Complications of Centroneuraxial Blocks

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Neurologic complications associated with spinal or epidural anesthesia can be due to toxic effects of the injected agent, incorrect placement of a needle or catheter causing direct neural tissue damage, infectious agents, or spinal cord compromise due to ischemia or mass effect. Adverse events related to the surgical procedure, positioning, or a patient's underlying medical condition can also present as "complications" of regional anesthesia. Anticipation and prevention of complications, along with their early diagnosis and treatment, are the most important factors in dealing with regional anesthetic risks. Several large studies have confirmed the rarity of permanent neurologic injury associated with this type of anesthesia (1,2).

INTRAOPERATIVE TECHNICAL PROBLEMS

Direct nerve trauma with a large-gauge, sharp-bevel needle injection of local anesthetic solutions intraneurally or injection of an inappropriate agent are avoidable causes of nerve injury. Large studies have documented the risk of peripheral nerve injury associated with surgery under general anesthesia. Scrupulous documentation of paresthesias elicited during the block as well as the presence or absence of pain during injection of local anesthetic can be helpful in determining the etiology of postoperative neurological complaints. Tourniquet duration and pressure, a description of the patient's position on the operating table including documentation of efforts to pad vulnerable anatomic sites, and a record of local anesthetic injections by the surgeon should also be part of the permanent anesthesia record.

Faulty equipment and technique can cause complications during the performance of a block. Attempted withdrawal of an epidural or intrathecal catheter through the needle can result in shearing of the catheter, leaving a portion of it in the epidural or intrathecal space. Surgical exploration is not recommended for pieces of catheter which are left in the epidural space, though the patient should be informed of the presence of the remnant. When the catheter breaks off at or just beneath the surface of the skin during removal the remnant may serve as a theoretical conduit for bacteria from the surface of the skin into the epidural space; efforts to retrieve the catheter are recommended. Inserting the catheter beyond the recommended 2–4 cm may result in coiling and subsequent knotting of the catheter in the epidural space. This problem will usually present as difficulty in removing the catheter; radicular pain during attempted removal may indicate a knot around a nerve root. Epidural catheters have a high tensile strength, so that it is sometimes possible to apply gentle, continuous traction on the catheter until the knot becomes attenuated enough to allow it to be removed intact.

NEURAXIAL HEMATOMA

Epidural hematomas present as neurologic deficits in the postoperative period due to cord compression. Epidural needles and catheters frequently (2.8%–11.5%) cause vascular trauma associated with minimal bleeding which usually resolves without sequelae. Patients with abnormal coagulation are at increased risk, although coincidental hematoma development is also possible. In a review of the literature between 1906 and 1994, Vandermeulen et al. (3) reported 61 cases of spinal hematoma associated with epidural or spinal anesthesia. In 42 of the 61 patients (68%), the spinal hematomas occurred in association with hemostatic abnormality. Twenty-five of the patients had received IV or subcutaneous heparin, whereas an additional five patients presumably received heparin, as they were undergoing a vascular surgery. In addition, 12 patients showed evidence of coagulopathy or thrombocytopenia or were treated with antiplatelet medications, oral anticoagulants, thrombolytics, or dextran 70 immediately before or after the spinal or epidural anesthetic. Needle and catheter placement was reported to be difficult in 15 (25%), or bloody in 15 (25%) patients. Thus, in 53 of the 61 cases (87%), either a clotting abnormality or needle placement difficulty was present.

Recommendations—Specific Anticoagulants

Fully anticoagulated patients are usually not candidates for central neural blockade; however, antiplatelet drugs are often self-administered for pain relief or prescribed for a variety of preventative or therapeutic reasons. Although the effects of most nonsteroidal antiinflammatory drugs (NSAIDs) are measured in days, the antiplatelet effects of aspirin may last for a week or longer. A large retrospective study suggesting that central neural blockade is safe in patients taking these medications has been confirmed prospectively (4). The bleeding time is not a good predictor of the risk of bleeding. Newer antiplatelet drugs such as ticlopidine and clopidogrel have not been extensively

studied. The relative risk of bleeding with these drugs is unknown; however, case reports of bleeding after the use of these drugs suggest that a waiting period of 5–10 days after discontinuation of these drugs would be prudent (5). COX-2 inhibitors do not have antiplatelet effects and therefore should not increase the risk of spinal hematoma.

Coumadin has a good safety profile when used postoperatively in orthopedic patients for antithrombosis (6). A small dose is usually given the day before surgery and continued in the postoperative period. A neuraxial technique can be used, but the catheter should be discontinued before an INR of 1.5. Patients on long term coumadin therapy should have a normalized INR before needle placement. The INR is a measure of Factor VII activity, which is the factor with the shortest half-life. Therefore, low levels of Factors II, IX, and X may persist when the INR is only slightly prolonged (representing normalization of Factor VII) when discontinuing coumadin therapy. Conversely, the early prolongation of INR in a patient beginning coumadin therapy can be associated with adequate levels of other factors. Individual factor levels may be helpful.

The safety of continuous epidural techniques in the presence of *standard heparinization* during major vascular surgery has been well documented (7). The recommendations for neuraxial blockade in the presence of unfractionated heparin include the following: 1) wait 1 h after needle or catheter placement to administer heparin; 2) wait 4–6 h after stopping heparin and check partial thromboplastin time (PTT) before needle placement; 3) follow PTT or ACT to avoid excessive heparin effect; 4) manage catheter removal with the same safety precautions used for placement. The use of neuraxial techniques in the presence of full heparinization during cardiopulmonary bypass has been studied, and with the limited data available, it has not been associated with a high risk of complications. However, this technique must be considered controversial; important clinical outcome advantages have not been demonstrated yet (8,9).

Several cases of spinal hematoma in patients receiving *fractionated low-molecular-weight heparin* (*LMWH*) who underwent epidural or spinal anesthesia have been reported (10). A total of 60 cases are in the Medwatch series as of 2002 (11). Enoxaparin is the most commonly prescribed LMWH in the United States; the recommended dosage is larger than that used in Europe, and a two dose per day regimen is recommended. The first dose of LMWH is given 10–12 h after surgery; however, this time interval may shorten as antithrombotic efficacy studies are completed. If LMWH has been administered, a waiting period of 10–12 h is recommended before block placement, and single shot spinal is considered the safest alternative. Removal of catheters should occur before the first postoperative dosing. The risk of bleeding has been shown to be lower with a single shot small needle (i.e., spinal). These recommendations are evolving as the use of these drugs expands, and new drugs are released. Addition of other anticoagulants, such as NSAIDS, increases the risk. Measurement of Xa activity has not been helpful in determining an appropriate course of action.

Spinal hematomas associated with indwelling epidural catheters and intrathecal bleeding with continuous spinal anesthesia in patients receiving *thrombolytic agents* (streptokinase, urokinase, t-PA) have been reported in the literature (12). Spinal and epidural anesthesia should be avoided in these patients. If a catheter is in place and these agents are given, the safest course would be to allow the effects of the thrombolytic agent to dissipate (at least 24 h) before removing the catheter.

Newer anticoagulants such as *hirudin* and the pentasaccharide *Fondaparinux* present serious risks of bleeding. Hirudin is primarily used in patients with heparin allergy and induces irreversible antithrombin activity. Fondaparinux is in FDA review and will be recommended for perioperative antithrombosis. This highly effective anti-Xa drug is the active portion of the heparin molecule. Present recommendation, based on the pharmacologic profile of this drug, is that no neuraxial technique be performed in its presence. Clinical experience may modify this recommendation.

Herbal medications enjoy widespread use in the surgical population (13). Three commonly used herbals are associated with anticoagulation activity: ginseng, garlic, and ginkgo. Both garlic and ginkgo have antiplatelet effects; ginseng has been shown to increase PT/PTT in animals (14). These herbals should be discontinued preoperatively (garlic 7 days, ginkgo 36 h, ginseng 24 h).

Allowing the local anesthetic to wear off before instituting continuous postoperative infusions, and using small-dose local anesthetic and narcotic infusions when appropriate, permit continuing evaluation of the patient's neurologic status during the postoperative period. The patient should be monitored closely for early signs of cord compression such as complaints of back pain or an increase in intensity of motor or sensory blockade, particularly the development of new paresis. If spinal hematoma is suspected, the treatment of choice is immediate decompressive laminectomy. Recovery is unlikely if surgery is postponed for more than 8–12 h. Recommendations for neuraxial blockade in the presence of anticoagulant therapy are present on the American Society of Regional Anesthesia (ASRA) web site (www.asra.com).

INFECTIOUS COMPLICATIONS

Dural puncture has been cited as a risk factor for *meningitis* in the septic patient. The presumed mechanisms include introduction of blood into the intrathecal space during needle placement and disruption of the normal protective mechanisms provided by the

Table 1. 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 yr	Elderly
Previous history	Infection[a]	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[a]—later obtunded	Abolished	Abolished
MRI/CT/Myelogram	Signs of extradural compression	Signs of extradural compression	Normal
Cerebrospinal fluid	Increased cell count	Normal	Normal
Blood data	Rise in sed rate	Abn coags	Normal

[a] Infrequent findings.

blood–brain barrier, but are unproven. In 1919, Weed et al. (15) demonstrated that dural puncture performed in septicemic rats invariably resulted in fatal meningitis. In the same year, Wegeforth and Latham (16) described 93 patients suspected of having meningitis who had a diagnostic lumbar puncture (LP) and blood cultures. Thirty-eight patients had proven meningitis; the other 55 (6 were bacteremic at the time of LP) had normal cerebrospinal fluid (CSF). Five of these six patients subsequently developed meningitis. These findings suggested that patients with bacteremia were at risk. The LPs in this study were performed during two epidemics of meningitis occurring at a military installation. These two historical studies provided support for the claim that LP during bacteremia was a risk factor for meningitis. Subsequent clinical studies reported conflicting results. Pray (17) reported that the incidence of meningitis in children who underwent a diagnostic LP during pneumococcal sepsis was no greater among patients who had normal CSF results than those who did not undergo diagnostic LP. Eng and Seligman (18) retrospectively reviewed the records of 1089 bacteremic patients, including 200 patients who underwent LP. There was no difference between the incidence of spontaneous and "LP induced" meningitis. Teele et al. (19) reviewed the records of 277 children with bacteremias from 1971 to 1980. Meningitis occurred in 7 of 46 (15%) children undergoing LP with normal CSF, but in only 2 of 231 (1%) children who did not undergo LP. These differences were statistically significant. In addition, children receiving antibiotics at the time of LP were less likely to develop meningitis.

Carp and Bailey (20) supported the finding that treatment with antibiotics may prevent LP-induced meningitis. Twelve of 40 bacteremic rats subjected to cisternal puncture with a 26-gauge needle developed meningitis. Neither bacteremic animals that were not subjected to dural puncture nor animals undergoing dural puncture in the absence of bacteremia developed meningitis. In humans, antibiotic therapy is often deferred until after cultures are obtained. 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 LP) and used a 26-gauge needle, producing a relatively large dural defect in the rat compared to a similar puncture site in humans. Finally, no local anesthetics, which are typically bacteriostatic, were injected. Human data are scarce, although epidural anesthesia has been extensively used in febrile pregnant patients with rare adverse infectious complications. The importance of a localized infection at a site distant from the site of needle insertion in the etiology of epidural or intrathecal infectious complications is unknown, but at best such an association is highly theoretical.

Epidural abscess formation after epidural or spinal anesthesia can be superficial, requiring limited surgical drainage and IV antibiotics, or occur deep in the epidural space with associated cord compression. 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 several days after neural blockade with clinical signs of severe back pain, local tenderness, and fever associated with leukocytosis. MRI is advocated as the most sensitive modality for evaluation of the spine when infection is suspected (21). Du Pen et al. (22) reported a 5.4% incidence (1:1700 catheter days) of infection during chronic epidural catheterization, which compared favorably with infection rates associated with other chronic catheters (e.g., Hickman).

Repeated applications of local anesthetics via an indwelling intrathecal catheter or by multiple single-shot spinal injections to improve on a patchy or failed block have been associated with *cauda equina syndrome*. Suggested precautions include 1) aspiration of CSF before and after drug injection; 2) evaluation of the

extent of sacral blockade to ascertain preferential distribution to that site; 3) limit the drug dosage to a maximum precalculated "safe" dosage; 4) if an injection is repeated, avoid reinforcement of the same drug distribution (change patient position, drug baricity, etc.); and 5) if CSF cannot be aspirated after injection, do not repeat with a "full" dose unless no sign of neural blockade (including the sacral area) is present (23) (Table 1).

POSTOPERATIVE NEUROLOGIC COMPLICATIONS

Persisting sensory blockade renders affected anatomical sites vulnerable to injury and interferes with the patient's ability to feel painful responses to surgically induced problems such as ischemia or compression of tissues due to overly tight casts or surgical dressings. Neurologic complications of regional anesthetics are usually discovered after the patient has left the recovery room. Persistent motor blockade during recovery from sensory anesthesia may indicate anterior spinal artery occlusion or spasm. Lack of recovery from spinal or epidural blockade in the expected time interval may indicate spinal cord compression due to epidural hematoma. Because early intervention, preferably <12 h, is the key to success in managing these potentially devastating complications, prompt diagnosis (MRI) and early surgical management is indicated.

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