Anesthesia for Post Anesthesia Care Nurses

Coleman, Harman, Koke, Sightler, Squires, Storm

STORM ANESTHESIA

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Introduction

Welcome to Storm Anesthesia's Anesthesia for Post Anesthesia Care nurses. This is a seven-video series on the basics on pharmacology and anesthesia techniques for the perianesthesia care nurse.

We, a group of five senior students from the University of South Carolina School of Medicine Nurse Anesthesia Program and one CRNA, have created this series in the hopes it will help the transition into the perianesthesia world. The series attempts to shine a bit of light on the techniques anesthesia uses during surgery, as well as explain the basics of the pharmacology behind our drug uses. This is by no means a series that will explain everything that happens during anesthesia, but our hope is that you, the perianesthesia nurse, will find our report a little less intimidating and a little more informative. After all, the better you understand the report, the better you can take care of the patient, and ultimately, this will increase the safety and satisfaction for both your patients and yourself.

The group consists of Alexandra Harman, BSN, RN; Braiden Sightler, BSN, RN; Jordan Coleman, BSN, RN, CCRN; Kelsey Squires, BSN, RN, CCRN; Victoria Koke, BSN, RN; and Michael Storm, DNAP, CRNA, CCRN.

The videos can be watched separately, but there are some references among the videos and the basics of the pharmacology along the way. Therefore, it may be beneficial to watch the series in order. Either way, have fun and don't forget to download the accompanying handouts. These handouts are the complete transcripts of the narrations and include all relevant pictures from the videos.

This video-series is sponsored by Storm Anesthesia and Palmetto Health Richland Anesthesia Department.

Enjoy and let's get started.

Michael Storm, DNAP, CRNA, CCRN
Editor
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Local Anesthetics

Victoria Koke, BSN, RN
University of South Carolina
School of Medicine
Graduate Program in Nurse Anesthesia
Class of 2018

Hello, my name is Victoria Koke. I am a registered nurse and a senior master's student in the anesthesia program at the University of South Carolina and today I would like to talk to you about Local Anesthetics.

Objectives

The objectives of this presentation are:

- Discuss the clinical use of local anesthetics
- Explain the physiological processes of how local anesthetics work
- Distinguish between commonly utilized local anesthetics
- Discuss some of complications of local anesthetic and the treatment of these complications

Local Anesthesia

- Local anesthesia is defined as the reversible loss of sensation in a limited area of the body without loss of consciousness by the use of a chemical agent
- This form of anesthesia can also be referred to or thought of as local analgesia as it blocks the pain transmission
- Local anesthesia has the ability to produce desensitization and analgesia of skin (topical anesthesia), tissues (infiltration and field block anesthesia), and regional structures (neuraxial, conduction and peripheral nerve block anesthesia)

Clinical Use of Local Anesthetics

Now let's examine some formal definitions associated with local anesthesia.

Conduction Block Anesthesia: local anesthetic injected in the immediate vicinity of a major nerve plexus (brachial plexus, lumbar plexus, and neuraxial anesthesia)

Field Block Anesthesia: local anesthetic injected in fanlike manner, into tissue surrounding an incision or puncture site

Infiltration Anesthesia: local anesthetic injected into the area of terminal nerve endings

Intravenous Anesthesia: general anesthesia produced by injection of local anesthetics and other anesthetic agents into the venous circulation to cause central nervous system depression

Neuraxial Blockade: a generic term that encompasses both spinal and epidural anesthesia

Peripheral Nerve Block: local anesthetic deposited in the immediate vicinity of an individual nerve to produce anesthesia

Topical Anesthesia: local anesthetic applied to skin or mucous membranes (pharyngeal cavity or urethra). Systemic absorption from the mucous membranes is rapid. Excessive dosing can lead to toxicity

Why Use Local Anesthesia

There are several reasons why one would opt to incorporate local anesthesia into their surgical process. It can provide effective analgesia. This is beneficial as it helps decease the use of post-operative narcotics and non-opioid analgesics. Consequently, the use of local anesthetics helps decrease post-operative pain thus aiding in patient satisfaction. Local anesthesia when used on its own without the addition of general anesthesia has the ability to reduce recovery time as one does not have to wait for a patient to fully wake up as they may not require any sedation at all to perform a procedure. This facet in conjunction with the formerly mentioned pain relief provided by local anesthetics can get individuals up and moving around sooner. The use of local anesthesia has the ability to decrease the body's stress response to surgical trauma by blunting pain and the pain response at the surgical site. Finally, the use of local anesthesia has the potential to reduce central sensitization.

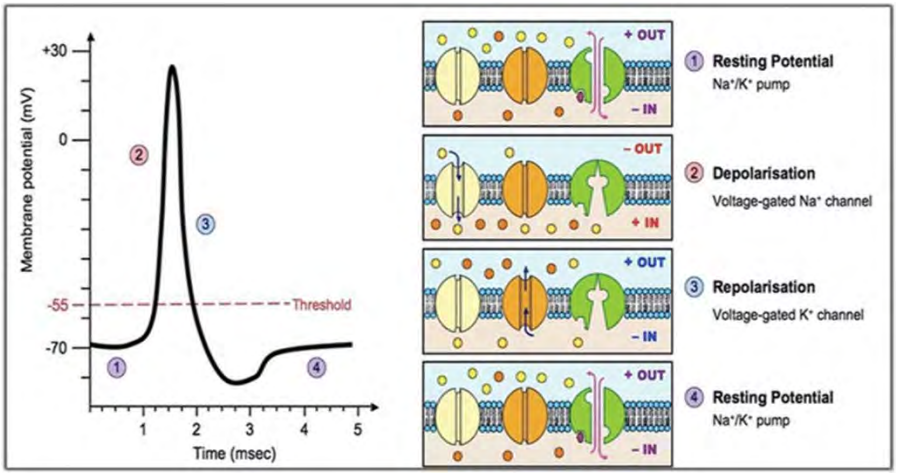
Central sensitization is a condition of the nervous system that is associated with the development and maintenance of chronic pain. When central sensitization occurs, the nervous system goes through a process called wind-up and gets regulated in a persistent state of high reactivity. Making an individual more susceptible to chronic pain which is harder to treat. Local anesthetics can disrupt the transmission of these nerve impulses thus helping reduce the development of central sensitization.

Advantages vs. Disadvantages

The use of local anesthetics is equipped with both advantages and disadvantages. Local anesthetics can be used as a stand-alone anesthetic/analgesic or in conjunction with general anesthesia. Local anesthesia when utilized by itself has a lower cost than general anesthesia. Additionally, the use of local anesthesia can decrease the use and thus cost of narcotics. It is suitable for awake patients so is a great option for outpatient surgery. Local anesthesia can decrease the time one spends immobilized therefore helping decrease the risk of Deep Vein Thrombosis (DVT) in the post-surgical setting. There are however some disadvantages to the use of local anesthetics. In order to achieve success with the use of a local anesthetic you must have a cooperative patient. For this reason, a patient may still require sedation in conjunction with the use of local anesthetics. Additionally, although infrequent, local anesthetics do have the ability to be irritants or cause some negative side effects such as lightheadedness, muscle twitch, or headache.

Mechanism of Action

Local anesthetics work at the level of the Na^+-channel to prevent action potentials from occurring. Depicted on the left in this graphic, is a single action potential. This process occurs at the axon. The axon is a long slender projection located on the neuron or nerve cell membrane.

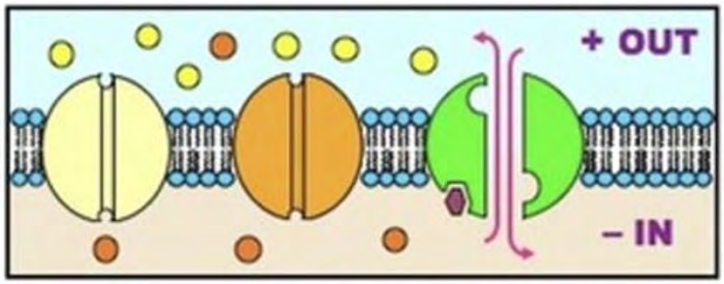


Each neuron has a designated negative resting membrane potential. The resting membrane potential is maintained by the sodium potassium pump, which helps balance the influx and efflux of positive sodium ions and negative potassium ions in and out of the cell.

Now let's look at a single action potential illustrated by this picture.

- **OUT** (top of picture) is outside the cell
- **IN** (bottom of picture) is inside the cell.

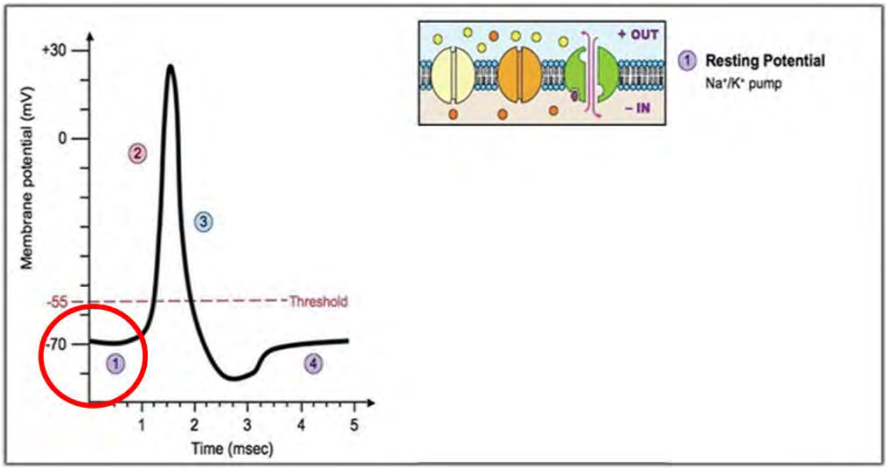
The large yellow circle is the sodium channel and the small yellow circles are the sodium ions. The large orange circle is the potassium channel and the small orange circles are the potassium ions.



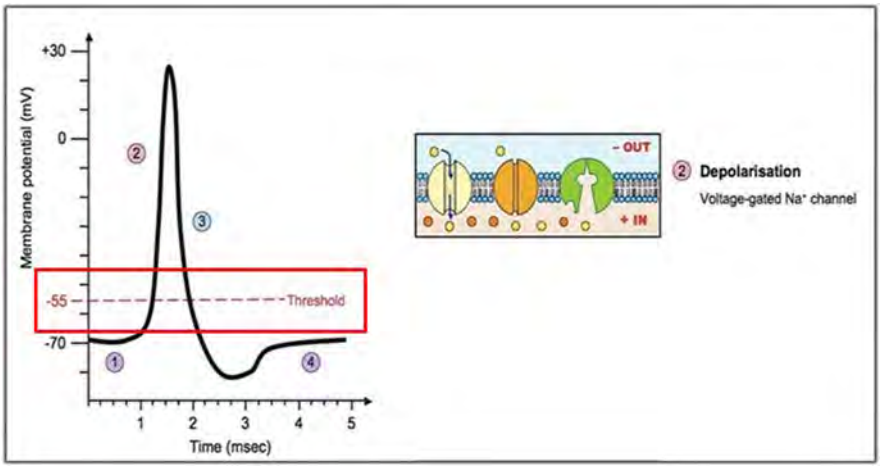
As you can see there is a preponderance of sodium ions outside the cell (yellow circles) compared to potassium ions and vice versa inside the cell; more potassium and less sodium. In this picture both the sodium and the potassium channels are closed. The only movement of ions happens through the Na/K-pump.

This pump is the large green circle. This energy-driven pump pumps sodium ions out of the cell and potassium ions into the cell. This action requires energy since the ions move against their concentration gradient; ie., sodium ions are plentiful outside the cell and the Na/K-pump moves even more sodium ions out of the cell and potassium ions are plentiful inside the cell, but still more are moved into the cell. Since this movement goes "uphill" so-to-speak it requires energy.

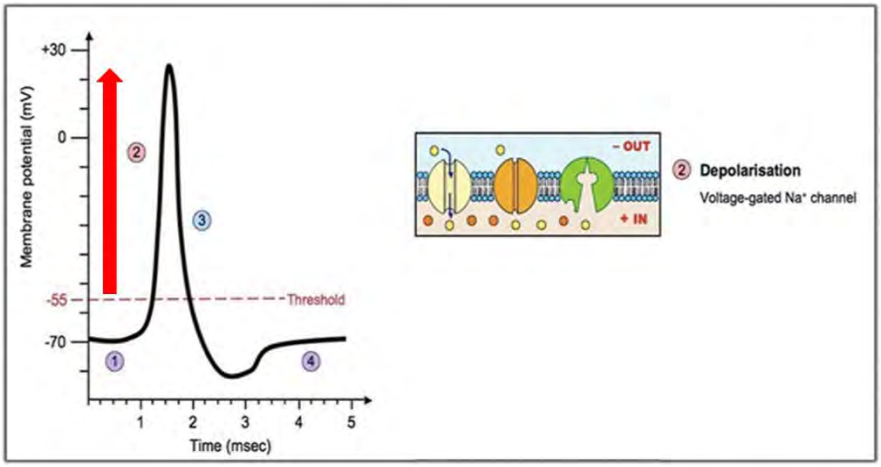
Now let's break down the steps of the action potential. In the graphic to the right, the resting membrane potential is -70 millivolts (mV) and is designated in this picture as step 1 of the action potential. As previously discussed, the resting membrane potential is maintained by the sodium/potassium pump which keeps this balance by regulating the movement of positive sodium ions and negative potassium ions in and out of the cell.



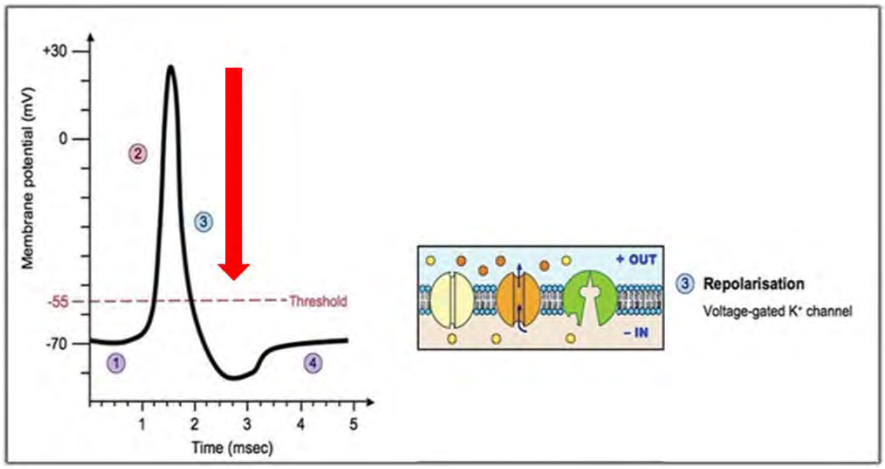
A stimulus such a pain, causes the influx of positive sodium ions into the cell. This alters the membrane potential, making it less negative. This is process is called depolarization. The influx continues until the neuron reaches its threshold. Threshold is the level at which an action potential can be triggered. In this example, the threshold of this neuron is depicted via the red dotted line and is –55 mV.



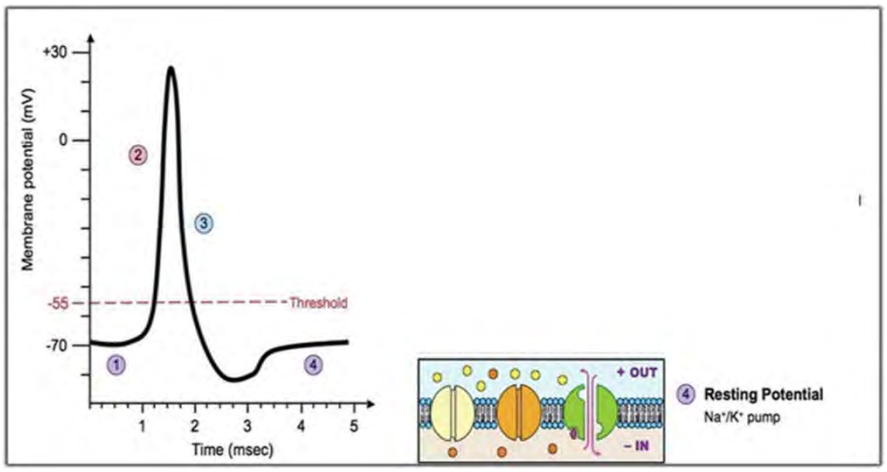
Upon reaching threshold, the action potential is set in motion and cannot stop until completion. The neuron rapidly depolarizes with the influx of sodium ions via voltage gated sodium channels until reaching its peak. This process is denoted in the picture above as step 2 of the action potential.



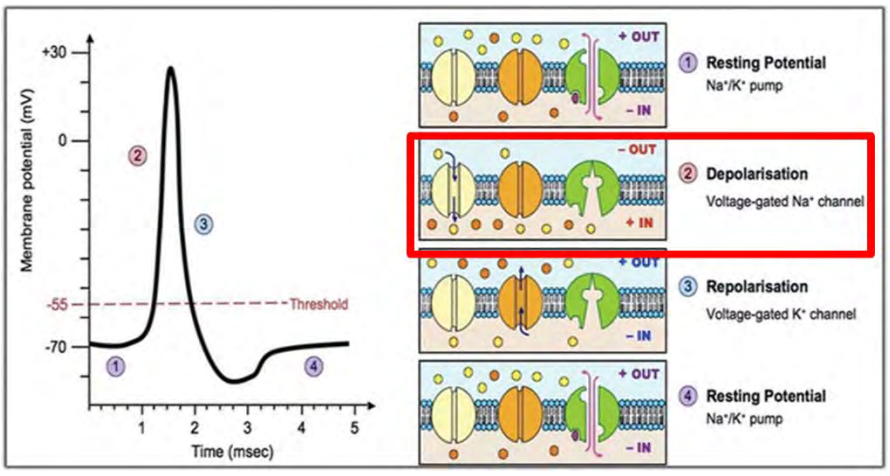
Upon reaching its peak, the membrane potential begins to repolarize or become more negative. This process occurs through the influx of potassium ions through voltage-gated potassium channels and is depicted above as step 3 of the action potential.



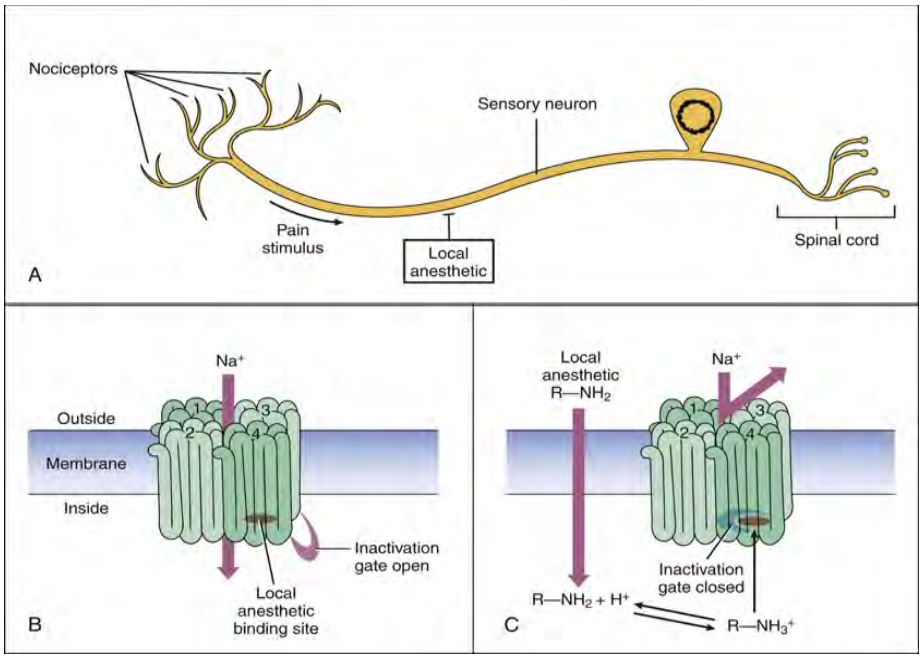
The neuron continues to repolarize until it falls below threshold once again and reaches its resting negative membrane state, donated by step 4 in the action potential. It then maintains this state via the sodium/potassium pump until another stimulus re-initiates the process.



Consequently, to prevent an action potential, local anesthetics must block voltage gated sodium channels. By blocking these channels, the neuron is prevented from depolarizing and thus prevented from reaching threshold, rendering it unable to trigger an action potential.

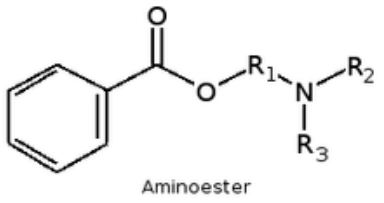


Depicted to the right is a larger scale view of the physiologic processes we discussed on the last slide. Box A, depicts a single sensory neuron in the body. Here we view the presence of nociceptors at the end of the neuron body. Nociceptors are a type of receptor located at the end of the neuron. Nociceptors sense and send signals of pain to the spinal cord and the brain. Local anesthetics achieve blockade impeding the transmission of the message of pain from nociceptors to the spinal cord and the brain. This is achieved by inhibiting the influx of Na^+ ions through the selective voltage gated Na^+ channels of the membrane as depicted in pictures B and C above. By inhibiting Na^+ ions there is an impairment of the generation of the action potential as a whole, and thus a stop of the conduction of nerve impulses. The prevention of these nerve impulses, prevents the message of pain from reaching the brain.

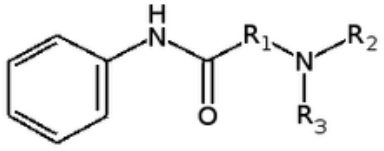


Classification

Local anesthetics can be classified by their chemical structure, duration of action and potency. In terms of chemical structure, local anesthetics can either be classified as an aminoester or an aminoamide. Aminoesters are derived from benzoic acid, whereas aminoamides are derived from aniline. The differences between these two chemical structures is depicted above in the picture to your left. Ester local anesthetics include: Procaine, Chloroprocaine, Benzocaine, Cocaine, and Tetracaine. Amide local anesthetics include: Bupivacaine, Mepivacaine, Ropivacaine, Levobupivacaine, Lidocaine, and Prilocaine. To quickly differentiate whether a local anesthetic is an ester or amide, note that there are two I's in the amide agents.



Aminoester



Aminoamide

In addition to chemical structure, local anesthetics can also be classified by their potency and duration of action.

Drug	Potency	Duration of Action*	Parenteral Uses	Topical Uses
Ester-type Drugs				
Procaine	Low	Short	Infiltration, nerve block, and spinal anesthesia	None
Chloroprocaine	Low	Short	Epidural, infiltration, and nerve block anesthesia	None
Benzocaine	Low	Medium	None	Dermal, laryngeal, and oral
Cocaine	Low	Medium	None	Laryngeal, nasal, and urogenital
Amide-type Drugs				
Lidocaine	Intermediate	Short	Epidural, infiltration, nerve block, and spinal anesthesia	Dermal, laryngeal, and oral
Mepivacaine	Intermediate	Short	Epidural, infiltration, nerve block, and spinal anesthesia	None
Prilocaine	Intermediate	Short	Infiltration anesthesia	Dermal
Etidocaine	Intermediate	Long	Infiltration and nerve block anesthesia	None
Bupivacaine	High	Medium	Epidural, infiltration, nerve block, and spinal anesthesia	None
Ropivacaine	High	Long	Epidural, infiltration, and nerve block anesthesia	None
*The duration varies with the dose and route of administration. short = 0.25-1.5 hours; medium = >1.5-5 hours; and long = > 5 hours.				

As indicated in this table: low potency short duration local anesthetics include drugs such as procaine and chloroprocaine.

Intermediate potency and medium duration of action local anesthetics include mepivacaine, lidocaine, and prilocaine.

High potency and long duration of action local anesthetics include bupivacaine, ropivacaine and etidocaine.

It is important to understand these distinctions when caring for your patient so that you can anticipate the onset of symptoms associated with the wearing-off of the local anesthetic as well as be proactive in treating pain.

Factors Effecting Activity and Potency

There are two major factors that affect the activity and potency of local anesthetics:

- lipid solubility
- protein binding

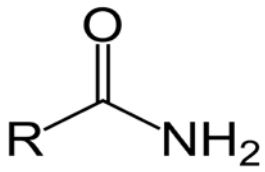
If a local anesthetic has increased lipid solubility it will have a decreased or slower onset of action. Local anesthetics that are highly protein bound have a longer duration of action. These concepts are important to keep in the back of your mind as we discuss individual local anesthetics as short, intermediate and long action as well as their potency as a drug.

Pharmacokinetics

Pharmacokinetics is the study of how drugs move in the body. This includes how drugs are absorbed, metabolized and excreted. As previously mentioned, drugs with greater lipid solubility and protein binding will have a slower onset and longer duration of action- this can also be thought of as lower systemic absorption. Ester and Amide local anesthetics are both metabolized and excreted in different ways in the body. Ester linked local anesthetics, are metabolized by non-specific plasma pseudocholinesterase. Esterase in present in the liver, Red Blood Cells (RBCs), and synovial fluid. Ester anesthetics are excreted as metabolites in the urine. Amide linked local anesthetics are almost exclusively metabolized in the liver by microsomal enzyme CYP-450. Similar to ester linked local anesthetics, amide linked local anesthetics can be excreted as metabolites in urine but are also excreted as metabolites in the bile.

Amides

Amides include: lidocaine, prilocaine, mepivacaine, bupivacaine, ropivacaine, levobupivacaine, and etidocaine. Amides are metabolized by the liver and rarely cause allergic reactions.

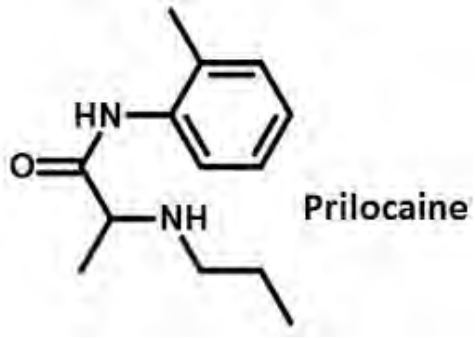


Lidocaine

Lidocaine is an intermediate acting local anesthetic. It has a quick onset is very potent, this is because lidocaine is highly protein bound. It is because of this strength that lidocaine can be accompanied by side effects of dizziness and sleepiness. To help combat these side effects, lidocaine is frequently administered with epinephrine to decrease the vasodilation and absorption of this drug. The co-administration of epinephrine also helps prolong the duration of action of local anesthetics and thus is commonly co-administered with the local anesthetic in use. Lidocaine which is typically effective between 40 and 90 minutes, can last up to 240 minutes when administered with epinephrine. The maximum dose of this medication without the use of epinephrine is 4 mg/kg and increases to 7 mg/kg with the use of epinephrine. Lidocaine, is metabolized by hepatic microsomal enzymes such as CYP450. This means that one should exercise caution when administering lidocaine to a patient who takes medications that either decrease hepatic blood flow or inhibit metabolic enzymes. Some of these drugs include propranolol, cimetidine and SSRIs. It is frequently utilized in spinal anesthesia as it possesses the ability to block both sensory and motor nerves equally well. Additional uses of lidocaine include the treatment of re-entry tachyarrhythmia.

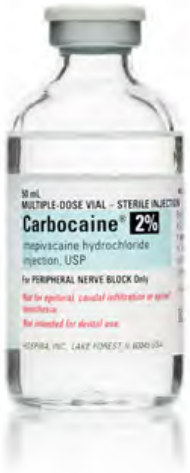
In this instance, the ischemic portion of ventricle is damaged and does not conduct aps as rapidly as healthy portion of heart, it will eventually lag behind to a point that it begins firing signals on its own very rapidly. IV lidocaine targets areas with faster depolarization (ischemic portion) and will block conduction to bring back into sequence with the rest of the ventricle.

Prilocaine



Prilocaine is a short acting, intermediate potency amide. This local anesthetic has a duration of action of approximately 60 minutes, or 380 minutes when administered with epinephrine. The maximum dose of prilocaine is 5 mg/kg, and increases to 7.5 mg/kg with the addition of epinephrine. It has less of a vasodilatory effect than lidocaine and is metabolized in the blood. Once absorbed in the blood, prilocaine, releases a chemical compound called ortho-toluidine. This substance converts hemoglobin to methemoglobin. Consequently, prilocaine has the ability to cause methemoglobinemia. Symptoms of methemoglobinemia include brown-gray cyanosis, tachypnea, metabolic acidosis, hypoxia, headache, irritability, and chocolate-colored blood. Methemoglobinemia can be treated with a methylene blue injection of 1-2 mg/kg (this dose can be repeated if necessary).

Mepivacaine



Mepivacaine is an intermediate-acting local anesthetic. This local anesthetic similar to lidocaine has moderate potency and toxicity, however it does not case the same vasodilatory effects as lidocaine. Mepivacaine can provide local anesthesia for 60-120 minutes and can last up to 360 minutes when administered with epinephrine. The maximum dose without epinephrine is 5 mg/kg and 7 mg/kg with epinephrine. Mepivacaine is metabolized by hepatic microsomal enzymes. Consequently, similarly to lidocaine be aware that patients taking medications also metabolized by these enzymes are at risk for having a prolonged duration of action of this local anesthetic. Mepivacaine is utilized in both epidural and caudal anesthesia. However, unlike lidocaine, Mepivacaine is not frequently utilized in spinal anesthesia as its use is associated with an increased incidence of Transient Neurological Symptoms. Transient neurological symptoms include intense pain in the buttocks and thighs that sometimes radiates to the lower extremities. It begins within 24 hours of spinal anesthesia and can last as long as ten days but typically resolves within 48 hours.

Bupivacaine

Bupivacaine is a long-acting local anesthetic, that is 4x more potent than lidocaine. As a result, the result of this potency the residual analgesia of this medication outlasts the anesthetic providing a longer course of pain relief for the patient. This medication provides a greater sensory block than motor block and is commonly utilized in both spinal and epidural anesthesia due to its long duration of action of 120-240 minutes and as long as 8 hours with the addition of epinephrine. The maximum dose of bupivacaine is 2.5 mg/kg or 3.2 mg/kg with epinephrine. The maximum single dose for bupivacaine is 175 mg and the suggested maximum 24-hr dose is 400mg. Like formerly mentioned amides, this local anesthetic is also metabolized by hepatic microsomal enzymes.

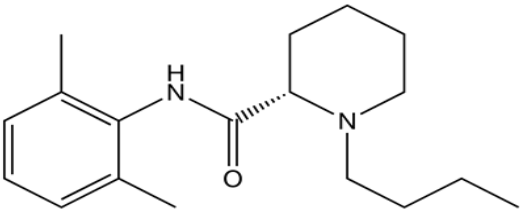
CAUTION: When utilizing this drug, one needs to be aware of the potential cardiovascular side effects at higher doses and accidental IV injection which could leave your patient with severe hypotension or a ventricular arrhythmia that might not be easy to correct due to Bupivacaine's long duration of action.

Ropivacaine

Ropivacaine is a long acting local anesthetic with intermediate potency. Its chemical structure is very similar to bupivacaine resulting in a similar projection for way this local anesthetic works in the body. However, ropivacaine is associated with less cardiotoxicity and less motor block than bupivacaine. Ropivacaine is frequently utilized in epidural anesthesia and can have a duration of action of approximately 3 hours or up to 6 hours when administered with epinephrine. The maximum dose of Ropivacaine is 2-3 mg/kg.

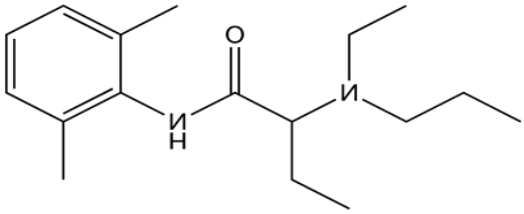
Levobupivacaine

Levobupivacaine is an intermediate acting local anesthetic. It has a similar chemical structure and drug profile to bupivacaine but is not associated with the cardio depressant effects that can sometimes accompany bupivacaine. It has a fast onset of less than 15 minutes and a duration of action that can last upwards of 9 hours, and as long as 12 hours with the addition of epinephrine. This local anesthetic is frequently injected in conjunction with an additive for additional pain relief such



as fentanyl, clonidine or preservative free morphine. The maximum dose of levobupivacaines without additive is 2 mg/kg or 400mg over the course of 24 hours.

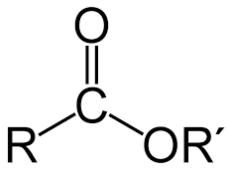
Etidocaine



Etidocaine is a long-acting, high potency local anesthetic. To achieve a sensory block with this local anesthetic it requires an extremely high concentration. It is utilized as a local injection, in peripheral nerve blocks and in epidurals due to its long duration of action of 4 hours and up to 8 hours with the addition of epinephrine. The maximum dose for Etidocaine is 2.5 mg/kg or 4 mg/kg when administered with epinephrine.

Esters

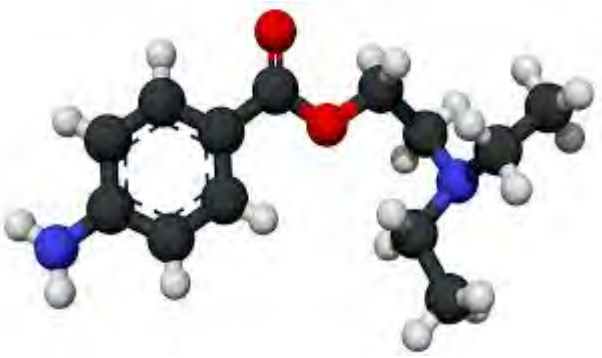
Now we will continue our discussion of local anesthetics, focusing on the ester local anesthetics. Esters include cocaine, procaine, chloroprocaine, tetracaine and benzocaine.



Cocaine

Cocaine was the first ester-class drug used as a clinical local anesthetic in 1884. It is a strong topical anesthetic and is utilized in anesthesia especially for its vasoconstrictive properties. For this reason, it was previously utilized the nasal mucosa before nasotrachael intubation and during nasal operations. Although still available, this local anesthetic is no longer frequently used due to the high risk for abuse potential and the availability of other vasoconstrictors such as Afrin Spray and Phenylephrine. Cocaine has a duration of action between 5 and 90 minutes. It is metabolized by the liver and its metabolites are excreted in the urine. This local anesthetic blocks the reuptake of catecholamines and consequently causes an accumulation of norepinephrine in the synaptic cleft. This leads to profound effects on the central nervous system. At low doses it will cause stimulation, restlessness, euphoria and talkativeness. In moderate doses it can cause tremors, convulsions and emesis and in high doses it can cause CNS stimulation (marked by elevated heart rate and blood pressure), followed by CNS depression and increased body temperature.

Procaine



Procaine was the first synthetic ester-class local anesthetic. Created in 1904, it is less potent and shorter acting than its amide counterpart: lidocaine. Procaine is reported to cause more allergic reactions than lidocaine. It is not effective topically but can be utilized in spinal anesthesia, peripheral nerve blocks and local infiltration anesthesia. To help slow absorption it is commonly administered with a vasoconstriction agent, this also aides in diminishing the chance for systemic toxicity when administering this drug. The duration of action of Procaine is 20-60 minutes and up to 90 minutes with the addition of epinephrine with a maximum dose of 8 mg/kg or 10 mg/kg when administered with epinephrine. Procaine is metabolized by plasma cholinesterase.

Chloroprocaine

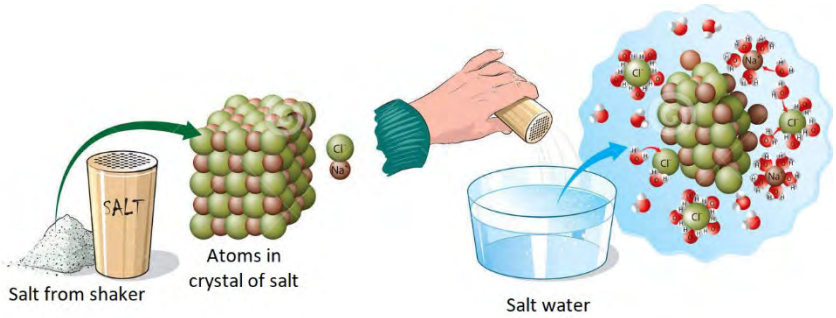
Chloroprocaine is a short-acting local anesthetic. It has a rapid onset and is utilized in local infiltration, peripheral nerve blocks and caudal/epidural anesthesia. Chloroprocaine provides a stronger sensory than motor blockade and can last up to 60 minutes or as long as 90 minutes with the addition of epinephrine. The maximum dose of Chloroprocaine is 10 mg/kg or 15 mg/kg with epinephrine. It is metabolized by plasma cholinesterase. Administration of this drug can be associated with thrombophlebitis, a condition in which a blood clot forms in the vein causing inflammation and pain. Symptoms included: redness, swelling, tenderness, warmth and tenderness at the effected site. Treatment includes: elevating the site, cold or warm compresses and anti-inflammatory medications.

Tetracaine

Tetracaine is a long acting, high potency local anesthetic. In fact, Tetracaine is ten times more potent than procaine. This medication is effective topically and causes an extensive motor and sympathetic blockade. For this reason, it is commonly utilized in corneal anesthesia. Its duration of action is 2-3 hours or 10 hours with epinephrine, the maximum dose is 1.5 mg/kg or 2.5 mg/kg with epinephrine and it is metabolized by plasma cholinesterase.

Benzocaine

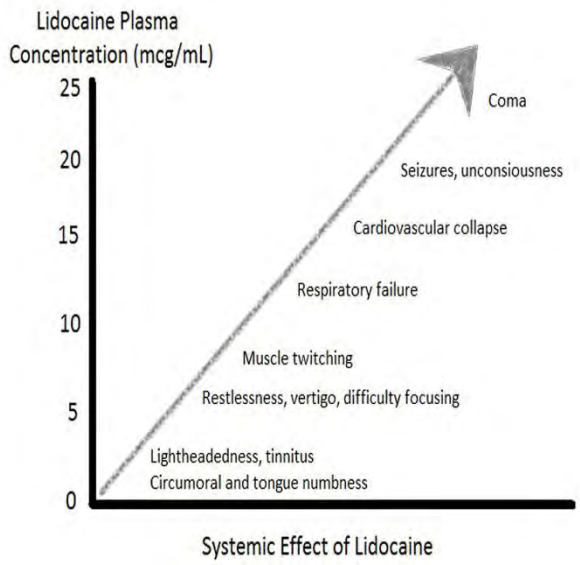
Our last local anesthetic is benzocaine. This local anesthetic is ONLY effective topically as it exists only in an nonionized form which is atypical of local anesthetics. Other local anesthetics are made of several elements that combine to make the drug. As a result, the drug carries neither a positive or negative charge. If a drug can be ionized, it can split into two parts. One of the parts carries a plus charge (+) while the other part has a negative charge (-).



A simple example is table salt or sodium chloride (NaCl) which can become ionized in water. NaCl is an inorganic compound. When table salt (NaCl) is placed in water it is able to ionize or divide into the ions of Sodium (Na^+) and chloride (Cl^-). Non-ionized drugs on the other hand, such as benzocaine are organic compounds that cannot separate or divide. The molecules are stuck in their structures together in such a way that they cannot separate into parts. It is for this reason that benzocaine is effective as a topical anesthetic as it will not be broken down into parts. The typical duration of action for this local anesthetic is 30-60 minutes.

Possible Side Effects

Some of the side effects of local anesthetics which can include Central Nervous System Toxicity, Cardiovascular Toxicity and Neural Damage.



Central Nervous System Toxicity

Hypercarbia and hypoxia can potentiate CNS toxicity. Therefore, it is important that after receiving a local anesthetic your patient maintains an appropriate oxygen saturation level. CNS toxicity can sometimes be avoided with the application of supplemental oxygen (02). Symptoms of CNS toxicity included: circumoral numbness (this is typically an early symptom), light-headedness,

tinnitus, visual disturbances, slurred speech, muscle twitching, irrational speech, apnea followed by cardiovascular depression, grand mal seizures which should be treated with versed and valium and finally coma. It is important to monitor your patient closely and notice these symptoms early on.

Cardiovascular Toxicity

Although uncommon, cardiovascular side effects are among the hardest complications associated local anesthetics to treat. This is because, local anesthetics competitively block the NA^+ channels of the heart. Cardiovascular toxicity is most commonly associated with the administration of bupivacaine and Tetracaine. Signs and symptoms include hypertension that leads to hypotension, premature ventricular complexes, prolonged PR interval and QRS complexes on EKG and ultimately cardiovascular collapse. The first line treatment is: PREVENTION. Local anesthetics should be initially injected in divided doses with frequent aspiration to prevent the possibility of cardiovascular toxicity. If cardiovascular toxicity should occur: hypotension should be treated with ephedrine. One should be prepared to provide advanced cardiac life support with the possibility of transcutaneous pacing. The use of 20% intralipid infusion to the site of regional anesthesia can also offer some protection from the progression of cardiovascular collapse.

Lipid Rescue

In the event of local anesthetic-induced cardiac arrest that is unresponsive to standard therapy such as ACLS protocol drugs in addition to standard cardio-pulmonary resuscitation, intralipid 20% should be given intravenously. Throughout the lipid rescue it is important to maintain CPR efforts with limited interruptions. Administer an intralipid 20% bolus at the rate of 1.5 ml/kg over one minute. This bolus dose can be repeated every 3-5 minutes up to a dose of 3ml/kg in total until circulation is restored. After the initial bolus continue with a intralipid infusion at a rate of 0.25 ml/kg/min. After circulation is restored continue the infusion increasing it to a rate of 0.5 ml/kg/min for drops in blood pressure.

Neural Toxicity

The spinal cord and roots are most prone to neural damage. Additional neural side effects include Transient radicular irritation and cauda equina syndrome. Transient radicular irritation is associated with the administration lidocaine. It typically presents within 24 hours and can last up to one week. Symptoms include pain in the buttock, lower back

pain and posterior thigh pain. Cauda equina syndrome (CES) is associated in descending frequency with: lidocaine, Tetracaine, bupivacaine, and ropivacaine. This is an injury to the lumbar-sacral plexus and sensory paresthesia. Symptoms include: bowel and bladder dysfunction and paraplegia.

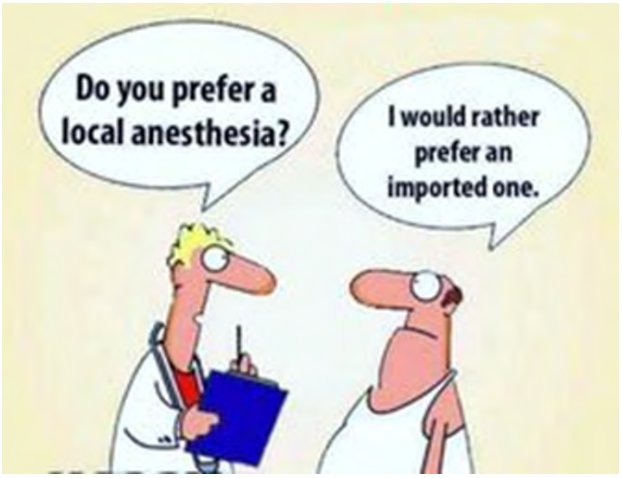
Conclusion

Local anesthetics are utilized in a wide variety of anesthesia including: conduction block anesthesia, field block anesthesia, infiltration anesthesia, neuraxial blockade, peripheral nerve blocks and topical anesthesia. Local anesthetics work by inhibiting the influx of NA+ through voltage-gated sodium channels, thus preventing action potentials in the body. Local anesthetics can be used to provide stand-alone analgesia or in conjunction with general anesthesia. The duration of action and potency of the local anesthetic varies with the drugs lipid solubility and affinity for protein binding. Local anesthetics are divided into two classes: amides and esters.

Amides include: prilocaine, lidocaine, mepivacaine, bupivacaine, ropivacaine, levobupivacaine and etidocaine. Esters include: cocaine, procaine, chloroprocaine, tetracaine and benzocaine. Although uncommon: CNS, cardiovascular and neural toxicity is possible with local anesthetics. Local anesthetics are widely used to provide analgesia in a wide array of patients and procedures.

Thank You

This concludes my presentation on local anesthetics. Thank you for your continued work in caring for our patients and your time.



Bibliography

Odom-Forren J. Drain's Perianesthesia Nursing - A Critical Care Approach. Vol 7th ed. St. Louis, MO: Elsevier; 2018.

Schick L, Windle PE. Perianesthesia Nursing Core Curriculum. Vol 3rd ed. St. Louis, MO: Elsevier; 2016.

Miller RD (ed). Miller's Anesthesia. Vol 7th ed. (Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish HP, Young WL, eds.). Philadelphia, PA: Churchill Livingstone, Elsevier; 2010.

Barash PG, Cullen BP, Stoelting RK, Cahalan MK, Stock MC, Ortega R. Clinical Anesthesia. Vol 7th ed. Philadelphia, PA: Lippincott Williams & Wilkis; 2013.

Flood P, Rathmell JP, Shafer S. Stoelting's Pharmacology & Physiology in Anesthetic Practice. Vol 5th ed. Philadelphia, PA: Wolters Kluwer Health; 2015.