in bacteremic subjects, the risk of central neuraxial block in patients with herpes simplex and human immunodeficiency virus, and the clinical studies investigating the risk of infection during chronic epidural catheterization in febrile and immunocompromised patients will be discussed. An understanding of these concerns will assist the clinician in the evaluation of the febrile patient for central neuraxial block.

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From the Department of Anesthesiology, Mayo Clinic College of Medicine, Rochester, MN.

Accepted for publication April 27, 2006.

Reprint requests: Denise J. Wedel, M.D., Department of Anesthesiology, Mayo Clinic, 200 First Street, Southwest, Rochester, MN 55905. E-mail: wedel.denise@mayo.edu

1098-7339/06/3104-0007$32.00/0
doi:10.1016/j.rapm.2006.04.003

Meningitis

Dural puncture has long been considered a risk factor in the pathogenesis of meningitis. Exactly how bacteria cross from the blood stream into the spinal fluid is unknown. The suggested mechanisms include introduction of blood into the intrathecal space during needle placement and disruption of the protection provided by the blood-brain barrier. However, lumbar puncture is often performed in patients with fever or infection of unknown origin. If dural puncture during bacteremia results in men-

ingitis, definite clinical data should exist. In fact, clinical studies are few and often outdated.

Initial laboratory and clinical investigations were performed over 80 years ago (Table 1). In 1919, Weed et al.[4] showed that lumbar or cisternal puncture performed during septicemia (produced by lethal doses of an intravenously administered gram-negative bacillus) invariably resulted in a fatal meningitis. In the same year, Wegeforth and Latham[5] reported their clinical observations on 93 patients suspected of having meningitis who received a diagnostic lumbar puncture. Blood cultures were taken simultaneously. The diagnosis was confirmed in 38 patients. The remaining 55 patients had normal cerebrospinal fluid (CSF). However, 6 of these 55 patients were bacteremic at the time of lumbar puncture. Five of the 6 bacteremic patients subsequently developed meningitis. It was implied, but not stated, that patients with both sterile blood and CSF cultures did not develop meningitis. Unfortunately, these lumbar punctures were performed during 2 epidemics of meningitis occurring at a military instillation, and it is possible that some (or all) of these patients may have developed meningitis without lumbar puncture. These 2 historic studies provided support for the claim that lumbar puncture during bacteremia was a possible risk factor for meningitis.

Subsequent clinical studies reported conflicting results. Pray[6] studied the incidence of pneumococcal meningitis in children who underwent a diagnostic lumbar puncture during pneumococcal sepsis. The incidence of meningitis was no greater among patients who were subjected to lumbar puncture, which produced normal CSF (8/30 patients or 27%), than among those who did not undergo diagnostic spinal tap (86/386 patients or 22%). Eng and Seligman[7] retrospectively reviewed the records of 1,089 bacteremic patients, including 200 patients who underwent lumbar puncture. The authors reported that the incidence of meningitis after lumbar puncture did not significantly differ from the incidence of spontaneous meningitis and concluded: "If lumbar puncture induced meningitis does occur, it is rare enough to be clinically insignificant."

However, not all studies have been as reassuring as those described earlier. In a review of meningitis associated with serial lumbar punctures to treat posthemorrhagic hydrocephalus in premature infants, Smith et al.[8] attempted to identify risk factors. Six of 22 (27%) infants undergoing multiple (2-33) therapeutic dural punctures during a period of 2 to 63 days developed meningitis. Bacteremia, a risk factor for meningitis in this report, was associated with central venous or umbilical artery catheters. However, 11 septic infants who underwent dural

Table 1. Meningitis After Dural Puncture

Author, Year	No. of Patients	Population	Microorganism(s)	Patients With Spontaneous Meningitis	Patients With Lumbar Puncture–Induced Meningitis	Comments
Wegeforth, 1919[5]	93	Military personnel	*N meningitidis* *S pneumoniae*	38 of 93 (41%)	5 of 93, including 5 of 6 bacteremic patients	LPs performed during meningitis epidemics
Pray, 1941[6]	416	Pediatric with bacteremia	*S pneumoniae*	86 of 386 (22%)	8 of 30 (27%)	80% of patients with meningitis <2 yrs of age
Eng, 1981[7]	1,089	Adults with bacteremia	Atypical and typical bacteria	30 of 919 (3.3%)	3 of 170 (1.8%)	Atypical organisms responsible for lumbar puncture–induced meningitis
Teele, 1981[9]	271	Pediatric with bacteremia	*S pneumoniae* *N meningitidis* *H influenzae*	2 of 31 (9%)	7 of 46 (15%)*	All cases of meningitis occurred in children <1 yr of age. Antibiotic therapy reduced risk
Smith, 1986[8]	11	Preterm with neonatal sepsis		0%	0%	

NOTE. Spontaneous meningitis = concurrent bacteremia and meningitis (without a preceding lumbar puncture) and lumbar puncture–induced meningitis = positive blood culture with sterile CSF on initial exam; subsequent positive CSF culture (same organism present in blood).

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*Significant association ($P < .001$).

puncture did not develop meningitis. The number of dural punctures, incidence of "difficult or traumatic" procedures, and use of antibiotics did not differ between infants who developed meningitis and those who did not. A causal relationship between the dural puncture and onset of meningitis was not clear. Teele et al.[9] retrospectively reviewed the records of 277 bacteremic children during a 10-year interval from 1971 to 1980. Meningitis occurred in 7 of 46 (15%) children with normal CSF obtained during a bacteremia. However, only 2 of 231 (1%) children who did not undergo lumbar puncture developed meningitis. These results were significantly different. In addition, children treated with antibiotics at the time of lumbar puncture were less likely to develop meningitis than children who were not treated until after lumbar puncture. The authors admitted that clinical judgment may have allowed the pediatricians to select the child in whom meningitis is developing before the CSF is diagnostic; these patients may appear more ill and thus suggest the performance of a lumbar puncture.

Prevention of lumbar puncture-induced meningitis with antibiotic therapy is supported by a more recent version of the Weed animal study. Carp and Bailey[10] investigated the association between meningitis and dural puncture in bacteremic rats. Twelve of forty rats subjected to cisternal puncture with a 26-gauge needle during an *Escherichia coli* bacteremia subsequently developed meningitis. Meningitis occurred only in animals with a blood culture result of ≥50 colony-forming units/mL at the time of dural puncture, a circulating bacterial count observed in patients with infective endocarditis. In addition, bacteremic animals not undergoing dural puncture as well as animals undergoing dural puncture in the absence of bacteremia did not develop meningitis. Treatment of a group of bacteremic rats with a single dose of gentamycin immediately before cisternal puncture eliminated the risk of meningitis; none of these animals developed a CNS infection.

This study shows that dural puncture in the presence of bacteremia is associated with the development of meningitis in rats and that antibiotic treatment before dural puncture reduces this risk. Unfortunately, this study did not include a group of animals that were treated with antibiotics after dural puncture. Because many surgeons defer antibiotic therapy until after cultures are obtained, the actual clinical scenario remains unstudied. There are several other limitations to this study. Although *E coli* is a common cause of bacteremia, it is an uncommon cause of meningitis. In addition, the authors knew the sensitivity to the bacteria injected, allowing for appropriate antibiotic coverage. The authors also performed a cisternal puncture (rather than lumbar puncture) and used a 26-gauge needle, producing a relatively large dural defect in the rat compared with humans. Finally, no local anesthetic was injected. Local anesthetic solutions are bacteriostatic, which may theoretically reduce the risk of meningitis in normal clinical settings. Although these results may apply to the performance of spinal anesthesia in the bacteremic patient, they do not apply to administration of epidural anesthesia in the febrile patient, which is associated with a higher incidence of vascular injury and typically involves placement of an indwelling foreign body.

Meningitis after spinal anesthesia has been only rarely reported. In a study evaluating the frequency of meningitis in patients undergoing spinal anesthesia, Kilpatrick and Girgis[11] retrospectively reviewed records of all patients admitted to the meningitis ward in Cairo, Egypt. During a 5-year period from 1975 to 1980, 17 of 1,429 patients admitted with a diagnosis of meningitis had a history of recent spinal anesthesia. The patients developed meningeal symptoms 2 to 30 days (mean 9 days) after spinal anesthesia and were symptomatic for 1 to 83 days (mean 15 days) before hospital admission. Ten of the 17 had positive CSF cultures: 8 were *Pseudomonas aeruginosa*, 1 was *S aureus*, and 1 was *Streptococcus mitis*. These organisms were not cultured from patients who had not had spinal anesthesia. Two additional patients with a history of recent spinal anesthesia showed evidence of tuberculous meningitis. The lack of positive CSF cultures was presumed to be a result of oral antibiotic therapy, which was present in over half of patients at the time of admission. However, all patients, including those with negative CSF cultures, were treated with antibiotic therapy. Four of the 17 patients died. These results suggest that meningitis occurring in patients with a history of recent spinal anesthesia is often caused by unusual or nosocomial organisms and that aggressive bacteriologic evaluation and antibiotic coverage is warranted.

Despite the report by Kilpatrick and Girgis,[11] most cases of meningitis associated with spinal anesthesia are reported as single cases or small case series. Older case reports often reported an association of meningitis with a break in sterilization techniques affecting patient preparation or reusable equipment.[12] More recent reports often describe unusual or nosocomial organisms, and diagnosis is frequently confounded by the use of broad-spectrum antibiotics before the onset of clinical symptoms.[11-13] Nosocomial infections are suspected when an unusual pathogen such as *Streptococcus salivarius*, commonly found in the oral cavity, skin, and gastrointestinal tract, causes meningitis. Three

recent reports describe a total of 5 patients undergoing minor orthopedic surgery who developed postspinal meningitis involving *S salivarius*.[14-16] In an additional case involving *S aureus*, a nasal swab from the anesthesiologist yielded a genotypically identical strain.[15] In all cases, appropriate aseptic techniques were used, including a mask; however, the suspected mechanism was droplet contamination of the spinal needle.

Videira et al.[17] reviewed spinal-related complications for the previous 3 years and initiated a survey of aseptic techniques in their hospital after identifying 2 cases of postspinal meningitis. They reported a total of 3 cases of postspinal meningitis (streptococcal strains in 2 patients and no growth in the third) in 38,128 spinal anesthetics and 0 cases in 12,822 patients receiving other regional or general anesthesia. These numbers did not differ statistically. However, they also noted a wide variability in the type and extent of aseptic techniques used by their anesthesia staff. Based on their findings, standardized recommendations for asepsis were implemented in their institution. Recommendations regarding meticulous antisepsis and sterile barriers such as masks and gowns may be reasonable but have no proven effect in diminishing this rare, iatrogenic event. Even when meningitis occurs temporally after spinal anesthesia, it is often difficult to establish a cause-and-effect relationship between spinal anesthesia and meningitis. The following case report describes a probable case of lumbar puncture–induced meningitis.[11] A 60-year-old man underwent kidney stone removal under general anesthesia. On postoperative day 6, the patient remained afebrile but was taken to the operating suite for transurethral clot evacuation. Spinal anesthesia was performed under aseptic technique. Cerebrospinal fluid was clear. Forty minutes later, shaking chills developed. Initial blood and urine cultures were negative. The following day, the patient became febrile and complained of headache and back pain and appeared confused. CSF examination revealed cloudy CSF with a leukocytosis (80% polymorphonucleocytes) and decreased glucose concentration consistent with bacterial infection but no growth on culture. Three days later, a repeat lumbar puncture was performed with similar results. A third lumbar puncture was performed 2 days later; culture yielded group D streptococcus (enterococci). Group D enterococci are unusual sources of meningitis. In this case, it is possible, although unlikely, that the patient was bacteremic before administration of the spinal anesthetic. It is more likely that the bacteria entered the blood stream during bladder irrigation (because bacteremia occurs in perhaps 60% of urologic procedures) and traversed the dura at the puncture site, similar to the animals in the study by Carp and Bailey.[10] However, despite the apparent temporal association, it is difficult to prove the presence of a prebacteremic dural puncture increased the risk of subsequent meningitis in this patient.

Bacterial meningitis can also present after epidural blockade with or without a localized epidural abscess.[18,19] Ready and Helfer[18] described 2 cases of meningitis after the use of epidural catheters in parturients. In the first case, a healthy 28-year-old parturient underwent lumbar epidural catheter placement for elective cesarean section. Epidural analgesia was provided for 48 hours postoperatively with an opioid infusion. At the time of catheter removal, a 4-cm erythematous indurated area, which was tender to palpation, was noted at the entry site. Three days later, the patient complained of severe headache, nuchal rigidity, and photophobia. An area of cellulitis was present at the epidural insertion site. CSF examinations revealed an elevated protein (308 mg/dL), decreased glucose (27 mg/dL), and 3,000 leukocytes/μL (73% polymorphonucleocytes). Culture of the CSF was positive for *Staphylococcus faecalis*. Urine and blood cultures were negative. There was no evidence of epidural abscess on magnetic resonance imaging (MRI) scan. Antibiotic therapy was initiated, and the patient recovered completely. Although it was thought the most likely source of the meningitis was the area of cellulitis surrounding the epidural catheter insertion site, the possibility of alternate causes could not be excluded.

In the second case, a lumbar epidural was placed in a healthy 25-year-old parturient. Delivery occurred uneventfully 50 minutes later, and the catheter was removed. No local inflammation was noted at the catheter insertion site. The patient reported a nonpositional headache and neck stiffness 24 hours later. Lumbar puncture revealed elevated protein (356 mg/dL), decreased glucose (5 mg/dL), and 4,721 leukocytes/μL (90% polymorphonucleocytes). CSF cultured positive for *Staphylococcus uberis* (a strain of α-hemolytic streptococcus). However, urine, blood, and vaginal cultures also grew the same organism. Antibiotic therapy was initiated, and recovery was complete. The short duration of the indwelling catheter, the lack of physical findings suggestive of infection at the catheter insertion site, and the presence of the organism in vaginal secretions, blood, and urine suggest that the source of the meningitis was most likely hematogenous spread of the infecting organism from the vagina.[18] The case reported by Berman and Eisele[13] and the 2 cases by Ready and Helfer[18] show that a cause-and-effect relationship should not be assumed between regional anesthesia and CNS infection but rather other possible sources should be investigated.

Epidural Abscess

Although infection has long been a concern of epidural anesthesia and analgesia, most cases of epidural catheter-induced spinal epidural abscess or meningitis appear as individual case reports or in retrospective reviews.[18,20,21] Although epidural catheters frequently colonize, clinical signs of epidural infection at the time of catheter removal or during follow-up are rare. Most epidural abscesses are not related to the placement of indwelling catheters but are believed to be related to infections of the skin, soft tissue, spine, or hematogenous spread to the epidural space. The incidence is generally reported to be extremely low, although 1 recent study suggests a possible increase.[22] Several studies have specifically examined the risk of epidural abscess in patients receiving epidural anesthesia and/or analgesia (Table 2). In a large retrospective review, epidural abscess from all causes accounted for 0.2 to 1.2 cases per 10,000 admissions to tertiary hospitals.[20] Of the 39 cases of epidural abscess occurring over a 30-year period from 1947 to 1974, *S aureus* (57%), streptococci (18%), and gram-negative bacilli (13%) were the most common pathogens. The source of infection was most often caused by osteomyelitis (38%), bacteremia (26%), and postoperative infection (16%). Only 1 of the 39 cases was related to an epidural catheter. In a more recent review, Ericsson et al.[21] reported 10 cases of epidural abscess. Four of these were associated with invasive spinal procedures including repeated lumbar punctures in the presence of meningitis (2 cases), epidural catheter (1 case), and a paravertebral anesthetic injection (1 case). In a retrospective study, Danner and Hartman[23] reported no spinal infections related to epidural anesthesia/analgesia. These authors were able to characterize the clinical course of epidural abscess as well as identify risk factors for neurologic recovery. Diagnosis was more difficult and often delayed in patients with chronic epidural abscesses because these patients were less likely to be febrile or have an elevated leukocyte count compared with patients with acute abscesses. However, rapid neurologic deterioration could occur in either group. In addition, earlier diagnosis and treatment improved neurologic outcome. Steroid administration and increased neurologic impairment at the time of surgery adversely affected outcome. Other retrospective reviews indicate a similarly low incidence of epidural abscess, with a Swedish study reporting none in 9,232 epidurals[24] and a German report of 2 cases in 13,000 procedures.[25] However, these reassuring studies notwithstanding, Wang et al.[22] present a differing view in the results of their 1-year prospective survey of Danish anesthesiologists. Seventy-eight percent of anesthesia departments participated, performing 17,372 epidural anesthetics. Twelve possible epidural abscesses were reported: 9 were subsequently determined to be spinal and epidural abscesses, 2 were subcutaneous infections, and 1 was a misplaced catheter. The 9 abscesses represented an incidence of 1:1,930 catheters and differed between university (1:5,661) and nonuniversity community hospitals (1:796). The epidural catheters in the affected cases were in situ for a mean of 11 days. Five of the 9 involved thoracic catheters, and 67% were placed for perioperative pain management. The majority (67%) had received LMWH as thromboprophylaxis before epidural catheter placement, and all but 1 patient was deemed immunocompromised. *S aureus* was the pathogen in 6 of the 9 cases; 2 patients had no bacterial growth. Several common factors, with undetermined significance, were noted in the affected patients; mean catheter times were longer than average, most patients were immunocompromised with chronic disease states, and perioperative antithrombotic agents were administered in the majority. The authors also pointed out that the overall neurologic outcome in these patients was grave, perhaps because of the insidious progression of symptoms and often late intervention.

Table 2. Differential Diagnosis of Epidural Abscess, Epidural Hemorrhage, and Anterior Spinal Artery Syndrome

	Epidural Abscess	Epidural Hemorrhage	Anterior Spinal Artery Syndrome
Age of patient	Any age	50% over 50 years	Elderly
Previous history	Infection*	Anticoagulants	Arteriosclerosis/hypotension
Onset	1-3 days	Sudden	Sudden
Generalized symptoms	Fever, malaise, back pain	Sharp, transient back and leg pain	None
Sensory involvement	None or paresthesias	Variable, late	Minor, patchy
Motor involvement	Flaccid paralysis, later spastic	Flaccid paralysis	Flaccid paralysis
Segmental reflexes	Exacerbated,* later obtunded	Abolished	Abolished
Myelogram/CT scan	Signs of extradural compression	Signs of extradural compression	Normal
Cerebrospinal fluid	Increased cell count	Normal	Normal
Blood data	Rise in sedimentation rate	Prolonged coagulation time*	Normal

*Infrequent findings.

Abscess formation after epidural or spinal anesthesia can be superficial, requiring limited surgical drainage and intravenous antibiotics, or occur deep in the epidural space with associated cord compression. The latter is fortunately a rare complication, but it requires aggressive, early surgical management to achieve a satisfactory outcome. Superficial infections present with local tissue swelling, erythema, and drainage, often associated with fever but rarely causing neurologic problems unless untreated. Epidural abscess formation usually presents days to weeks after neural block with clinical signs of severe back pain, local tenderness, and fever associated with leukocytosis (Table 3). Radiologic evidence of an epidural mass in the presence of a neurologic deficit is diagnostic. MRI without contrast is advocated as the most sensitive modality for evaluation of the spine when infection is suspected.[19,26,27] Surgical intervention within 12 hours is associated with the best chance of neurologic recovery.

The anesthesiologist is frequently faced with the management of the parturient with suspected chorioamnionitis, approximately 8% of whom are bacteremic. Bader et al.[28] investigated the use of regional anesthesia in women with chorioamnionitis. Three hundred nineteen women were identified from a total of 10,047 deliveries. Of the 319 women, 100 had blood cultures taken on the day of delivery. Eight of these had blood cultures consistent with bacteremia. Two hundred ninety-three of the 319 patients received a regional anesthetic; in 43 patients, antibiotics were administered before needle or catheter placement. No patient in the study, including those with documented bacteremias, had infectious complications. In addition, mean temperatures and leukocyte counts in patients who received blood cultures showed no significant differences between bacteremic and nonbacteremic groups. Goodman et al.[29] also retrospectively reviewed the hospital records of 531 parturients who received epidural or spinal anesthesia and were subsequently diagnosed with chorioamnionitis. Blood cultures were drawn in 146 patients; 13 were positive. Antibiotics were administered before the regional block was placed in only 123 patients, whereas nearly one third of patients did not receive antibiotic therapy in the entire peripartum period. As with the study by Bader et al.[28] leukocytosis, fever, abdominal tenderness, or foul-smelling discharge were not predictors of positive blood cultures. There were no infectious complications. These authors continue to administer spinal and epidural anesthesia in patients with suspected chorioamnionitis because the potential benefits of regional anesthesia outweigh the theoretical risk of infectious complications. However, the small number of patients with documented bacteremias in both studies defies a definitive statement regarding the risk of CNS infections in patients suspected of chorioamnionitis undergoing regional anesthetic techniques. Epidural-related infections are extremely rare in the obstetrical patient; Scott and Hibbard[30] report a single epidural abscess in 505,000 epidurals for obstetrical analgesia and anesthesia over a 4-year period in the United Kingdom. Relatively short catheter durations, antibiotic administration in select patients, and the lack of immunocompromise in this generally healthy population might be factors.

Strafford et al.[31] reviewed 1,620 pediatric patients who received epidural analgesia for postoperative pain relief. The authors concluded that for terminally ill patients, the risk of infection with long-term epidural catheterization is acceptable but recommended careful monitoring to avoid serious neurologic sequelae.

Jakobsen et al.[32] examined the records of 69 patients with localized infections who had a total of 120 catheters placed, undergoing on average 4 epidural anesthetics with catheters left in place for a mean of 9 days. On 12 occasions, the catheter was removed because of local infection, no specific therapy was instituted, and the infection resolved. There was 1 case of spondylitis not believed to be related to epidural catheterization. The retrospective nature of this study, as well as the small number of patients limit the conclusions, but it suggests that placing an epidural catheter in a chronically infected patient may not be associated with a high risk of epidural infection.

Factors Affecting Bacterial Colonization During Epidural Analgesia

The low frequency of significant epidural infection (1-2 cases per 10,000 hospital admissions[20]) associated with epidural catheter placement is especially notable when compared with the frequency of intravenous catheter-related septicemia, which approaches 1%, or greater than 50,000 cases annually. Several factors may contribute to the low incidence of epidural space infections, including meticulous attention to aseptic technique, careful monitoring of catheter insertion site, antibiotic prophylaxis, and perhaps the use of bacterial filters. However, because these interventions are commonly initiated in patients with indwelling central venous catheters, additional factors unique to epidural anesthesia and analgesia, such as the bactericidal effect of local anesthetic solutions may also contribute significantly. Bupivacaine and lidocaine have been shown to inhibit the growth of a variety of microorganisms in culture.[33] Unfortunately, the

Table 3. Infectious Complications After Regional Anesthesia

Author, Year	No. of Patients	Population	Neuraxial Techniques	Antibiotic Prophylaxis	Duration of Indwelling Catheter	Complications
Kane, 1981[1]	115,000	Surgical and obstetric	65,000 Spinal 50,000 Epidural	Unknown	Unknown	3 Meningitis (all after spinal anesthesia)
DuPen, 1990[34]	350	Cancer and AIDs patients	Permanent (tunneled) epidural analgesia	No	4-1,460 days	30 Insertion site infections, 19 deep track or epidural space infections; Treated with catheter removal and antibiotics, 15 uneventfully replaced
Scott, 1990[30]	505,000	Obstetrical	Epidural	Unknown	Unknown	1 Epidural abscess; laminectomy with partial recovery
Bader, 1992[28]	319	Parturients with chorioamnionitis	General (26), epidural (224), spinal (29), local (50) anesthesia	Yes (13%)	Surgical	None
Strafford, 1995[31]	1,620	Pediatric surgical	Epidural analgesia	No	2.4 days median	3 Positive epidural catheter tip cultures 1 Candida colonization of epidural space (along with necrotic tumor)
Goodman, 1996[29]	531	Parturients with chorioamnionitis	Spinal (14), epidural (517) anesthesia and analgesia	Yes (23%)	>24 h in (64 patients)	None
Dahlgren, 1995[24]	18,000	All indications and ages of patients	Spinal (8,768) and Epidural (9,232)	Unknown	Unknown	None
Kindler, 1996[25]	13,000	4,000 Obstetrical 9,000 Surgical	Epidural	Unknown	Unknown	2 Epidural abscess, both requiring laminectomy
Auroy, 1997[2]	71,053	Surgical	Spinal (40,640) Epidural (30,413)	Unknown	Unknown	None
Aromaa, 1997[44]	720,000	Surgical	Epidural (170,000) Spinals (550,000)	Unknown	Unknown	4 Meningitis 2 Epidural abscess 2 Discitis 2 Superficial skin infections
Wang, 1999[22]	17,372	Surgical, cancer & trauma	Epidural	Unknown	11 days mean 6 days median	9 Epidural abscess; 7 required laminectomy; complete recovery in 6 of 10 patients 2 Subcutaneous infections
Moen, 2004[45]	1,710,000	Pain, surgical and obstetrical	Spinal (1,260,000) Epidural (450,000)	Unknown	2d-5wk	29 Meningitis; partial sequelae in 6 patients 13 Epidural abscess, laminectomy performed in 6 patients; 4 of 5 patients with deficits did not recover

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bactericidal effect decreases significantly with concentrations of local anesthetic typically used to provide analgesia, whereas opioid solutions do not exhibit any ability to inhibit bacterial growth. In addition, the growth of *S aureus* and coagulase-negative staphylococcus, the most commonly identified pathogens in epidural infections, is inhibited only at higher concentrations of local anesthetic, such as solutions of 2% lidocaine and 0.5% bupivacaine. Therefore, although it appears that local anesthetic solutions are unlikely to prevent epidural infections in most patients receiving epidural analgesia, it is possible that in immunocompromised patients local anesthetics may inhibit the growth of more fastidious organisms, even at low concentrations. Further clinical studies are needed to investigate the in vivo bactericidal effects of dilute local anesthetic solutions.

The catheter hub, catheter insertion site, and hematogenous spread are 3 major routes of entry for microorganisms into the epidural space, with the catheter hub accounting for nearly half of the sources.[34-36] A bacterial filter placed at the catheter hub acts as a physical barrier for bacteria present in the infusing solution and should theoretically reduce the incidence of epidural colonization. However, studies of epidural catheter tip cultures have reported mixed results, and cases of epidural infection after hub colonization despite the use of filters have been reported.[34,36,37] Possible explanations for hub-related epidural infections in patients with bacterial filters include a reduced antimicrobial effectiveness with prolonged use and direct contamination of the hub during filter-changing techniques. De Cicco et al.[38] reported a positive trend between the number of filter changes and the rate of positive hub cultures. These data suggest that continued attention to aseptic technique is warranted throughout the period of epidural catheterization and that the use of bacteriologic filters is alone unlikely to be efficacious in preventing epidural colonization and infection.[39]

Several studies have evaluated the risk factors for the development of epidural space infections in patients with indwelling epidural catheters. Darchy et al.[40] studied 75 patients in the intensive care unit receiving epidural analgesia (median 4 days). Nine patients had local (catheter insertion site) infections, including 4 patients with epidural catheter (local inflammation with positive epidural catheter culture) infections, representing a frequency of 2.7 local (catheter insertion site) infections and 1.2 epidural infections per 100 days of epidural catheterization. Staphylococcus epidermidis was the most frequently cultured microorganism. All catheters were removed on the appearance of a discharge at the catheter insertion site, and antibiotic therapy was not specifically prescribed. The presence of both local erythema and discharge was associated with positive epidural catheter cultures. Concomitant infection at other sites, antibiotic therapy, and duration of indwelling epidural catheter were not significant risk factors for epidural infections. The authors recommended a meticulous daily inspection of the catheter insertion site and immediate removal of the catheter if both erythema and local discharge are present.

Anesthetic Management

These studies and epidemiologic data provide guidance in the administration of spinal or epidural anesthesia in the febrile patient. However, as with all clinical judgments, the decision to perform a regional anesthetic technique must be made on an individual basis considering the anesthetic alternatives, the benefits of regional anesthesia, and the risk of CNS infection (which may theoretically occur in any bacteremic patient).

Numerous clinical and laboratory studies have suggested an association between dural puncture during bacteremia and meningitis. However, much of these data need to be interpreted with caution. The clinical studies are limited to pediatric patients who are historically at high risk for meningitis. Many of the original animal studies used bacterial counts that were far in excess of those noted in humans in early sepsis, making CNS contamination more likely.[4,41] Despite these conflicting results, many experts suggest that, except in the most extraordinary circumstances, central neuraxial block should not be performed in patients with untreated systemic infection.

Available data suggest that patients with evidence of systemic infection may safely undergo spinal anesthesia, provided appropriate antibiotic therapy is initiated before dural puncture and the patient has shown a response to therapy, such as a decrease in fever.[10,42] Although few data exist on the administration of epidural anesthesia in the patient with a treated systemic infection, the studies by Bader et al.[28] and Goodman et al.[29] are reassuring.

Available data suggest that spinal anesthesia may be safely performed in patients at risk for low-grade transient bacteremia after dural puncture. Once again, little information exists concerning the risk of epidural anesthesia in patients suspected of developing an intraoperative transient bacteremia (such as during a urologic procedure). However, short-term epidural catheterization is most likely safe, as suggested by large retrospective reviews that included a significant number of obstetric and urologic patients.[1,2]

Conservatively, all patients with an established local or systemic infection should be considered at risk for developing infection of the CNS. Patients should be observed carefully for signs of infection when a continuous epidural catheter is left in place for prolonged periods. It is probably advisable to remove an epidural catheter in patients with either known or suspected untreated bacteremia postoperatively. In addition, injection of local anesthetic or insertion of a catheter in an area at high risk for bacterial contamination, such as the sacral hiatus, may also increase the risk for abscess formation, emphasizing the importance of meticulous aseptic technique. A delay in diagnosis and treatment of major CNS infections of even a few hours significantly worsens neurologic outcome. Bacterial meningitis is a medical emergency. Mortality is approximately 30% even with antibiotic therapy. Meningitis presents most often with fever, severe headache, altered level of consciousness, and meningismus. The diagnosis is confirmed with a lumbar puncture. Lumbar puncture should not be performed if epidural abscess is suspected because contamination of the intrathecal space may result. CSF examination in the patient with meningitis reveals leukocytosis, a glucose level <30 mg/dL, and a protein level >150 mg/dL. In addition, the anesthesiologist should consider atypical organisms in patients suspected of meningitis after spinal anesthesia.

The clinical course of epidural abscess progresses from neuraxial discomfort and root pain to weakness (including bowel and bladder symptoms) and eventually paralysis.[23,43] The initial back pain and radicular symptoms may remain stable for hours to weeks. However, the onset of weakness often progresses to complete paralysis within 24 hours. Although the diagnosis was historically made with myelogram, noncontrast MRI imaging is currently recommended. However, when MRI capabilities are not available, computed tomography myelography may also be used. A combination of antibiotics and surgical drainage remains the treatment of choice. As with spinal hematoma, neurologic recovery is dependent on the duration of the deficit and the severity of neurologic impairment before treatment.[23]

Recommendations

Recommendations are as follows:

1. Serious central neuraxial infections such as arachnoiditis, meningitis, and abscess after spinal or epidural anesthesia are rare (Grade B).
2. The decision to perform a regional anesthetic technique must be made on an individual basis considering the anesthetic alternatives, the benefits of regional anesthesia, and the risk of CNS infection (which may theoretically occur in any bacteremic patient) (Grade C).
3. Despite conflicting results, many experts suggest that, except in the most extraordinary circumstances, central neuronal block should not be performed in patients with untreated systemic infection (Grade C).
4. Available data suggest that patients with evidence of systemic infection may safely undergo spinal anesthesia, provided appropriate antibiotic therapy is initiated before dural puncture and the patient has shown a response to therapy, such as a decrease in fever (placement of an indwelling epidural (or intrathecal) catheter in this group of patients remains controversial) (Grade A).
5. Available data suggest that spinal anesthesia may be safely performed in patients at risk for low-grade transient bacteremia after dural puncture (Grade B).
6. Epidural catheters should be removed in the presence of local erythema and/or discharge; there are no convincing data to suggest that concomitant infection at remote sites or the absence of antibiotic therapy are risk factors for infection.
7. A delay in diagnosis and treatment of major CNS infections of even a few hours may significantly worsen neurologic outcome (Grade B).

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