

## Complications of central neural blockade\*

Denise J. Wedel

### ABSTRACT

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. The anesthesiologist is in a unique position to participate as a knowledgeable consultant in all phases of the evaluation and treatment of perioperative complications (*Rev Mex Anest* 1998;21:176-181)

**Key Words:** Anesthesia: spinal, epidural; Complications: neural blockade, Anesthesia

### RESUMEN

**Complicaciones del Bloqueo Neural Central.** Las complicaciones neurológicas asociadas con anestesia espinal o epidural, pueden deberse a efectos tóxicos del agente inyectado, colocación incorrecta de la aguja o daño directo al tejido nervioso por el catéter, agentes infecciosos o compromiso debido a isquemia o efecto de masa. Efectos adversos, relacionados al procedimiento quirúrgico, posición o condición médica subyacente, también pueden presentarse como "complicaciones" de la anestesia regional. La anticipación y prevención de las complicaciones así como su diagnóstico temprano, son los factores más importantes en el reconocimiento de los riesgos de la anestesia regional. El anestesiólogo se encuentra en una posición única para participar como consultante en el reconocimiento y diagnóstico de todas las fases de evaluación y tratamiento de estas complicaciones perioperatorias (*Rev Mex Anest* 1998;21:176-181).

**Palabras Clave:** Anestesia: espinal, epidural; Complicaciones: bloqueo neural, anestesia.

### PREOPERATIVE EVALUATION

THE SERIOUS risks of regional techniques are often overstated. When compared to general anesthesia, there is no evidence that regional anesthesia is associated with a higher incidence of complications. Several studies have examined the safety of central neural blockade (spinal, caudal and epidural) in large groups of patients, all confirming the rarity of permanent neurologic injury associated with this type of anesthesia<sup>1-3</sup>.

Patient refusal and infection at the site of needle placement are absolute contraindications to

regional anesthesia. Almost any significant medical condition will affect the decision to perform neural blockade or the choice of technique and local anesthetic. Some conditions have well-documented risks, such as spinal anesthesia in the presence of significant aortic stenosis. Others are less clear and require careful preoperative assessment by the anesthesiologist to choose the best regional technique.

Patients with progressive neurological diseases such as multiple sclerosis or ALS may develop new neurologic lesions coincidentally following a regional anesthetic. Subsequent symptoms may be difficult to differentiate from nerve injuries resulting from the surgical procedure or anesthetic. The risk of neurologic complications associated with the anesthetic and surgical procedure, as well as the risk of stress-related disease progression should be fully docu-

Professor of Anesthesiology, Mayo Clinic, Rochester, Minnesota, USA. \*Presented at: 3rd Panamerican Symposium on Regional Anesthesia and Pain. Cancun, Mexico, November 1997.

mented in the patient's chart (Table 1). Pre-existing stable neurological lesions, for example a hemiparetic motor deficit following a cerebrovascular accident or peripheral neuropathy associated with diabetes mellitus, which are appropriately evaluated and documented are not contraindications to neural blockade.

A careful medication history should be obtained from each patient prior to neural blockade. Drugs affecting hemodynamic responses and coagulation are especially important in planning a regional approach.

## 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. Careful intraoperative positioning of the blocked lower extremities is also important. Large studies have documented the risk of peripheral nerve injury associated with surgery under general anesthesia<sup>4</sup>. Coincidental nerve injuries unrelated to neural blockade have been reported in patients undergoing regional anesthesia as well.

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. Because of the risk of deep infection, efforts to retrieve the catheter are recommended. Inserting the catheter beyond the recommended 2 to 4 cm may result in coiling and subsequent knotting of the catheter in the epidural space. This problem will usually present as difficulty in re-

**Table I.** Causes of Neurological Sequelae Unrelated to Anesthesia

---

---

Patient positioning
Surgical retractors
Surgical trauma
Tourniquet pressure
Cast or dressing application
Undiagnosed neurological diseases

---

moving the catheter, which will gradually attenuate as traction is applied. Epidural catheters are made from materials of 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 pulled intact through the structures overlying the epidural space. If radicular pain is elicited during attempted removal, the catheter may be knotted around a nerve root.

## POSTOPERATIVE NEUROLOGIC COMPLICATIONS

The patient who has undergone central neural blockade requires monitoring until the block shows signs of resolution. Residual effects of sedative and general anesthetic agents can result in difficulty evaluating the extent of neural blockade. Persisting sensory blockade renders affected anatomical sites vulnerable to injury, requiring careful positioning and padding, particularly if the local anesthetic is long-acting.

A block of the somatic nerves 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. Since early intervention is the key to success in managing these potentially devastating complications, prompt diagnosis, preferably by nuclear magnetic resonance scan, is recommended followed by early surgical management if indicated.

Rare serious neurologic problems are reported in association with continuous epidural techniques. These include fistula formation, epidural abscess, lumbar root compression secondary to epidural air, transient unilateral anterior spinal cord syndrome secondary to catheter irritation and spasm, and masking of a compartment syndrome following free fibular transfer. In some cases, removal or replacement of the catheter may resolve the problem. Radiographic procedures are often helpful in making the diagnosis and choosing an appropriate intervention.

## HEMORRHAGIC COMPLICATIONS

### *Epidural Hematoma*

Epidural hematomas may present as neurologic deficits in the postoperative period due to cord compression. Epidural needles and catheters frequently (2.8 to 11.5%) cause vascular trauma associated with minimal bleeding which usually resolves without sequelae. Patients with abnormal coagulation are theoretically at increased risk for development of epidural hematomas following even minor trauma. Reports of spontaneous hematoma formation in anticoagulated as well as in normal patients illustrate the risk of coincidental hematoma development as well.

In a review of the literature between 1906 and 1994, Vandermeulen et al.<sup>5</sup> reported 61 cases of spinal hematoma associated with epidural or spinal anesthesia. In 42 of the 61 patients (68%), the spinal hematomas occurred in patients with evidence of hemostatic abnormality. Twenty-five of the patients had received IV or subcutaneous heparin, while additional five patients were presumably administered heparin, as they were undergoing a vascular surgical procedure. In addition,<sup>12</sup> patients had 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 broody in 15 (25%) patients. Thus, in 53 of the 61 cases (87%), either a clotting abnormality or needle placement difficulty was present.

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 wide variety of preventative or therapeutic reasons. While the effects of most NSAIDs are measured in days, the antiplatelet effects of aspirin may last for a week or

longer after ingestion. A large retrospective study suggesting that central neural blockade is safe in patients taking these medications has been confirmed prospectively<sup>6,7</sup>. Appropriate preoperative evaluation of such patients should include determination of a history of abnormal bleeding or bruising bacterial site in the skin or subcutaneous tissues, or from which might indicate further evaluation. The bleeding time nosocomial source in the hospitalized patient, is not a good predictor of the risk of bleeding.

The safety of inserting epidural catheters prior to heparinization in patients undergoing major vascular surgery has been reported<sup>8</sup>. Also to the safety of low molecular weight heparin<sup>9</sup> and perioperative coumarin in orthopedic patients<sup>10</sup>. However, several recent cases of spinal hematoma in patients receiving LMWH who underwent epidural or spinal anesthesia have been reported<sup>11</sup>. Enoxaparin is the only LMWH presently approved for use in the US, and the recommended dosage is larger than that used in Europe resulting in a higher rate of postoperative hemorrhagic complications. Ten to twelve hours is the recommended safe time interval following this agent for placement of centroneuraxis blocks or removal of catheters.

While block placement with subsequent heparinization appears relatively safe, the risk of spinal hematoma in patients who receive thrombolytic therapy is less well defined. Spinal hematomas associated with indwelling epidural catheters and intrathecal bleeding with continuous spinal anesthesia in patients receiving thrombolytic agents have been reported in the literature<sup>12</sup>. Because of the high risk of hemorrhage associated with this treatment, spinal and epidural anesthesia should be avoided in patients who will receive thrombolytic therapy.

When the coagulation status is affected by any of these agents, allowing the local anesthetic to wear off prior to instituting continuous postoperative infusions, and consideration of the use of narcotic infusions when appropriate, permit ongoing evaluation of the patient's neurologic status during the postoperative period. The patient should be monitored closely in the perioperative period 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 hours.

## INFECTIOUS COMPLICATIONS

Neurological complications of central neural blockade due to infection are extremely rare. Possible risk factors include underlying sepsis, diabetes, depressed immune status, steroid treatment, localized bacterial growth or infection, and long term catheter maintenance.

Bacterial infection in the vicinity of the spinal cord can present as meningitis or cord compression secondary to abscess formation. The source can be exogenous due to contaminated equipment or drugs, or endogenous secondary to a bacterial source present in the patient. Indwelling catheters may also be contaminated from a superficial bacterial site in the skin or subcutaneous tissues, or from nosocomial source in the hospitalized patient.

Continuous catheters can theoretically serve as wicks for the spread of infection from the surface of the skin to the epidural space.

### *Meningitis*

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 blood-brain barrier but are unproven. Lumbar puncture is often performed in patients with fever or infection of unknown origin.

In 1919 Weed et al<sup>13</sup> demonstrated that lumbar or cisternal puncture performed during septicemia (produced by IV injection of a gram-negative bacillus) invariably resulted in fatal meningitis. In the same year Wegeforth and Latham<sup>14</sup> described 93 patients suspected of having meningitis who received a diagnostic lumbar puncture with simultaneous blood cultures. The diagnosis was confirmed in 38 patients. The remaining 55 patients (six of whom were bacteremic at the time of lumbar puncture) had normal CSF. Five of the six bacteremic patients subsequently developed meningitis. These findings suggested that patients with both sterile blood and CSF cultures did not develop meningitis, while patients with bacteremia were at risk. The lumbar punctures in this study were performed during two epidemics of meningitis occurring at a military installation. It is possible that some (or all) of these patients may have developed meningitis without lumbar puncture. However, these two historical studies provided support for the claim that lumbar puncture during bacteremia was a risk factor for meningitis<sup>15</sup>.

Subsequent clinical studies reported conflicting results. Pray's reported that the incidence of meningitis in children who underwent a diagnostic lumbar puncture during pneumococcal sepsis was no greater among patients who had normal CSF results than those who did not undergo diagnostic spinal tap. Eng and Seligman<sup>16</sup> retrospectively reviewed the records of 1089 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.

In a review of meningitis associated with serial lumbar punctures to treat post-hemorrhagic hydrocephalus in premature infants, Smith et al<sup>17</sup> attempted to identify risk factors. Six of 22 (27%) infants undergoing multiple (2 to 33) therapeutic aural punctures during a period of two to 63 days developed meningitis. Bacteremia, a risk factor for meningitis in this report, was associated with central venous or umbilical artery catheters. However,<sup>11</sup> septic infants who underwent aural puncture did not develop meningitis. The number of aural 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 aural puncture and onset of meningitis was not clear. Teele et al.<sup>18</sup> reviewed the records of 277 children with bacteremias during a ten year interval from 1971-80.

Meningitis occurred in 7 of 46 (15%) children undergoing lumbar puncture with normal CSF. However, only 2 of 231 (1%) children who did not undergo lumbar puncture developed meningitis.

These differences were statistically significant. In addition, children receiving 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 observed that clinical judgement might have resulted in lumbar punctures being performed more commonly in children in whom meningitis was more likely based on clinical signs and symptoms. The finding that treatment with antibiotics may prevent lumbar puncture - induced meningitis was supported by Carp and Bailey<sup>19</sup> who investigated the association between meningitis and aural puncture in bacteremic rats. Twelve of forty rats subjected to cisternal puncture with a 26-gauge needle during an *E. Coli* bacteremia subsequently developed meningitis. Neither bacteremic animals which were not subjected to aural puncture nor animals undergoing aural puncture in the absence of bacteremia, developed meningitis. Treatment of a group of bacteremic



rats with a single dose of gentamicin immediately prior to cisternal puncture appeared to eliminate the risk of meningitis.

Unfortunately, this study did not include a group of animals that were treated with antibiotics after aural puncture. In humans antibiotic therapy is often deferred until after cultures are obtained. There are several other limitations to this study. While *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 utilized a 26-gauge needle, producing a relatively large aural 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<sup>20</sup>. 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**

Abscess formation following 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. The latter is fortunately a rare complication, but it requires aggressive, early surgical management in order 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 several days after neural blockade with clinical signs of severe back pain, local tenderness, and fever associated with leukocytosis. Radiologic evidence of an epidural mass in the presence of variable neurologic deficit are diagnostic. Magnetic resonance imaging is advocated as the most sensitive modality for evaluation of the spine when infection is suspected<sup>21</sup>. Surgical intervention within 12 hours is associated with the best chance of neurologic recovery. Injection of epidural steroids and underlying disease processes associated with immunocompromise theoretically increase the risk of infection. A large study of the use of epidural catheters for management of pain (postoperative and chronic) in children<sup>22</sup> reported no infectious complica-

tions in the postoperative group and one epidural infection in a patient with osteosarcoma which had metastasized to the epidural space. Du Pen et al.<sup>23</sup> 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). Patients should be observed carefully for signs of infection when a continuous epidural catheter is left in place for prolonged periods. 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.

Chronic adhesive arachnoiditis resulting in the devastating complication of cauda equine syndrome can be caused by a variety of etiologic factors including bacteria, direct cord trauma, distilled water, blood, ischemia, contaminants, direct local anesthetic toxicity, additives to local anesthetics such as bisulfite, and the accidental injection of neural toxins. Clinical signs including bowel and bladder dysfunction, sensory loss in the perineum, and variable lower extremity paresis, can present slowly over days to weeks. The variable nature of the complaints and onset can result in a delay in diagnosis. CSF laboratory examination and radiographic studies may not be helpful in determining the etiology of this problem, but should be performed to rule out other anatomical or infectious causes. A cystometrogram will often show increased bladder volume and reduced sensation of urgency. Electromyography may also be helpful in determining the extent of involvement and confirming the clinical findings. This rare complication has been linked with the use of spinal microcatheters where greater than normal doses of local anesthetic were administered because of initial inadequate blockade<sup>24</sup>. The authors concluded that though the etiology was unclear, the neural damage might have been caused by a combination of maldistribution of relatively high doses of local anesthetic to the sacral nerve roots. These catheters have been removed from the market for re-evaluation.

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 may be a potentially unsafe practice. 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<sup>25</sup>.

## ANTERIOR SPINAL ARTERY SYNDROME

Anterior spinal artery thrombosis or spasm causes a syndrome consisting primarily of lower extremity paresis with a variable sensory deficit, usually diagnosed in the postoperative period as the neural blockade resolves. The etiology of this problem is uncertain, though direct trauma to the anterior artery and ischemia secondary to hypotension or vasoconstrictor agents may be causative factors. The clinical presentation can be difficult to differentiate from other hemorrhagic or infectious causes of cord compression. Patient factors such as advanced age and a history of peripheral vascular disease may also be important etiologic factors. While the addition of vasoconstrictors to intrathecal local anesthetics has been implicated as a theoretical cause, spinal cord perfusion studies do not show a deleterious effect of epinephrine<sup>26</sup>.

Major neurologic complications of regional anesthesia are rare, but can be devastating to the patient and anesthesiologist. Prevention and management begin in the preoperative period with a careful evaluation of the patient's medical history and appropriate preoperative patient education including a frank discussion of risks and advantages of available anesthetic techniques. Preparation for the intraoperative period requires availability of appropriate monitoring and resuscitation equipment as well as careful management of the technical and pharmacologic aspects of the nerve block. Postoperative complications can occur hours to days after the completion of the surgical procedure. Anticipation of these problems and timely postoperative evaluations are critical to early diagnosis and management. Most major neurologic complaints benefit from a multispecialty approach involving neurology, radiology, internal medicine, and surgery to assist in appropriate evaluation and treatment.

## REFERENCES

1. Usubiaga JE. Neurological complications following epidural anesthesia. *Int Anesthesiol Clin* 1975;13: 1.
2. Phillips OC, Ebner H, Nelson AT. Neurologic complications following spinal anesthesia with lidocaine: a prospective review of 10,440 cases. *Anesthesiology* 1969;30:284-9.
3. Kane RE. Neurologic deficits following epidural or spinal anesthesia. *Anesth Analg* 1981;60:151.
4. Dhuner K-G. Nerve injuries following operations: a survey of cases occurring during a six-year period. *Acta Anaesthesiol Scand* 1950;11:289-90.
5. Vandermeulen EP, Van Aken H, Vermeylen J. Anticoagulants and spinal-epidural anesthesia. *Anesth Analg* 1994;79:165-77.
6. Horlocker TT, Wedel DJ, Offord KP. Does preoperative antiplatelet therapy increase the risk of hemorrhagic complications associated with regional anesthesia? *Anesth Analg* 1990;70:631-4.
7. Horlocker TT, Wedel DJ, Schroeder DR, Rose SH, Elliott BA, McGregor DG, Wong GY. Preoperative anti platelet therapy does not increase the risk of spinal hematoma associated with regional anesthesia. *Anesth Analg* 1995;80:303-9.
8. Baron HC, LaRaja RD, Rossi G, Atkinson D. Continuous epidural anesthesia in the heparinized vascular surgical patient: a retrospective review of 912 patients. *J Vasc Surg* 1987;6:144-6.
9. Bergqvist D, Lindblad B, Mätzsch T. Low molecular weight heparin for thromboprophylaxis and epidural/spinal anaesthesia is there a risk? *Acta Anaesthesiol Scand* 1992;36:605-9.
10. Horlocker TT, Wedel DJ, Schlichting JL. Postoperative epidural analgesia and oral anticoagulant therapy. *Anesth Analg* 1994;79: 89-93.
11. Hynson JM, Katz JA, Bueff HU. Epidural hematoma associated with enoxaparin. *Anesth Analg* 1996;82:1072-5.
12. Rabito SF, Ahmed S, Feinstein L, Winnie AP. Intrathecal bleeding after the intraoperative use of heparin and urokinase during continuous spinal anesthesia. *Anesth Analg* 1996;82:409-11.
13. Weed LH, Wegeforth P, Ayer JB, Felton LD. The production of meningitis by release of cerebrospinal fluid during an experimental septicemia. *JAMA* 1919;72:190-3.
14. Wegeforth P, Latham JR. Lumbar puncture as a factor in the causation of meningitis. *Am J Med Sci* 1919;158:183-202.
15. Pray LG. Lumbar puncture as a factor in the pathogenesis of meningitis. *Am J Dis Child* 1941;295: 62-8.
16. Eng RHK, Seligman SJ. Lumbar puncture-induced meningitis. *JAMA* 1981;245:1456-9.
17. Smith KM, Deddish RB, Ogata ES. Meningitis associated with serial lumbar punctures and post-hemorrhagic hydrocephalus. *J Pediatrics* 1986;109:1057-60.
18. Teel DW, Dashefsky B, Rakusan T, Klein JO. Meningitis after lumbar puncture in children with bacteremia. *N Engl J Med* 1981;304:1079-81.
19. Carp H, Bailey S. The association between meningitis and aural puncture in bacteremic rats. *Anesthesiology* 1992;76:739-42.
20. Bader AM, Gilbertson L, Kirz L, Datta S. Regional anesthesia in women with chorioamnionitis. *Regional Anesth* 1992;17:84-6.
21. Mamourian AC, Dickman CA, Drayer BP, Sonntag VKH. Spinal epidural abscess: three cases following spinal epidural injection demonstrated with magnetic resonance imaging. *Anesthesiology* 1993;78:204-7.
22. Stafford MA, Wilder RT, Berde CB. The risk of infection from epidural analgesia in children: A review of 1620 cases. *Anesth Analg* 1995;80:234-8.
23. Du Pen SL, Peterson DG, Williams A, Bogosian AJ. Infection during chronic epidural catheterization: Diagnosis and treatment. *Anesthesiology* 1990;73:905-9.
24. Rigler ML, Drasner K, Krejcie TC, Yelich SJ, Scholnick FT, DeFontes J, Bohner D. Cauda equine syndrome after continuous spinal anesthesia. *Anesth Analg* 1991; 72: 275-81.
25. Drasner K, Rigler ML. Repeat injection after a "failed spinal": at times, a potentially unsafe practice. *Anesthesiology* 1991;75:713-4.
26. Kozody R, Palahniuk RJ, Wade JG, Cumming MO. The effect of subarachnoid epinephrine and phenylephrine on spinal cord blood flow. *Can Anaesth Soc J* 1984;31:503.