

Physiologic Effects of Neuraxial Blockade

Normal physiologic manifestations of neuraxial blockade such as hypotension are not necessarily complications but normal physiological effects of neuraxial blockade. A thorough understanding of these effects will allow the anesthesia provider to anticipate alterations and treat the patient in a timely manner, preventing complications. A brief review of the mechanism of action, somatic and autonomic blockade as well as cardiovascular, respiratory, gastrointestinal, renal, and metabolic/endocrine effects will be discussed.

Neuraxial Blockade Mechanism of Action, Spread, Uptake & Elimination

Placement of local anesthetics in the epidural space is at a physiologic distance from the intended targets of spinal nerves and nerve roots. Several barriers to the spread of local anesthetic result in larger volumes of local anesthetic being administered compared to the volume used for spinal anesthesia. Barriers include dura mater, systemic absorption, and epidural fatty tissue. Dura mater acts as a modest barrier; the majority of local anesthetic solution is absorbed systemically through the extensive venous system in the epidural space. Epidural fat acts as a reservoir. The remaining local anesthetic reaches the spinal nerve and nerve roots. The degree of horizontal and vertical spread is dependent upon the volume of local anesthetic administered. Since the epidural space is a potential space, higher volumes of local anesthetics must be administered to allow spread of the local anesthetic to nerve roots in the required areas for the proposed surgical procedure. In addition, a larger volume and dose of local anesthetic is required due to large mixed nerves found in the epidural space, penetration of the arachnoid and dura mater, absorption of local anesthetic into tissue and fat, and absorption of local anesthetic by epidural veins (peak blood concentration occurs in 10-30 minutes after a bolus). Local anesthetics absorbed in veins are diluted by blood. The pulmonary system acts as a temporary buffer protecting against toxicity. Subsequent distribution occurs to the vessel rich organs, muscle, and fat. Amides will bind to α -1 globulins which have a high affinity but become saturated rapidly. Amides are metabolized by the liver and excreted by the kidneys. Esters are metabolized by pseudocholinesterase so rapidly that there are rarely significant plasma levels.

Local anesthetics placed in the subarachnoid space will effectively block sensory, autonomic, and motor impulses by interacting with the anterior/posterior spinal nerve roots and the dorsal root ganglion as they pass through the CSF. Blockade of the anterior nerve root fibers result in blockade of efferent motor and autonomic transmission. Neural blockade of posterior nerve root fibers results in the blockade of somatic and visceral impulses. Spinal anesthesia is achieved with a small dose and volume of local anesthetic resulting in dense sensory and motor block. Uptake and elimination of local anesthetics is affected by the concentration of local anesthetic, surface area of neuronal tissue exposed, lipid content of neuronal tissue, and blood flow to the tissue. Concentration of local anesthetic is highest at the point of injection, and as the local anesthetic travels away from the site of injection it is diluted by CSF and absorbed into tissue.

Somatic Blockade

Neuraxial anesthesia effectively stops the transmission of painful sensation and abolishes the tone of skeletal muscle, enhancing operating conditions for the surgeon. Sensory blockade involves somatic and visceral painful stimulation. Motor blockade involves skeletal muscles. Neuraxial anesthesia results in a phenomenon known as differential blockade. This effect is due to the activity of local anesthetics and anatomical factors. Local anesthetic factors include the concentration and duration of contact with the spinal nerve root. As the local anesthetic spreads out from the site of injection the concentration becomes less, which may in turn effect which nerve fibers are susceptible to blockade. Anatomical factors are related to various fiber types found within each nerve root. Small myelinated fibers are easier to block than large unmyelinated fibers. In general, the differential blockade found after neuraxial blockade is as follows: sympathetic blockade is 2-6 dermatome segments higher than sensory and sensory blockade is generally 2 dermatome levels higher than motor.

Autonomic Blockade

Neuraxial blockade effectively blocks efferent autonomic transmission of the spinal nerve roots, producing a sympathetic block and a partial parasympathetic block. Sympathetic fibers are small, myelinated, and easily blocked. During neuraxial blockade, the anesthesia provider will observe a sympathetic block prior to sensory, followed by motor. The sympathetic nervous system (SNS) is described as thoracolumbar since sympathetic fibers exit the spinal cord from T1 to L2. The parasympathetic nervous system (PNS) has been described as craniosacral since parasympathetic fibers exit in the cranial and sacral regions of the CNS. It should be noted that neuraxial blockade does not affect the vagus nerve (10th cranial nerve). The end result of neuraxial blockade is a decreased sympathetic tone with an unopposed parasympathetic tone. This imbalance will result in many of the expected alterations of normal homeostasis noted with the administration of epidural and spinal anesthesia.

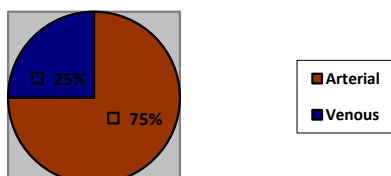
Cardiovascular Effects

Neuraxial blockade can impact the cardiovascular system by causing the following changes:

- Decrease in blood pressure (33% incidence of hypotension in non-obstetric populations)
- Decrease in heart rate (13% incidence of bradycardia in non-obstetric populations)
- Decrease in cardiac contractility

Vigilance is required by the anesthesia provider. Risk factors for bradycardia during spinal anesthesia include patients with a baseline heart rate of less than 60, ASA I (healthy patients), prolonged P-R interval, beta blocker administration, and a > than T5 level. Risk factors for hypotension include a > T5 level, > 40 years of age, a baseline SBP < 120 mmHg, and a lumbar puncture at or above L2-L3. Risk factors for epidural anesthesia are most likely similar to spinal anesthesia, however the risk factors for epidural anesthesia and bradycardia or hypotension have not been clearly delineated at this time.

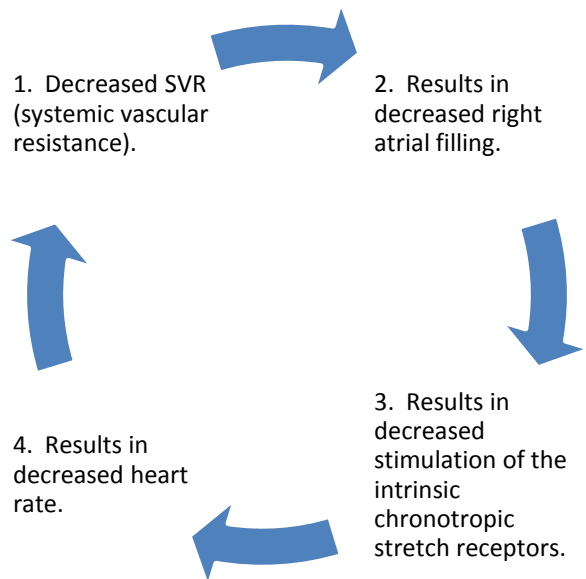
Sympathectomy is the term used to describe blockade of sympathetic outflow. Nerve fibers that affect vasomotor tone of the arterial and venous vessels arise from T5-L1, which is generally within the area that the anesthesia



provider wants to block with neuraxial blockade. The sympathetic dermatome ranges from 2-6 levels higher than the sensory dermatome level. Sympathectomy is directly related to the height of the block and results in venous and arterial vasodilatation. The venous system contains about 75% of the total blood volume while the arterial system contains about 25%. Dilation of the venous system is predominantly responsible for decreases in blood pressure since the arterial system is able to maintain much of its vascular tone.

Total peripheral vascular resistance in the normal patient (normal cardiac output and normovolemic) will decrease 15-18%. In the elderly the systemic vascular resistance may decrease as much as 25% with a 10% decrease in cardiac output.

Heart rate may decrease during a high block due to blockade of the cardioaccelerator fibers (T1-T4). Heart rate may also decline as a result of a decrease in SVR, decreased right atrial filling, and decreases in the intrinsic chronotropic stretch receptor response.



There are no set criteria on how low the blood pressure should be allowed to decline after neuraxial blockade. It largely depends on age and co-existing diseases (i.e. cardiovascular disease, renal dysfunction, etc.). It is not unreasonable to allow a modest decline (<20%) and treat a decline of > 20%. It has been found that spinal blockade has some protective effects during a decline in blood pressure. Total body oxygen consumption decreases in response to the extent of spinal blockade, providing a margin of safety. Severe hypotension may be the result of vasodilatation, bradycardia, and decreased contractility. Aggravating factors such as a head up position or the weight of a gravid uterus on venous return in the parturient may cause further declines in blood pressure. Occasionally sudden cardiac arrest may be seen with spinal anesthesia due to unopposed vagal stimulation.

Anticipation of these effects is essential. Using an appropriate “dose” of local anesthetics is essential. Volume loading the patient with 10-20 ml/kg of crystalloid fluid or appropriate amount of colloid immediately prior and during the administration of a spinal anesthetic may be helpful. The patient’s cardiac function and medical history should be taken into account prior to this measure. Left uterine displacement is essential for the parturient. Trendelenburg position may help increase blood pressure by autotransfusion. Care must be taken not to extend the neuraxial blockade even higher. Generally a spinal is “set” 10-15 minutes after administration. Bradycardia should be rapidly treated with atropine (0.5 – 1 mg IVP). Hypotension should be treated with phenylephrine, a direct acting alpha adrenergic agonist, which increases venous tone and causes arterial constriction. If

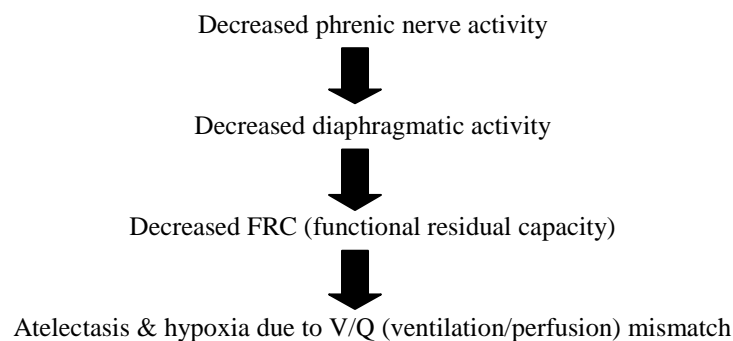
hypotension is associated with bradycardia, then phenylephrine may not be the best choice. Phenylephrine may cause reflex bradycardia! Ephedrine has a direct beta adrenergic effect, increasing heart rate and contractility as well as some indirect vasoconstriction (α). This may be a better choice in this situation. Profound hypotension and bradycardia, which persists despite treatment, should be treated with epinephrine in a dose of 5-10 mcg IVP. Epinephrine should be repeated and/or the dose increased until the desired response is achieved.

Respiratory Effects

Neuraxial blockade plays a very minor role in altering pulmonary function. Even with high thoracic levels of blockade, tidal volume is unchanged. There is a slight decrease in vital capacity. This is the result of relaxation of the abdominal muscles during exhalation. The phrenic nerve is innervated by C3-C5 and is responsible for the diaphragm. The phrenic nerve is extremely hard to block, even with a high spinal. In fact, apnea associated with a high spinal is thought to be related to brainstem hypoperfusion and not blockade of the phrenic nerve. This is based on the fact that spontaneous respiration resumes after hemodynamic resuscitation has occurred.

The risk and benefits of neuraxial anesthesia should be carefully weighed for the patient with severe lung disease. Patients with chronic lung disease depend on intercostal and abdominal muscles to aid their inspiration and exhalation. Neuraxial blockade may reduce the function of these muscles, having a detrimental impact on the patient's ability to breathe, as well as affect the ability to clear secretions and cough. For procedures above the umbilicus, a pure regional anesthetic may not be beneficial for the patient with chronic lung disease. However, postoperative analgesia with thoracic epidurals has been found to be helpful to the patient with severe lung disease undergoing a thoracic or abdominal procedure. Thoracic and abdominal surgical procedures are associated with decreased phrenic nerve activity resulting in decreased diaphragmatic function and FRC (functional reserve capacity). This can lead to atelectasis and hypoxia due to ventilation/perfusion mismatching. Thoracic epidural analgesia has been found to decrease the incidence of pneumonia, respiratory failure, improve oxygenation, and decrease the amount of time that the patient may require for postoperative ventilation.

Consequences of thoracic and abdominal surgical procedures



Gastrointestinal Effects

Since sympathetic outflow originates at T5-L1, neuraxial blockade results in a sympathectomy with a predominance of parasympathetic nervous system effects. The end result is a small, contracted gut with peristalsis. Hepatic blood flow decreases in relation to decreases in mean arterial pressure but does not differ significantly from other anesthetic techniques. Postoperative epidural analgesia enhances the return of gastrointestinal function.

Renal Effects

Neuraxial blockade has little effect on the blood flow to the renal system. Autoregulation maintains adequate blood flow to the kidneys as long as perfusion pressure is maintained. Neuraxial blockade effectively blocks sympathetic and parasympathetic control of the bladder at the lumbar and sacral levels. Urinary retention can occur due to the loss of autonomic bladder control. Detrusor function of the bladder is blocked by local anesthetics. Normal function does not return until sensory function returns to S3. Risk factors for prolonged blockade of the detrusor muscle include the use of long acting local anesthetics, age > 50 years, volume of fluids administered, and surgical procedure. Prolonged blockade of the detrusor muscle may lead to bladder over distention and urinary retention. This should be taken into consideration if no urinary catheter will be placed. If possible, short acting medications should be used. The anesthesia provider should monitor the amount of intravenous fluids administered to prevent over distention of the bladder. The patient with a history of an enlarged prostate is at risk for urinary retention. Patients should be monitored for urinary retention.

Metabolic and Endocrine Effects

Surgery produces a host of neuroendocrine responses related to inflammatory response and activation of somatic and visceral afferent nerve fibers. This response results in the release of adrenocorticotropic hormone, cortisol, epinephrine, norepinephrine, vasopressin, and activation of the renin-angiotension-aldosterone system. The release of these substances has the following clinical manifestations: hypertension, tachycardia, hyperglycemia, protein catabolism, depressed immune response, and alteration in renal function. As noted earlier, neuraxial blockade can effectively block this response. For intra-abdominal surgery, it may only partially suppress its effects. For lower extremity surgery, it can totally suppress these effects. To be effective, neuraxial blockade should be extended into the postoperative period. The effect of neuraxial blockade is beneficial by reducing catecholamine release, decreasing stress related arrhythmias, and decreasing the incidence of ischemia.

Substances Released in Response to Surgical Trauma

- adrenocorticotropic hormone
- cortisol
- epinephrine
- norepinephrine
- vasopressin
- activation of the renin-angiotension-aldosterone system

Clinical Manifestations of Neuroendocrine Response

- hypertension
- tachycardia
- hyperglycemia
- protein catabolism
- depressed immune response
- alteration of renal function

Epidural Specific Effects

The overall systemic effects of spinal anesthesia are the same for epidural anesthesia. The main difference is related to the amount of local anesthetic required to produce anesthesia. Since larger doses are used, the patient's blood levels of local anesthetic concentration may get high enough to produce adverse systemic effects. It is important to closely monitor the total dose of local anesthetic that is administered. In addition, the speed of sympathectomy is reduced, allowing the anesthesia provider to respond to alterations in hemodynamics.

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