Blood and Pus: Hemorrhagic and Infectious Complications of Neuraxial Anesthesia

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Spinal Hematoma

The actual incidence of neurologic dysfunction resulting from hemorrhagic complications associated with neuraxial blockade is unknown; however, the incidence cited in the literature is estimated to be less than 1 in 150,000 epidural and less than 1 in 220,000 spinal anesthetics (Tryba 1993). In a review of the literature between 1906 and 1994, Vandermeulen et al. (Vandermeulen 1994) reported 61 cases of spinal hematoma associated with epidural or spinal anesthesia. In 87% of patients, a hemostatic abnormality or traumatic/difficult needle placement was present. More than one risk factor was present in 20 of 61 cases. Importantly, although only 38% of patients had partial or good neurologic recovery, spinal cord ischemia tended to be reversible in patients who underwent laminectomy within eight hours of onset of neurologic dysfunction.

The need for prompt diagnosis and intervention in the event of a spinal hematoma was also demonstrated in a review of the American Society of Anesthesiologists (ASA) Closed Claims project, which noted that spinal cord injuries were the leading cause of claims in the 1990’s (Cheney 1999). Spinal hematomas accounted for nearly half of the spinal cord injuries. Risk factors for spinal hematoma included epidural anesthesia in the presence of intravenous heparin during a vascular surgical or diagnostic procedure. Importantly, the presence of postoperative numbness or weakness was typically attributed to local anesthetic effect rather than spinal cord ischemia, which delayed the diagnosis. Patient care was rarely judged to have met standards (1 of 13 cases) and the median payment was very high.

It is impossible to conclusively determine risk factors for the development of spinal hematoma in patients undergoing neuraxial blockade solely through review of the case series, which represent only patients with the complication and do not define those who underwent uneventful neuraxial analgesia. However, large inclusive surveys that evaluate the frequencies of complications (including spinal hematoma), as well as identify subgroups of patients with higher or lower risk, enhance risk stratification. In the series by Moen et al. (Moen 2004) involving nearly 2 million neuraxial blocks, there were 33 spinal hematomas. The methodology allowed for calculation of frequency of spinal hematoma among patient populations. For example, the risk associated with epidural analgesia in women undergoing childbirth was significantly less (1 in 200,000) than that in elderly women undergoing knee arthroplasty (1 in 3600, p<0.0001). Likewise, women undergoing hip fracture surgery under spinal anesthesia had an increased risk of spinal hematoma (1 in 22,000) compared to all patients undergoing spinal anesthesia (1 in 480,000).

Overall, these series suggest that the risk of clinically significant bleeding varies with age (and associated abnormalities of the spinal cord or vertebral column), the presence of an underlying coagulopathy, difficulty during needle placement, and an indwelling neuraxial catheter during sustained anticoagulation (particularly with standard heparin or LMWH). They also consistently demonstrate the need for prompt diagnosis and intervention.

Practice guidelines or recommendations summarize evidence-based reviews. However, the rarity of spinal hematoma defies a prospective-randomized study, and there is no current laboratory model. As a result, the consensus statements developed by the American Society of Regional Anesthesia and Pain Medicine represent the collective experience of recognized experts in the field of neuraxial anesthesia and anticoagulation (Horlocker, 2003). They are based on case reports, clinical series, pharmacology, hematology, and risk factors for surgical bleeding. An understanding of the complexity of this issue is essential to patient management.

Oral Anticoagulants

Few data exist regarding the risk of spinal hematoma in patients with indwelling epidural catheters who are anticoagulated with warfarin. The optimal duration of an indwelling catheter and the timing of its removal also remain controversial. To date, only three studies have evaluated the risk of spinal hematoma in patients with indwelling spinal or epidural catheters who receive oral anticoagulants perioperatively. Odoom and Sih (Odoom 1983) performed 1000 continuous lumbar epidural anesthetics in vascular surgical patients who were receiving oral anticoagulants preoperatively. The thrombotest (a test measuring factor IX activity) was decreased (but not below 10% activity) in all patients prior to needle placement. Heparin was also administered intraoperatively. Epidural catheters remained in place for 48 hours postoperatively. There were no neurologic complications. While these results are reassuring, the obsolescence of the thrombotest as a measure of anticoagulation combined with the unknown coagulation status of the patients at the time of catheter removal limit the usefulness of these results. Therefore, except in extraordinary circumstances, spinal or epidural needle/catheter placement and removal should not be performed in fully anticoagulated patients.
There were no symptomatic spinal hematomas in two smaller series with a total of nearly 700 patients undergoing neuraxial block in combination with warfarin anticoagulation perioperatively (Horlocker 1994; Odoom 1983; Wu 1996). In both studies, epidural catheters were left indwelling approximately two days. The mean international normalized ratio (INR) at the time of catheter placement was 1.4, although in a small number of patients the INR was therapeutic (2.0-3.0). A large variability in patient response to warfarin was also noted, demonstrating the need for close monitoring of the coagulation status (Horlocker 1994). A large series of patients is required to confirm these results. However, the small number of hematomas reported and widespread use of warfarin thromboprophylaxis (at least in the United States) in patients administered neuraxial block suggests that patients receiving oral anticoagulants may safely undergo regional techniques with appropriate monitoring of the level of anticoagulation.

Intravenous and Subcutaneous Standard Heparin

The safety of neuraxial techniques in combination with intraoperative heparinization is well documented, providing no other coagulopathy is present. In a study involving over 4000 patients, Rao and El-Etr (Rao 1981) demonstrated the safety of indwelling spinal and epidural catheters during systemic heparinization during vascular surgery. However, the heparin was administered at least 60 minutes after catheter placement, level of anticoagulation was closely monitored, and the indwelling catheters were removed at a time when circulating heparin levels were relatively low. A subsequent study in the neurologic literature by Ruff and Dougherty (Ruff 1981) reported spinal hematomas in 7 of 342 patients (2%) who underwent a diagnostic lumbar puncture and subsequent heparinization. Traumatic needle placement, initiation of anticoagulation within one hour of lumbar puncture and concomitant aspirin therapy were identified as risk factors in the development of spinal hematoma in anticoagulated patients. Subsequent studies using similar methodology have verified the safety of this practice, provided the monitoring of anticoagulant effect and the time intervals between heparinization and catheter placement/removal are maintained.

There have been continued discussions regarding the relative risk (and benefit) of neuraxial anesthesia and analgesia in the patient undergoing complete heparinization for cardiopulmonary bypass. A review has recommended certain precautions to be taken, including delay of surgery for 24 in case of traumatic needle/catheter placement (Chaney 1997). Although there were no spinal hematomas reported in the small series of patients who have undergone epidural block for cardiac surgery, investigators repeatedly observe that this technique remains controversial because of the degree of anti-coagulation required and the associated risk of permanent spinal cord damage from an epidural hematoma. Such a risk must be balanced by clinical advantages if the technique is to be justified (Horlocker 2003).

Low-dose subcutaneous standard (unfractionated) heparin is administered for thromboprophylaxis in patients undergoing major thoracoabdominal surgery and in patients at increased risk of hemorrhage with oral anticoagulant or low molecular weight heparin (LMWH) therapy. A review of the literature by Schwander and Bachmann (Schwander 1991) noted no spinal hematomas in over 5000 patients who received subcutaneous heparin in combination with spinal or epidural anesthesia. There are only three cases of spinal hematoma associated with neuraxial blockade in the presence of low-dose heparin, two of which involved a continuous epidural anesthetic technique (Vandermeulen 1994). It is important to note that while the ACCP guidelines are more often recommending thrice daily dosing of subcutaneous heparin (due to patient co-morbidities and increased risk of thromboembolism), the safety of neuraxial block in these patients is unknown.

Low Molecular Weight Heparin

Extensive clinical testing and utilization of LMWH in Europe over the last ten years suggested that there was not an increased risk of spinal hematoma in patients undergoing neuraxial anesthesia while receiving LMWH thromboprophylaxis perioperatively (Bergqvist 1992; Vandermeulen 1994). However, in the five years since the release of LMWH for general use in the United States in May 1993, over 60 cases of spinal hematoma associated with neuraxial anesthesia administered in the presence of perioperative LMWH prophylaxis were reported to the manufacturer (Horlocker 1998; Horlocker 2003). Many of these events occurred when LMWH was administered intraoperatively or early postoperatively to patients undergoing continuous epidural anesthesia and analgesia. Concomitant antiplatelet therapy was present in several cases. The apparent difference in incidence in Europe compared to the United States may be a result of a difference in dose and dosage schedule. For example, in Europe the recommended dose of enoxaparin is 40 mg once daily (with LMWH therapy initiated 12 hours preoperatively), rather than 30 mg every twelve hours. However, timing of catheter removal may also have an impact. It is likely that the lack of a trough in anticoagulant activity associated with twice daily dosing resulted in catheter removal occurring during significant anticoagulant activity. Importantly, there are no data to suggest that the risk of spinal hematoma is increased with certain LMWH formulations (Horlocker 1998). The incidence of spinal hematoma in patients undergoing neuraxial block in combination with LMWH has been estimated at 1 in 40,800 spinal anesthetics and 1 in 3100 continuous epidural anesthetics (Schroeder 1998). It is interesting in that the frequency of spinal hematoma in this series is similar to that reported by Moen et al (Moen 2004) for women undergoing total knee replacement with epidural analgesia.
The indications and labeled uses for LMWH continue to evolve. Indications for thromboprophylaxis as well as treatment of thromboembolism or MI have been introduced. These new applications and corresponding regional anesthetic management warrant discussion (Geerts 2008). Several off-label applications of LMWH are of special interest to the anesthesiologist. LMWH has been demonstrated to be efficacious as a “bridge therapy” for patients chronically anticoagulated with warfarin, including parturients, patients with prosthetic cardiac valves, a history of atrial fibrillation, or preexisting hypercoagulable condition. The doses of LMWH are those associated with DVT treatment, not prophylaxis, and are much higher. An interval of at least 24 hours is required for the anticoagulant activity to resolve.

Antiplatelet Medications

Antiplatelet medications are seldom used as primary agents of thromboprophylaxis. However, many orthopedic patients report chronic use of one or more antiplatelet drugs. Although Vandermeulen et al (Vandermeulen 1994) implicated antiplatelet therapy in 3 of the 61 cases of spinal hematoma occurring after spinal or epidural anesthesia, several large studies have demonstrated the relative safety of neuraxial blockade in both obstetric, surgical, and pain clinic patients receiving these medications (CLASP, 1994; Horlocker 2002; Horlocker 1995). In a prospective study involving 1000 patients, Horlocker et al (Horlocker 1995) reported that preoperative antiplatelet therapy did not increase the incidence of blood present at the time of needle/catheter placement or removal, suggesting that trauma incurred during needle or catheter placement is neither increased nor sustained by these medications. The clinician should be aware of the possible increased risk of spinal hematoma in patients receiving antiplatelet medications who undergo subsequent heparinization (Ruff 1981). Ticlopidine and clopidogrel are also platelet aggregation inhibitors. These agents interfere with platelet-fibrinogen binding and subsequent platelet-platelet interactions. The effect is irreversible for the life of the platelet. Ticlopidine and clopidogrel have no effect on platelet cyclooxygenase, acting independently of aspirin. However, these medications have not been tested in combination. Platelet dysfunction is present for 5–7 days after discontinuation of clopidogrel and 10–14 days with ticlopidine. Platelet glycoprotein IIb/IIIa receptor antagonists, including abciximab (Reopro®, epifibatide (Integrilin®) and tirofiban (Aggrastat®), inhibit platelet aggregation by interfering with platelet-fibrinogen binding and subsequent platelet-platelet interactions. Time to normal platelet aggregation following discontinuation of therapy ranges from eight hours (epifibatide, tirofiban) to 48 hours (abciximab). Increased perioperative bleeding in patients undergoing cardiac and vascular surgery after receiving ticlopidine, clopidogrel and glycoprotein IIb/IIIa antagonists warrants concern regarding the risk of anesthesia-related hemorrhagic complications.

Table1. Neuraxial Anesthesia and Anticoagulation

<table>
<thead>
<tr>
<th>Warfarin</th>
<th>Discontinue chronic warfarin therapy 4–5 days before spinal procedure and evaluate INR. INR should be within the normal range at time of procedure to ensure adequate levels of all vitamin K-dependent factors. Postoperatively, daily INR assessment with catheter removal occurring with INR&lt; 1.5</th>
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<tr>
<td>Antiplatelet medications</td>
<td>No contraindications with aspirin or other NSAIDs. Thienopyridine derivatives (clopidogrel and ticlopidine) should be discontinued 7 days and 14 days, respectively, prior to procedure. GP IIb/IIIa inhibitors should be discontinued to allow recovery of platelet function prior to procedure (8 hours for tirofiban and epifibatide, 24–48 hours for abciximab).</td>
</tr>
<tr>
<td>Thrombolytics/ fibrinolytics</td>
<td>There are no available data to suggest a safe interval between procedure and initiation or discontinuation of these medications. Follow fibrinogen level and observe for signs of neural compression.</td>
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<tr>
<td>LMWH</td>
<td>Delay procedure at least 12 hours from the last dose of thromboprophylaxis LMWH dose. For &quot;treatment&quot; dosing of LMWH, at least 24 hours should elapse prior to procedure. LMWH should not be administered within 24 hours after the procedure. Indwelling epidural catheters should be maintained with caution and only with once daily dosing of LMWH and strict avoidance of additional hemostasis altering medications, including ketorolac.</td>
</tr>
<tr>
<td>Unfractionated SQ heparin</td>
<td>There are no contraindications to neuraxial procedure if total daily dose is less than 10,000 units. For higher dosing regimens, manage according to intravenous heparin guidelines.</td>
</tr>
<tr>
<td>Unfractionated IV heparin</td>
<td>Delay needle/catheter placement 2–4 hours after last dose, document normal aPTT. Heparin may be restarted 1 hour following procedure. Sustained heparinization with an indwelling neuraxial catheter associated with increased risk; monitor neurologic status aggressively.</td>
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NSAIDs= nonsteroidal antiinflammatory drugs; GP IIb/IIIa= platelet glycoprotein receptor IIb/IIIa inhibitors; INR= international normalized ratio; LMWH= low-molecular-weight heparin; aPTT= activated partial thromboplastin time. Adapted from: (Horlocker, 2003).

Anesthetic Management of the Anticoagulated Patient

The decision to perform spinal or epidural anesthesia/analgesia and the timing of catheter removal in a patient receiving thromboprophylaxis should be made on an individual basis, weighing the small, though definite risk of spinal hematoma with the benefits of regional anesthesia for a specific patient. Alternative anesthetic and analgesic techniques exist for patients considered to be at an unacceptable risk. The patient’s coagulation status should be
optimized at the time of spinal or epidural needle/catheter placement, and the level of anticoagulation must be carefully monitored during the period of epidural catheterization (Table 1).

It is important to note that patients respond with variable sensitivities to anticoagulant medications. Indwelling catheters should not be removed in the presence of a significant coagulopathy, as this appears to significantly increase the risk of spinal hematoma (Moen 2004; Vandermeulen 1994). In addition, communication between clinicians involved in the perioperative management of patients receiving anticoagulants for thromboprophylaxis is essential in order to decrease the risk of serious hemorrhagic complications. The patient should be closely monitored in the perioperative period for signs of cord ischemia. If spinal hematoma is suspected, the treatment of choice is immediate decompressive laminectomy. Recovery is unlikely if surgery is postponed for more than 10-12 hours; less than 40% of the patients in the series by Vandermeulen et al. (Vandermeulen 1994) had partial or good recovery of neurologic function.

**Meningitis and Epidural Abscess**

Bacterial infection of the central neuraxis may present as meningitis or cord compression secondary to abscess formation. 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 neural axis may present as meningitis or cord compression secondary to abscess formation. The infectious source for meningitis and epidural abscess may result from distant colonization or localized infection with subsequent hematogenous spread and CNS invasion. The anesthetist may also transmit microorganisms directly into the CNS by needle/catheter contamination through a break in aseptic technique or passage through a contiguous infection. An indwelling neuraxial catheter, though aseptically sited, may be colonized with skin flora and consequently serve as a source for ascending infection to the epidural or intrathecal space.

Historically, the frequency of serious CNS infections such as arachnoiditis, meningitis, and abscess following spinal or epidural anesthesia was considered to be extremely low- cases were reported as individual cases or small series (Baker 1975; Ready 1989). However, recent epidemiologic series from Europe suggest that the frequency of infectious complications associated with neuraxial techniques is increasing (Ericsson 1990; Moen 2004). In a national study conducted from 1997 to 1998 in Denmark, Wang et al (Wang 1999) reported the incidence of epidural abscess after epidural analgesia was 1:1930 catheters. Patients with epidural abscess had an extended duration of epidural catheterization (median 6 days, range 3-31 days). In addition, the majority of the patients with epidural abscess were immunocompromised. Often the diagnosis was delayed; the time to first symptom to confirmation of the diagnosis was a median of five days. *S. aureus* was isolated in 67% of patients. Patients without neurologic deficits were successfully treated with antibiotics, while those with deficits underwent surgical decompression, typically with only moderate neurologic recovery. It is difficult to determine why the frequency of symptomatic epidural abscess was so high in this series. Since perioperative antithrombotic therapy was involved in most cases, it is possible that the epidural abscesses were infected “micro” epidural hematomas, but this is not strongly supported by the diagnostic imaging studies and neurosurgical findings.

In the series by Moen et al (Moen 2004) there were 42 serious infectious complications. Epidural abscess occurred in 13 patients; nine (70%) were considered immunocompromised as a result of diabetes, steroid therapy, cancer or alcoholism. Six patients underwent epidural block for analgesia following trauma. The time from placement of the epidural catheter to first symptoms ranged from 2 days to 5 weeks (median 5 days). Although prevailing symptoms were fever and severe backache, five developed neurologic deficits. All seven positive cultures isolated *S. aureus*. Overall neurologic recovery was complete in 7 of 12 patients. However, four of the five patients with neurologic symptoms did not recover. Meningitis was reported in 29 patients for an overall incidence of 1:53,000. A documented perforation of the dura (intentional or accidental) occurred in 25 of 29 cases. In the 12 patients in whom positive cultures were obtained, alpha-hemolytic streptococci were isolated in 11 patients and *S. aureus* in one.

These large epidemiologic studies represent new and unexpected findings regarding the demographics, frequency, etiology and prognosis of infectious complications following neuraxial anesthesia. Epidural abscess is most likely to occur in immunocompromised patients with prolonged durations of epidural catheterization. The most common causative organism is *S. aureus*, which suggests the colonization and subsequent infection from normal skin flora as the pathogenesis. Delays in diagnosis and treatment result in poor neurologic recovery, despite surgical decompression. Conversely, patients who develop meningitis following neuraxial blockade typically are healthy and have undergone uneventful spinal anesthesia. Furthermore, the series by Moen et al (Moen 2004) validates the findings of individual case reports of meningitis after spinal anesthesia- the source of the pathogen is mostly likely to be the upper airway of the proceduralist. While the frequency of serious infectious complications is much higher than
reported previously, the results may be due to differences in reporting and/or clinical practice (asepsis, perioperative antibiotic therapy, duration of epidural catheterization)

**Meningitis after Dural Puncture and Neuraxial Anesthesia**
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 presumed mechanisms include introduction of blood into the intrathecal space during needle placement and disruption of the protection provided by the blood-brain barrier. Initial investigations were performed over 80 years ago (Weed 1919; Wegeforth 1919). Subsequent clinical studies reported conflicting results regarding the causal relationship between dural puncture during bacteremia and meningitis. However, the protective effect of antibiotic administration prior to lumbar puncture was suggested (Carp 1992; Teele 1981).

**Epidural Abscess after Epidural Anesthesia**
Several relevant studies have specifically examined the risk of epidural abscess in patients receiving epidural anesthesia and/or analgesia. Bader et al. (Bader 1992) 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 prior to 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. 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.

The safety of epidural analgesia in 75 patients admitted to the intensive care unit was prospectively evaluated by Darchy et al (Darchy 1996). There were no epidural abscesses. However, five of nine patients with positive cultures of the catheter insertion site also had positive catheter tip cultures (epidural catheter infection); *Staphylococcus epidermidis* was the most commonly cultured microorganism. Local infection of the catheter site was treated with catheter removal, but antibiotic therapy was not specifically prescribed. Concomitant infection at other sites, antibiotic prophylaxis, and duration of epidural analgesia were not risk factors for epidural-analgesia related infections. The authors noted that the presence of both erythema and local discharge is a strong predictor of local and epidural catheter infection.

Epidural anesthesia and analgesia in a patient with a known systemic or localized infection remains controversial. Jakobsen et al (Jakobsen 1995) retrospectively reviewed the records of 69 patients with abscesses or wound infections who underwent epidural catheter placement for surgical debridement over a seven year-period. Several patients had more than one catheter inserted. Catheters were left indwelling for a mean of nine days. On 12 occasions (eight patients) the catheter was removed because of local infection. None of the patients demonstrated signs or symptoms of neuraxial infection. The authors concluded that epidural anesthesia is relatively safe for patients requiring repeat surgical treatment of localized infection. In contrast, Bengtsson et al. (Bengtsson 1997) reported three epidural catheter-related infections in patients with cutaneous wounds over a four year-period. All patients were treated with antibiotic therapy; one patient underwent transcutaneous drainage of an epidural abscess. However, there were no neurologic deficits. It is difficult to determine the actual risk of epidural abscess in patients with chronic localized infections who undergo epidural catheter placement due to the small number of patients studied and the rarity of this complication. Therefore, the clinician must maintain vigilance in neurologic monitoring to assure early recognition and treatment.

**Neuraxial Blockade in the Immunocompromised Patient**
Large series have demonstrated that patients with immunodeficiencies are at increased risk for infectious complications compared to those with intact immune function. However, there are few investigations which have evaluated the frequency of meningitis or epidural abscess within a specific immunodeficient population (Ericsson 1990; Horlocker 2006; Moen 2004).

**Herpes Simplex Virus**
Herpes simplex virus type 2 (HSV-2) infection is an incurable, recurrent disease characterized by asymptomatic periods alternating with recrudescence of genital lesions. The primary infection is associated with viremia and can be accompanied by a variety of symptoms, including fever, headache, and rarely aseptic meningitis. In contrast, recurrent or secondary infections present as genital lesions without evidence of viremia. When obstetric patients present for delivery with evidence of active HSV-2 infection, cesarean section is recommended to avoid exposing the neonate to the virus during
vaginal delivery. Neuraxial block in these patients is controversial because of the theoretical potential of introducing the virus into the CNS. However, there are little data to support these concerns.

**Table 2. Infectious Complications following Neuraxial Anesthesia in the Immunocompromised Patient**

- The attenuated inflammatory response within the immunocompromised patient may diminish the clinical signs and symptoms often associated with infection and result in a delay in diagnosis and treatment.
- The range of microorganisms causing invasive infection in the immunocompromised host is much broader than that affecting the general population and includes atypical and opportunistic pathogens.
- Early and effective therapy is paramount in optimizing neurologic outcome; consultation with an infectious disease specialist is advised.
- Prolonged antibiotic therapy (weeks-months) is often required because of persistent and immunologic deficiencies.
- Since eradication of infection is difficult once established, prevention of infection is paramount in caring for immunocompromised patients.

From: Horlocker, et al 2006, with permission

**Human Immunodeficiency Virus**

The risk of performing neuraxial block in patients infected with human immunodeficiency virus (HIV) is largely undetermined. Approximately 40% of patients with the diagnosis of acquired immune deficiency syndrome (AIDS) have clinical signs of neuropathy, and 70% to 80% have neuropathic changes present at autopsy. Since the virus infects the CNS early in the disease, it is unlikely that neuraxial block would result in new CNS transmission. However, the neurologic symptoms associated with HIV infection such as aseptic meningitis, headache, and polyneuropathy would be indistinguishable from those related to regional technique. Hughes et al. (Hughes 1995) reported safe administration of neuraxial block to 18 HIV-infected parturients. The patients studied showed no postpartum change in immune, infectious or neurologic status. Avidan et al. (Avidan 2002) and Bremerich et al. (Bremerich 2003) also reported a low complication rate for parturients with HIV infection on antiretroviral therapy who underwent spinal anesthesia. However, in all three series (with a combined total of 117 patients), the patients were relatively healthy and in the early stage of their disease. The effects of anesthesia on patients with more advanced disease are unreported.

**Aseptic Technique**

Although previous publications have repeatedly recommended meticulous aseptic technique, only recently have standards for asepsis during the performance of regional anesthetic procedures been defined (Hebl 2006) (Table 3).

Handwashing remains the most crucial component of asepsis; gloves should be regarded as a supplement to- not a replacement of handwashing (Saloojee 2001). The use of an antimicrobial soap reduces bacterial growth and reduces the risk of bacteria being released into the operative field should gloves become torn or punctured during the procedure. An alcohol-based antiseptic provides the maximum degree of antimicrobial activity and duration. Prior to washing, all jewelry (rings, watches, etc) should be removed; higher microbial counts have been noted in health care workers who do not routinely remove these items before handwashing. Sterile gloves protect not only patients from contamination, but also health care workers from blood-borne pathogens and are required by the Occupational Safety and Health Administration (Hebl 2006). Glove leaks are more likely to occur with vinyl compared to latex gloves (24% vs. 2), with contamination of the health care workers’ hands noted following the leaks in 23% of cases (Olsen 1993). Conversely, the use of gowns does not further reduce the likelihood of cross contamination in an intensive care unit setting compared to gloves alone. At this time, there are insufficient data to make recommendations regarding routine use for single injection or temporary neuraxial/peripheral catheter placement. However, placement of an indwelling permanent device, such as a spinal cord stimulator, warrants the same asepsis as a surgical procedure, including gowns, hats, and antibiotic pretreatment (Hebl 2006; Rathmell 2006).

Surgical masks, initially considered a barrier to protect the proceduralist from patient secretions and blood, are now required by the Center for Disease Control (http://www.cdc.gov/ncidod/dhqp/pdf/guidelines/Isolation2007.pdf) due to the increasing number of cases of post spinal meningitis, many of which result from contamination of the epidural or intrathecal space with pathogens from the operator’s buccal mucosa (Moen 2004; Schneeberger 1996; Trautmann 2002).
Antiseptic Solutions

Controversy still exists regarding the most appropriate and safe antiseptic solution for patients undergoing neuraxial and peripheral techniques. Povidone iodine and chlorhexidine gluconate (with or without the addition of isopropyl alcohol) have been most extensively studied (Birnbach 1998; Kiniron 2001). In nearly all clinical investigations, the bactericidal effect of chlorhexidine was more rapid and more effective (extending its effect hours following its application) than povidone iodine. The addition of isopropyl alcohol accelerates these effects. Chlorhexidine is effective against nearly all nosocomial yeasts, and bacteria (gram-positive and gram-negative); resistance is extremely rare. It also remains effective in the presence of organic compounds, such as blood. It must be noted that chlorhexidine-alcohol labeling contains a warning against use as a skin preparation prior to lumbar puncture. The FDA has not formally approved chlorhexidine for skin preparation prior to lumbar puncture because of the lack of animal and clinical studies examining the neurotoxic potential of chlorhexidine, nor due to a number of reported cases of nerve injury. Indeed, it is important to note that there are no cases of neurotoxicity with either chlorhexidine or alcohol (Hebl 2006). Therefore, as a result of its superior effect, alcohol-based chlorhexidine solutions are considered the antiseptic of choice for skin preparation before any regional anesthetic procedure (Hebl 2006).

Anesthetic Management of the Infected or Febrile Patient

In summary, several clinical and laboratory studies have suggested an association between dural puncture during bacteremia and meningitis. The data are not equivocal, however. The clinical studies are limited to pediatric patients who are historically at high-risk for meningitis. Many of the original animal studies utilized bacterial counts that were far in excess of those noted in humans in early sepsis, making CNS contamination more likely. Despite these conflicting results, it is generally recommended that except in the most extraordinary circumstances, central neuronal block should not be performed in patients with untreated bacteremia. Patients with evidence of systemic infection may safely undergo spinal anesthesia, if antibiotic therapy is initiated prior to dural puncture, and the patient has demonstrated 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; patients should be carefully selected and monitored for evidence of epidural infection (Wedel 2006).

The attenuated inflammatory response within the immunocompromised patient, including patients with HSV and HIV, may diminish the clinical signs and symptoms often associated with infection. Likewise, the range of microorganisms causing invasive infection in the immunocompromised host is much broader than that affecting the general population and includes atypical and opportunistic pathogens. Consultation with an infectious disease specialist is advised to facilitate initiation of early and effective therapy (Horlocker 2006).

Meticulous aseptic technique, including hand-washing with chlorhexidine, wearing of mask and sterile gloves by the proceduralist, skin asepsis with chlorhexidine and antibiotic pretreatment for the placement of permanent devices, is critical to the prevention of infectious complications related to regional anesthesia (Hebl 2006).

All patients with an established local or systemic infection should be considered at risk for developing infection of the CNS. A delay in diagnosis and treatment of even a few hours significantly worsens neurologic outcome. Bacterial meningitis is a medical emergency. Mortality is approximately 30%, even with antibiotic therapy. The clinical course of epidural abscess progresses from spinal ache and root pain, to weakness (including bowel and bladder symptoms) and eventually paralysis. 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, radiologic examination such as CT scan, or more preferably MRI, is currently recommended. A combination of antibiotics and surgical drainage remains the treatment of choice. As with spinal hematomas, neurologic recovery is dependent on the duration of the deficit and the severity of neurologic impairment before treatment.

References

Table 3. Variables That May Influence Infectious Complications

| Site of Catheter Placement (thoracic vs. lumbar vs. caudal) |
| Choice of Antiseptic and Technique of Application |
| Choice of Barrier Protection (masks, gloves, gowns) |
| Timing and Selection of Perioperative Antibiotics |
| Duration of Neuraxial or Peripheral Catheterization |
| Use of Bacterial Filters |
| Dressing Types (transparent vs. dry gauze dressing; use of antiseptic dressings) |

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