Complications of Neuraxial Blockade

Several complications are associated with neuraxial blockade. Complications can be divided into several categories and include: exaggerated physiological responses, needle/catheter placement, and medication toxicity. Overall there is a low incidence of serious complications related to the administration of neuraxial blockade. However, complications may be temporary or permanent. Use of epidural anesthesia may have a higher incidence of complications when compared to spinal anesthesia. The patient population most affected is obstetrics.

Adverse or Exaggerated Physiological Responses

This category includes high neural blockade, cardiac arrest, and urinary retention. As noted earlier, there are normal physiologic manifestations that occur with neuraxial blockade. Vigilance, knowledge, preparation, and anticipation can reduce complications.

High Neural Blockade

High neural blockade can occur with either epidural or spinal anesthesia. This complication may be due to the administration of excessive doses of local anesthetic, failure to reduce doses in patients susceptible to excessive spread (i.e. elderly, pregnant, obese, or short patients), increased sensitivity, and excessive spread. When dosing a spinal or epidural, it is important to monitor the patients' vital signs and block level. Use of alcohol wipes as well as pin prick testing every few minutes will help track the blocks progression. Incremental dosing of epidurals allows the anesthesia provider to determine if the block is progressing more rapidly than anticipated. With hyperbaric spinal techniques, changing the patients' position may slow down excessive spread. Prevention is based on careful consideration in the dosing of the neuraxial block, anticipation of potential complications, and continual monitoring of the blocks progression.

Initial symptoms include the following:

- dyspnea
- numbness or weakness of the upper extremities (i.e. tingling in the fingers)
- nausea will usually precede hypotension (hypoperfusion of the brain is responsible for nausea)
- mild to moderate hypotension

At this point, change the patients' position if a hyperbaric spinal technique is used, stop the administration of epidural local anesthetics, apply supplemental oxygen, open up the intravenous fluids, treat hypotension with ephedrine or phenylephrine, and treat tachycardia/bradycardia. Carefully choose the vasopressor. For example, if the patient is hypotensive and bradycardic, then ephedrine should be used. The administration of phenylephrine in the patient who is experiencing bradycardia may increase the patient's blood pressure, yet worsen bradycardia due to reflexive vasoconstriction. Phenylephrine is the medication of choice if the patient is tachycardic and hypotensive. Refractory bradycardia and/or hypotension should be rapidly treated with epinephrine (starting with small 5-10 mcg doses). Epinephrine should be repeated and doses increased until the desired effect is obtained.

If the block has spread to cervical dermatomes the following will be noted:

- severe hypotension
- bradycardia
- respiratory insufficiency

Additional spread may lead to unconsciousness and apnea. Treatment includes the A, B, C's:

- Airway and breathing- supplemental oxygen, maintenance of a patent airway by intubation, and mechanical ventilation if necessary.
- Circulation- aggressive intravenous fluid administration, Trendelenburg position and vasopressors. If ephedrine and phenylephrine are not adequate to treat hypotension, then treat the patient with epinephrine. Early and aggressive treatment may avoid cardiac arrest! Bradycardia should be treated with atropine. Dopamine infusions may be considered.

Once the patient has been successfully treated and stabilized, surgery can often proceed. The decision to proceed is based on individual circumstances such as severity and time spent hypotensive, indications of myocardial ischemia, etc. Respiratory compromise associated with a high spinal is often transient.

Cardiac Arrest and Hypotension during Neuraxial Blockade

Cardiac arrest can occur with epidural and spinal anesthetics. However, cardiac arrest is more common with spinal anesthesia and may be as high as 1:1,500. Most cardiac arrests are preceded by bradycardia. In several cases, young healthy patients have suffered this complication. There are several key points in the prevention of this potentially devastating complication:

- Hydrate the patient with fluid prior to a block. In healthy adult patients 1 liter of fluid will help replace the fasting deficit. For healthy obstetric patients undergoing cesarean section, a pre-loading dose of 1.5 liters is helpful in reducing the incidence of hypotension and bradycardia. It is important to do this within 15 minutes of the block since 2/3rds of intravenous crystalloid solution administered will leave the intravascular space.
- Aggressively treat bradycardia with atropine, followed by ephedrine and epinephrine. Young healthy patients with high vagal tones are at risk for cardiac arrest during spinal anesthesia. A spinal anesthetic will produce a sympathectomy with unopposed vagal stimulation. Error on the conservative side and treat changes. In reviews it has been found that delays in the treatment of bradycardia may have lead to cardiac arrest.
- Consider the risk factors for bradycardia during spinal anesthesia. Risk factors include the following: baseline heart rate of < 60 bpm, ASA class I, use of beta blockers, sensory level > T6, age < 50 years, and a prolonged PR interval.

Urinary Retention

Urinary retention is the result of local anesthetic blockade of S2-S4, which decreases bladder tone and inhibits normal voiding reflexes. In addition, neuraxial opioids may contribute to urinary retention. Urinary retention is more common in elderly males with a history of prostrate hypertrophy. A urinary catheter should be used for moderate to lengthy procedures. Careful assessment in the postoperative period is important to detect urinary retention. Prolonged urinary retention may also be a sign of serious neurological injury.

Complications Associated with Needle or Catheter Insertion

Complications in this category include inadequate anesthesia/analgesia, intravascular injection, total spinal anesthesia, subdural injection, backache, postdural puncture headache, neurological injury, spinal or epidural hematoma, meningitis, arachnoiditis, epidural abscess, and sheering of the epidural catheter.

Inadequate Anesthesia or Analgesia

The rate of block failure is relatively low. However, the anesthesia provider must be prepared to supplement a marginal block or convert to a general anesthetic. The rate of block failure decreases as experience with spinals and epidurals increase. Inadequate anesthesia associated with spinal anesthesia may be associated with the following:

- Outdated or improperly stored local anesthetics. For example, tetracaine will lose potency when stored for long periods in a warm environment.
- Once free flowing CSF is noted, the clinician must be careful not to move the needle before and during injection. It is helpful to confirm aspiration of CSF before injection of local anesthetic solution, midway through the injection, and after the injection. This should decrease the risk of placing the local anesthetic in an area other than the subarachnoid space.
- Even with free flowing CSF it is possible that the opening of the spinal needle is not entirely in the subarachnoid space. This will result in a partial subdural injection and a partial spinal block.

Epidural anesthesia is more subjective. With a spinal anesthetic CSF confirms that the needle is in the correct anatomical space. The administration of epidural anesthesia relies on the "loss of resistance" or "hanging drop" technique. Either technique may lead to false positives. In addition, anatomy varies among patients. The spread of local anesthetics in the epidural space is less predictable. Anatomical factors include the following:

- The spinal ligament may be soft, resulting in never achieving a "good" loss of resistance. This can occur in young adults and women who are in labor.
- If slightly off the midline, the anesthesia provider may encounter a "soft" feeling. This is because the needle may be in the paraspinous muscle and not firmly in the spinal ligaments.
- Block failure may occur if the catheter is inserted into the subdural space or a vessel. Horner's syndrome, a high spinal, or absence of anesthesia can occur with subdural placement of local anesthetics. Local anesthetic toxicity can occur if the epidural catheter was inadvertently placed into a vessel. The epidural catheter may also be placed into the subarachnoid space. This is why it is essential to perform a test dose and slowly dose the epidural.
- Septations within the epidural space may cause a barrier to spread of local anesthetic, resulting in "patchy" anesthesia. Additional local anesthetic, with the "spared" areas dependent, will often correct this problem.
- L5, S1, and S2 are large nerve roots. Their large size may prevent penetration of local anesthetic. This problem can be corrected by elevating the head of the bed and adding local anesthetic. This position change places these nerve roots in a dependent position, allowing for additional local anesthetic penetration.

- Even a great epidural may not prevent visceral pain. This is due to the visceral afferent fibers that travel with the vagus nerve. Increasing the level of the block may alleviate this. In addition, supplementation of the block with intravenous opioids and sedative medications should be considered.

Other causes of a failed epidural anesthetic include the following:

- Not waiting long enough. Allow enough time for the medication to work. The onset of epidural anesthesia is slower than spinal anesthesia.
- The catheter is inserted too far, resulting in a unilateral block. During an unilateral block the tip of the catheter has either exited the epidural space or is off to one side. Pulling the catheter back 1-2 cm and adding local anesthetic with the non affected side down will generally take care of this problem.

Inadvertent Intravascular Injection

The risk of serious complications related to an intravascular injection, when performing a spinal anesthetic, is almost non-existent due to the small amount of local anesthetic required to induce anesthesia. The risk of serious complications lies with the administration of epidural or caudal anesthesia. This is due to the relatively large amounts of local anesthetic administered. Toxicity affects the central nervous and cardiovascular systems. Several factors affect the potential response to large doses of local anesthetics and include type of local anesthetic, rate of injection, and acid-base balance. The first factor is the type of local anesthetic. Local anesthetics vary in their potential to cause toxicity. The least to most toxic local anesthetic is as follows:

chloroprocaine< lidocaine< levobupivacaine < ropivacaine < bupivacaine

Rate of injection is a second factor. The faster the injection rate the smaller amount of local anesthetic medication required to cause signs and symptoms of toxicity. Acid base balance is a third factor. Patients that are experiencing respiratory acidosis (decreased pH and increased CO2) will have a lower threshold for convulsions. On the other hand, metabolic acidosis (increased pH and increased CO2) does not lower the threshold as significantly. Hypercarbia will increase cerebral blood flow which allows the brain to have a greater uptake of local anesthetic molecules. Ion trapping occurs due to a decrease in pH and an increase in the cationic form of the local anesthetic which keeps the molecules from diffusing across nerve membranes. Finally, hypercarbia/acidosis decreases the ability of local anesthetics to bind with proteins which increases the amount of local anesthetic available for diffusion into the CNS.

Cardiovascular effects include alteration of electrical and mechanical activity and direct effects on the cardiac muscle and vascular smooth muscle. Local anesthetics that are very lipid soluble, extensively protein bound, and potent are more likely to cause cardiotoxic reactions (bupivacaine, tetracaine, and etidocaine) than agents that are less lipid soluble, protein bound, and potent (lidocaine, mepivacaine, and prilocaine).

Electrical conduction- inhibition of NA channels will depress electrical conduction, rate of depolarization of Purkinje's fibers, action potential duration, and refractory period. Bupivacaine has the greatest depressant effect on the electrical conduction system.

Cardiac dysrhythmias- caused by primarily bupivacaine followed by etidocaine and ropivacaine. The result includes ventricular fibrillation. Lidocaine, mepivacaine, or tetracaine rarely cause ventricular fibrillation.

Myocardial contractility- there is a dose dependent negative effect on the inotropic actions of the heart. The greatest degree of depression occurs with bupivacaine, tetracaine, and etidocaine. Moderate depression occurs with lidocaine, mepivacaine, and prilocaine. The least potent local anesthetics include procaine and chloroprocaine.

Signs and symptoms associated with high blood concentrations of local anesthetics include the following:

- Hypotension
- Arrhythmias
- Cardiovascular collapse
- Seizures
- Unconsciousness

Prevention includes the use of a test dose prior to the injection of local anesthetic, careful aspiration prior to injection, incremental dosing, and vigilant monitoring for early signs and symptoms of an intravascular injection. Early symptoms include an increase in heart rate (if using an epinephrine containing solution), tinnitus, a funny or metallic taste, and subjective changes in mental status. If the patient experiences early symptoms, stop the administration of local anesthetics. Anticipate impending complications such as seizures, hypotension, and cardiac arrest. The use of lipids in the treatment of local anesthetic toxicity has shown promise. There are currently no established methods and research continues. For updates please refer to http://lipidrescue.squarespace.com. Prepare the appropriate medications and equipment. Next, re-evaluate the placement. If there is any doubt about proper placement, simply remove the epidural catheter and once symptoms have abated, replace the catheter.

Total Spinal- **covered earlier**

Subdural Injection

The subdural space is a potential space found between the dura and arachnoid mater. It contains a small amount of serous fluid and extends intracranially. Local anesthetics can travel higher in the subdural space than in the epidural space. The small dose of local anesthetic, associated with a spinal anesthetic, may result in a failed spinal. Larger doses, associated with epidural analgesia, may result in Horner's syndrome. Still larger doses, associated with epidural anesthesia, may be associated with a total spinal. Treatment is the same as with high neuraxial blockade (i.e. supportive measures such as intubation, mechanical ventilation, and cardiovascular support). Prevention is more difficult since aspiration will generally be negative. However, with slow and incremental dosing, a higher and faster progression of the anesthetic will be noted than one normally expects.

Backache

Up to 30% of patients that undergo general anesthesia will complain of a backache. A large number of patients suffer from chronic back pain. This is generally not a contraindication to neuraxial techniques. The patient should be aware that spinal or epidural anesthesia may result in some discomfort. Anytime a needle goes through anatomical structures there is an inflammatory response. This may result in spasms and is generally short lived. Use of ice and anti-inflammatory medications will help. Symptoms may continue for up to a few weeks. Though backaches are common, they should not be dismissed. Back pain is an early sign of serious complications such as epidural/spinal hematoma and abscess formation. Careful investigation of the signs and symptoms will help you determine if it is a benign complication or a sign and symptom of a more serious problem.

Postdural Puncture Headache

Postdural puncture headaches can occur after a spinal anesthetic, epidural "wet tap", epidural catheter that is threaded or migrates into the subarachnoid space, or after a successful epidural in which the tip of the needle indented or scratched the dura enough to cause a subsequent leak. The development of a headache is due to leakage of CSF through the "hole" in the dura. Subsequently, there is a decrease in intracranial pressure as the CSF leaks out. When the patient assumes an upright position, there is traction on the dura, tentorium, and blood vessels, resulting in pain. Traction placed upon the 6th cranial nerve may result in diplopia and tinnitus.

Symptoms include the following:

- Headache, associated with a sitting or standing position, which is reduced or relieved by laying down flat.
- Characteristics of the headache include bilateral, frontal, retro-orbital, and/or occipital with radiation to the neck. It may be described as throbbing or constant and associated with nausea and photophobia.
- Onset is generally 12-72 hours after the procedure. Occasionally it may be immediate.
- Untreated, it will usually resolve in a week.

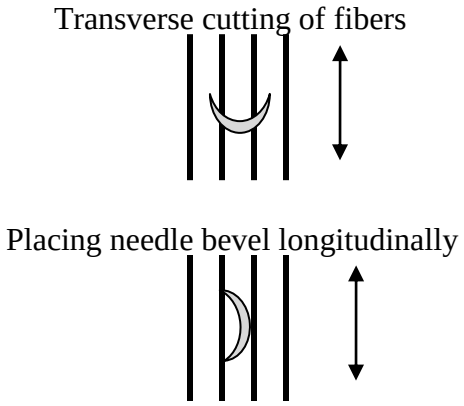
Differential diagnosis is essential to rule out meningitis. The two key differentiating signs and symptoms include the postural component of the headache and lack of a fever. Additional tests include a normal white blood cell count and a negative cerebral spinal fluid tap. A spinal tap would be necessary only in circumstances where the diagnosis is unclear. Most postdural puncture headaches can be diagnosed based on the postural component, lack of fever, and normal white blood cell count.

Factors that are within the clinicians control that can reduce the incidence of postdural puncture headaches include:

- Use of small gauge needles
- Use of pencil point needles
- Longitudinal orientation of the bevel of cutting needles

As noted above the choice of the spinal needle impacts the incidence of post dural puncture headache:

- An increased incidence is associated with needle size, needle type, and patient population.
- The larger the needle, the higher the incidence of postdural puncture headache. Use the smallest needle possible.
- Cutting point needles are associated with a higher incidence of postdural puncture headache when compared to pencil point needles of the same size. If possible, use a pencil point needle for spinal anesthetics.
- Cutting point needles have an increased incidence of postdural puncture headache if inserted in a manner that transects the fibers of the dura in a transverse fashion. If using a cutting needle, place the bevel of the needle so that it is longitudinal to the fibers. In the past it was thought that fibers were longitudinal in nature. Recently it has been found that fibers are random in nature. However, when a cutting needle is orientated in a longitudinal manner there is a decrease in postdural puncture headaches. This is because tension is placed on the fibers in a longitudinal manner. Tension will then pull the fibers "open" and allow additional CSF to "leak" out.



Tension is applied in a longitudinal manner which exerts an influence on the amount of CSF that may "leak" out of the dura.

- Pencil points, in the past, were thought to decrease postdural puncture headaches due to causing less trauma to the dural fibers. In reality they cause more trauma than cutting needles. However, this trauma is advantageous since an inflammatory reaction results in localized edema which actually reduces the size of the defect and minimizes the amount of CSF that leaks out.
- Patients with the following characteristics are at increased risk:
    - ✓ Less than 50 years of age
    - ✓ Female (this is contended by Longnecker)
    - ✓ Pregnant

Wet Tap After Epidural Needle Insertion

Some have advocated the use of prophylactic measures to treat a postdural puncture headache before signs and symptoms appear. Prophylactic measures include the administration of an epidural blood patch, epidural dextran, or epidural administration of preservative free saline. A wet tap with a 17 gauge epidural needle results in a 50% incidence of postdural puncture headache. Half of the patients will not experience a postdural puncture headache. In addition, prophylactic epidural blood patches performed less than 24 hours after dural puncture have a 71% failure rate. After 24 hours there is a failure rate of 4%. The conservative approach, to a "wet" tap, would be to wait and see if

symptoms develop. Otherwise, 50% of the patients would receive an unnecessary treatment with attendant risk of complications.

Conservative Treatment Measures

Signs and symptoms associated with a postdural puncture headache can be debilitating. Conservative measures of treatment should be instituted first. An optimal regime has not been established despite years of study. Conservative measures include the following:

- Supine position- theoretically this decreases hydrostatic pressure and reduces the amount of CSF leaking from the dural puncture. However, there is no evidence that bed rest will reduce the duration of headache. Early ambulation should be encouraged, if tolerated, to reduce complications. Bed rest should be encouraged for patients who do not tolerate an upright position. A supine position reduces the intensity of the headache.
- Hydration- theoretically encourages the production of CSF. Dehydration may contribute to the severity of symptoms that the patient experiences. Patients should be hydrated intravenously and/or encouraged to take fluids by mouth. Studies however do not show a decrease in the duration of postdural puncture headache.
- Caffeine- theoretically helps decrease signs and symptoms of a headache by vasoconstriction of the cerebral vessels. Once again, this may help decrease the symptoms but does not reduce the number of patients that will require an epidural blood patch. Intravenous caffeine may be administered in a dose of 500 mg. In practice, oral caffeine intake is encouraged. An oral dose of 300 mg has been shown to decrease the intensity of postdural puncture headache.

Common beverages and caffeine content.

Beverage	Caffeine Content
Regular Coke™	34 mg/12 oz
Coffee (brewed)	80-135 mg/ 7 oz
Coffee (instant)	65-100 mg/ 7 oz
Tea (black)	70 mg/ 6 oz
Tea (green)	35 mg/ 6 oz

- Analgesic medication administration may decrease the severity of the signs and symptoms. Medications include acetaminophen or non steroidal anti-inflammatory medications such as ibuprofen.
- Stool softeners and a soft diet will help decrease Valsalva straining and leakage of CSF.

Epidural Blood Patch

An epidural blood patch is the definitive treatment for post dural puncture headache. It will successfully resolve 70-95% of all postdural puncture headaches on the first treatment. A subsequent epidural blood patch will resolve symptoms in 70-95% of those who did not respond to the first treatment. An epidural blood patch is generally offered after 12-24 hours of conservative treatment. An epidural blood patch is not without risk. It is essential that to check the patients' coagulation status and past history. Ensure that anticoagulants have not been administered in the post partum/postoperative period. In addition, ensure that the patient is not bacteremic or septic.

An epidural blood patch involves the injection of 10-20 ml of the patients own blood at the level of dural puncture. Alternatively, the anesthesia provider may choose one space below the site of dural puncture. The injected blood will stop leakage of CSF by mass effect or by coagulating and "plugging" the hole. Inform the patient of the risks and benefits. The risks are essentially the same as with any neuraxial technique. In addition, there is the increased risk of meningitis or infection since blood, which can be contaminated, is being taken out of the body and then placed in an area that has breached the blood brain barrier.

Accessing the epidural space is the same as performing an epidural anesthetic. The process is as follows:

1. Assemble the supplies: mask, sterile gloves, epidural tray, additional betadine and alcohol. The assistant will require sterile gloves, a mask, a sterile syringe to draw blood, sterile needle for venipuncture, and a tourniquet.
2. Prior to locating the epidural space, identify a suitable vein for the blood draw as well as an alternative vein. Prep the area with betadine and consider draping the area with sterile towels.
3. Perform the usual steps for locating the epidural space. Once the epidural space is located, have the assistant aseptically withdraw 15-20 ml of blood. Ensure that contamination does not occur. Breaking sterile technique may put the patient at risk for significant complications!
4. Place 15-20 ml of blood into the epidural space. The patient may experience pressure but should not experience acute or sharp pain.
5. The patient should remain supine for a period of 1-2 hours.
6. The patient should avoid heavy lifting or straining for 48 hours, as this may dislodge the epidural blood patch, resulting in the return of the postdural puncture headache.

Neurological Injury

This can be a transient or permanent complication. Fortunately neurological injury is extremely rare with an incidence of 0.08-0.16%. Avoid trauma to the nerve roots or spinal cord. Appropriate anatomical landmarks should be identified prior to the initiation of neuraxial blockade. Measures include the following:

- Document pre-procedural neurological deficits. Does the patient suffer from neuropathy, chronic/acute low back pain, sensory, and/or motor deficits?
- Document conditions that may contribute to postoperative neurological deficits such as peripheral vascular disease, diabetes, intervertebral disk disease, and spinal/neurological disorders.
- Subarachnoid techniques should be performed below L2 in adults and L3 in children.
- Multiple attempts increase the incidence of trauma. Avoid this by carefully positioning the patient, noting anatomical landmarks, take your time, and be deliberate when performing neuraxial techniques. Don't be afraid to ask for help from another anesthesia provider if difficulties are encountered.
- Remove and redirect the needle when encountering a paresthesia. The incidence of encountering a paresthesia has been estimated to be 6%.

- If pain is encountered during needle insertion, catheter insertion, or injecting medication, immediately stop. Direct injury to a spinal nerve root may cause permanent injury.
- Document the presence of paresthesia or pain during neuraxial blockade. Document the subjective description and if it was transient or prolonged. Alternatively, if the block was placed without a paresthesia or pain, document it.

If a patient experiences a neurological deficit after neuraxial blockade, rule out an epidural hematoma or abscess. Neurological deficits may occur related to surgical positioning, improper positioning in the postoperative period, or as a result of direct trauma related to the surgical procedure. Obstetric patients are at risk for neurological deficits related to cesarean section or normal vaginal delivery. Carefully document the signs and symptoms that the patient is experiencing. For example, is the patient experiencing a peripheral neuropathy in the distribution of the neuraxial block? Often these symptoms are transient. Is the patient experiencing severe symptoms such as sharp back and leg pain? This may indicate the formation of a spinal/epidural hematoma or transient neurological symptoms. Is the patient experiencing a progression of numbness, motor weakness, and/or sphincter dysfunction? This may indicate the development of a spinal/epidural hematoma. Trauma and subsequent damage to the conus medullaris will result in isolated sacral dysfunction.

Symptoms include the following:

- Paralysis of the biceps femoral muscle
- Sensory loss of the posterior thigh, perineal area, or great toes
- Bowel and/or bladder dysfunction

Postpartum neurological deficits unrelated to neuraxial techniques include lateral femoral cutaneous neuropathy, foot drop, and even paraplegia.

After an initial evaluation it is reasonable to request a neurological consult.

Spinal or Epidural Hematoma

Trauma during neuraxial techniques to epidural veins is usually benign and self limiting. The incidence of neuraxial hematoma formation for epidurals is approximately 1:150,000 and 1:220,000 for spinal anesthesia. The following factors increase the risk for developing a spinal or epidural hematoma:

- Anticoagulant use or disease processes that affect coagulation
- Multiple attempts during neuraxial blockade
- Formation of a hematoma after epidural catheter removal

Bleeding in the subarachnoid/epidural space will result in the compression of neural tissue. Due to anatomical factors, it is not possible to apply pressure to compress blood vessels and stop the bleeding. Compression of neuraxial structures result in ischemia and subsequent injury. The onset of symptoms is generally rapid and include the following:

- Sharp back and leg pain
- Progression of numbness and motor weakness
- Sphincter dysfunction

Rapid diagnosis is essential. An MRI or CT scan can identify an epidural or spinal hematoma. Surgical decompression occuring within 4-6 hours of onset to avoid permanent injury.

Meningitis and Arachnoiditis

Meningitis is a rare complication. The incidence of meningitis has been estimated to be 1:50,000 for spinal anesthetics and 1:90,000 for epidural anesthetics. However, it is essential that sterile technique is maintained during neuraxial blockade. Unfortunately, there are providers that perform lumbar puncture without a mask. A recent review article found that most cases of post neuraxial blockade bacterial meningitis are due to contamination of the puncture site by aerosolized mouth particles. Strains of viridans streptococcus, commonly found in the mouth, are the dominant causative agents resulting in post dural puncture meningitis. The anesthesia provider should wear a surgical mask, and change the mask between patients. To a lesser extent, meningitis can be caused by skin bacteria and from endogenous sites of infection. Care should be taken when securing indwelling catheters since they can become colonized with organisms. The presentation of meningitis may mimic a post dural puncture headache. Signs and symptoms of meningitis may include headache, neck pain, fever, and alteration in the level of consciousness.

Arachnoiditis is a rare complication. It was more common in the past when there were no disposable trays. In the past, needles were cleaned with solutions that caused chemical meningitis and neurological dysfunction. Chemical arachnoiditis can occur from inadvertent injection of steroids into the subarachnoid space. If performing epidural steroid injections and there is a question of possibly being in the subarachnoid space, stop the procedure and have the patient come back a week later. Lumbar arachnoiditis may occur following surgical procedures or trauma in the lumbar region.

Epidural Abscess

The formation of an epidural abscess is rare, with an incidence ranging from 1:6,500-1:500,000 cases. Patients can develop an epidural abscess independent of neuraxial blockade. Risk factors include back trauma, intravenous drug use, and neurological surgical procedures. Epidural abscesses associated with neuraxial anesthesia are generally due to an indwelling epidural catheter. Signs and symptoms develop between 5 days and several weeks.

Four stages have been suggested:

1. Back or vertebral pain intensified by percussion over bony vertebrae. The patient with back pain and fever should alert the anesthesia provider to the possibility of an epidural abscess.
2. Nerve root and radicular pain
3. Motor, sensory, and/or sphincter dysfunction
4. Progression to paralysis or paraplegia

The prognosis is dependent upon when an epidural abscess is diagnosed. If an epidural abscess is suspected, the epidural catheter should be immediately removed and the tip cultured. The epidural insertion site should be inspected for signs and symptoms of infection. If drainage can be expressed from the site, it should be sent for analysis. In addition, blood cultures should be sent for analysis. If an epidural abscess is suspected, a neurological consult should be sought. The most common causative agents are staphylococcus aureus and staphylococcus epidermidis. Antibiotic coverage

should be immediately instituted. A MRI or CT can confirm or rule out the diagnosis. Additional treatment for a confirmed epidural abscess includes a decompression laminectomy.

Prevention of this complication is important.

- Sterile technique is essential when inserting epidural catheters. Hat, mask, sterile gloves, hand washing, a sterile field, and proper preparation of the skin should take place.
- If there is any doubt that sterility has been violated, stop, start over.
- If the epidural catheter has been disconnected, the anesthesia provider must use their clinical judgment to decide to aseptically reattach the infusion or remove the catheter.
- Reduce catheter manipulation
- Maintain a closed system at all times.
- Use the bacterial filter that comes with the epidural kit.
- Remove the epidural catheter after 96 hours. If the epidural is required for a period longer than 96 hours, remove the current catheter and replace with another every 96 hours.

Sheering off the Tip of an Epidural Catheter

Never attempt to withdraw an epidural catheter back through the needle. Pull both the needle and catheter out at the same time. When removing an epidural catheter, use steady pressure. Do not stretch or jerk the catheter. If difficulty in removing the catheter is encountered, have the patient curl up in a ball in a lateral decubitus position. This position should maximize the intervertebral space and allow for catheter removal. Steady, gentle pressure should allow the catheter to be removed in its entirety.

If the epidural catheter sheers or breaks off in the epidural space, it should be left in place. Observe the patient for complications. If the catheter breaks outside of the epidural space, in superficial tissue, it should be surgically removed. A remnant of an epidural catheter in superficial tissue can result in infection.

Complications Associated with Medication Toxicity

Allergic Reactions

The incidence of allergic reactions to local anesthetics is very low. Based on the chemical structure of local anesthetics esters are more likely to cause an allergic reaction when compared to amides. Esters are metabolized into PABA which is a known allergen. For amides that are in multi-dose vials a preservative known as methylparaben may be used. Structurally similar to PABA, methylparaben may be responsible for allergic reactions. In practice multi-dose vials of local anesthetic should be avoided and practitioners should strive to use preservative free preparations. Reactions to local anesthetics may be often related to vagal reactions, toxicity related to intravascular injection or a relative overdose, and side effects related to the use of epinephrine which may cause tachycardia, flushing, and tachypnea among other common signs and symptoms related to its administration. Of anesthetics that are used the most common type of agent that results in an allergic reaction are muscle relaxants > thiopental > propofol > etomidate = ketamine =

benzodiazepines > local anesthetics. No allergic reactions have been documented to occur with volatile anesthetics.

Anaphylactic reactions involve a number of mediators. It is an exaggerated response of the immune system to an antigen whether it be a protein, metabolite, substance, etc. This response is very complex and a short summary will be presented. Histamine, leukotrienes, BK-A, and platelet activating factor act to increase vascular permeability and contraction of smooth muscle. H-1 acts to contract bronchial smooth muscles while H-2 causes vasodilation, mucous production, and enhanced contractility of the heart. BK-A actives to divide bradykinin and kininogen which increases vascular permeability, vasodilates vessels, and causes contraction of smooth muscles. Hageman factor may cause coagulation. A number of other mediators will cause additional tissue damage through inflammatory cells.

The end result of this exaggerated response is:

- Airway- angioedema of upper airway, bronchospasm, and edema of the lower airway. Signs and symptoms include bronchospasm, cough, dyspnea, pulmonary edema, laryngeal edema, and hypoxia.

- Vascular- increased permeability allows edema to occur resulting in hypovolemia and shock. Primary symptom will be hypotension and shock.

- Heart- hypoperfusion and hypoxemia results in arrhythmias and myocardial ischemia. Coronary vasoconstriction may occur. Tachycardia and arrhythmias are common.

- Other vital organs- resulting shock and lactic acidosis leads to additional ischemic trauma.

- The effect of mediators will manifest dermatologically as urticaria, facial edema, and pruritus.

Treatment includes the following:

- Stop the administration of the suspected medication
- Administer 100% O2 and consider intubation if the patient is not already intubated.
- Epinephrine administered in doses of 0.01-0.5 mg IV or IM
- Administer fluids rapidly to combat the hypovolemia and shock (1-2 L of crystalloid)
- Diphenhydramine in a dose of 50-75 mg IV
- Rantidine or cimetidine IV
- Hydrocortisone up to 200 mg IV or alternatively methylprednisolone in a dose of 1-2 mg/kg IV.

Systemic Toxicity- has been covered earlier under inadvertent intravascular injection.

Transient Neurological Symptoms

Transient neurological symptoms, or TNS, in the past has been called transient radicular irritation. It is a relatively new diagnosis first described in 1993. Signs and symptoms of TNS include low back pain with radiation to the legs. Signs and symptoms occur after the spinal anesthetic has regressed and normal sensation has returned. It can occur between 1 and 24 hours and generally subsides after several days. There are no sensory or motor symptoms. All local anesthetics have been associated with TNS. There is an occasional report of TNS after epidural anesthesia. The incidence is highest with spinal lidocaine. This has prompted many in the anesthesia community to abandon the use of lidocaine as a spinal anesthetic. On the other hand, it has left the anesthesia community with few short acting local anesthetic agents. Procaine is often too short lived. Prilocaine has a relative high incidence of nausea and vomiting. Mepivacaine has a similar profile to lidocaine, both for duration and the incidence of TNS.

The exact mechanism is not known. It is theorized that lidocaine is more neurotoxic to the unsheathed nerve than other local anesthetics. Two factors contributing to the incidence of TNS include positioning and early ambulation. TNS is more common in patients that have received a spinal anesthetic and are then placed in a lithotomy position. This position may cause lumbosacral nerve root stretching, decreasing perfusion, and making nerves more susceptible to toxic effects of local anesthetics. Knee arthroscopies are one of the most common surgical procedures associated with TNS.

Prevention largely lies with avoiding lidocaine, which has the highest incidence. Bupivacaine is an excellent alternative. However if only lidocaine is available the anesthesia provider must weigh the risk/benefit since TNS is a transient complication. It is important to closely monitor positioning when placing a patient in lithotomy position. Patients undergoing ambulatory surgical procedures or procedures in the lithotomy position should be informed that this complication can occur. Treatment is symptomatic and short lived.

Cauda Equina Syndrome

Cauda equina syndrome has been associated with continuous spinal catheter techniques and 5% lidocaine. Cauda equina syndrome is permanent and associated with sphincter dysfunction, sensory/motor deficits, and paresis. Sensory deficits generally occur in a peripheral nerve pattern due to the maldistribution of hyperbaric lidocaine and subsequent neurotoxicity. Concerning neurotoxicity of local anesthetics lidocaine = tetracaine > bupivacaine > ropivacaine. The patient will experience pain similar to nerve root compression. Cauda equina syndrome has been reported after single shot spinal anesthetics as well as spinal catheter techniques. Cauda equina syndrome can rarely occur after epidural anesthesia.

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