Avoiding the pitfalls of epidural anesthesia in obstetrics

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The benefits of epidural anesthesia for the relief of pain associated with childbirth has long been recognized. Although epidurals are widely used for pain relief during the labor process, side effects and complications associated with epidurals do occur. The purpose of this review is to summarize the benefits, as well as the disadvantages of epidurals, in an effort to minimize those complications.

As with all areas of medicine, the expansion of scientific knowledge in the practice of obstetrical anesthesia has been enormous. The most important of these advances is the accumulation of knowledge about the physiology of the labor process and its effect on the mother and fetus. Mothers today can deliver their babies painlessly, but it must be remembered that there are potential risks associated with obstetrical anesthesia. This review will discuss those risks as well as the basic guidelines for reducing or eliminating complications.

Lumbar epidural anesthesia has been very popular and has even been suggested as the "ideal" anesthetic for pain relief in obstetrics. Segmental epidural anesthesia can block pain pathways for each stage of labor as well as for cesarean section if required. Today's mother wants to be awake and participating in her delivery. Epidural anesthesia makes this possible.

Certainly, not all women in labor require or desire pain relief. Nevertheless, labor can be very stressful to mother and baby. Unrelieved pain of labor can result in additional increase in maternal oxygen consumption, hyperventilation, respiratory alkalosis, increased catecholamines and metabolic acidosis. Not only does the unrelieved pain of labor tax a mother's strength, it has also been shown to be deleterious to the fetus as well. Maternal hypocapnia, a result of hyperventilation, shifts the O₂ dissociation curve to the left, impairing the release of O₂ to maternal tissue as well as to the fetus. It has also been suggested, although controversial, that maternal hypocapnia combined with increased catecholamines causes uteroplacental vasoconstriction (thereby decreasing uteroplacental blood flow) resulting in neonatal depression. These responses can be attenuated with pain relief provided by epidural anesthesia.

Although an epidural utilized for labor can accomplish more than just relief of maternal pain, it is not without risk. Complications often appear unexpectedly and may be associated with errors in judgement, the use of an unsuitable drug or incorrect patient management. Most complications can be avoided by adhering to safe basic principles of epidural anesthesia. It must be remembered that obstetrical complications can be threatening not only to a mother but also to her child. Early recognition of complications is imperative so that treatment can be instituted without delay. (See Table I.)
Figure 1
Physiologic changes secondary to pain in labor

<table>
<thead>
<tr>
<th>PAIN</th>
<th>Anxiety</th>
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<tbody>
<tr>
<td>&quot;STRESS&quot;</td>
<td>Increased Release:</td>
</tr>
<tr>
<td>Increased Oxygen Consumption</td>
<td>- Endorphin</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>- Lipotropin</td>
</tr>
<tr>
<td>Respiratory Alkalosis</td>
<td>ACTH Secretion</td>
</tr>
<tr>
<td>Hypocalcaemia</td>
<td>Increased Cortisol Release</td>
</tr>
<tr>
<td>Disorientation</td>
<td>Increased ADH Release</td>
</tr>
<tr>
<td>Paresthesia</td>
<td>Tetany</td>
</tr>
<tr>
<td>Gastric Inhibition</td>
<td>Increased Autonomic Activity</td>
</tr>
<tr>
<td>Lipolysis</td>
<td>Increased Catecholamine Release</td>
</tr>
<tr>
<td>Increased Free Fatty Acids</td>
<td>Decreased Placental Perfusion</td>
</tr>
<tr>
<td>Metabolic Acidemia</td>
<td>Increased Gastrin Release</td>
</tr>
<tr>
<td>Fetal Acidosis</td>
<td>Increased Gastric Acidity</td>
</tr>
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</table>

There are concerns over the use of epinephrine in a test dose in the obstetrical patient because of the possible deleterious effects on uterine blood flow; but, generally speaking, it is accepted that 1:200,000 epinephrine has little effect on the healthy fetus.

If epinephrine is not used, the patient should complain of numbness of the tongue or circumoral tissue, lightheadedness, dizziness, visual or auditory disturbances such as ringing in the ears or a metallic taste in the mouth if an adequate milligram dose of local anesthetic is administered intravenously. As the concentration of local anesthetic increases in the blood, seizure activity may develop. Central nervous system (CNS) signs and symptoms usually precede cardiovascular (CV) toxicity.

Even though the cardiovascular system seems to be approximately three times more resistant to the effects of local anesthetics as compared to the CNS, all local anesthetics do have the potential to depress the myocardium. Anesthetists are now more aware of the CV effects of local anesthetics due to recent problems associated with bupivacaine cardiotoxicity, whereby, CV collapse may precede CNS toxicity. Bupivacaine cardiotoxicity was first suggested by Albright in 1979. Since the introduction of bupivacaine in the United States in 1973, there have been 44 reported maternal cardiac arrests associated with the use of bupivacaine. Of those 44 cardiac arrests, 30 resulted in death and an additional 7 survived with CNS damage. Since the majority of these cases occurred with the use of 0.75% bupivacaine, the Food and Drug Administration recommended, in August, 1984, that 0.75% bupivacaine should no longer be used in obstetrics.

Complications associated with epidural anesthesia

In pregnancy, due to an increase in maternal blood volume and compression of the inferior vena cava by the gravid uterus, there is engorgement of the intervertebral plexus and epidural veins making venous puncture or cannulation by the epidural needle or catheter possible. Intravascular injection of local anesthetics is a major cause of acute systemic toxicity in pregnant mothers. Signs and symptoms are directly related to the type of the local anesthetic as well as volume, concentration and rate of injection. (See Figure 2.)

Mild signs and symptoms of toxicity should be detected by a "test dose" which usually consists of 3 cc of local anesthetic and may include epinephrine. The standard dose of epinephrine in a test dose is 15-20 μg. An intravascular injection of 15-20 μg of epinephrine should be detected within 45-90 seconds and usually results in palpitations, increased heart rate, or increased blood pressure.


Figure 2
Relationship of signs and symptoms of local anesthetic toxicity to plasma concentrations of lidocaine

### Table I
Complications associated with epidural anesthesia

<table>
<thead>
<tr>
<th>Complications</th>
<th>Method to decrease</th>
<th>Treatment of complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
<td>Preload with 1 L lactated Ringer's solution. Left uterine displacement.</td>
<td>Increase fluids.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Additional uterine displacement.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vasopressor (Ephedrine vasopressor of choice 5-10 mg IV).</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Oxygen.</td>
</tr>
<tr>
<td>Delayed systemic toxicity</td>
<td>Bear in mind recommended safe doses. Monitoring of plasma blood levels. Use of low dose infusion.</td>
<td>See acute toxicity.</td>
</tr>
<tr>
<td>Total spinal</td>
<td>Use of test dose prior to every redose. Wait 5 minutes to ensure no signs of spinal anesthesia before administration of therapeutic dose. If there is questionable cerebrospinal fluid (CSF), test with blood or urine test strip for glucose. Monitor vital signs for 20 minutes.</td>
<td>Assisted ventilation and oxygenation via intubation if indicated. Treatment of hypotension with rapid infusion of fluids, epedrine, and Trendelenburg position with left uterine displacement.</td>
</tr>
<tr>
<td>Subdural injection</td>
<td>Suspect a subdural injection if no aspiration of CSF and after a 3 cc test dose there is widespread anesthesia. Wait 5 minutes for test dose.</td>
<td>Treatment of hypotension with fluids and left uterine displacement. Epedrine if necessary. Replace epidural catheter.</td>
</tr>
<tr>
<td>Horner's syndrome</td>
<td>There may not be any method to reduce incidence but possibly rate or volume of injection could influence cephalad spread.</td>
<td>Usually benign and no treatment necessary.</td>
</tr>
</tbody>
</table>

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<th>Complications associated with epidural anesthesia</th>
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<tbody>
<tr>
<td><strong>Allergic reaction</strong></td>
<td>If patient allergic to ester, use amide or vice versa. If question of allergy, have patient tested predelivery. Ask if allergic to suntan lotion.</td>
<td>Have resuscitative equipment readily available.</td>
</tr>
<tr>
<td><strong>“Wet tap” or post lumbar puncture headache (PLPH)</strong></td>
<td>Stabilize epidural needle with left hand. Use positive pressure technique so dura is &quot;tented&quot; as epidural space is entered. Enter epidural space with needle at tangential angle. Do not advance needle while patient is contracting.</td>
<td>Inform patient. Redo at another interspace. a. Use test dose. b. Administration of remainder of dose slowly in fractional doses. Make postops to detect headaches.</td>
</tr>
<tr>
<td></td>
<td><em>Conservative treatment</em> Bedrest, fluids, analgesics, abdominal binder.</td>
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<td></td>
<td>Saline injection 40-60 cc of saline into epidural space through catheter.</td>
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<tr>
<td></td>
<td><em>Epidural blood patch</em> Injection of 10-20 cc of patient’s blood into epidural space via sterile technique. Repatch occasionally if necessary. Injection of 15-20 ml of autologous blood before removing catheter.</td>
<td></td>
</tr>
<tr>
<td><strong>Backache</strong></td>
<td>Avoid trauma to tissues with epidural needle. Do not persist if difficulty is encountered and patient complains. Pay close attention to patient positioning.</td>
<td>Analgesics or heat.</td>
</tr>
<tr>
<td><strong>Shearing of catheter</strong></td>
<td>Never withdraw the catheter through the epidural needle. Never force a catheter if resistance is encountered. Remove needle and catheter as one unit if unable to thread catheter.</td>
<td>Inform patient that this is usually not a serious problem. If unable to remove catheter, leave in place and obtain an x-ray to determine location. Surgical removal if necessary.</td>
</tr>
<tr>
<td><strong>Trauma to spinal cord or nerve roots</strong></td>
<td>Placement of needle below L-2. Listen to any complaints of pain. Avoid &quot;lurch and search&quot; technique. Remove needle and/or catheter if there is severe paresthesia.</td>
<td></td>
</tr>
<tr>
<td><strong>Anterior spinal artery syndrome</strong></td>
<td>Avoid hypotension. Avoid epinephrine. Avoid injection of more than 20 cc. Fractionate dose.</td>
<td>Treatment of hypotension with fluids, left uterine displacement and vasopressor (ephedrine).</td>
</tr>
<tr>
<td><strong>Unilateral block or unblocked segment</strong></td>
<td>Do not advance epidural catheter more than 2-3 cm into epidural space.</td>
<td>Pull back catheter 1 cm and retest. Increase volume and concentration of local anesthetic. Carbonation of local anesthetic. Turn patient to affected side.</td>
</tr>
<tr>
<td><strong>Epidural hematoma</strong></td>
<td>Avoid epidural in patients with coagulopathy or patient on anticoagulant therapy. Document reversal of anticoagulant therapy with lab tests.</td>
<td>Constant surveillance for S&amp;S of spinal cord compression, neurologic deficit or severe back pain. Myelogram, CT scan. Laminectomy for decompression of spinal cord.</td>
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There are a number of possible mechanisms associated with bupivacaine cardiotoxicity. The work by Clarkson and Hondeghem demonstrated that although both lidocaine and bupivacaine produce a fast blockade within the sodium channels, bupivacaine has a slower reversal. The blockade of sodium channels slows conduction causing an increased PR interval and widened QRS, as evidenced by the ECG. In addition, Coyle and associates have shown that bupivacaine may also affect the calcium slow channels as well. This could explain the refractoriness to resuscitation and electro-mechanical dissociation associated with the cardiotoxicity.

Being highly lipid soluble and highly protein bound, the use of bupivacaine should decrease systemic toxicity. However, in the event of an unintentional intravascular injection with the development of metabolic acidosis, protein binding may be decreased, thus, increasing the intracellular drug concentration by ion trapping. Protein binding alterations in pregnancy have also been suggested as a possible means of bupivacaine toxicity.

In contrast to the acute systemic toxic reaction, a delayed systemic toxic reaction to local anesthetics may be observed. This is usually due to an overdose of drug. Signs and symptoms may not occur for 20 minutes to several hours. The anesthetist must bear in mind the total dose of local anesthetic being injected.

One of the most commonly tested-for complications of epidural anesthesia is the perforation of the arachnoid with a resultant injection of a large amount of local anesthetic into the cerebral spinal fluid (CSF). Blockade of cervical and/or phrenic nerve roots (total spinal) becomes a complication when it is not diagnosed or treated properly. Signs and symptoms of a total spinal include apnea, profound hypotension, unconsciousness and dilation of the pupils. This situation is worsened when the parturient assumes the supine position producing aortocaval occlusion and decreased venous return to the heart.

Ideally, the anesthetist would like to visualize placement of the epidural catheter midline in the posterior aspect of the epidural space. Since, however, the epidural catheter is placed blindly, improper placement can occur. One such situation is placement of the catheter between the dura and the arachnoid or the subdural space. Even though no CSF is aspirated through the catheter, a 3 cc test dose of local anesthetic can produce an unusually wide spread of anesthesia since the subdural space is compressed as a result of venous engorgement and the block extends intracranially.

Horner's syndrome is a result of a high cervical sympathetic blockade from increased spread of local anesthetic producing ptosis, miosis, and anhidrosis (reduced sweating), and has been reported even when sensory anesthesia has not extended above T7. This neurological finding is usually associated with upward diffusion of local anesthetic in the epidural space and symptoms should dissipate after the effects of the local anesthetic have worn off.

If an epidural vein is lacerated during insertion of the epidural catheter, the bleeding usually stops. An epidural hematoma can form, however, if a patient has had unusual trauma from an epidural needle, is on anticoagulant therapy or has a coagulopathy. (See Figure 3.) Hematomas in the epidural space can cause rapid compression of the spinal cord resulting in permanent nerve damage unless there is prompt surgical intervention.

**Figure 3**

*Complications of epidural block*

- Anterior Spinal Artery – Spasm or Thrombosis
- Injection into Spinal Nerve
- Epidural Abscess
- Injection into Spinal Cord


Infection resulting from epidural placement is extremely rare. The mechanisms for the transmission of bacteria include a break in aseptic epidural technique or transmission from a lesion in the skin, subcutaneous tissue or from the blood. The signs and symptoms of a rapidly developing, acute, epidural infection include severe back pain, local overlying tenderness, fever and leukocytosis. Nuchal rigidity and elevated protein in CSF may also be present.

The most common and troublesome complication associated with an epidural is the post-lumbar...
puncture headache (PLPH) which results from the loss of CSF through a hole created by needle puncture in the dura. The incidence of a PLPH in obstetric patients is related to needle size and is as high as 70-80% with a 16-gauge needle and only 2% with a 25-gauge spinal needle. The patient usually complains of a frontal or occipital headache which is aggravated by standing or sudden movements and is relieved when the patient assumes the supine position. It may be accompanied by nausea and vomiting, dizziness, visual disturbances, as well as auditory problems. The patient with a PLPH suffers severe discomfort, inconvenience and a prolonged hospital stay since duration of headache may last up to 14 days.

Many patients believe they are allergic to local anesthetics because they have experienced a systemic toxic reaction or a vasovagal reaction. Although the amino ester agents have been shown to produce allergic type reactions because they are derivatives of para-aminobenzoic acid, the amino amide groups are rarely associated with an allergic response. The incidence of a backache after a successful and atraumatic epidural block is no higher than that in a normal surgical patient receiving general anesthesia. A backache occurs in procedures where there is a flattening of the normal lumbar curve and is further aggravated by the lithotomy position. In the obstetrical population, the incidence of backache is 30-40%, but this occurs regardless of the type of anesthesia utilized and is probably due to lordosis seen in pregnancy.

Shearing an epidural catheter tip is almost always the result of attempting to withdraw the catheter before the needle is removed. In some postures, the lamina or spinal ligaments may grip the catheter making removal difficult. If too much catheter is inserted, the catheter may become looped and even knotted.

Trauma to the spinal cord or nerve roots is rare but remains one of the primary concerns of the anesthetist. Cord damage should not occur if correct anatomical landmarks are used and the block is performed below L1 (Figure 4). Trauma to the nerves usually results in severe pain to the patient.

The anterior spinal artery syndrome is characterized by predominant motor weakness in the legs and is caused by ischemia of the anterior 2/3 of the lower spinal cord. Spinal cord blood flow is largely determined by inflow pressure and outflow resistance, therefore, epidural anesthesia can be a cause or contributing factor in anterior spinal artery syndrome if hypotension is severe or uncorrected, especially if epinephrine has been used.

About the same time anesthetists were alerted to the possible cardiotoxicity associated with bupivacaine, neurotoxicity from accidental subarachnoid injection of 2-chloroprocaine was also reported. Because of the persistence of the neurological deficit associated with its use, it was suggested that injections of large volumes of a low pH solution could cause neurotoxicity. Foldes used 3.3% of chloroprocaine in the 1950s for spinal anesthesia in over 200 patients without neurological sequelae. The small volume of local anesthetic solution used by Foldes did not contain the preservative sodium bisulfite and the pH was higher. The possible mechanism for the cause of neurotoxicity was tested through various animal experiments. Although the data was conflicting, it led investigators to look at interaction of low pH, high bisulfite concentration and large volumes with neurotoxicity.

Large volumes injected into the subarachnoid space can increase intrathecal pressures which, in turn, can decrease vascular supply to the spinal cord. The vascular supply does not seem to be compromised when total volumes are less than 20 cc. Rapid injections, however, have been associated with convulsions in experimental animals.

Plain 2-chloroprocaine, as demonstrated by Foldes, is a safe local anesthetic. Experiments have shown that nerves exposed to 2-chloroprocaine at different pH concentrations recovered but when bisulfite was administered, the nerve block was persistent. At a low pH, bisulfite seems to act as a reducing agent rather than as an antioxidant, and, thus, causes acidification of nerve axoplasm.
How to decrease the pitfalls associated with epidural anesthesia

Pitfalls or complications associated with epidural anesthesia can be decreased by selecting the appropriate patient. Not every patient in labor is a candidate for an epidural. There are patients in whom an epidural should be avoided (Table II).

<table>
<thead>
<tr>
<th>Table II</th>
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<tbody>
<tr>
<td>Contraindications for elective epidural</td>
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<tr>
<td><strong>Major</strong></td>
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<tr>
<td>Patient refusal</td>
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<tr>
<td>Major coagulation defects</td>
</tr>
<tr>
<td>Uncorrected hypovolemia</td>
</tr>
<tr>
<td>Infection in area of needle insertion or severe systemic infection</td>
</tr>
<tr>
<td>Allergy</td>
</tr>
<tr>
<td><strong>Controversial</strong></td>
</tr>
<tr>
<td>Herpes simplex virus type 2</td>
</tr>
<tr>
<td>Backache</td>
</tr>
<tr>
<td><strong>Technical problems</strong></td>
</tr>
<tr>
<td>Severe scoliosis</td>
</tr>
<tr>
<td>Morbid obesity</td>
</tr>
<tr>
<td>Inability to flex back</td>
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</table>

Some parturients refuse to have a needle placed in their backs because of fear. An epidural is not an acceptable alternative if one refuses a spinal. The risk of unintentional dural puncture is 0.5-2.5% and neurological sequelae can also occur.

As noted previously, a hematoma can occur if a patient has a bleeding diathesis or is on anticoagulant therapy. There is no evidence that minidose heparin increases the risks of hemorrhagic complications; but, generally, regional anesthesia is felt to be contraindicated unless the coagulation profile is documented as normal. The reversal of heparin occurs approximately four hours after the last dose but should be confirmed with appropriate laboratory tests (Table III). Common laboratory tests to rule out a coagulopathy include: bleeding time, platelet count, partial thromboplastic time, prothrombin time, and thrombin time.

There seems to be much controversy over the use of an epidural if the patient has chorioamnionitis since sepsis is a contraindication for epidural anesthesia. Although there are no case reports of epidural abscess associated with chorioamnionitis, one needs to approach these patients with caution. If the patient's temperature and white blood count are elevated, the diagnosis of chorioamnionitis needs to be made and the patient treated with an appropriate antibiotic before an epidural can be considered.

Epidural anesthesia should be used in patients with neurological disease only after careful consultation. The neurological defect must be stable and well documented in the patient's chart. The patient must be aware of risks and benefits and these, too, should be documented on the patient's chart.

Since hypotension is a secondary side effect of epidural anesthesia and is aggravated by hypovolemia, any volume deficit must be corrected before a patient is a candidate for epidural anesthesia.

The patient with a documented allergy to ester local anesthetics should receive amide local anesthetics or vice versa. Since cross allergies do not extend between amides and esters, it is rare that a patient would be allergic to all the "caines." However, a proven drug allergy would be a contraindication to the use of local anesthetics.

The use of epidural anesthesia in the patient with herpes simplex virus type 2 (HSV-2) is controversial. Marx does not recommend the use of epidural anesthesia due to the possibility of shedding the HSV-2 virus from the skin and the risk of dissemination into the ganglia. Ravindran, on the other hand, reported the use of epidural anesthesia in 30 patients without associated complications.

Since symptoms of severe backache are more likely to occur after delivery, regardless of the type of anesthetic technique utilized, caution needs to be used when making patient selection. Backache is often related to improper positioning of the patient, but it can also occur from trauma to underlying ligaments and tissue during epidural placement.

Other conditions in which an epidural may be more difficult include the patient with severe scoliosis, morbid obesity or inability to flex the back.

A patient receiving an epidural must be able to communicate the required history, be cooperative and display the ability to understand the risks and benefits of regional anesthesia. If laboratory tests are required, results should be available. Preload...
hydration with 1,000 cc of a nondextrose-containing solution should precede the block to compensate for the segmental vasodilatation, which occurs as a secondary effect from an epidural. Adequate monitoring equipment must also be available. Epidural anesthesia should only be administered in an area supplied with proper resuscitation equipment and drugs.

Once the patient has been properly prepared, the anesthetist must adhere to the "Do's" of good technique. Do examine the back and observe for any abnormalities, signs and symptoms of infection, or spina bifida occulta observed by a tuft of hair or a fat pad. Lumbar epidural blocks are performed below L₁ where the spinal cord ends and are usually placed at the L₂-₃, L₃-₄ or L₄-₅ interspace.

Do position the patient correctly in a position which is as comfortable to her as possible. Most laboring women are already on their side and can be positioned comfortably in the lateral decubitus position. The sitting position can be utilized but remember that CSF pressures will be higher in a sitting position, and this could increase the likelihood of a "wet tap." The sitting position may be helpful in the obese patient where palpation of the spinous process will be more difficult. Regardless of the position selected, an assistant needs to be present to help with the patient positioning. Finally, the anesthetist must assume a comfortable posture while performing the block.

Since placement of an epidural catheter should be a sterile technique, do remember to wear a cap, mask and gloves after completing a thorough hand washing. A large area of the back should be prepped with Betadine® or an iodine paint, but take care not to produce a chemical contamination of the epidural space by slopping prep solution on needles, etc. Draping should be adequate. Resist temptation to be rushed. Remember, too, that the epidural catheter is a possible introductory site for contamination and that the injection port must also be kept sterile.

Identification of landmarks is necessary but do avoid using excessive time or pressure in doing this as it only causes the patient to have less confidence in the anesthetist. Skin infiltration with local anesthetic is necessary and a locator needle (20-gauge, 1½-inch needle usually included on the epidural tray) may be helpful if deeper infiltration is required or desired.

In identifying the epidural space, do keep the stylet flush with the needle so the epidural needle does not become plugged with tissue debris. This also ensures a smoother introduction. Do use a prefilled glass syringe with 3 cc of solution or air for loss of resistance technique. The continuous positive pressure technique allows the dura to be pushed away or "tented" when the epidural space is entered. The use of 3 cc of local anesthetic provides early identification of subarachnoid block injection as well as providing analgesia before the catheter is advanced. Air has also been used in the loss of resistance technique. The "hanging drop" technique seems less reliable since a negative pressure is not always observed in the pregnant woman.

After advancement of the epidural catheter, do use a test dose. Negative aspiration does not ensure that the catheter is not in a blood vessel, subdural or subarachnoid space. A test dose must precede each and every therapeutic dose whenever a catheter is refilled. It has been proposed that an effective test dose should contain a milligram dose of local anesthetic capable of producing evidence of intravascular or spinal anesthesia. Dosing of an epidural should be done between contractions, since there is extradural venous distension during contractions. If epinephrine is used in the test dose, the standard dose is 15-20 µg with 3 cc local anesthetic. Epinephrine in a test dose can readily be determined by increased pulse rate when ECG monitoring is utilized. However, recent studies questioned both the safety and efficacy of the practice in obstetrics where a positive epinephrine response can be difficult to detect in a laboring mother.

After a satisfactory test dose and therapeutic dose, do evaluate and determine if the epidural is working. The patient either feels relief of does not. If no analgesia is present after 15 minutes, the epidural catheter is probably not in the right place. It is possible that there could be a leak in the catheter allowing escape of local anesthetic outside the epidural space. If bilateral analgesia is present but the patient is still uncomfortable, check the progress of labor. It is possible that lower segments may not have been blocked with the initial dose and the dose needs to be increased.

If the level of analgesia is inadequate, a missed or unblocked segment may be the problem. The most frequent complaint associated with an unblocked segment is in the area of the skin supplied by L₁. The etiology is unknown. L₁, the thinnest of the lumbar nerve roots, should not be unblocked due to poor penetration. L₁ supplies the ileohypogastric nerve, which is not formed until after the root leaves the epidural space, and could explain the difficulty in blocking this root. Spread of local anesthesia through the lower segments may be irregular. The largest spinal nerve roots are L₅, S₁, and S₂, and these may be more difficult to penetrate. If a nerve is weakly blocked, this may be corrected by increasing the concentration of the local anesthetic, turning the patient to the affected side or by
carbonating lidocaine or 2-chloroprocaine (1 mEq sodium bicarbonate to 10 ml of local), which will increase the base form of the local anesthetic and provide a faster diffusion and faster onset.\textsuperscript{11-12}

Advancement of too much epidural catheter may be the cause of a unilateral block.\textsuperscript{10} With the engorgement of the epidural veins, flow of local anesthetic solution may be impeded to the other side. This may be corrected by withdrawing the catheter 1-2 cm. Again, a test dose must precede a therapeutic dose whenever a catheter is moved. Another possible cause of a unilateral block is the presence of a congenital adhesion, which may produce a septum in the posterior epidural space.

Problems with the epidural catheter can be avoided by not advancing the catheter too far. Advancement of an epidural catheter more than 2-3 cm may cause the catheter to kink, curl up or to move out an intervertebral foramina. Check the patency of the epidural catheter before taping. If the catheter is kinked, try withdrawing it a centimeter. Don’t remove the catheter through the needle or remove the stylet from the catheter through the needle until the needle is out of the patient’s back, since this increases the possibility of shearing the catheter.

Removal of the epidural catheter can sometimes create a problem. The patient’s back should be flexed. If resistance is met — don’t force! It may be helpful to have the patient move from side to side. If unable to remove the catheter, leave the catheter in place and obtain an x-ray to determine the location of the catheter. A knot in the catheter directly under the skin can possibly be made small enough by tightening of the catheter so the catheter can be pulled under the skin. Otherwise, surgical removal is necessary.

When a portion of the epidural catheter is sheared off in the patient, do inform the patient. This is usually not a serious problem and she can be reassured that often surgical implants of similar material are intentionally placed in the patient. Obtain a neurological consultation and have the catheter removed if paresthesia develops. Documentation of removal of an intact catheter at the end of delivery is appropriate.

The diagnosis of a “wet tap” with an epidural needle is easily made with visualization of a “gush” of fluid through the hub of the needle. Options available to the anesthetist are to inform the patient, remove the needle and try at another interspace or to pull back slowly until the fluid stops.\textsuperscript{7}

If an epidural is replaced after a “wet tap,” small incremental doses after the test dose should be used in an attempt not to raise epidural pressures, which could shunt fluid into the subarachnoid space.\textsuperscript{10} Introduction of the needle tangentially so that the dural fibers are separated rather than cut also allows better sealing of the wound. If, during the top up dose, the solution aspirated through the catheter is in question, it can be tested for positive glucose using Chemstrip BC\textsuperscript{®} which has been successful in detecting CSF even in a 1:20 solution.\textsuperscript{13}

Since epidural veins lie laterally, gross blood in the epidural needle should be a clue that the needle needs to be repositioned more midline at another interspace. A lacerated vein may increase vascular absorption of the local anesthetic. Blood in the catheter can sometimes be cleared by withdrawing the catheter a few millimeters and then injecting normal saline. If this does not clear, the epidural catheter must be replaced.\textsuperscript{8}

Intravascular injection can be decreased by “steering clear” of the epidural veins and by choosing the midline approach.\textsuperscript{2} It is helpful to open up the epidural space by injecting a test dose of solution through the needle. The patency of the epidural catheter needs to be checked before aspiration. Aspiration should be done with a gentle “twisting” technique using a 5 cc glass syringe rather than a 20 cc syringe which can create too much negative “pulling” pressure. After negative aspiration, a 3 cc test dose should be administered and if appropriate, 1:200,000 epinephrine can be used. The remainder of the therapeutic dose can be administered in fractionated 5 cc doses after a 5-minute period to ensure that no signs and symptoms of toxicity or subarachnoid block have occurred.

Pay close attention to any signs and symptoms of CNS toxicity. The purpose of a test dose is to prevent serious complications since CNS toxicity is dose related. Oxygen needs to be administered if any signs and symptoms of cerebral irritation occur since hypoxia worsens metabolic acidosis and cardiovascular depression. It must also be remembered that left uterine displacement is mandatory in the pregnant woman to ensure adequate venous return (Figure 5), and that hypotension must be aggressively treated with fluids and/or vasopressor such as ephedrine. If seizures occur, it may become necessary to stop them since ventilation may be difficult in the paralized, convulsing patient. The selection of drug utilized to stop seizures is controversial. While thiopental has been used without showing any significant alteration of protein binding, diazepam has been recently shown to worsen the cardiotoxic effects of bupivacaine.\textsuperscript{14} Succinylcholine may be worrisome because of the release of potassium, which may worsen bupivacaine cardiotoxicity.

Drug therapy for cardiac toxicity or CPR (cardiopulmonary resuscitation) associated with local
anesthetic toxicity has not been established. Lido-
caine, a membrane-stabilizing drug, raises the
threshold of defibrillation contrasted to bretylium,
has been shown to lower the defibrillator threshold.
Bretylium may be useful in the treatment of ven-
tricular arrhythmias associated with bupivacaine
cardiotoxicity.

To avoid problems with high blood levels of
local anesthetic, bear in mind the recommended
safe dose for the local anesthetic being used. Cumu-
lative overdose can lead to serous complications. A
preventative measure might be switching to a low
dose infusion.

Trauma to the spinal cord is not likely if place-
ment of the epidural is made below L1. Pay close
attention to any complaints of pain which the pa-
tient may communicate. Pain can be a warning sign
since the touching of a nerve root or spinal cord will
be accompanied by severe pain. Avoid a “lurch and
search” technique with an epidural needle. If land-
marks are not easily palpated, a locator needle can
be used.

Hypotension, a secondary effect of regional
anesthesia, can be avoided by preloading with ap-
proximately 1 L of lactated Ringer’s solution and
avoidance of aortocaval compression. Whenever hy-
potension occurs, it needs to be vigorously treated
with further fluid resuscitation, left uterine dis-
placement and vasopressor therapy. Ephedrine is
the vasopressor of choice since it has the least effect
on uterine blood flow.2

To avoid compromising spinal cord blood sup-
ply, local anesthetics should be fractionated rather
than given in a bolus, and total injection should not
exceed 20 cc during initial injection. If a suspicious
wet tap occurs, it may be best to avoid the use of
2-chloroprocaine because of the problems of associ-
ated neurotoxicity.

Conducting a thorough and informative post-
epidural interview cannot be overemphasized. An
anesthetic is not complete until such an interview is
obtained. The reasons for a postepidural interview
are twofold. First, it gives the anesthetist time to
make a thorough evaluation of any complications
that might not have presented at the time the
epidural was administered and to follow up on any
known complications. Second, it lets the patient
know there is interest and concern for her well-
being. The anesthetist needs to rule out and/or
identify a PLPH, epidural abscess or hematoma,
backache or any neurological complications which
may present themselves in the postdelivery period
so they may be appropriately treated.

Conservative treatment for a PLPH consists of
hydration with 3 L fluid over a 24-hour period,
alagesics for pain, bed rest, the use of an abdomi-
nal binder to increase intra-abdominal pressure,
and the use of intravenous caffeine.3 A saline bolus
injection of 40-60 cc in the epidural space may be
helpful as a temporary measure. The most effective
and instantaneous method of relief of PLPH is the
epidural blood patch in which 10-20 cc of blood is
withdrawn (using a sterile technique) from the pa-
tient and injected into the epidural space. Utilizing
this technique, 89-100% of patients have obtained
relief. Occasionally, a repatch may be required if
the headache persists after 24 hours. Transient side
effects of an epidural blood patch include backache,
neck ache, paresthesia in legs or abdominal cramps
as a result of too rapid an injection. DiGiovanni
reported more than 1,000 cases of epidural blood
patch since 1960 with no apparent complications.3

An epidural abscess will develop rapidly and,
as mentioned earlier, is associated with local overly-
ing tenderness, severe back pain, fever and leuko-
cytosis.4 More than 50% of patients will also have
nuchal rigidity. Spinal decompression is the treat-
ment and unless this is performed, paraplegia will
develop. Epidural hematoma also requires decom-
pression since it can produce compression of the
spinal cord.

Following an epidural, a patient may have a
prolonged blockade which can last up to 48 hours.
This may be due to injections of concentrated solutions in epidural fat which can create a reservoir of local anesthesia. This type of block is reversible and usually has no untoward effects. Permanent neurological complications are extremely rare with an incidence of less than 1:11,000-1:20,000.2

Conclusion

Labor is a process that involves many physiologic changes for both mother and baby. The challenge for the anesthetist is to reduce the pain associated with childbirth and thereby decrease the deleterious effect of pain without interfering with the labor process. Epidural anesthesia can clearly accomplish this goal. An epidural must not be thought of as a purely benign procedure, as complications can often result. Complications may occur even in the hands of skilled anesthetists, but a vigilant anesthetist who follows basic guidelines can significantly reduce or eliminate many of these complications.

REFERENCES


AUTHORS

After graduating from the Cincinnati General Hospital School of Nurse Anesthesia in 1972, Carolyn Nicholson, CRNA, BSEd, spent one year in postgraduate training in obstetrical anesthesia. She has continued with her interest in obstetrics by lecturing and producing articles for publication. In 1986, she was recipient of the first AANA Clinical Practitioner Award. She is a member of the AANA Journal Editorial Advisory Board. Mrs. Nicholson is currently the obstetrics clinical and didactic instructor at the University of Cincinnati School of Nurse Anesthesia. She received her BSEd from the University of Cincinnati in 1986.

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