

## CHAPTER 3: NEURAXIAL ANESTHESIA

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### SPINAL ANESTHESIA

It is one of the easiest and most reliable techniques of regional anesthesia. The very small doses of local anesthetics used to produce spinal anesthesia are devoid of direct systemic effects.

In 1885 James Corning, an American neurologist, was the first person to use cocaine intrathecally to treat some neurological conditions. Augustus Bier, a German surgeon, was the first person to use intrathecal cocaine to produce surgical anesthesia. In a classic paper published in 1899, he described the failed attempt by his assistant Hildebrandt, to perform a spinal anesthesia on him, and his successful spinal on Hildebrandt. Both of them became the first patients suffering from post dural puncture headaches.

#### Anatomy

The spinal canal has a protective sheath composed of three layers. From the outside to the inside they are: duramater, arachnoid and pia mater. The potential space between the dura and arachnoid is called *subdural space*. The cerebrospinal fluid (CSF) flows between the arachnoid and pia mater in the space called *subarachnoid space*.

The spinal cord begins cranially at the foramen magnum, as a continuation of the medulla oblongata. It terminates caudally at the *conus medullaris*, which in the adult corresponds to the level of the lower border of L1, and in the young child to the upper border of L3. From this end, a prolongation of the pia mater called the *filum terminale* attaches the spinal cord to the coccyx. The *dural sac* itself ends at the level of the second sacral vertebra.

The spinal cord is composed of a core of gray matter surrounded by white matter. The gray matter on cross section has an H shape, with ventral (motor) and dorsal (sensory) horns. The white matter is described as having anterior, lateral and posterior white columns.

There are 31 pairs of spinal nerves; each one being formed by two roots, a ventral or motor root and a dorsal or sensory root. The dorsal root has the dorsal root ganglion. Because the spinal cord of an adult is shorter than the vertebral column, the spinal nerves descend a variable distance in the spinal canal before exiting through the intervertebral foramen. The most distal lumbar and sacral nerves travel the longest distance inside the spinal canal, forming what is known as the *cauda equina*. As the spinal nerve pierces the dural sac, it draws with it a dural sleeve. The spinal nerves exit through the intervertebral foramen, formed between two vertebrae. There are 8 cervical nerves. The first cervical nerve exits through the occipital bone and C1, the 8<sup>th</sup> cervical nerve exits between C7 and T1. Distal to T1 each spinal nerve exits below the corresponding vertebra.

The vertebral column has a series of curvatures in the anteroposterior plane. The cervical and lumbar curvatures have an anterior convexity (lordosis) and the thoracic and sacral have posterior convexity (kyphosis). These curvatures play a role in the spread of the local anesthetic solution, as we will review later.

The blood supply to the spinal cord comes from one anterior spinal artery and two posterior spinal arteries. These arteries anastomose to form longitudinal vessels, reinforced by segmental arteries that enter the vertebral canal through the intervertebral foramina. The anterior two thirds of the spinal cord are supplied by the anterior spinal artery reinforced in the neck by branches of the vertebral artery.

In the thoracic region the anterior spinal artery receives only a few radicular arteries from the aorta. In the lumbar region a large branch called *radicularis magna or artery of Adamkiewicz*, reinforces the anterior spinal artery. It arises 78% of the times on the left side, and typically enters the spinal canal through a single intervertebral foramen between T8 and L3. This important branch is at risk of damage during retroperitoneal dissections (e.g., surgery on the distal aorta), which could lead to ischemia of the spinal cord. A case of transient paraplegia after neurolytic celiac plexus block on a pancreatic cancer patient was reported in 1995 by Wong and Brown. The proposed mechanism was reversible arterial spasm post injection of ethanol solution.

## **Planes between the surface of the skin and subarachnoid space**

The needle used to perform a diagnostic spinal tap or a spinal anesthesia needs to cross the skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, duramater and arachnoid, before reaching the subarachnoid space and CSF. The space between the ligamentum flavum and duramater is the epidural space.

## **Cerebrospinal fluid CSF**

It is primarily formed in the choroid plexus of the cerebral ventricles. The CSF flows from the lateral ventricles to the third and fourth ventricles, and from there to the cisterna magna. It flows then around the brain and spinal cord, within the subarachnoid space. The CSF is absorbed into the venous system of the brain by the villi in the arachnoid membrane. CSF is formed and reabsorbed at a rate of 0.3-0.4 mL/min.

The CSF volume in the brain is between 100-150 mL. The volume of CSF below T12, where most of the spinal anesthetics are performed is, according to Hogan and collaborators, widely variable among individuals, ranging from 28-80 mL. CSF volume is decreased with increased abdominal pressure, like the one accompanying pregnancy and obesity. Therefore, increased abdominal pressure could potentially lead to higher spread of a neuraxial blockade.

### Composition of cerebrospinal fluid and serum in humans

	CSF	Serum
Sodium (mEq/L)	141	140
Potassium (mEq/L)	2.9	4.6
Calcium (mEq/L)	2.5	5.0
Magnesium (mEq/L)	2.4	1.7
Chloride (mEq/L)	124	101
Bicarbonate (mEq/L)	21	23
Glucose mg/100mL)	61	92
Protein (mg/100mL)	28	7000
pH	7.31	7.41
Osmolality (mOsm/kg H <sub>2</sub> O)	289	289

#### Site of action

The nerve root is the main site of action for both spinal and epidural anesthesia. In spinal anesthesia the concentration of local anesthetic in CSF is thought to have minimal effect on the spinal cord itself.

#### Indications

Abdominal and lower extremity procedures are the most common. It has been used for lumbar spine surgery. Saddle blocks are frequently used for rectal surgery.

#### Baricity

It is the result of dividing density of the local anesthetic solution by that of the CSF. The density of CSF has a mean value of 1.0003. If the baricity is 1.0 it is by definition isobaric; if greater than 1 it is hyperbaric and if less than 1 it is hypobaric.

##### 1. Hypobaric solutions

Tetracaine is the local anesthetic most frequently used for hypobaric spinal anesthesia. Solutions of 0.1% to 0.33% tetracaine in water are reliably hypobaric in all patients. The most common uses of hypobaric solutions are for rectal procedures in jackknife position and for hip surgery injecting in lateral position with the surgical side up.

##### 2. Isobaric solutions

Tetracaine and plain bupivacaine diluted with CSF make good isobaric solutions. These solutions stay very close to the point of injection.

##### 3. Hyperbaric solutions

The easiest, safest and most widely used way of providing spinal anesthesia. The solution is rendered hyperbaric by adding glucose. Gravity and patient's position determines the

spread. In supine position L3 and T6 are the highest points of the spine and subsequently they become the limits for spread.

## **Determinants of local anesthetics spread in the spinal fluid**

### **1. Major factors**

- Baricity acting together with gravity
- Position of patient (except isobaric solutions)
- Dosage, rather than volume or concentration

Baricity is the main factor that determines local anesthetic spread in the subarachnoid space. It obviously works in conjunction with gravity and patient position.

When plain local anesthetics are used, total dose is more important than injected volume or concentration. Van Zundert et al reported in 1996, that a 70 mg dose of plain subarachnoid lidocaine produced the same quality of spinal block over a wide range of concentrations and volumes. Sheskey et al in 1983 demonstrated similar sensory levels with 10 mg of plain bupivacaine, at different concentrations and volumes. However, doses of 15-20 mg of plain bupivacaine produced higher sensory levels of spinal (T2-T4 level) than 10 mg (T5-T8 level). When hyperbaric bupivacaine or tetracaine solutions are used, similar levels of spinal blocks are obtained at different doses, when the concentration is maintained constant. In the case of hyperbaric bupivacaine, it seems that this applies as long as the dose is higher than 7.5 mg. Above this dose the level is determined by baricity acting along with the curvatures of the spine, patient position, and gravity. In general, the higher the spread the shorter the duration of the sensory blockade, because the concentration of the drug decreases from the point of injection.

### **2. Minor factors**

- Level of injection
- Increased abdominal pressure (obesity and pregnancy)
- Patient height (only at extremes)
- Coughing
- Direction of needle bevel can affect spread of isobaric preparations. The bevel should be directed toward the desired region.

### **3. No effect**

- Addition of vasoconstrictors
- Barbotage (aspirating and injecting technique to produce CSF turbulence)
- Age
- Gender

## **Type of techniques**

### **Sitting, midline approach**

Sitting position is commonly used for neuraxial blocks. It may be the preferred position in patients whose midline may be difficult to determine, like obese patients. The position of the iliac crest is frequently used to determine the L4-L5 interspace. However, accumulation of adipose tissue around the patient mid section, could lead to a higher-than-desired level for needle placement.

The Closed Claims Project shows cases of spinal cord injury by the spinal needle, in which the level of needle placement was grossly underestimated. I suggest instead using the upper end of the intergluteal sulcus to determine the position of the sacral hiatus. In adults the L5-S1 interspace is around 10 cm (4 inches) cephalad to this point (height of the sacrum). This measurement in adults should always be distal to the termination of the spinal cord at L1.

Using a hyperbaric solution in the sitting position, and leaving the patient in that position for at least 5 minutes, produces a saddle block. However, up to 20 minutes is necessary to wait, in the desired position, to achieve any appreciable "saddle" or "lateralized" distribution blockade.

### **Lateral position**

It is the position of choice in many institutions. The patient lies on his/her side. It is more comfortable for the patient and decreases the risk for accidental fall and vasovagal problems. The technique otherwise is similar to sitting position

### **Paramedian approach**

In some elderly patients, with calcified ligaments, it is difficult to advance the thin spinal needle through the midline. The lateral approach is a good alternative in those cases. The spinous process is identified and the point of entrance is marked about 2 cm paramedian. The needle is directed slightly medial and cephalad.

### **Taylor Approach**

Usually the L5-S1 interspace is the larger. A spinal technique through it is known as Taylor approach. The entrance point is 1 cm medial and 1 cm caudal to the posterior superior iliac spine directing the needle cephalad and toward the midline.

### **Anesthesia duration**

The local anesthetic used and the rate at which it is removed from the subarachnoid space determines duration. Elimination is entirely dependent on vascular absorption and does not involve metabolism of local anesthetics within the subarachnoid space. Absorption occurs in the subarachnoid space itself and in the epidural space (local anesthetics cross the dura both ways).

### **Side effects and Complications**

#### **1. Hypotension**

It is the most frequent seen side effect. It is mainly the result of venous pooling with decreased cardiac output secondary to sympathetic blockade. There is also a small component of arteriolar dilation. However systemic blood pressure does not decrease proportionally because of compensatory vasoconstriction, especially in the upper extremities with intact sympathetic innervation. Even with total sympathetic blockade after spinal anesthesia the decrease in systemic vascular resistance is less than 15%. This is because arterioles retain intrinsic tone and do not dilate maximally.

The magnitude of the blood pressure decrease depends on the extent of sympathetic blockade, intravascular volume, and cardiovascular status. Preloading the patient with 250-500 mL, while frequently used, is unsupported by the evidence.

A mild vasopressor like ephedrine in 5-10 mg increments and fluid are all that is usually necessary to treat hypotension. Ephedrine is usually the drug of choice because it produces vasoconstriction and increases cardiac output.

Phenylephrine is a good second choice especially if tachycardia is present. It causes vasoconstriction, and it could decrease the cardiac output.

Trendelenburg position can alleviate the venous pooling, but may produce an even higher spinal level. Elevating the legs with the patient sitting at 30-45 degrees is a good compromise.

## 2. Bradycardia

When the sympathetic block reaches T2 level, the cardioaccelerator fibers are blocked and the vagus action is unopposed. The extent to which heart rate decreases in response to total sympathetic block during spinal usually is moderate (10-15%). However **severe bradycardia** and **asystole** have been reported in normal patients during otherwise uneventful spinal anesthesia. It can occur even in the absence of hypotension and can occur after 30-45 minutes of spinal.

The Bezold-Jarisch reflex has been implicated. This reflex would be triggered by decreased venous return to the heart producing a paradoxical hypervagal response. **Early recognition and treatment is essential.** Ephedrine, atropine and in some cases epinephrine are indicated along with fluid replacement.

## 3. Total spinal

Spinal anesthetic that involves the cervical region. It is manifested by respiratory arrest, bradycardia, hypotension and unconsciousness. The respiratory arrest most likely is a manifestation of ischemia of the medullary respiratory center secondary to intense hypotension and drop in cardiac output (complete sympathetic blockade) severe enough to compromise cerebral circulation.

Block of phrenic nerve is not a likely cause. Management involves ABC with control of the airway, ventilator support, use of vasopressors, and atropine and fluid replacement as needed.

## Miscellaneous physiologic effects

### 1. Respiratory

Arterial gases are usually unaffected in patients breathing room air. Tidal volume, maximum inspiratory volumes and negative intrapleural pressure during inspiration are unaffected, despite intercostal muscle paralysis with high thoracic levels. This is because diaphragmatic activity remains intact.

Expiratory volumes and total vital capacity are significantly diminished in high thoracic spinal, as are maximum intrapleural pressures during forced expiration, and coughing. This is mainly due to paralysis of abdominal muscles.

## 2. **Hepatic**

Hepatic blood flow decreases to the extent of hypotension to a degree similar than after general anesthesia. Spinal anesthesia has not proven to be an advantage or disadvantage in patients with liver disease. For intraabdominal surgery the decrease in hepatic perfusion is mainly due to surgical manipulation.

## 3. **Renal**

Renal blood flow as cerebral blood flow is autoregulated through a wide range of arterial pressure. In the absence of renal vasoconstriction renal blood flow does not decrease until mean arterial pressure decreases below 50 mm Hg. Thus, in the absence of severe hypotension, renal blood flow and urinary output remain unaffected during spinal anesthesia. Loss of autonomic bladder control results in urinary retention. This is more frequent in males.

## 4. **Endocrine and metabolic**

Spinal anesthesia, but not general anesthesia, blocks the hormonal and metabolic stress response associated with surgery. This response involves increases in ACTH, cortisol, epinephrine, norepinephrine and vasopressin as well as activation of the renin-angiotensin-aldosterone system. However this effect seems to wear off along with the spinal anesthesia, producing metabolic and hormonal responses similar than after general anesthesia for the same operation.

## 5. **Gastrointestinal**

The small intestine contracts during spinal and sphincters relax due to unopposed vagus nerve activity. The combination of contracted gut and complete relaxation of abdominal muscle provide good surgical conditions.

## **Other effects and complications**

### 1. **Nausea**

Frequent side effect due to imbalance of sympathetic and parasympathetic visceral tone. Hypotension, bradycardia or hypoxia must be rule out. Antiemetics like ondansetron or droperidol are usually effective.

### 2. **Post dural puncture headache (PDPH)**

PDPH is due to CSF leak through the dural puncture site. The subsequent loss of CSF pressure produces stretching of the meningeal coverings of intracranial nerves whenever the upright position is assumed.

The pain characteristics, involving exacerbation in the upright position and relief in the recumbent position, remain the main diagnostic tool. It is more frequent in females, in younger patients and during pregnancy. The size and type of needle are proven factors. Pencil point needles significantly reduce the risk.

Spinal needles are either cut-bevel (Quincke-type) or pencil-point (Whitacre-type). It has been usually accepted that the collagen fibers of the duramater are oriented longitudinally and that the bevel of a cutting needle should be oriented vertically to reduce trauma to the dural fibers. This concept has been challenged by Reina and collaborators. They found that dural fibers are arranged in laminae with fibers in all different directions and not necessarily longitudinal. They also showed that pencil point needles produce a more traumatic lesion in the dura than cutting-point needles. They hypothesized that a more traumatic lesion may stimulate more inflammation than a cleaner cut does. The inflammatory response and edema would then limit the leakage of CSF. This observation agrees with the surprisingly low incidence of PDPH after continuous spinal anesthesia with an 18-gauge epidural needle and a 20-gauge epidural catheter. The catheter might act as foreign object producing an inflammatory reaction.

This low incidence can also, at least in part, reflect the fact that continuous spinal are more frequently performed in older patients. Older age is accompanied by a decreased risk of PDPH.

#### In the issue of PDPH:

- Pencil point needles less than or equal to 22 gauge and cut-bevel needles less than or equal to 27 gauge produce an incidence of PDPH of approximately 1%.
- Continuous spinal with 20 gauge catheters is not likely to produce PDPH in an older patient population.
- Obstetric patients undergoing spinal anesthesia with small pencil point needles show a 3-4% rate of post dural puncture headache. Conservative treatment involves bed rest, IV or oral fluids, acetaminophen and NSAIDs. Hydration and caffeine stimulates production of CSF.
- Epidural blood patch with 15-20 mL of autologous blood, injected at the same original puncture level or one space below, is a very effective treatment. The effect can be immediate or be delayed by a few hours. A single blood patch is about 90% effective.

### 3. **Transient neurological symptoms (TNS)**

Usually appears 12- 24 hrs after surgery and consist of mild to moderate pain or sensory abnormalities in the lower back, buttocks or lower extremities. It resolves between 6 hrs and 4 days. No patient with TNS has ever been reported to develop neurological deficits or motor weakness. If present, other more serious diagnosis must be ruled out: epidural hematoma, nerve root damage, cauda equine syndrome. The first report appeared in the literature in 1993 when Schneider et al published a series of 4 patients with buttocks pain after spinal.

Prospective, randomized studies have shown:

- A higher (but variable) incidence after lidocaine spinal. Decreasing the concentration of lidocaine to 0.5% does not appear to change this incidence.
- Its incidence seems related to other factors like: lithotomy (30-36%), knee arthroscopy (18-22%), whereas the risk after supine position appears to be relatively low (5 to 8%).



The cause for TNS is not well understood and could represent a mild and reversible form of neuropathy. Many possible causes have been postulated: local anesthetic toxicity, needle trauma, neural ischemia secondary to sciatic nerve stretching, patient positioning, small gauge, pencil-point needles promoting local anesthetic pooling, muscle spasm, early mobilization, etc. Because of the low incidence of TNS after bupivacaine spinal, we could be reasonably sure that TNS is not the result of the subarachnoid block per se, the needle or the position for it.

Even though neurotoxicity is frequently mentioned as possible cause for TNS, a case can be made against it. Cauda equina syndrome (CES) is known to result from local anesthetic toxicity; however the factors that increase CES (e.g., higher doses/concentration of local anesthetics and the addition of vasoconstrictors), do not have an effect on TNS.

We know that TNS is mostly associated with lidocaine spinal, lithotomy position, knee arthroscopy and ambulatory surgical status (obesity could be a contributing factor) and that it is very rarely associated with bupivacaine spinal.

We also know that decreasing the concentration of lidocaine from 5% to 0.5% does not decrease the incidence of TNS and that hyperosmolarity, hyperbaricity and addition of glucose ARE NOT contributing factors.

First line of treatment is reassurance, NSAIDs, comfortable positioning and heating pad. A second line of treatment can include narcotics and muscle relaxants like cyclobenzaprine. Trigger point injections have been used with reported success.

Eliminating lidocaine from subarachnoid block probably is not warranted at this point. However do not use it for ambulatory surgery in lithotomy position or knee arthroscopy (high risk). On the other hand, the incidence of TNS after inguinal hernia with lidocaine spinal is only 8%, after C-section is 0-8% and after tubal ligation is 3%, similar to non-pregnant patients undergoing surgery in the supine position. Bupivacaine, even in small doses, increases discharge time. Perhaps the combination of small doses of bupivacaine plus narcotics is the best possible approach.

#### **4. Cauda equina syndrome**

It is a rare but devastating complication resulting in perineal anesthesia and possible loss of bowel and bladder control. Most of the reported cases have been associated with the use of continuous spinal with microcatheters (30-gauge and smaller) along with use of 5% hyperbaric lidocaine. Low flow rates promoting pooling of concentrated drug around the sacral roots have been postulated as the reason for this condition. In 1992 the FDA issued a safety alert that resulted in the withdrawal of these catheters from the US market. The incidence of CES increases with increased concentration of local anesthetics as well as the addition of vasoconstrictors. There have been reports of cauda equina syndrome after epidural anesthesia.

#### **5. Back pain**

As many as 40% of patients may complain of this annoying side effect. It is postulated to be the result of stretching of the ligaments following the relaxation of back muscles. This is

similar to what is seen in up to 25-30% of patients receiving general anesthesia in the supine position. It can also be the result of localized inflammatory response with muscle spasm.

Rest, local heat and NSAIDs are the treatment of choice.

## **6. Hearing loss**

Transient minor hearing loss has been described after spinal anesthesia. The risk seems larger with larger-gauge needles and it might be the result of temporary decrease in CSF pressure with traction of intracranial nerves.

The problem is mild but well documented with audiometry. It resolves on its own.

## **7. Infection**

Abscess or meningitis is rare. The development of meningitis after lumbar puncture in

bacteremic patients is a concern. Animal models suggest that perioperative use of antibiotics eliminates this risk. Lumbar puncture in patients infected with HIV is controversial.

Neuraxial techniques including blood patch have been performed on these patients without apparent problems. The risk has to be evaluated on an individual basis.

## **Spinal anesthesia in the outpatient setting**

A few years ago spinal anesthesia was favored for same day surgery patients. However, widely available, poorly-soluble general anesthetic agents and LMA have decreased its use. Home readiness involves short duration and in many institutions, ability to void. Duration is a function of the agent and dose used. The spread of the agent dictates the duration at a given dermatome. It is likely that the more segments blocked by a given dose (more spread) the shorter the duration at any given segment. Hyperbaric solutions and isobaric solutions injected rapidly with the bevel turned caudad concentrate around the sacral roots and can delay sensory motor recovery and the ability to void. On a milligram basis, isobaric preparations injected rapidly with the bevel facing cephalad are more likely to improve home readiness and voiding. Procaine and very small doses of bupivacaine plus narcotics have been used in the outpatient setting with variable success.

## **Intrathecal adjuncts**

### **1. Epinephrine**

It prolongs duration, but also prolongs the recovery time and voiding time. Thus it should

not be used in the ambulatory setting.

## 2. **Fentanyl**

The lipophilic synthetic opioids appear to improve the quality of the block without prolonging recovery. Ben-David et al in 1997 showed that 5 mg of hyperbaric bupivacaine was inadequate in 27% of cases of spinal for knee arthroscopy. Adding 10 mcg of fentanyl reduced the failure rate to zero.

Fentanyl produces pruritus in about 50% of the patients. Serotonin inhibitors (like ondansetron) are being used to treat this side effect too. Respiratory effects are unlikely with doses below 25 mcg.

## 3. **Morphine**

The use of hydrophilic intrathecal narcotics is accompanied by a longer lasting analgesia, but also by a higher rate of complications. Among them are: delay respiratory depression (4-6 hrs after the injected dose), increased nausea and vomiting, pruritus and delayed voiding.

## 4. **Clonidine and neostigmine**

They potentiate spinal local anesthetics and produce postoperative analgesia, but they produce unacceptably high rates of hypotension and sedation (clonidine) and protracted vomiting (neostigmine).

## EPIDURAL ANESTHESIA

**It is technically more difficult to perform than spinal and because larger doses of local anesthetics are used it has the potential for systemic toxicity. On the other hand, it offers a greater degree of flexibility in the extent and duration of anesthesia.**

### Anatomy

**The spinal epidural space extends from the foramen magnum to the end of the dural sac at the level of S2. It is bounded anteriorly by the vertebral bodies and posteriorly by the laminae and ligamentum flavum. The epidural space outlines the spinal canal immediately superficial to the dura. In the cervical region the epidural space is smaller and it is wider in the lumbar area. A volume of local anesthetic about 10 times larger is required to produce lumbar epidural anesthesia than for equivalent subarachnoid blockade. Smaller volumes are sufficient for the thoracic space. The epidural space is filled with connective tissue, fat and veins, which can become enlarged during pregnancy. The spinal nerves travel through this space surrounded by a sheath of dura.**

### **Characteristic of an epidural blockade**

Epidural anesthesia produces a band of segmental anesthesia spreading cephalad and caudad from the site of injection. Epidural anesthesia has a slower onset and usually it is not as dense as spinal. This characteristic can be used as an advantage to obtain a more pronounced

differential blockade. Dilute concentrations can spare the motor fibers while still able to produce sensory analgesia. This is commonly employed in labor epidural analgesia.

### **Factors affecting the spread of local anesthetics in the epidural space**

In general 1-2 mL of local anesthetic is needed per every segment to be blocked. Thus, to achieve a T4 level from an L4-5 injection 12-24 mL of local anesthetic is needed.

1. Dose and volume: The total dose and the volume affect the height of the block. The effect of volume is linear but it plateaus at about 20 mL, after which there is a greater loss through intervertebral foramina, especially in younger patients.
2. Age: As opposed to spinal, age is a major factor in the spread of epidural anesthesia with smaller volumes producing a higher spread in older patients. This may be due to the narrowing of the intervertebral foramina with age.
3. The site of injection influences the spread. Volumes as small as 6-8 mL of solution injected at the thoracic level can produce anesthesia due to smaller volume of the epidural space.
4. Body weight: heavier patients have smaller volume requirements.
5. Height: Plays a small role with taller patients requiring higher volumes.
6. Gravity: is not a very important factor, as sitting position does not appear to enhance sacral spread.

### **Techniques**

#### **Lumbar epidural**

It is the most common site for epidural anesthesia. The midline or paramedian approach can be used. A block below the termination of the spinal cord at L1 should be safer. An accidental dural puncture (“wet tap”) could result in spinal cord damage at higher levels.

#### **Thoracic epidural**

It is technically more challenging and has a greater risk for spinal cord injury. It is rarely used as the primary anesthetic. Many people prefer the paramedian approach in the thoracic level, because of the extreme obliquity of the thoracic spinous processes.

#### **Epidural needles**

The Tuohy needle is the most commonly used. A typical needle is 17-18 gauge, 3.5 inches long. It has a blunt bevel with a gentle curve of 15-30° at the tip. The blunt tip helps push the dura away, “tenting” it, after the ligamentum flavum has been pierced.

#### **Epidural catheters**

They provide the means for continuous infusion. Usually they are 19-20 gauge in size. The needle bevel is directed in the desired direction (not a guarantee for catheter final location) and the catheter is advanced 2-6 cm. A short insertion increases the chance for accidental dislodgement. The farther in, the greater the chance of unilateral epidural and other complications (bloody tap, catheter knotting). Four to five cm is a good compromise.

#### **Test dose**

It is important because of the large doses of LA injected into the epidural space. The classic test dose is 3 mL of 1.5% lidocaine (45 mg) with 1:200,000 of epinephrine (15 mcg). The

45 mg of lidocaine, if intrathecal, should produce spinal anesthesia. The 15 mcg of epinephrine, if intravascular, should produce at least a 20% increase in heart rate within 30 sec or 30 beats between 20-40 sec (Barash's, 5<sup>th</sup> edition, 2006). In patients who are beta blocked the heart rate increase may not happen and an increase in systolic pressure of 20 mmHg or more may be more reliable (Barash's 5<sup>th</sup> edition, 2006). The use of epinephrine as a test dose in obstetrics is controversial. Some suggest instead the use of only 30 mg of lidocaine or 5 mg of bupivacaine.

### **Activating an epidural, Incremental dosing**

After a negative test dose most of practitioners will inject incremental doses of 5 mL at a time. This technique helps decrease the risk of systemic toxicity in case of catheter migration (intravascular or intrathecal).

### **Termination of action**

It is related to type of drug and degree of spread. It is commonly described as the time it takes to a two-segment regression of sensory blockade. The approximate time for two-segment regression (sensory) for chlorprocaine is 50-70 minutes, for lidocaine is 90-150 minutes and for bupivacaine is 200-260 minutes.

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