Basic Guide to Anesthesia for Developing Countries

Daniel D. Moos

Volume 2

The intent of this manual is to be freely used, copied, and distributed in Developing Countries for the teaching and promotion of basic anesthesia knowledge/skills.

The purpose of this manual is to provide developing countries with a copyright free basic anesthesia manual. This manual can be freely copied and translated into a native language for the promotion of basic anesthesia knowledge/skills. Contributors with credited pictures and illustrations have graciously given permission for their material to be used for this specific purpose. The author and publishers of this manual cannot accept liability from the use of this material or errors in translation. It is up to each translator to ensure that the translation is correct. Knowledge about the art and science of anesthesia continues to change. It is up to each anesthesia provider to continue to learn and upgrade their knowledge. This manual only contains basic knowledge and is not a replacement for more comprehensive anesthesia information.

“Every prudent man acts out of knowledge.”
Proverbs 13:15

Soli Deo Gloria

Acknowledgements

This project would not have been possible without the help of many.

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Thank you.

Introduction

This is the second volume of Basic Guide to Anesthesia for Developing Countries. This volume contains three distinct sections: regional anesthesia; obstetric anesthesia; and trauma. Each section contains information for basic and safe care. For advanced and in-depth discussions I would refer the reader to other sources. This is simply a primer.

This journey began in 2004 with a short trip to Afghanistan. I realized that there was an absence of basic anesthesia material in a native language. As time passed I learned that another country, Cambodia, lacked a basic anesthesia text in Khmer. Since 2004 I have worked hard to produce a basic text that would be freely available without copyright restrictions for translation and use in a native language.

It is my prayer that this manual will increase knowledge and improve care of all surgical patients who undergo care under our watchful eyes. Administering anesthesia is a lifelong learning process. This manual is designed to introduce a basic foundation of knowledge to the trainee as well as to serve as a basic review for those who are practicing the art and science of anesthesia.

Every effort was made to ensure that the material and information contained in this manual is correct and up-to-date. The publishers and authors cannot accept liability from errors that may occur from the use of this material.

Please feel free to contact me at moosd@charter.net with comments, questions, recommendations for future editions, and any concerns.

Daniel D. Moos

Resources

A number of resources are available for the anesthesia provider in developing countries.

Books

Anaesthesia at the District Hospital, 2nd Edition. Michael B. Dobson. Published by the World Health Organization in collaboration with the World Federation of Societies of Anesthesiologists. This manual was published to help guide medical officers in small hospitals. It contains a wealth of practical and useful information. This book is available in English.

Internet Resources

Manuals

Safe Anaesthesia. Lucille Bartholomeusz, 3rd edition updated and revised by Jean Lees**.** This manual is available at http://www.worldanaesthesia.org . This 700+ page manual contains comprehensive information concerning anesthesia. Individual chapters may be downloaded. This manual is available in English.

Basic Guide to Anesthesia for Developing Countries Volume 1, Daniel D. Moos. This manual is available at http://www.worldanaesthesia.org and http://ifna-int.org . This manual can be freely downloaded, copied, and translated for the promotion of basic anesthesia knowledge and skills. The manual contains 230 pages of information which includes: medical math, documentation, fluid management/replacement, medications, preparation, positioning/monitoring, airway management, basic CPR, cardiac arrest, recovery basics, and pediatric anesthesia.

Basic Guide to Resuscitation for Developing Countries. Daniel D. Moos. This manual is available at http://www.worldanaesthesia.org and http://ifna-int.org . This manual can be freely downloaded, copied, and translated for the promotion of basic resuscitation techniques. Additional information concerning basic resuscitation may be obtained at http://erc.edu (European Resuscitation Council) and http://americanheart.org (American Heart Association).

Primary Trauma Care is an excellent resource for basic trauma care. It is available at **http://www.primarytraumacare.org .** This thirty-nine page manual is available in English, Chinese, Spanish, French, Indonesian, Mongolian, Farsi, and Vietnamese.

Education

World Anaesthesia Online can be accessed at http://www.nda.ox.ac.uk/wfsa/. This web site is dedicated to the promotion of anesthesia knowledge and skills in the developing world. Update in Anaesthesia is "An educational journal aimed at providing practical advice for those working in isolated or difficult environments." The majority of the updates that are available online are in English. A small number of updates are available online in Russian and French. The print version is available in English, Russian, French, Mandarin, and Spanish.

World Anaesthesia can also be accessed at http://www.neda.ox.ac.uk/wfsa/ . World Anaesthesia is a newsletter of the World Federation of Societies of Anaesthesia. The newsletter allows "for the exchange of views & ideas on advancing the specialty of anaesthesia in the developing world." It is available in English.

International Organizations

International Federation of Nurse Anesthetists (IFNA) was founded in 1989 and currently has 34 country members. The IFNA is an international organization whose mission is in part dedicated to the advancement of educational standards and practices of anesthesia. The IFNA website is located at http://www.ifna-int.org .

Additional resources concerning the IFNA include:

Caulk R, Ouellette S M. The International Federation of Nurse Anesthetists A Professional Study and Resource Guide For The CRNA. AANA Publishing 2001; Chapter 19: 381-406.

McAuliffe M. Countries where anesthesia is administered by nurses. AANAJ 64 (5), 469-479.

Henry B, McAuliffe M. Practice and education of nurse anesthetists. Bulletin of the World Health Organization.The International Journal of Public Health. 77 (3), 267-270.

World Federation of Societies of Anesthesiologists (WFSA) was founded in 1955 and currently has 122 country members. The objectives of the WFSA is the improve/disseminate knowledge concerning the standards of anesthesia, pain treatment, trauma management and resuscitation to all countries of the world. The WFSA website can be located at http://anaesthesiologists.org .

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Local Anesthetics

Chapter One

Local Anesthetics

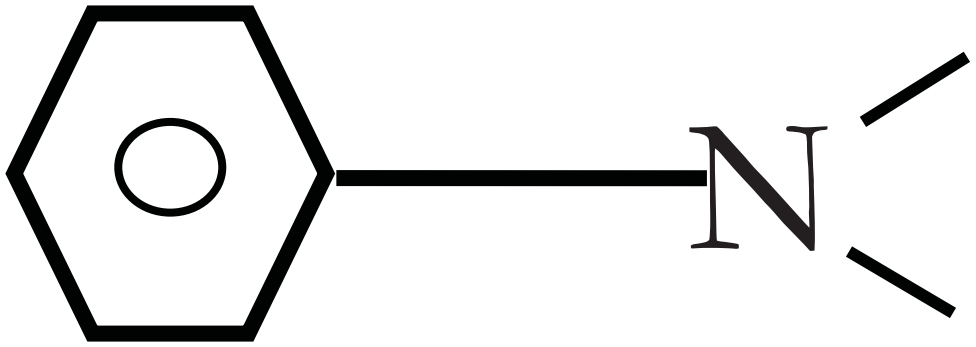
Local anesthetics produce a reversible loss of sensation in a portion of the body. Local anesthetics may be used as the sole form of anesthesia, in combination with general anesthesia, and/or to provide postoperative analgesia.

Indigenous natives of Peru chewed on leaves of Eryroxylon coca, the source of cocaine, to decrease fatigue and promote a feeling of well being. In 1884, Koller introduced cocaine as a topical anesthetic for the cornea. There were two problems with cocaine, physical dependence and toxicity. In 1905, Einhorn introduced the prototypical ester local anesthetic, procaine. In 1943, Lofgren introduces lidocaine, the prototypical amide local anesthetic.

Chemistry

The basic chemical structure of a local anesthetic molecule consists of 3 parts:

1. Lipophilic group- an aromatic group, usually an unsaturated benzene ring.
2. Intermediate bond- a hydrocarbon connecting chain, either an ester (-CO-) or amide (-HNC-) linkage. The intermediate bond determines the classification of local anesthetic.
3. Hydrophilic group- a tertiary amine and proton acceptor.



Lipophilic Group	**Intermediate Bond**	**Hydrophilic Group**
Benzene Ring	Ester or Amide Linkage (-CO- ester or –HNC- amide)	Tertiary Amine & Proton Acceptor

Amide and ester local anesthetics follow different paths of metabolism. Ester local anesthetics are more likely to cause an allergic reaction. (See Biotransformation & Excretion).

Amides	Esters
Bupivacaine	Benzocaine
Etidocaine	Chloroprocaine
Levobupivacaine	Cocaine
Lidocaine	Procaine
Mepivacaine	Tetracaine
Prilocaine	
Ropivacaine	

Structure Activity Relationships (Potency, Duration, & Onset)

The intrinsic potency, duration, and onset of action for a local anesthetic are dependent upon:

1. Lipophilic-hydrophobic balance
2. Hydrogen ion concentration

Lipophilic-Hydrophobic Balance

The term "lipophilic" means "fat" loving, expressing the tendency of the local anesthetic molecule to bind to membrane lipids. The term "hydrophobic" means fear of water. The lipid membrane is a hydrophobic environment. The term "hydrophobicity" is often used to describe the physiochemical property of local anesthetics. Potency as related to local anesthetics correlates with lipid solubility. In clinical practice, the potency of a local anesthetic is affected by several factors including:

- Hydrogen ion balance
- Fiber size, type, and myelination
- Vasodilator/vasoconstrictor properties (affects rate of vascular uptake)
- Frequency of nerve stimulation
- pH (an acidic environment will antagonize the block)
- Electrolyte concentrations (hypokalemia and hypercalcemia antagonizes blockade)

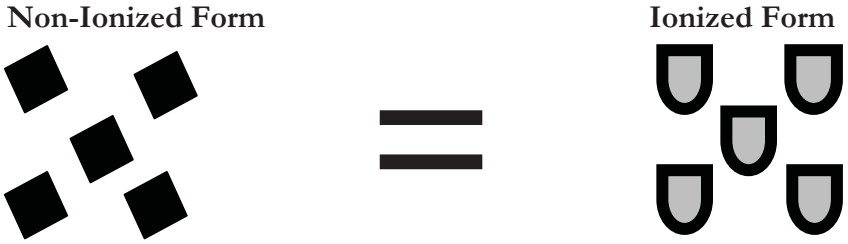
Duration of action is associated with lipid solubility. Highly lipid soluble local anesthetics generally have a longer duration of action due to decreased clearance by localized blood flow and increased protein binding.

Local Anesthetic	Potency and Lipid Solubility/Duration of Action
AMIDES	
Bupivacaine/Levo-Bupivacaine	4/4
Etidocaine	4/4
Ropivacaine	4/4
Mepivacaine	2/2
Lidocaine	2/2
Prilocaine	2/2
ESTERS	
Tetracaine	4/3
Cocaine	2/2
Procaine	1/1
Chloroprocaine	1/1

1= least; 4= greatest

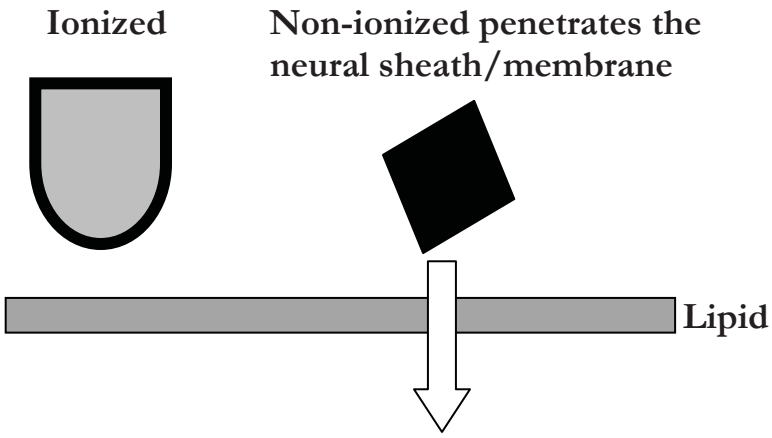
Hydrogen Ion Concentration

Local anesthetics are weak bases, containing a positive charge on the tertiary amine at a physiologic pH. Local anesthetics exist in equilibrium between the basic uncharged (non-ionized) form, which is lipid soluble, and the charged (ionized) cationic form, which is water soluble. The measurement pKa expresses the relationship between the non-ionized and ionized concentrations. Specifically, pKa is the pH at which the ionized and non-ionized forms of the local anesthetic are equal.

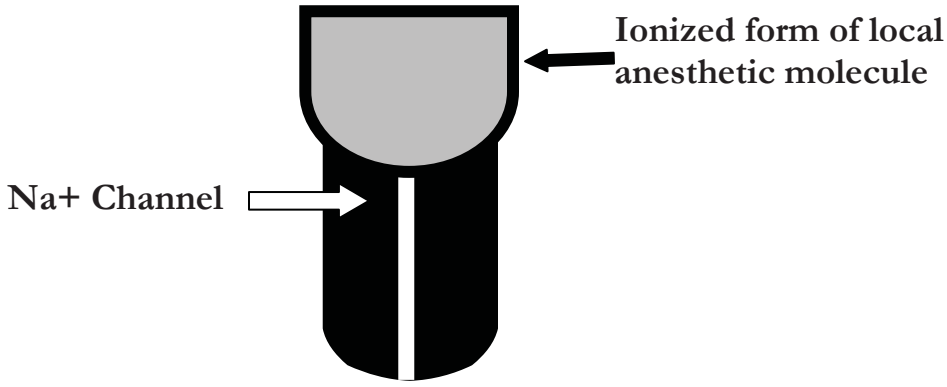


pKa = pH at which ionized and non-ionized forms of local anesthetic are equal.

Local anesthetics are weak bases and contain a higher ratio of ionized medication compared to non-ionized. Increasing the concentration of non-ionized local anesthetic will speed onset. In general, local anesthetics with a pKa that approximates physiologic pH have a higher concentration of non-ionized base resulting in a faster onset. On the other hand, a local anesthetic with a pKa that is different from physiologic pH will have more ionized medication which slows onset. For example, the pKa for lidocaine is 7.8 and 8.1 for bupivacaine. Lidocaine is closer to physiologic pH than bupivacaine. Lidocaine has a greater concentration on non-ionized local anesthetic than bupivacaine which results in a faster onset. Non-ionized and ionized portions of local anesthetic solution exert distinct actions. Lipid soluble, non-ionized form of the local anesthetic penetrates the neural sheath and membrane. In the cell, the non-ionized and ionized forms equilibrate. The ionized form of the local anesthetic binds with the sodium channel. Once "bound" to the sodium channel, impulses are not propagated along the nerve.



Ionized- binds with the sodium channel



Local Anesthetic	pKa
AMIDES	
Bupivacaine and levo-Bupivacaine	8.1
Ropivacaine	8.1
Lidocaine	7.8
Prilocaine	7.8
Etidocaine	7.7
Mepivacaine	7.6
ESTERS	
Chloroprocaine	9.0
Procaine	8.9
Cocaine	8.7
Tetracaine	8.2

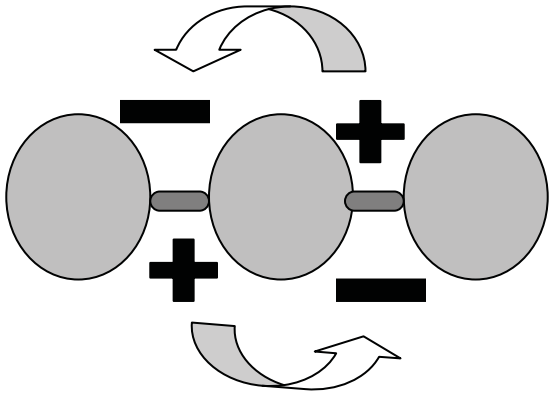
Clinically, onset of action is not the same for all local anesthetics with the same pKa. This is due to the intrinsic ability of the local anesthetic to diffuse through connective tissue. Local anesthetics with a pKa closest to the physiological pH generally have a higher concentration of non-ionized molecules and a more rapid onset. Two notable exceptions are chloroprocaine and benzocaine. Chloroprocaine has a high pKa and rapid onset. Benzocaine does not exist in an ionized form and exerts its effects by alternate mechanisms.

Clinical Implications of Hydrogen Ion Concentration

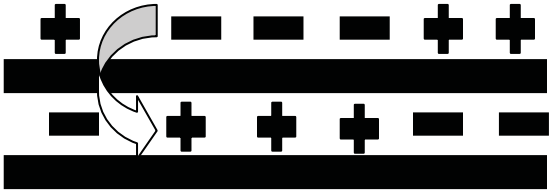
Local anesthetics are prepared as a water soluble hydrochloride salt and generally have a pH of 6-7. If the commercial preparation contains epinephrine, the solution must be acidic to create a stable environment. The corresponding pH is in the range of 4-5. Commercial preparations with epinephrine have less free base, slowing the onset of action. To enhance clinical onset, carbonated solutions of epinephrine containing local anesthetics have been used instead of HCL solutions. Alternatively, adding sodium bicarbonate to commercial preparations of epinephrine containing local anesthetic solutions can hasten the onset. One (1) ml of 8.4% sodium bicarbonate should be added to each 10 ml of lidocaine or mepivacaine, and 0.1 ml of 8.4% of sodium bicarbonate should be added to each 10 ml of bupivacaine. Increasing the volume of sodium bicarbonate added the local anesthetic preparation may lead to precipitation. Altering the pH to a more basic solution will increase the amount of non-ionized compared to ionized which will speed onset. Sodium bicarbonate increases the amount of free base, increases onset, improves the quality of the block, and decreases pain associated with subcutaneous infiltration.

Peripheral Nerve Anatomy

Axolemma- the peripheral nerve axon cell membrane.
Non-myelinated nerves- contain axons within a single Schwann cell (i.e. autonomic postganglionic efferent and nociceptive afferent C fibers).
Large motor and sensory fibers are enclosed in many layers of myelin.
Myelin insulates the axolemma and speeds conduction to the nodes of Ranvier. The nodes of Ranvier are interruptions in the myelin, allowing current regeneration. High concentrations of Na+ channels are located at the nodes of Ranvier.

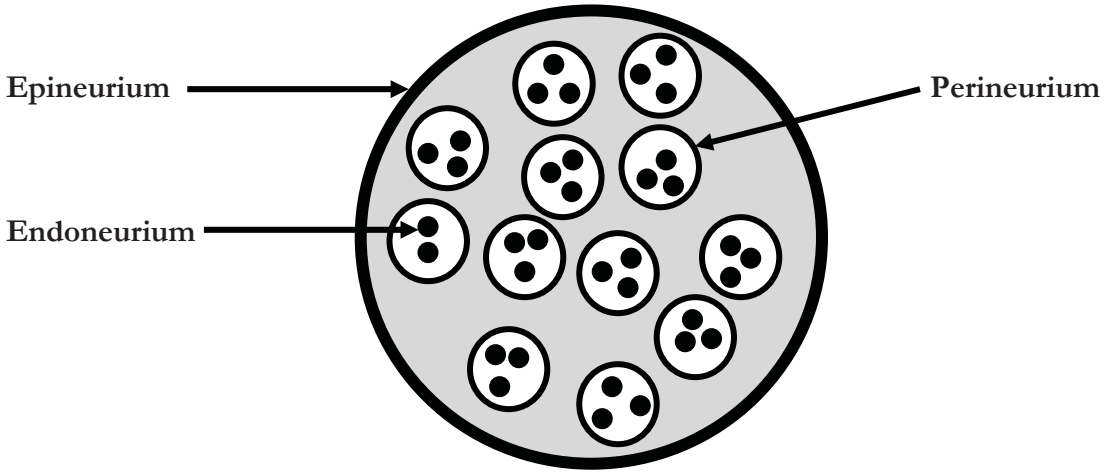


Non-myelinated fibers have Na+ channels distributed along the axon.



A peripheral nerve contains several axon bundles called fascicles.
Endoneurium is the connective tissue that covers an individual nerve.
Perineurium is the connective tissue that covers each fascicle.
Epineurium is the connective tissue covering the entire nerve.

Schematic Illustration of Nerve



Nerve Conduction Physiology

The neural membrane contains a voltage difference of +60 mV (inner) to -90 mV (outer). At rest the neural membrane is impermeable to Na+ ions, and selectively permeable to K+ ions. The Na+/K+ pump maintains the ion gradient. The K+ to Na+ gradient is constant at 30:1. Within the cell, the concentration of K+ is kept at 30, and outside the cell it is maintained at 1. Sodium, on the other hand, is at higher concentrations outside the cell.

At Rest

Outside Cell

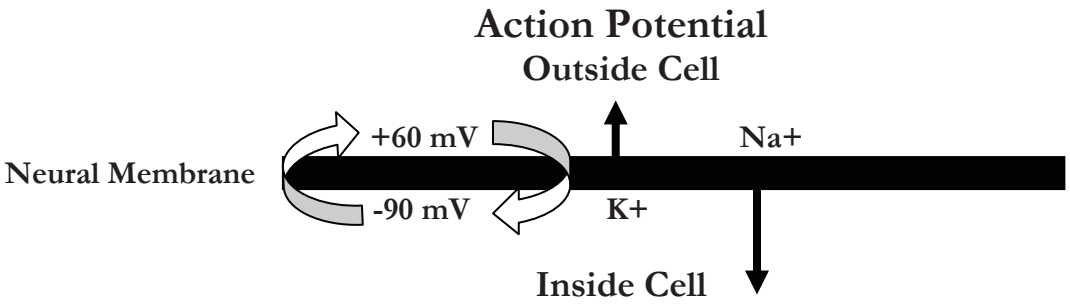
Neural Membrane

-90 mV **K+ concentration low; Na+ concentration high**

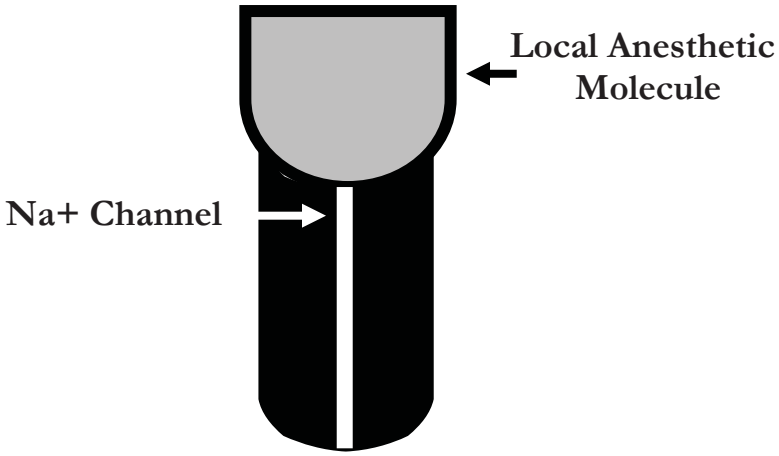
+ 60 mV **K+ concentration high; Na+ concentration low**

Inside Cell

During an action potential, the nerve membrane switches its permeability from K+ to Na+, changing the membrane potential from -90 to +60 mV (negative to positive) and back again.

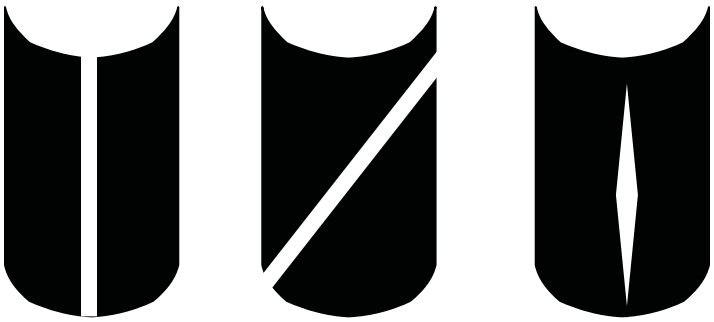


Local anesthetics produce a conduction block of neural impulses, preventing the passage of Na+ through Na+ channels.



Local anesthetics DO NOT alter the resting membrane potential. The Na+ channel acts as a receptor for local anesthetic molecules. Local anesthetics are stereospecific. Their action is dependant on the conformational state of the Na+ channel. Local anesthetics bind more readily to the Na+ channel during depolarization. This may occur during the "open" and "inactivated" state. Local anesthetics may bind during the resting state but not as readily as during the "open" or "inactivated" state.

Conformational State of Na+ Channel



Open (activated) **Inactivated** **Closed** (resting)

In the "inactivated" state, local anesthetics stabilize the Na+ channel. Local anesthetic molecules may bind within the Na+ channel as well as block the external opening. This action prevents permeability to Na+, slowing the rate of depolarization. The rate of depolarization is slowed; threshold potential is not met or propagated along the nerve membrane. Repeated depolarization increases the number of local anesthetic molecules bound to Na+ channels by increasing the number of available binding sites. Local anesthetic disassociation from inactivated channels occurs at a slower rate compared to resting channels.

Fiber Types

There are several classifications of nerve fibers. The classification of a nerve fiber impacts its sensitivity to local anesthetics. The order of susceptibility to blockade by fiber type is as follows: (least susceptible to most susceptible) small myelinated fibers (Aα motor) < Aα type Ia; Aα type Ib; Aβ type II; Aγ < Aδ sensory fibers < small, non-myelinated C fibers, and partially myelinated B fibers.

Fiber Type	Function	Diameter (mm)	Speed of Conduction	Local Anesthetic Sensitivity*	Myelination
Aα	Motor	12-20	Fast	1	Yes
Aα	Proprioception	12-20	Fast	2	Yes
Aα	Proprioception	12-30	Fast	2	Yes
Aβ	Touch Pressure/Proprioception	5-12	Medium	2	Yes
Aγ	Motor	3-6	Medium-Slow	2	Yes
Aδ	Pain Cold Temperature Touch	2-5	Medium-Slow	3	Yes
B	Preganglionic autonomic fibers	<3	Medium-Slow	4	Some
C (dorsal root)	Pain Warm and Cold Touch	0.4-1.2	Slow	4	No
C (sympathetic)	Postganglionic sympathetic fibers	0.3-1.3	Slow	4	No

(* local anesthetic sensitivity= 1 is the least sensitive and 4 is most sensitive)

Summary of Impulse Blockade by Local Anesthetics

1. Local anesthetic is deposited near a nerve. A portion of the local anesthetic is removed due to tissue binding and circulation. If the local anesthetic is an ester, a portion of the deposited local anesthetic will be removed by local hydrolysis in addition to tissue binding and circulation. The remaining local anesthetic penetrates the nerve sheath.
2. Local anesthetic penetrates the axon membranes and axoplasm. This step is dependant on pKa and lipophilicity.
3. Local anesthetic binds to Na+ channels preventing their opening by inhibiting conformational changes resulting in activation. Local anesthetics may also bind to the channel pore and block the passage of Na+.
4. During onset, impulse blockade is incomplete. Partially blocked fibers are inhibited by repetitive stimulation. The reverse is true during recovery.
5. The primary route for local anesthetics is the hydrophobic route, within the axon membrane.
6. Onset is due to the slow diffusion of local anesthetic molecules into the nerve, NOT by binding to ions, which occurs at a faster rate. Recovery occurs in reverse.

Pharmacokinetics

Pharmacokinetics involves the medication/body interaction. It is how the body handles medication. Principles of pharmacokinetics include: absorption, distribution, metabolism, and elimination. The local anesthetic blood concentration is determined by:

- Amount of local anesthetic injected
- Absorption rate
- Site of injection
- Rate of tissue distribution
- Rate of biotransformation
- Excretion rate

Patient related factors include:

- Age
- Cardiovascular status
- Hepatic function

Systemic Absorption of Local Anesthetics

Systemic absorption of local anesthetics is determined by:

- Site of injection
- Dose and volume
- Addition of vasoconstrictor
- Pharmacologic profile of the local anesthetic

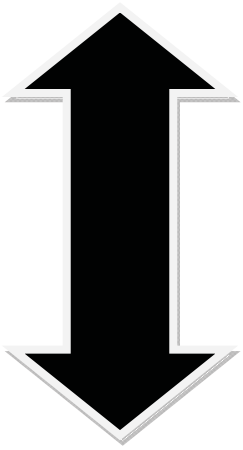
1. Site of Injection

Site of injection impacts blood levels of local anesthetic. Areas of high vascularity result in greater uptake and higher blood concentrations. The uptake of local anesthetic from greatest to least is as follows:

IV> tracheal> intercostal> caudal> paracervical> epidural> brachial> sciatic> subcutaneous

Uptake of Local Anesthetics Based on Regional Anesthetic Technique

Result in Highest Blood Concentrations

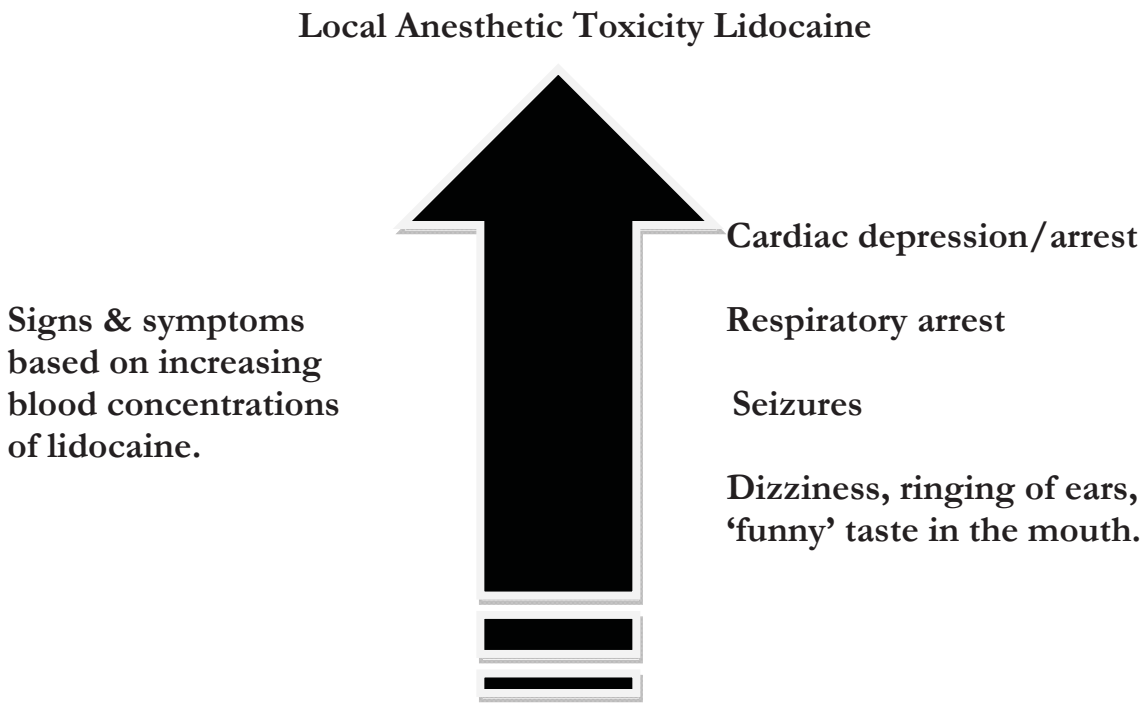


Intravenous
Tracheal
Intercostal
Caudal
Paracervical
Epidural
Brachial
Sciatic
Subcutaneous

Lower Blood Concentrations of Local Anesthetic

Clinically, the site of injection plays an important role in toxicity. For example, 400 mg of plain lidocaine in the intercostal space may lead to peak blood concentrations of 7 mcg/ml. This may result in CNS toxicity. In contrast, 400 mg of plain lidocaine in the brachial plexus will yield blood levels of 3 mcg/ml, which is not toxic.

Toxicity associated signs and symptoms may vary among local anesthetics. For example, with lidocaine there is a larger disparity in blood concentrations required to cause CNS signs and symptoms compared to concentrations that result in cardiovascular collapse. With bupivacaine there is a small difference in the blood concentrations that may result in CNS signs and symptoms and concentrations that result in cardiovascular collapse. Often seizures occur at the same time as cardiovascular collapse. Ropivacaine is similar to bupivacaine with respect to onset and duration. However, blood concentrations of ropivacaine required to cause cardiovascular collapse are much higher than bupivacaine. Ropivacaine has a larger margin of safety. In addition, metabolism plays a role in toxicity. Amides have a high rate of first pass metabolism as the local anesthetic passes through the liver. Slow absorption from tissue is less likely to result in toxicity. Toxicity is often the result of intravenous/intra-arterial injection or overdose.



2. Dose and volume

Blood concentrations of local anesthetics correspond proportionally to the total dose. Higher blood concentrations are associated with large volumes of dilute local anesthetic compared to the same dose in a smaller volume.

3. Vasoconstrictor

Epinephrine, in concentrations of 5-10 mcg/ml, is commonly used to decrease the absorption of local anesthetics. A 5 mcg/ml (1:200,000) dose of epinephrine will significantly reduce the peak blood levels of lidocaine and mepivacaine. Epinephrine does not affect the vascular absorption of etidocaine and bupivacaine in the epidural space. However, the addition of epinephrine does significantly reduce the vascular absorption of etidocaine and bupivacaine when utilized for peripheral nerve blocks. Benefits of decreased absorption include increased neuronal uptake, enhanced quality of analgesia/anesthesia, prolonged duration of action, and decreased risk of toxicity.

A concentration of 1:200,000 (5 mcg/ml) is commonly used for peripheral nerve blocks to reduce vascular absorption. To add epinephrine to local anesthetic solutions use a 1mg/ml (1:1000) ampoule of epinephrine. Take the total volume of local anesthetic, divide it in half, and move the decimal point two places to the left. For example, 40 ml of 1% lidocaine, divide 40 by 2 and 20 is the result. Next, move the decimal point two places to the left. The result is 0.20. This is the amount of epinephrine added to the local anesthetic solution to yield a 1:200,000 concentration. To check the calculation, multiply 5 mcg/ml by 40 ml, which equals 200 mcg. It is important to always check the concentration of epinephrine and the total dose added to the local anesthetic.

A second technique for adding epinephrine to local anesthetic preparations is detailed below:

- 1:200,000 epinephrine concentration would equal 5 mcg/ml.
- Dilute epinephrine using a 10 ml syringe. Draw up 1 ml of 1:1000 epinephrine (1 mg per ml) and 9 ml of normal saline.
- Mix it by tilting the syringe back and forth.
- The concentration of epinephrine is now 100 mcg per ml.
- Add epinephrine to the local anesthetic solution (see table below).

1:200,000 Epinephrine Concentration	
Volume of Local Anesthetic	**Amount of Epinephrine Added to Local Anesthetic Solution**
20 ml	100 mcg of epinephrine
30 ml	150 mcg of epinephrine
40 ml	200 mcg of epinephrine
50 ml	250 mcg of epinephrine

- Always label the syringe of epinephrine. Once the epinephrine is added to the local anesthetic, discard what remains. Epinephrine can be lethal and should be discarded to avoid inadvertent administration.
- Epinephrine containing local anesthetics should never be injected into end organs such as ears, nose, penis, fingers, or toes. Epinephrine may cause vasoconstriction and subsequent necrosis of tissue.

4. Pharmacologic Profile

Individual local anesthetics exhibit different rates of absorption. For example, it has been found that during brachial plexus blockade, lidocaine is absorbed faster than prilocaine, and bupivacaine is absorbed more rapidly than etidocaine. In general, local anesthetics that are highly tissue bound are absorbed at a slower rate. In addition, absorption is dependant on the individual local anesthetics intrinsic ability to cause vasodilatation.

Distribution of Local Anesthetics

A two compartment model describes the systemic distribution of local anesthetics. The rapid disappearance phase (α phase) is related to uptake by rapidly equilibrating tissue (tissue with high vascular perfusion which include the brain, lung, liver, kidney, and heart). The slow phase of disappearance (β phase) is the function of the individual local anesthetics distribution to muscle tissue and the gut.

Local anesthetics are distributed to all tissues. Higher concentrations of local anesthetics are found in highly perfused organs compared to tissues that receive lower rates of perfusion. The pulmonary system is responsible for extraction of local anesthetics. As local anesthetics are transported through the pulmonary vasculature, levels are greatly reduced. The largest reservoir for local anesthetics is the skeletal muscle.

Biotransformation and Excretion of Local Anesthetics

The metabolism of local anesthetics is dependent upon their classification: ester vs. amide. Ester local anesthetics undergo extensive hydrolysis in the plasma by pseudocholinesterase enzymes (plasma cholinesterase or butyrylcholinesterase). Ester hydrolysis is rapid, resulting in water soluble metabolites which are excreted in the urine. The ester that is an exception is cocaine. In addition to ester hydrolysis cocaine is partially metabolized in the liver (N-methylation). Patients with pseudocholinesterase deficiency are at risk for toxicity (genetic or liver disease). This is due to slowed metabolism and accumulation of the ester local anesthetic. Procaine and benzocaine are metabolized to p-aminobenzoic acid (PABA), which has been associated with allergic reactions. Benzocaine may result in methemoglobinemia. When ester local anesthetics are placed in the CSF, metabolism does not occur until there has been vascular absorption of the local anesthetic. CSF does not contain esterase enzymes.

Amide local anesthetics are metabolized primarily by microsomal P-450 enzymes in the liver (N-dealkylation and hydroxylation) and, to a lesser extent, in other tissues. The rate of metabolism among amides varies.

prilocaine> lidocaine> mepivacaine> ropivacaine> bupivacaine.

Prilocaine metabolites include o-toluidine derivatives, which can accumulate after large doses (>10 mg/kg), resulting in the conversion of hemoglobin to methemoglobinemia. Treatment for methemoglobinemia includes the administration of methylene blue. Methylene blue is generally available in a 1% solution. 1-2 mg/kg should be administered over 5 minutes. Methylene blue reduces methemoglobin to hemoglobin.

The excretion of amide local anesthetics occurs in the kidneys. Less than 5% of the unchanged medication is excreted by the kidneys.

Patient Alterations in the Pharmacokinetics of Local Anesthetics

Age is one factor that alters the pharmacokinetics of local anesthetics. Changes in half life have been demonstrated for the elderly and newborns. In both populations, lidocaine has been found to have an increased half life. Newborns have an immature hepatic enzyme system, whereas the elderly have decreased hepatic blood flow. The second factor that affects the pharmacokinetics of local anesthetics includes any disease process (i.e. hepatitis) that diminishes hepatic blood flow or impairs the livers ability to produce enzymes. This may result in elevated levels of amide local anesthetics in these patients compared to patients with normal liver function.

Clinical Pharmacology

General Considerations

General considerations related to local anesthetics include the following:

- Anesthetic potency
- Onset of action
- Duration of action
- Differential sensory/motor blockade

Potency, onset, and duration were covered earlier under structure activity relationships. For completeness they will be briefly covered under clinical pharmacology.

Anesthetic Potency

The primary factor related to potency is the hydrophobicity (lipid solubility) of the local anesthetic. Local anesthetics penetrate the nerve membrane and bind to Na+ channels, which are hydrophobic. Additional factors include:

- Fiber size, type, and myelination
- Hydrogen ion balance
- Vasodilator/vasoconstrictor properties (affects the rate of vascular uptake)
- Frequency of nerve stimulation
- pH (acidic environment will antagonize the block)
- Electrolyte concentrations (hypokalemia and hypercalcemia antagonizes blockade).

Onset of Action

In the individual nerve, onset is related to the unique physiochemical property of the local anesthetic. Clinically, the onset of action is related to pKa, dose, and concentration.

- pKa – when pKa approximates the physiologic pH a higher concentration of non-ionized base is available, increasing onset of action.
- Dose- the higher the dose of local anesthetic administered, the faster the onset.
- Concentration- higher concentrations of local anesthetic will result in a more rapid onset.

Duration of Action

Duration of action is dependant on individual local anesthetic characteristics. Local anesthetics are classified as follows:

- Short acting: procaine and chloroprocaine
- Moderate acting: lidocaine, mepivacaine, prilocaine
- Long acting: tetracaine, bupivacaine, etidocaine, ropivacaine, levobupivacaine

Duration of action is influenced by peripheral vascular effects that local anesthetics exhibit. Local anesthetics exhibit a biphasic effect on vasculature smooth muscle. At low, sub-clinical doses, vasoconstriction is noted. With larger, clinically relevant doses, vasodilatation is seen. The degree of vasodilatation varies among individual local anesthetics. For example, lidocaine> mepivacaine> prilocaine. The effect of local anesthetics on vascular tone and regional blood flow is complex and dependant on the following:

- Concentration
- Time
- Type of vascular bed

Ropivacaine is unique among local anesthetics since it exhibits a vasoconstrictive effect at clinically relevant doses.

Differential Sensory/Motor Blockade

Local anesthetics have the ability to produce varying degrees of inhibition for sensory and motor activity. For example, bupivacaine and etidocaine are both potent, long acting local anesthetics. Bupivacaine exhibits a more potent sensory than motor block. Etidocaine exhibits an equally effective sensory and motor block. Ropivacaine, on the other hand, exhibits a potent sensory block similar to bupivacaine but motor blockade appears less intense.

Factors Affecting Local Anesthetic Activity Clinically

Dose

An increase in the dose of a local anesthetic will increase the likelihood of a successful block while decreasing the time to onset. An increase in the volume of local anesthetic will be beneficial in the anatomical spread of anesthesia.

Addition of Vasoconstrictors

Epinephrine is the most commonly used vasoconstrictor. The usual dose and concentration is 5 mcg/ml or 1:200:000. Norepinephrine and phenylephrine have been used as vasoconstrictors but do not exhibit properties that make them superior to epinephrine. Epinephrine acts to decrease vascular absorption, reduces blood concentration of local anesthetics, and decreases the risk of toxicity thus allowing more local anesthetic molecules to reach the nerve membrane. As more molecules reach the nerve membrane, there is an increase in the depth and duration of local anesthetic blockade. Epinephrine prolongs the duration of blockade for most short to moderate acting local anesthetics. The addition of epinephrine for neuraxial blockade has the added benefit of activating endogenous analgesic mechanisms through α-Adrenergic receptors. This may increase the intensity of analgesic action. The addition of vasoconstrictors is controversial. Some advocate that vasoconstrictors may result in nerve injury due to decreased blood flow. Epinephrine containing

local anesthetics should never be injected into end organs such as ears, nose, penis, fingers, or toes. Epinephrine may cause vasoconstriction and subsequent necrosis of tissue.

Site of Injection

The anatomical location of blockade influences onset and duration. Location affects the rate of diffusion, vascular absorption, and the amount of local anesthetic administered. Subarachnoid blockade exhibits the most rapid onset and shortest duration of action. Rapid onset, within the subarachnoid space, occurs because nerve roots are not covered with a sheath. The short duration of action is related to the small dose and volume of local anesthetic used to produce anesthesia. Brachial plexus blockade, in contrast, has slower onset and longer duration of action. Local anesthetics are deposited in the sheath around the brachial plexus. Diffusion must take place before reaching the site of action. The long duration of action is related to slow vascular absorption, large doses, and increased exposure of neural tissue to local anesthetics.

Carbonation and pH adjustment

In the isolated nerve, adding sodium bicarbonate, or carbon dioxide, may accelerate the onset of action. The addition of bicarbonate increases the pH, which in turn, increases the amount of local anesthetic in the uncharged base form. This theoretically accelerates the rate of diffusion across the sheath and membrane, resulting in a faster onset. Controversy exists concerning the clinical utility of pH adjustment. Studies remain ambiguous concerning the use of sodium bicarbonate to improve the speed of local anesthetic induced anesthesia.

Mixtures of Local Anesthetics

Clinicians occasionally will combine local anesthetics to achieve quick onset and long duration. Often the clinician will combine a local anesthetic with a fast onset with a local anesthetic that has a long duration of action to achieve this goal. Clinical trials have yielded mixed results. Chloroprocaine and bupivacaine, in the brachial plexus region, have achieved a quick onset/prolonged duration. However, when used for epidural anesthesia it was found that the duration of action was shorter than if bupivacaine was administered alone. Clinically there are few advantages to this technique. It should be noted that when mixing local anesthetics the risk of toxicity remains. Care should be exercised not to exceed the maximum dose. Toxicities of local anesthetics are not independent, but additive! A solution containing 50% of the toxic dose of local anesthetic A, and 50% of the toxic dose of local anesthetic B, will have the same implications as 100% of the toxic dose of either local anesthetic alone.

Pregnancy

Hormonal changes during pregnancy are primarily responsible for the enhanced potency of local anesthetics. Mechanical factors such as dilated epidural veins, which decrease the volume of the epidural or subarachnoid space, may play a minor role in later stages of pregnancy. The spread and depth of epidural/spinal anesthesia is greater in the pregnant patient when compared to the patient

who is not pregnant. It has been found that the spread of local anesthetics are more extensive with epidural anesthesia as early as the first trimester. There is a correlation between progesterone levels and the mg per segment requirement of lidocaine required for the parturient. Based on current research, the dose of local anesthetics should be reduced for the pregnant patient by 30%, regardless of the trimester of pregnancy.

Medication Interactions with Local Anesthetics

Ester Local Anesthetics	Succinylcholine- may potentiate the effects since both are dependent on pseudocholinesterase for metabolism.
Ester Local Anesthetics	Cholinesterase inhibitors such as neostigmine and pyridostigmine can lead to a decrease in the metabolism of ester local anesthetics.
Ester Local Anesthetics	Decreased pseudocholinesterase activity during pregnancy and postpartum period.
Local Anesthetics in General	Opioids and alpha adrenergic agonists potentiate the analgesic effects of local anesthetics.
Local Anesthetics in General	Potentiate the effects of non-depolarizing muscle relaxant blockade.
Chloroprocaine (epidural)	May interfere with the analgesic effects of subarachnoid opioids.
Lidocaine	Cimetidine and propranolol decrease hepatic blood flow and lidocaine clearance. This acts to increase the risk of systemic toxicity.

Infiltration and Topical Local Anesthetics

Infiltration Anesthesia/Postoperative Analgesia

On occasion, anesthesia providers will place field blocks either as supplementation for a marginal regional anesthetic block or as the sole form of anesthesia. In addition, anesthesia providers should be knowledgeable about this form of anesthesia/analgesia for our surgeon and patient's sake. It is not uncommon to be asked by the surgeon, "How much can I inject?" As the 'expert' in local anesthetics, the anesthesia provider may be called upon to share their knowledge with surgical colleagues.

It is essential to know maximum local anesthetic dosages for plain and epinephrine containing local anesthetic solutions. Knowledge of the local anesthetic concentration/dose and the patients' weight will allow for rapid calculation of the maximum dose and volume of local anesthetic that can be safely administered. In addition to the maximum dose based on mg/kg, there is a total maximum dose regardless of weight. Avoidance of toxic dosages is essential.

Basic facts about infiltration:

- Almost any local anesthetic can be used for infiltration anesthesia.
- Onset is almost immediate for intradermal and subcutaneous administration.
- Epinephrine will prolong the duration of action of all local anesthetics, but is most pronounced with lidocaine.

- The conscious patient will experience some discomfort during infiltration due to the acidic nature of these solutions.
- Epinephrine containing local anesthetics should never be injected into end organs such as ears, nose, penis, fingers, or toes. Epinephrine may cause vasoconstriction and subsequent necrosis of tissue.

Commonly administered local anesthetics for infiltration

Plain local anesthetics (maximum doses based on 70 kg)

Local Anesthetic	Type	Concentration %	Max dose	Max dose mg/kg	Duration
Lidocaine	amide	0.5-1.0	300	4.5	30-60 minutes moderate duration
Mepivacaine	amide	0.5-1.0	300	4.5	45-90 minutes moderate duration
Bupivacaine	amide	0.25-0.5	175	2.5	120-240 minutes long duration
Ropivacaine	amide	0.1-1	200	3	120-360 minutes long duration

Local anesthetics with epinephrine (1:200,000) for infiltration

Local Anesthetic	Type	Concentration %	Max dose	Max dose mg/kg	Duration
Lidocaine	amide	0.5-1.0	500	7	120-360 minutes moderate duration
Mepivacaine	amide	0.5-1.0	500	7	120-360 minutes moderate duration
Bupivacaine	amide	0.25-0.5	225	3	180-420 minutes long duration

Topical Anesthesia

Several local anesthetics can be used for topical anesthesia. The most common local anesthetics include:

- Lidocaine
- Dibucaine
- Tetracaine
- Benzocaine
- EMLA (eutectic mixture of local anesthetic)

Topical local anesthetics provide effective, short term analgesia when applied to mucous membranes and abraded skin. Lidocaine and tetracaine sprays can be used for endotracheal anesthesia prior to

intubation. EMLA is a preparation used to provide cutaneous anesthesia through intact skin. EMLA is a mixture of 2.5% lidocaine and 2.5% prilocaine. The risk of methemoglobinemia is very rare. EMLA is effective in anesthetizing the skin in preparation for the placement of intravenous needles and skin grafting procedures. To be effective, EMLA must be placed under an occlusive dressing for 45-60 minutes.

Topical anesthesia is used in the emergency room for repairing lacerations. TAC is a mixture of 0.5% tetracaine, 1:200,000 epinephrine, and 10-11.8% cocaine. It is safe to use on skin, but should not be used on mucous membranes since rapid absorption may lead to toxicity. The maximum dose for adults is 3-4 ml. For the pediatric population, a dose of 0.05 ml/kg is considered safe. Concerns about cocaine toxicity, abuse, or diversion has led to the creation of an equally effective preparation, LET. LET is a preparation of lidocaine, epinephrine, and tetracaine. In the past ENT surgeons used cocaine for vasoconstriction and anesthesia; however this practice is rapidly being replaced by the use of oxymetazoline or phenylephrine in combination with a local anesthetic, such as 2-4% lidocaine. Dilute solutions should be used in children. Of concern to the anesthesia provider is the systemic absorption of phenylephrine, which can result in hypertension and reflex bradycardia. Oxymetazoline has a larger margin of safety and is absorbed less systemically.

Common Topical Preparations

Anesthetic	Concentration %	Form	Area of use
Benzocaine	1.5 20 20	Cream Ointment Aerosol	Skin and mucous membrane Skin and mucous membrane Skin and mucous membrane
Cocaine	4.0	solution	Ear, nose, throat
Dibucaine	0.25-1.0 0.25-1.0 0.25-1.0 0.25 2.5	Cream Ointment Aerosol Solution suppositories	Skin Skin Skin Ear Rectum
Lidocaine	2-4 2 2.5-5 2 10 10	Solution Jelly Ointment Viscous Suppositories aerosol	Oropharynx, trachea, nose Urethra Skin, mucous membranes Oropharynx Rectum Gingival mucosa
Tetracaine	0.5-1.0 0.5-1.0 0.25-1.0	Ointment Cream solution	Skin, rectum, mucous membranes Skin, rectum, mucous membranes Nose, tracheobronchial tree
EMLA	Lidocaine 2.5 Prilocaine 2.5	cream	Intact skin
TAC	Tetracaine 0.5 Epinephrine 1:200,000 Cocaine 11.8	solution	Cut skin
LET	Lidocaine 4 Epinephrine 1:200,000 Tetracaine 0.5	solution	Cut skin

Methemoglobinemia and Benzocaine

Benzocaine administration to the mucous membranes can result in the relatively uncommon but potentially fatal complication of methemoglobinemia. The anesthesia provider may encounter methemoglobinemia by assisting in airway management in another department, or in the OR when using this local anesthetic to anesthetize the upper airway.

Benzocaine made its debut into clinical use in 1900. It is used solely as a topical anesthetic. Benzocaine is the most commonly implicated local anesthetic associated with methemoglobinemia. The incidence of methemoglobinemia has been reported as high as 1 in 7,000 exposures. Up to 35% of topical benzocaine, when applied to mucous membranes, can be absorbed systemically. Inflamed areas of the mucous membranes absorb benzocaine at a higher rate. One of the problems with the administration of topical benzocaine sprays is estimating how much local anesthetic has been delivered. Application of topical benzocaine to mucous membranes should be limited to 1 second. Clinicians often fail to realize the significant absorption rate of benzocaine. In addition, clinicians may use multiple sprays or spray for longer than 1 second. In a review of benzocaine induced methemoglobinemia it was found that 46.4% of the cases reported had more than 1 spray of benzocaine.

Methemoglobinemia

Hemoglobin contains four heme groups (Fe+2) located on the surface of the molecule. Heme has the ability to reversibly bind with oxygen. Methemoglobin (MHb) is a form of hemoglobin that is unable to bind with oxygen. The ferrous irons (Fe+2) of the heme are oxidized to a ferric iron (Fe+3). The ferric heme is unable to bind with oxygen, resulting in a diminished ability to deliver oxygen to tissue.

Schematic diagram demonstrating the formation of methemoglobinemia

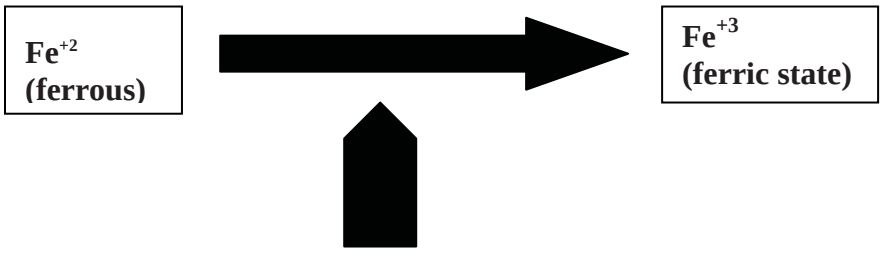


Illustration from Methemoglobinemia and Benzocaine. DD Moos, JD Cuddeford. Gastroenterology Nursing. 2007:30:5

Signs and Symptoms of Methemoglobinemia

Signs and symptoms are dependent on the levels of MHb. Patients with anemia and cardiopulmonary disorders may exhibit signs and symptoms earlier. When levels of MHb reach 10% or greater, the patient may appear cyanotic. MHb levels of 15% or greater may demonstrate: cyanosis, headache, weakness, dizziness, lethargy, and tachycardia. Levels between 10-20% are usually well tolerated. At levels of 45% or greater, signs and symptoms may include dyspnea, cyanosis, seizures, coma, dysrhythmias, and heart failure. At levels 70% or greater, mortality can occur.

Diagnosis

Methemoglobinemia should be considered in any patient who develops cyanosis after the use of topical pharyngeal anesthesia. Pulse oximetry readings will be inaccurate and not reflect the degree of hypoxia the patient is experiencing. Readings may range from 80-85% regardless of the severity of methemoglobinemia. A MHb level greater than 10% will result in an oximetry reading that is unreliable. Co-oximetry is able to differentiate between oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and MHb. The gold standard for confirming a diagnosis of methemoglobinemia is co-oximetry. This is available with most, but not all, ABG determinations. It is important to request co-oximetry when sending blood samples to the laboratory, if available.

Treatment

Patients who become cyanotic or hypoxic after the application of benzocaine should have supplemental oxygen placed. If their condition improves, then further evaluation for cardiopulmonary problems should be considered. If their condition does not improve and methemoglobinemia is suspected, then an arterial blood gas (ABG) with co-oximetry should be sent for evaluation. Methylene blue administration is not recommended until the presence of MHb is confirmed by co-oximetry, if available. Methylene blue, 1-2 mg/kg, is the treatment of choice for methemoglobinemia and should be administered over 5 minutes. Methylene blue accelerates the capacity of NADPH MHb reductase to reduce MHb. Reported side effects of methylene blue include: dizziness, confusion, restlessness, headache, abdominal pain, nausea and vomiting, dyspnea, hyper/hypotension, and diaphoresis. If the patient's condition improves after the administration of methylene blue, the patient should be monitored for the reoccurrence of symptoms. Methylene blue will not improve methemoglobinemia related to G-6-deficiency, NADPH methemoglobinemia, and cytochrome b5 reductase deficiency. Patients with a G-6-deficiency require transfusion or dialysis for treatment and methylene blue administration should be avoided. Patients with NADPH deficiency may acquire hemolytic anemia with the administration of methylene blue. For patients with no contraindications, repeated doses of methylene blue may be required. A second dose may be repeated in an hour. The total dose should not exceed 7 mg/kg since excessive methylene blue administration can result in methemoglobinemia. After initial treatment, the patient should be transferred to the intensive care unit for monitoring. Additional MHb levels should be measured at

hours 2 and 8 after the initial dose of methylene blue to monitor the patient for rebound methemoglobinemia.

Summary of Common Local Anesthetics

Local Anesthetic	Type	Onset of Action	Duration	Clinical Use
Procaine	Ester	Slow	Short	Spinal
Bupivacaine	Amide	Moderate	Long	Peripheral Nerve Blocks Infiltration Spinal Epidural
Ropivacaine	Amide	Moderate	Long	Peripheral Nerve Blocks Epidural
Chloroprocaine	Ester	Fast	Short	Peripheral Nerve Blocks Epidural
Etidocaine	Amide	Fast	Long	Peripheral Nerve Blocks Infiltration Epidural
Lidocaine	Amide	Fast	Moderate	Peripheral Nerve Blocks Infiltration Spinal Epidural Bier Block
Mepivacaine	Amide	Fast	Moderate	Peripheral Nerve Blocks Infiltration
Prilocaine	Amide	Fast	Moderate	Peripheral Nerve Blocks Infiltration Bier Block

Practical Application

A.) The surgeon wishes to use 1% plain lidocaine to infiltrate along the incision in a 4 kg pediatric patient. How much can he use?

- What about 0.5% plain lidocaine?
- What about 0.5% lidocaine with epinephrine?
- What about 0.5% plain bupivacaine?
- What about 0.5% bupivacaine with epinephrine?

B.) The surgeon wishes to use 1% plain lidocaine to infiltrate the wound in a 55 kg adult patient. How much can he inject?

- What about 0.5% plain lidocaine?
- What about 0.5% lidocaine with epinephrine?
- What about 0.5% plain bupivacaine?

- What about 0.5% bupivacaine with epinephrine?

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Introduction to Neuraxial Blockade

Chapter Two

Introduction to Neuraxial Blockade

Neuraxial blockade encompasses both spinal and epidural anesthesia. Neuraxial blockade offers several advantages to the patient when compared to general anesthesia. These include:

- Decreased incidence of nausea and vomiting
- Decreased blood loss
- Decreased incidence of graft occlusion
- Improved mobility following major knee surgery
- Superior pain control in the immediate postoperative period
- Decreased alteration in the patient's cardiopulmonary physiological status
- Improved patient satisfaction (especially in elderly)
- Less immunosuppression
- An alternative to general anesthesia for patients with a history of malignant hyperthermia
- An alternative for patient's that may not tolerate a general anesthetic
- Less cognitive impairment (especially in the elderly)
- Enhances flexibility/options for anesthetic care

Considerations

There are several factors that the anesthesia provider should consider when deciding on which anesthetic techniques to present to the patient. Examine the patients back for surgical scars, scoliosis, skin lesions, and surface anatomy that may make neuraxial blockade difficult. There are no routine preoperative tests for healthy patients undergoing neuraxial blockade. However, patients with a history of medications/medical conditions that may increase the risk of bleeding should have coagulation studies and platelet counts drawn. The patient should be assessed for thrombocytopenia prior to the initiation of neuraxial techniques. If your setting does not have the ability to perform coagulation studies/platelet counts the following signs and symptom may indicate bleeding tendencies:

- Blood in the urine
- Bleeding around the gums
- Petechiae (small purple colored spots on the skin)

In addition the patient should be carefully questioned:

- Do you bruise easily?
- Do you bleed easily?
- Do you have problems with forming a blood clot?

Generic Indications for Neuraxial Blockade

A careful review of the patient's history will yield valuable information, enabling the anesthesia provider to make an informed decision on the anesthetic technique. Neuraxial blockade may be a suitable option. Neuraxial blockade may be performed as the sole anesthetic (with or without sedation), combined with general anesthesia to decrease anesthetic requirements, or used for postoperative analgesia. Specific indications for epidural and spinal anesthetics will be covered under each technique. General considerations include the following:

- Suitability for the type of surgery being performed
- Surgeon's preference
- Experience in performing neuraxial blockade
- Physiological condition of the patient
- Is the patient mentally prepared to accept neuraxial blockade and temporary loss of motor/sensory function?
- No known contraindications to neuraxial blockade

When obtaining informed consent, include all the options and risks/benefits for each anesthetic technique (i.e. general v.s. neuraxial blockade). It is acceptable to present what may be the best choice to the patient. It is important to explain why, based on co-morbidities. The final decision is the patients. Most patients are quite accepting of the anesthesia providers' opinion, if presented in a manner that can be clearly understood. Never try to scare a patient into a neuraxial block. Be gentle and objective when presenting options. An explanation is often sufficient to help the patient make an informed decision.

Share with the patient specific complications/risks associated with neuraxial blockade. General risks include the following:

- Toxicity of local anesthetics (with epidural techniques)
- Transient or chronic paresthesia
- Nerve damage
- Intra-arterial injection, seizures, or cardiac arrest
- Block failure and the need to supplement or convert to general anesthesia

The acceptance of neuraxial blockade will provide the anesthesia provider with a cooperative patient which is essential to success. Carefully explain the procedure and what the patient should expect.

Contraindications for Neuraxial Blockade

Absolute Contraindications:

- Patient refusal
- Inability to guarantee sterility of medications/equipment

- Infection at the site of injection
- Coagulopathy (acquired, induced, genetic)
- Severe hypovolemia. Hypovolemia should be corrected prior to spinal anesthesia. A spinal anesthetic in a severely hypovolemic patient may lead to cardiac arrest.
- Increased intra-cranial pressure (i.e. brain tumor or recent head injury)
- Severe aortic stenosis
- Severe mitral stenosis
- Ischemic hypertrophic sub aortic stenosis
- Severe uncorrected anemia
- An allergy to local anesthetics. Ensure that it is a "true" allergy. Some patients may report symptoms such as dizziness, nausea, etc. during dental anesthesia. Ask the patient if they had trouble breathing, a rash, and other symptoms that would indicate a "true" allergy. If the patient had a true allergic reaction to a local anesthetic, identify which local anesthetic. Ester local anesthetics have a higher incidence of allergic reactions, related to their metabolism to PABA. Amide local anesthetics have a very low incidence of allergic reactions. There are no cross reactions between amides and esters. A true allergy is an absolute contraindication to a neuraxial blockade with the offending local anesthetic or others in the same class.

Relative Contraindications:

- Sepsis (may spread infection to subarachnoid/epidural space)
- Uncooperative patient (dementia, psychosis, emotional instability)
- Preexisting neurological deficits (hard to differentiate natural progression versus neurological trauma related to neuraxial blockade)
- Demyelinating lesions (i.e. multiple sclerosis may be exacerbated by the stress of surgery, temperature changes, or natural progression. However, it may be difficult to differentiate these potential causes from the use of spinal anesthesia.)
- Stenotic valvular heart lesions
- Severe spinal deformity

Controversial:

- Prior back surgery
- Inability to communicate with the patient
- Complicated surgeries that may involve a prolonged amount of time to perform, major blood loss, and maneuvers that may compromise respiration.

Neuraxial Blockade and Anticoagulation

Recent advances in pharmacology, the formulation and continued evolution of thromboembolism prophylaxis, and increased use of regional anesthesia have created the need for formalized guidance. The American Society of Regional Anesthesia and Pain Medicine (ASRA) have formulated guidelines to assist the anesthesia provider in caring for the patient on anticoagulants. For updates, the ASRA web site can be accessed at asra.com.

Current medications prescribed for thromboprophylaxis for total joint replacement include the following:

Unfractionated heparin

Low molecular weight heparin (LMWH)

- Ardeparin sodium (Normoflo®)
- Dalteparin sodium (Fragmin®)
- Danaparoid sodium (Orgaran®)
- Enoxaparin sodium (Lovenox®)
- Tinzaparin (Innohep®)

Warfarin sodium

Current medications prescribed for thromboprophylaxis for general surgery include the following:

Unfractionated heparin

LMWH

- Dalteparin sodium (Fragmin®)
- Enoxaparin sodium (Lovenox®)

Current medications prescribed for acute coronary syndrome and thrombembolism prophylaxis include the following:

- Enoxaparin sodium (Lovenox®)
- Dalteparin sodium (Fragmin®)
- Tinzaparin (Innohep®)

The major complication of anticoagulant therapy is bleeding. Bleeding can occur in the following anatomical areas: intraspinal, intracranial, intraocular, retroperitoneal, and mediastinal. This may result in hospitalization, transfusion, and death. Factors increasing the risk of bleeding while on anticoagulants include the following: intensity of anticoagulant effect, increased age, female gender, concomitant use of aspirin, history of gastrointestinal bleeding, and duration of anticoagulant treatment.

Spinal and Epidural Anesthesia and Hematoma Formation

Hematoma formation may be the result of a spontaneous bleed or trauma induced by a needle. The epidural space is at particular risk for bleeding due to the rich epidural venous plexus. The anatomy surrounding the spinal cord is relatively fixed. As a result, excessive bleeding into the epidural space may lead to compression, ischemia, nerve trauma, or paralysis. A bleed into the intrathecal space is generally less devastating, secondary to dilution by the cerebral spinal fluid.

In relation to epidural or spinal anesthesia, the risk of epidural hematoma formation is rare. The true incidence is unknown. It has been estimated that the incidence of epidural hematoma formation related to epidural anesthesia is between 1:150,000 and 1:190,000. The estimated incidence for spinal anesthesia is 1:220,000. There is a relationship between regional anesthesia and patients that receive anticoagulant medications during surgery. The incidence of epidural hematoma formation increases to 33:100,000 for epidural anesthesia and 1:100,000 for spinal anesthesia.

Risk Factors for the Development of Epidural/Spinal Hematoma

There are several risk factors for the development of an epidural/spinal hematoma related to the administration of spinal and epidural anesthesia. These factors include the following:

- Anatomic abnormalities of the spinal cord or vertebral column
- Vascular abnormalities
- Pathological or medication related alterations in homeostasis
- Alcohol abuse
- Chronic renal insufficiency
- Difficult and traumatic needle placement
- Epidural catheter removal

Signs and Symptoms of Epidural/Spinal Hematoma

The anesthesia provider should maintain a high index of suspicion when he/she encounters the following signs and symptoms following neuraxial anesthesia/analgesia administration:

- Low back pain (sharp and may radiate)
- Sensory and motor loss (numbness and tingling/motor weakness long after the block should have worn off)
- Bowel and bladder dysfunction
- Paraplegia

In the past, persistent low back pain was thought to be the classic symptom for epidural hematoma. Recent research indicates that the first symptoms may include sensory or motor loss, bowel and bladder dysfunction, numbness and tingling, prolonged motor weakness, and paraplegia.

Diagnostic Testing, Treatment and Outcomes

Diagnosis of an epidural hematoma is made by MRI (preferred), CT scan (may miss a small hematoma), and myelogram. Treatment is emergency decompressive laminectomy with hematoma evacuation. This must be done within 8-12 hours after the onset of signs and symptoms. The outcome is generally poor. There are three factors that affect the patient's recovery from this devastating complication: size and location of the hematoma, speed of development, and severity/nature of pre-existing neurological problems.

General Recommendations Related to the Perioperative Use of Anticoagulants

Specific recommendations will be reviewed for each classification of anticoagulant. The ASRA does provide some general guidelines concerning perioperative use of anticoagulants.

- Concurrent use of coagulation altering medications may increase the risk of bleeding without altering coagulation tests.
- When providing postoperative analgesia with an epidural catheter, the anesthesia provider should utilize opioids or dilute concentrations of local anesthetic to allow for neurological evaluation.
- Remove epidural catheters at the lowest point of anticoagulant activity. Do not administer additional doses of anticoagulant immediately after epidural catheter removal.
- In high risk cases, the patient should be monitored for neurological complications for 24 hours post epidural catheter removal.
- Frequent evaluation of neurological status of the patient should occur to aid in early detection of an epidural hematoma.

Anticoagulants

Common anticoagulants that may be encountered include the following:

- Antiplatelet medications
- Oral anticoagulants
- Standard heparin
- LMWH
- Thrombolytic and fibrinolytic therapy
- Herbal preparations
- New anticoagulants

Antiplatelet Medications

1. Aspirin:

Mechanism of action: blocks cyclooxygenase. Cyclooxygenase is responsible for the production of thromboxane A2, which induces platelet aggregation causing vasoconstriction.

Duration of action: irreversible effect on platelets. The effect will last for the life of the platelet (7-10 days). Long term use of large doses may lead to a decrease in prothrombin production lengthening the PT (prothrombin time).

2. NSAIDS:

Mechanism of action: inhibits cyclooxygenase by decreasing tissue prostaglandin synthesis.

Duration of action: reversible. Duration of action depends on half life of the medication and ranges from 1 hour to 3 days.

ASRA recommendations related to aspirin and NSAIDs: either medication alone should not increase the risk of epidural/spinal hematoma. However, dosages should be scrutinized and the duration of therapy should be taken into consideration. There are no laboratory tests that are accepted for preoperative testing. This includes bleeding time. A normal bleeding time does not necessarily indicate normal platelet function. On the other hand, an abnormal bleeding time does not necessarily indicate abnormal clotting function. Careful consideration should be given to other medications/conditions that may affect platelet function. Conditions that should increase the anesthesia providers' concern would include a history of bruising easily, history of excessive bleeding, female gender, and increased age.

3. Thienopyridine Derivatives- ticlopidine (Ticlid®) and clopidogrel (Plavix®):

Mechanism of action: Thienopyridine derivatives interfere with platelet membrane function by inhibition of adenosine diphosphate (ADP) induced platelet-fibrinogen binding.

Duration of action: Thienopyridine derivatives exert an irreversible effect on platelet function for the life of the platelet.

The ASRA recommends the discontinuation of ticlopidine 14 days prior to neuraxial blockade. Clopidogrel should be discontinued 7 days prior to neuraxial blockade. There are no accepted preoperative tests for these two medications.

4. Platelet GP IIb/IIIa inhibitors- abciximab (Reopro®), eptifibatide (Integrilin®) and tirofiban (Aggrastat®):

Mechanism of action: reversibly inhibits platelet aggregation by preventing the adhesion of ligands to glycoprotein IIb/IIIa, including plasminogen and von Willebrand factor.

Duration of action: time to normal platelet aggregation for abciximab is 24-48 hours. For eptifibatide and tirofiban normal platelet function should occur in 4-8 hours.

The ASRA recommends that neuraxial blockade should not be administered until there is normal platelet function. GP IIb/IIIa inhibitors are contraindicated within 4 weeks of surgery. If a GP IIb/IIIa inhibitor is administered postoperatively, after a spinal/epidural anesthetic, there should be careful neurological monitoring of the patient.

Oral Anticoagulants

1. Warfarin (Coumadin®)

Mechanism of action: inhibits vitamin K formation. Depletion of the vitamin K dependent proteins (prothrombin and factors VII, IX and X) occurs.

Duration of action: onset is 8-12 hours with a peak at 36-72 hours.
ASRA recommendations concerning patients using warfarin include the following:

- Evaluate patients for use of concurrent medications that affect clotting, in addition to warfarin.
- Warfarin should be stopped 4-5 days before surgery. A PT and INR should be measured prior to the initiation of neuraxial blockade.
- If the patient has received warfarin preoperatively; PT and INR should be measured if warfarin was administered more than 24 hours prior to surgery or a second dose has been administered.
- Patient's receiving postoperative low dose warfarin and epidural analgesia should have PT/INR monitored daily. Epidural catheters should be removed only when INR is < 1.5. Neurological testing should be performed routinely during epidural analgesia and continued for 24 hours after catheter removal if the INR is >1.5. In patients with an INR of >3.0 with an indwelling epidural catheter, the dose of warfarin should be held.

2. Standard Heparin

Mechanism of action: binds with antithrombin III, neutralizing the activated factors of X, XII, XI and IX.

Duration of action: for IV heparin the elimination half life is 56 minutes.

For patients receiving heparin, the ASRA has the following recommendations:

- No contraindication to use of neuraxial blocks in patients receiving mini-dose, subcutaneous heparin. The administration of subcutaneous heparin should be held until after block administration. Patient should be screened for concurrent medications that may impact clotting.

- Patients on heparin for more than 4 days should have a platelet count checked prior to the administration of neuraxial blockade secondary to the risk of heparin induced thrombocytopenia.

Precautions for vascular surgery and heparin use are as follows:

- Do not use neuraxial techniques in patients with coagulopathies.
- Heparin administration should be delayed for 1 hour after neuraxial access.
- Indwelling catheters should be removed 2-4 hours after the last dose and reevaluation of coagulation status. Heparin should not be reinitiated until at least 1 hour has passed.
- Patients receiving postoperative analgesia with local anesthetics should be monitored for hematoma formation.
- If a 'bloody tap' is encountered communicate with the surgeon. No data currently supports mandatory cancellation of the surgical case.

3. LMWH- ardeparin (Normiflo®), dalteparin (Fragmin®), enoxaparin (Lovenox®), tinzaparin (Innohep®), danaparoid (Organran®):

Variables associated with hematoma formation in patients receiving neuraxial anesthetics and LMWH include: female gender, the elderly, traumatic needle/catheter placement, and an indwelling catheter present during LMWH administration. The risk of epidural hematoma in patients on LMWH has been estimated to be 1:3000 for continuous epidural anesthesia and 1:40,000 for spinal anesthesia.

Mechanism of action: derived from standard heparin but the fragments are 1/3rd the size of heparin molecules. LMWH affects factor X. It does not alter the patient's PTT. Currently there are no laboratory measures of its action.

General ASRA recommendations:

- Assess the patient for concurrent medications that may alter coagulation.
- "Bloody tap" does not necessitate the cancellation of surgery. Communicate with the surgeon. LMWH initiation should be delayed for 24 hours.

Preoperative LMWH considerations:

- LMWH should be held for 10-12 hours prior to neuraxial blockade for normal dosing.
- Careful consideration should be given to total dosing and timing of LMWH. A delay of 24 hours prior to the initiation of neuraxial blockade should occur in the following dosing regimens: enoxaparin 1 mg/kg every 12 hours or 1.5 mg/kg every 24 hours; dalteparin 120 U/kg every 12 hours or 200 U/kg every 24 hours; tinzaparin 175 U/kg every 24 hours.

Postoperative LMWH considerations:

- Twice daily dosing- the first dose should not be administered until after 24 hours postoperatively. Indwelling catheters should be removed prior to the initiation of LMWH. If a continuous technique is used, then the catheter should be removed the next day with the first dose of LMWH occurring at a minimum of 2 hours after catheter removal.
- Single daily dosing- the first dose may be given 6-8 hours postoperatively. The second dose should occur at least 24 hours after the first dose. An indwelling epidural catheter should be removed 10-12 hours after the last dose of LMWH. Additional doses of LMWH should not occur for at least 2 hours after catheter removal.

Thrombolytic and Fibrinolytic Medications

Original recommendations related to the use of these medications were to avoid therapy if neuraxial puncture occurred in the last 10 days. The patient should be queried as to whether they received these medications recently. There is no data that details the length of time that neuraxial puncture should be withheld. If a patient has received a neuraxial block and fibrinolytic/thrombolytic therapy is unexpectedly initiated in the postoperative patient, the patient should be monitored closely for neurological complications. There are no recommendations for the removal of indwelling catheters in patients who unexpectedly receive thrombolytic/fibrinolytic therapy.

Herbal Preparations

Of concern to the anesthesia provider is the side effect of bleeding in the patient who consumes herbal preparations.

Mechanism of action: varies with the preparation.

- Garlic, ginger, feverfew: inhibit platelet aggregation
- Ginseng: antiplatelet components
- Alfalfa, chamomile, horse chestnut, ginseng: contain a coumadin component
- Vitamin E: reduces platelet thromboxane production
- Ginko: inhibits platelet activating factor

The risk for epidural/spinal hematoma is unknown. Surgical patients should be advised to stop herbal products 5-7 days before surgery. One of the crucial aspects of preoperative assessment is the concomitant use medications that alter coagulation. In addition, the patient should be screened for bleeding tendencies.

New Anticoagulants

New medications are continually being developed. New thrombin inhibitors such as bivalirudin and lepirudin have no specific recommendations. Caution should be maintained. Careful assessment of confounding medications, patient history, and risk and benefits ratio should be assessed.

Fondaparinux (Arixtra®) is an antithrombotic medication used for DVT prophylaxis. It binds with antithrombin III and neutralizes factor Xa. Peak effect is in 3 hours and half life is 17-21 hours. Its effects are irreversible. Extreme caution should be used with this medication until further clinical experience can help guide the anesthesia provider in the timing of neuraxial blockade. A black box warning similar to that of LMWH is included.

Bivalirudin and lepirudin are two new thrombin inhibitors. Bivalirudin is used in interventional cardiology and lepirudin is used to treat heparin-induced thrombocytopenia. There are no current recommendations for neuraxial blockade.

Anticoagulation and Peripheral Nerve Blocks/Plexus Blocks

It has been recommended that the ASRA guidelines to neuraxial blockade be applied to plexus and peripheral nerve blocks. This appears to be rather restrictive. Good clinical judgment should guide the anesthesia provider's decision making. The most serious complication of non-neuraxial regional techniques in anticoagulated patients is hemorrhage. Case reports highlight major bleeding occurring with psoas compartment and/or lumbar sympathetic blocks. In patients with neurological deficits, it has been found that complete recovery occurred in 6-12 months. The key to the reversal of neurological deficits is the fact that the bleeding occurs in an expandable site as opposed to bleeding associated with neuraxial blockade.

Current Recommendations for Spinal/Epidural Anesthesia and Anticoagulants

Classification	Medications	Recommendations	Laboratory
Antiplatelet's	Aspirin/NSAIDS Ticlopidine Clopidogrel Abciximab Eptifibatide Tirofiban	None DC 14 days before DC 7 days before Avoid	None None None None
Anticoagulants	Warfarin	DC 4-5 days before Monitor patient for 24 hours post spinal, epidural or removal of catheter	PT/INR prior to needle placement or catheter removal; INR <1.5
Heparin	Subq heparin IV heparin	Delay until after block Delay until 1 hour after block; remove catheter 2-4 hours after last dose.	>4 days check plt count Measure PTT
LMWH	Ardeparin Dalteparin Enoxaparin Tinzaparin Danaparoid	*Preop: block 10-12 hrs after last dose; high dose delay 24 hrs. (enoxaparin) *Postop: Twice daily dose delay 1st dose for 24 hrs; 2 hr delay after catheter removal. Once daily dose 1st dose 6-8 hrs post op; remove catheter 10-12 hr after last dose and wait 2 hrs till next dose. (enoxaparin)	None
Herbal Preparations	Garlic Ginkgo Ginseng Ginger Feverfew Vitamin E	DC 5-7 days before surgery	None
New Anticoagulants	Bivalirudin Lepirudin Fondaparinux	Unknown; assess risk Extreme caution; atraumatic needle placement; no catheters	None None

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Neuraxial Blockade Anatomy, Landmarks, Physiologic Effects, & Complications

Chapter Three
Neuraxial Blockade Anatomy, Landmarks, Physiologic Effects, & Complications

Knowledge of anatomy and landmarks are essential to the safe administration of neuraxial blockade. The anesthesia provider should know intimately what structures they will be encountering/traversing with their needle before attempting neuraxial techniques. Landmarks are important in the identification of appropriate area's to insert the needle. This valuable knowledge will allow the anesthesia provider to confidently administer spinal/epidural anesthesia, as well as enhance patient safety.

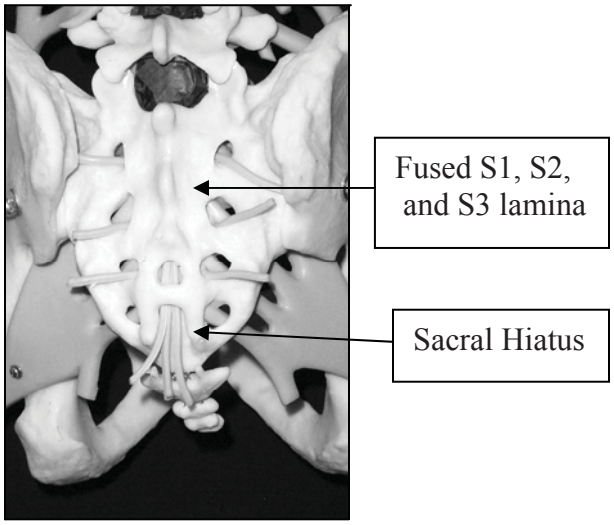
Anatomy

Vertebral Column

The bony vertebral column provides:

- structural support
- protection of the spinal cord and nerves
- mobility

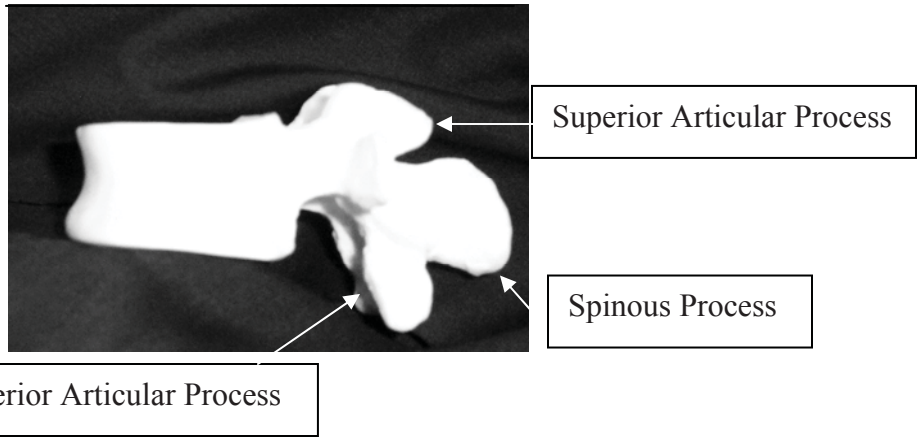
The vertebral column consists of 7 cervical, 12 thoracic, and 5 lumbar vertebrae as well as the sacrum, and coccyx. The first cervical vertebra is called the atlas. The atlas has a unique anatomical structure that allows for articulations to the base of the skull and second cervical vertebrae. The second cervical vertebra is called the axis. Each of the 12 thoracic vertebrae articulates with a corresponding rib.



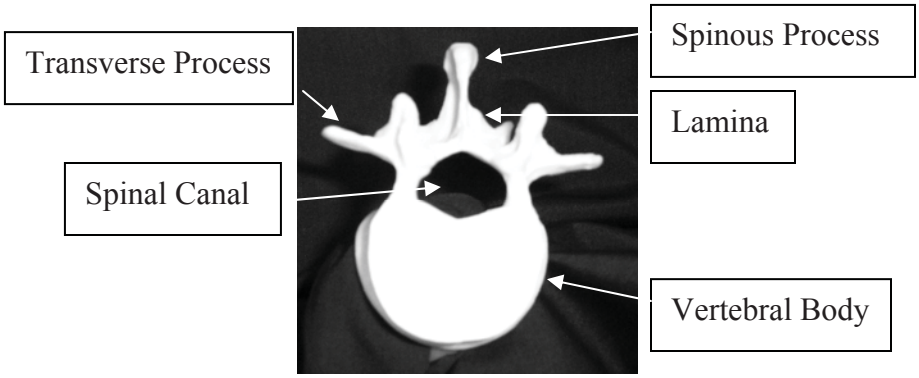
Sacral vertebrae are fused into one bone but retain individual intervertebral foramina. The lamina of a portion of S4 and S5 usually does not fuse, forming the sacral hiatus.

Individual vertebral anatomy consists of the pedicle, transverse process, superior and inferior articular processes, and spinous process. Each vertebral body is connected to the other by intervertebral disks. There are 2 superior and 2 inferior articular processes (synovial joints) on each vertebrae. Articular processes allow for articulation to the vertebrae above and below. Pedicles have notches superiorly and inferiorly, allowing the spinal nerve to exit the vertebral column.

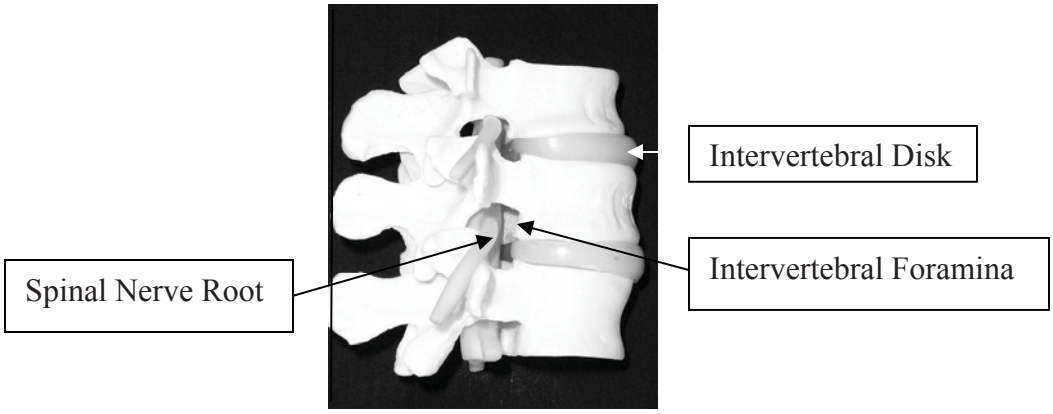
Side View of the Lumbar Vertebrae



Top View of Lumbar Vertebrae

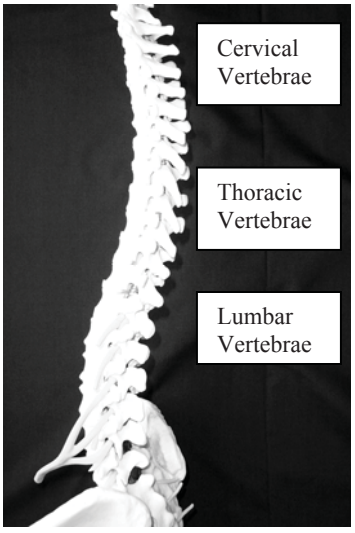


Side View of Lumbar Vertebrae



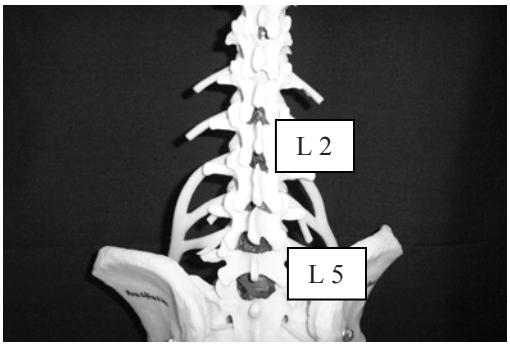
The bony spinal canal contains the following boundaries:

- Anterior boundary- vertebral body
- Lateral boundary- pedicles
- Posterior boundary- spinous process and laminae



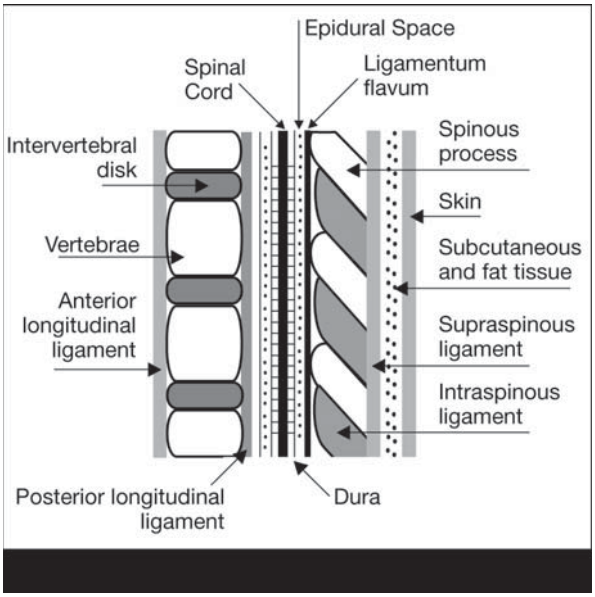
The angle of the transverse process in the lumbar and thoracic vertebrae impacts how the anesthesia provider will orientate the needle when performing an epidural. The spinous process in the lumbar region is almost horizontal with flexion; in the thoracic region they are angled in a slightly caudad direction.

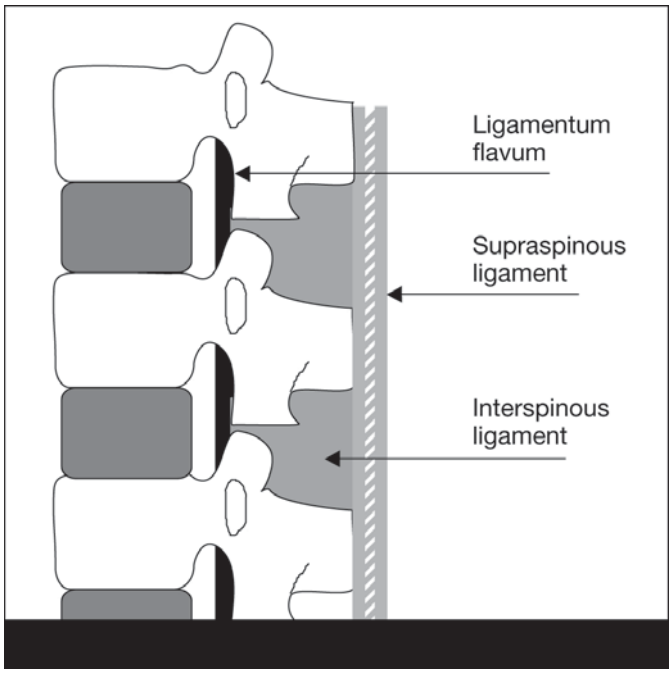
Interlaminar spaces differ in size. In general, the higher up the vertebral column, the smaller the interlaminar space. This is important to remember. If the anesthesia provider is having trouble placing a spinal/epidural at L2-L3, moving down a space will provide a larger intervertebral space, increasing success.



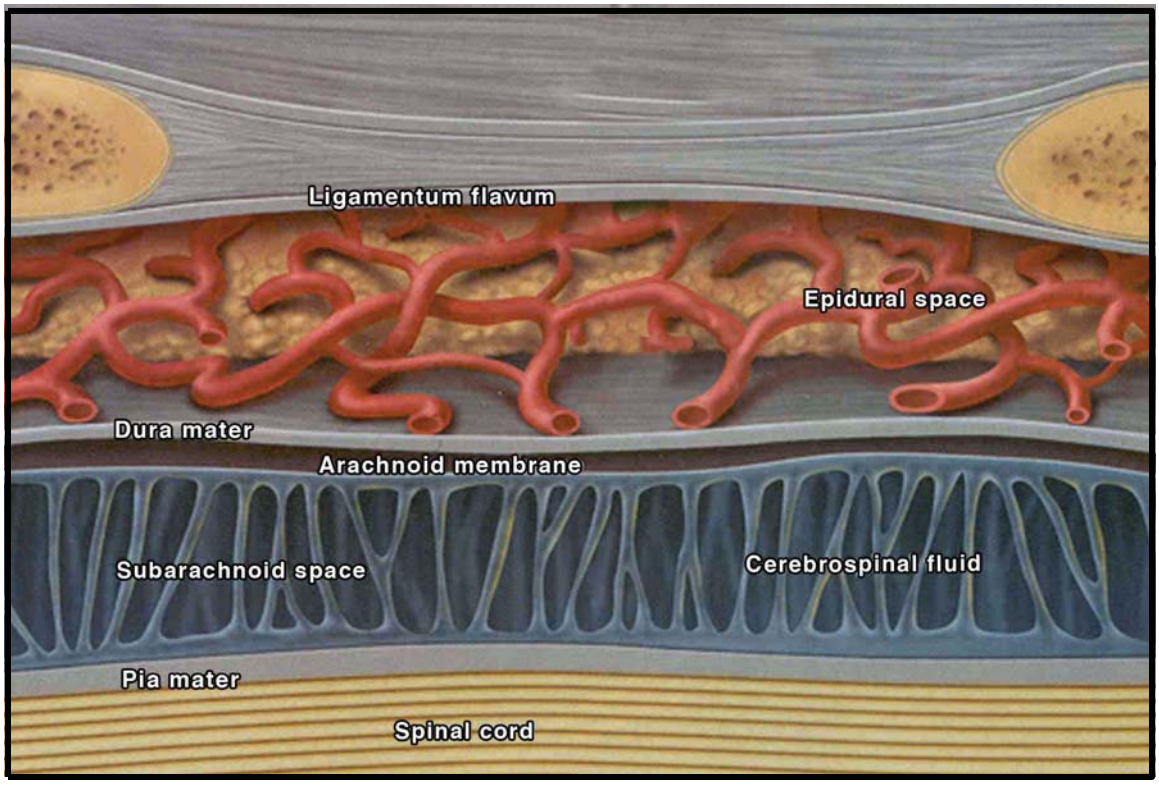
Supportive Structures

Ligaments maintain the shape of the vertebral column and provide support. Vertebral bodies and disks are connected and supported on the ventral side by anterior and posterior longitudinal ligaments.





On the dorsal side of the vertebral column the ligamentum flavum, interspinous ligament, and supraspinous ligament provide support. These dorsal ligaments are structures that the anesthesia provider will pass through when placing a needle for neuraxial blockade. With experience the anesthesia provider will be able to identify these structures through tactile feel.



Adapted with permission from "Unintended subdural injection: a complication of epidural anesthesia- a case report", AANA Journal, vol. 74, no. 3, 2006.

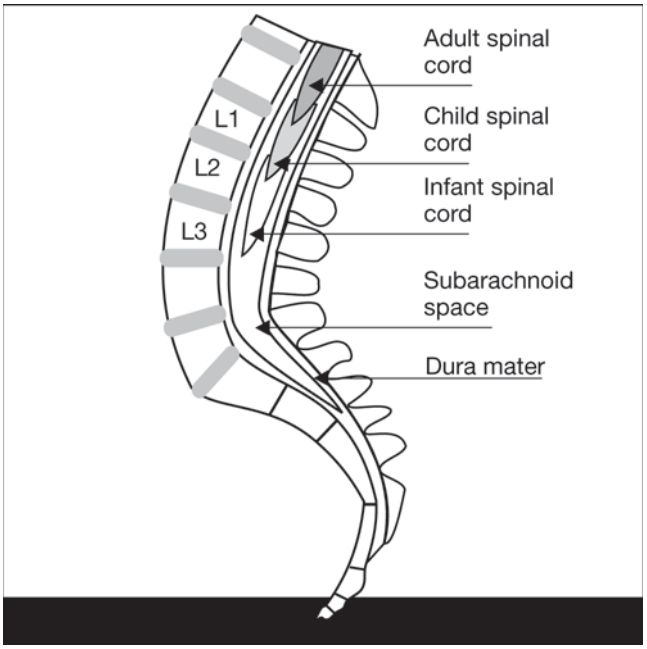
Blood Supply

The spinal cord and nerve roots access their blood supply from a single anterior spinal artery and paired posterior arteries. The vertebral artery at the base of the skull forms the anterior spinal artery and travels down the spinal cord supplying 2/3rds of the anterior spinal cord. Posterior spinal arteries are formed by the posterior inferior cerebellar arteries and travel down the dorsal surface of the spinal cord medial to the dorsal nerve roots. The two posterior spinal arteries supply 1/3rd of the posterior cord. Additional blood flow is supplied by anterior and posterior spinal arteries from intercostal and lumbar arteries. The artery of Adamkiewicz is a radicular artery arising from the aorta. It is a large, unilateral artery, generally found on the left side, providing blood supply to the lower anterior 2/3rds of the spinal cord. Injury to this structure can result in anterior spinal artery syndrome.

Subarachnoid Space

The subarachnoid space is a continuous space containing cerebral spinal fluid (CSF), spinal cord, and conus medullaris. It is in direct communication with the brainstem through the foramen magnum and ends with the conus medullaris at the sacral hiatus. The subarachnoid space extends from the cerebral ventricles down to S2. Asepsis and sterile technique are essential! Since the anesthesia provider places a needle directly into the subarachnoid space, infectious microbes can

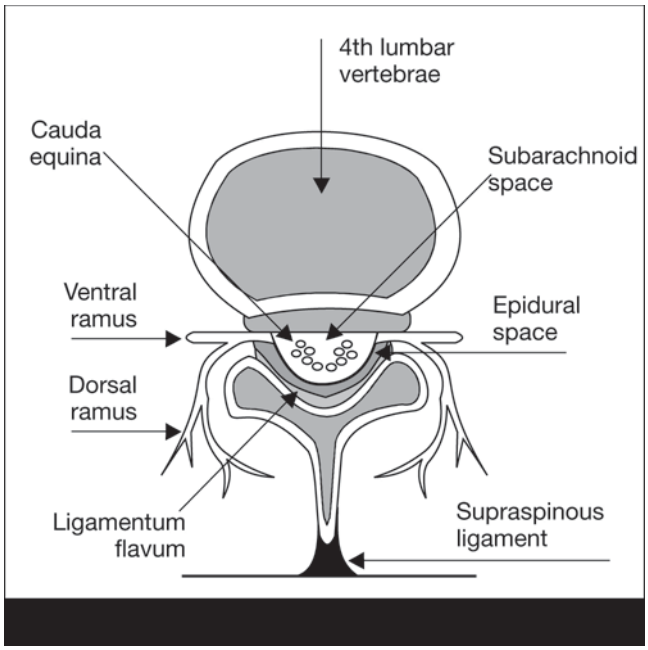
easily be introduced. If there is any doubt about potential or actual contamination, stop, then start again with fresh sterile supplies.



The spinal cord effectively ends at L1 in the adult and L3 in the infant. However, there are anatomical variations that influence the level that the spinal cord ends and the conus medullaris begins. In the adult, it is generally safe to place a spinal needle below L2, unless there is a known anatomical variation. Needle trauma to the cauda equina is unlikely. Individual nerves of the cauda equina are in a fluid environment and not likely to be pierced by a needle.

The anterior and posterior spinal nerve roots join each other and exit the intervertebral foramina, forming spinal nerves from C1-S5. At the level of the cervical vertebrae, the spinal nerves rise above the foramina, resulting in 8 cervical spinal nerves but only 7 cervical vertebrae. At T1 and below, each spinal nerve exits below the foramina. At L1 the spinal nerves form the cauda equina and course down the spinal canal until they exit their respective foramina. A dural sheath covers most nerve roots for a small distance after they exit the foramina.

Spinal nerve roots vary in size and structure from patient to patient. This may play a role in the quality of neuraxial blockade between patients when similar techniques are used. Dorsal roots are responsible for sensory blockade. They are larger than the anterior root that is responsible for motor blockade. Even though the dorsal root is larger, it is blocked more easily than the smaller anterior root. This is due to the organization of the dorsal root into bundles which expose a larger surface area to local anesthetic solutions. Thus, sensory nerves are blocked easier than motor.



CSF is a clear fluid that fills the subarachnoid space. The total volume of CSF in the adult varies between 100-150 ml. CSF volume within the subarachnoid space is approximately 25-35 ml and is continually produced at a rate of 450 ml per day. It is reabsorbed into the bloodstream through the arachnoid villi and granulations. The specific gravity of CSF ranges from 1.003-1.009, playing a crucial role in choosing the baracity of local anesthetic solution. CSF volume also plays a role in patient to patient variability in relation to block height and motor/sensory regression and accounts for 80% of patient variability. Body weight is the only patient measurement that coincides with CSF volume. This becomes important when administering neuraxial blockade to the obese and during pregnancy. In these patient populations the amount of CSF is usually less.

Three membranes surround the spinal cord within the vertebral column. Starting at the spinal cord and moving out they are:

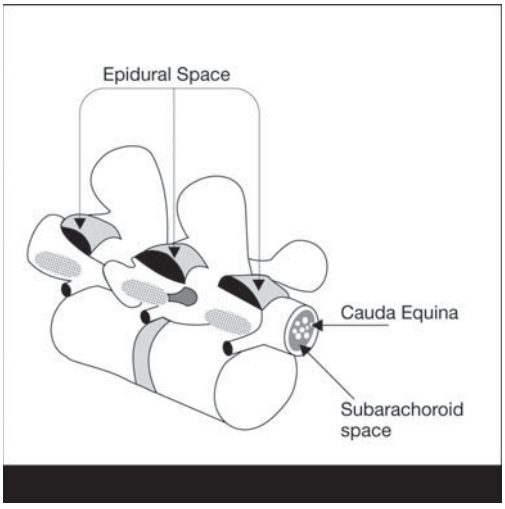
- pia mater
- arachnoid mater
- dura mater

Pia mater is highly vascular and covers the spinal cord and brain. The filum terminale is an extension of the pia mater, attaching to the periosteum of the coccyx. The arachnoid mater is non vascular and attached to the dura mater, functioning as the principal barrier to the migration of medications in and out of the CSF. Dura mater, the outermost membrane, is a fibrous and elastic membrane. It is an extension of cranial dura mater, extending from the foramen magnum to S2. The subdural space is a potential space found between dura and arachnoid mater. This space contains a small amount of serous fluid, which acts as a lubricant allowing the two surfaces to glide over each other during movement. Inadvertent injection of local anesthetics into this space may result in a failed spinal anesthetic or high neuraxial blockade after injection of an epidural anesthetic. Aspiration before injection may yield a small amount of serous fluid or be negative prior to the initiation of epidural anesthesia. With an epidural catheter present there is a risk of migration into this space.

Epidural Space

The epidural space extends from the foramen magnum to the sacral hiatus. It is segmented, not uniform in distribution. The epidural space surrounds the dura mater anteriorly, laterally, and posteriorly. Boundaries of the epidural space are as follows:

- anterior- posterior longitudinal ligaments
- lateral- pedicles and intervertebral foramina
- posterior- ligamentum flavum



The epidural space contains the following structures:

- fat
- nerve roots
- areolar tissue
- lymphatics
- blood vessels

As patients age, adipose tissue in the epidural space diminishes, and intervertebral foramina decrease in size. A decrease in adipose tissue results in decreased local anesthetic requirements in the elderly.

Posterior to the epidural space is the ligamentum flavum, which extends from the foramen magnum to the sacral hiatus. The ligamentum flavum is not one continuous ligament. It is composed of a right and left ligamenta flava which meet in the middle, forming an acute angle with a vertebral opening. The two ligamenta flava may or may not be fused in the middle at variable levels within the same patient. The ligamentum flavum varies in respect to thickness, distance to dura, skin to surface distance, and the size of the vertebral canal. The ligamentum flavum also varies in thickness from cephalad to caudad. It is thicker in the lumbar region compared to the thoracic region.

Site	Skin to ligament in cm	Thickness of ligament in mm
Thoracic		3.0-5.0
Lumbar	3.0-8.0	5.0-6.0

Anatomical structures located posterior to the ligamentum flavum are the:

- lamina and spinous processes
- interspinous ligament
- supraspinous ligament

The supraspinous ligament extends from the occipital protuberance to the coccyx, joining the vertebral spines together.

Unilateral Anesthesia and Epidurals

Varied anatomy of the epidural space may lead to a non uniform spread of local anesthetic solution, resulting in the uncommon but frustrating problem of unilateral anesthesia. Anatomical causes include a dorsomedian band in the midline of the epidural space, presence of epidural septa, or the presence of a midline posterior epidural fat pad.

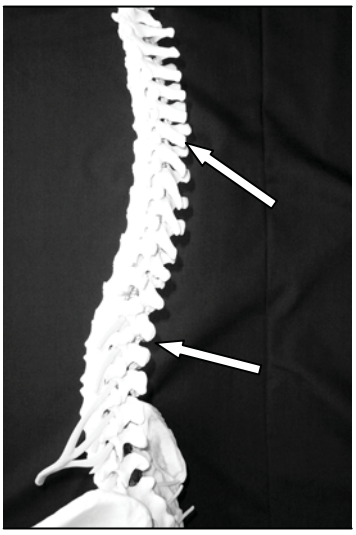
Surface Anatomy

Surface anatomy is important to help identify the correct area to place a neuraxial block.

When locating the midline the following should be noted:

- Spinal processes are generally palpable and define midline.
- If the anesthesia provider is unable to palpate the spinous process, identifying the gluteal crease may help identify midline. This will not be accurate if the patient has scoliosis or other deformities of the spine.

- Spinous processes in the cervical and lumbar areas are almost horizontal with flexion. Needle placement will be in a slightly cephalad direction. In the thoracic area the spinous processes are slanted in a caudad direction. With flexion, the anesthesia provider will need to direct the needle more cephalad.

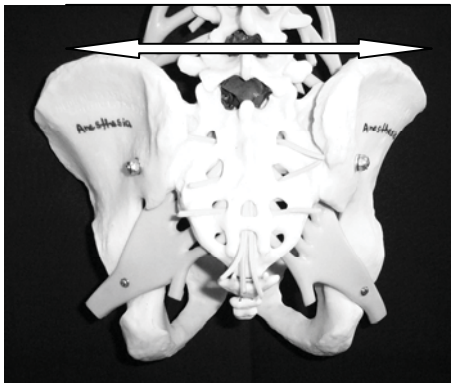


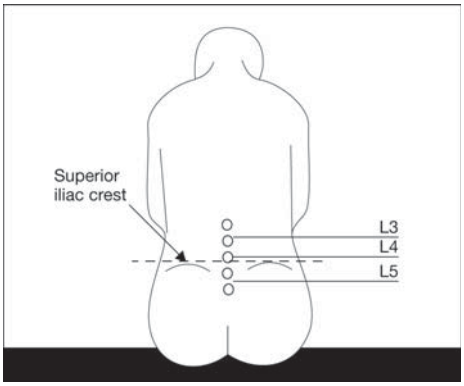
Anatomical landmarks can be identified by noting prominent vertebrae and landmarks:

- C2 is the first palpable spinous process
- C7 is the most prominent vertebrae
- The tip of the scapula, when the patients arms are at their side, corresponds with T7

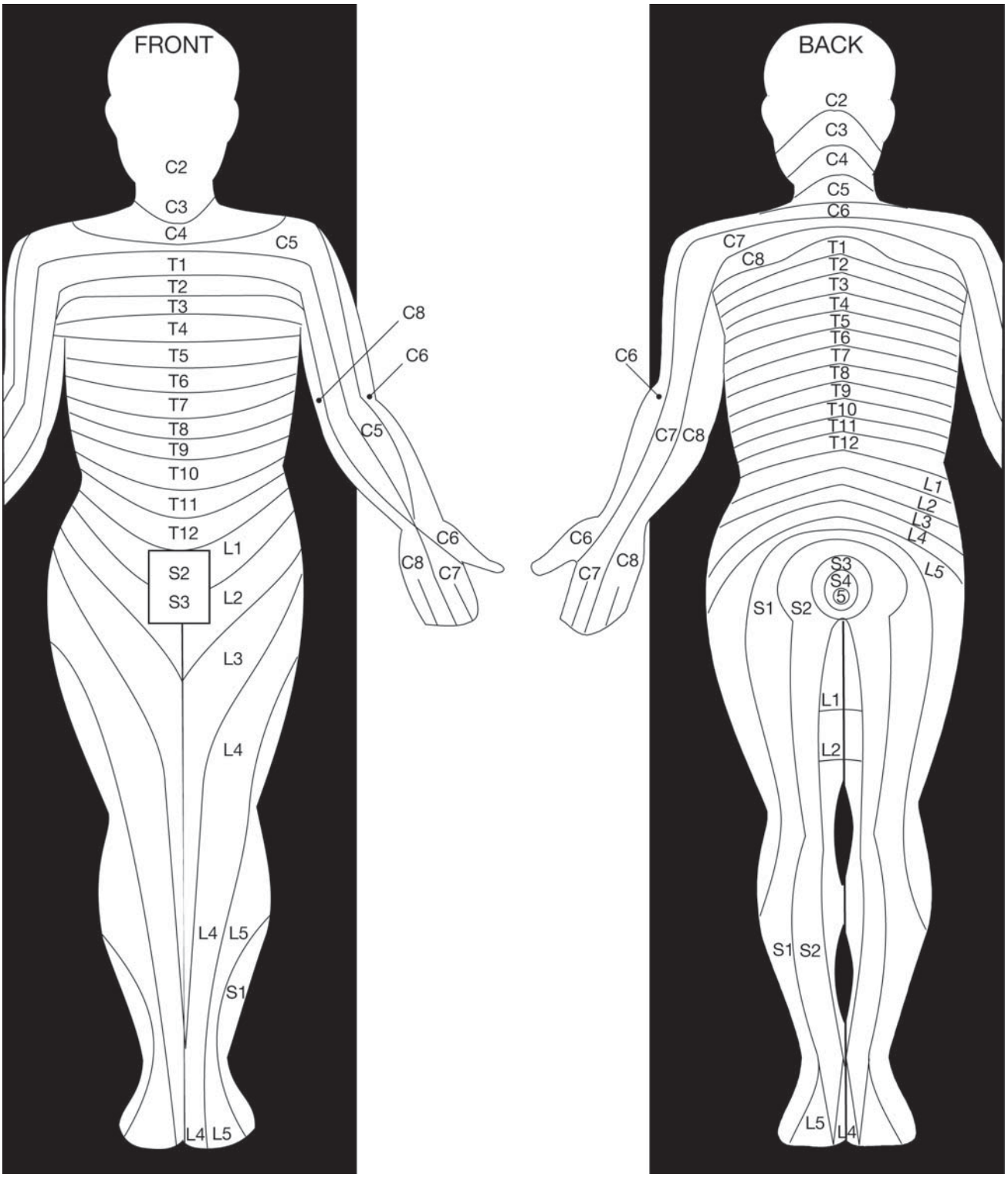
These landmarks are helpful in finding the "correct" level to place thoracic epidurals. It is helpful to count down and up to identify the correct level for placement.

A line drawn from the top of the iliac crest is known as "Tuffier's" line. This line generally crosses the body of L4 or the L4-L5 interspace. A line drawn across the posterior superior iliac spine will generally cross S2.





Dermatome Levels



This is the most common anatomical configuration. Variation may occur among patients.

Assessment of Neuraxial Blockade Level

Differential blockade occurs due to anatomy and the mechanism of action of local anesthetics. Local anesthetics injected into the subarachnoid/epidural space block transmission at spinal nerve roots. Blockade of nerve transmission is dependant on the concentration that reaches the site of action and the duration of contact. As local anesthetic spreads and distance increases, a smaller concentration of local anesthetic is available to reach nerve roots. Spinal nerve roots contain several nerve fiber types. In general, small myelinated fibers are more susceptible to blockade than larger unmyelinated fibers. With a neuraxial block there is a difference between sympathetic, sensory, and motor block level. The sympathetic level is generally two to six dermatome levels higher than the sensory level. The sensory level is approximately two dermatome levels higher than the motor level.

Knowledge of key dermatome levels assists the anesthesia provider in assessing the level of neuraxial blockade. An alcohol wipe is useful to assess the level of sympathectomy by measuring the patients' ability to perceive skin temperature sensation. A blunt needle is useful in the assessment of the sensory level. It should be sharp enough to cause a "pin prick" sensation but not so sharp as to break the patients skin. The use of the spinal needle stylet can be used. Pinching the patient can also be used. The table below will help determine if the level of blockade achieves the minimum level required for a proposed surgical procedure. When reviewing the required sensory levels, it seems odd that the sensory level is higher than where the surgical procedure actually takes place. For example, why is the level for lower extremity surgery with a tourniquet four levels higher than a surgical procedure without a tourniquet? Especially when the dermatome map indicates that sensation from the hip down entails the dermatome levels of L1-S1! The answer lies in the function of the afferent autonomic nerves. Afferent autonomic nerves innervate visceral sensations and viscerosomatic reflexes at spinal segments that are higher than the skin dermatome level of the proposed surgical intervention.

Operative Site	Level
Intraabdominal Procedures (other than lower abdominal)	T4
Lower Intraabdominal Procedures	T6
Lower extremities with a tourniquet Testicular and ovarian surgical procedures	T8
Hip surgery Vaginal or uterine surgical procedures Bladder and prostate surgical procedures	T10
Lower extremity surgery without a tourniquet	T12

The table below will help correlate surface anatomy, sensory dermatome levels, and anticipated systemic effects.

Surface Anatomical Area	Dermatome Level	Systemic Effects
Fifth finger (digit)	C8	Blockade of all cardioaccelerator fibers (T1-T4)
Inner aspect of arm and forearm	T1-T-2	Some degree of cardioaccelerator fiber blockade
Apex of axilla	T3	Possible cardioaccelerator fiber blockade
Nipple	T4-T5	Possible cardioaccelerator fiber blockade
Bottom of xiphoid process	T7	Possible splanchnic blockade (T5-L1)
Umbilicus	T10	Sympathetic nervous system blockade
Inguinal ligament area	T12	Sympathetic nervous system blockade is limited to the legs
Lateral foot	S1	

It is important to remember key surface anatomical levels to determine if neuraxial blockade is sufficient. This will allow time to administer general or alternative methods of anesthesia prior to skin incision.

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 mylelinated 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, mylelinated, 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 (10^{th} cranial nerve). The end result of neuraxial blockade is a

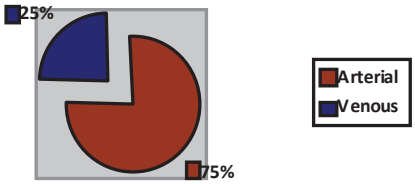
decreased sympathetic tone with an unopposed parasympathetic tone. This imbalance will result in many of the expected alterations of normal homeostasis noted with the administration of epidural and spinal anesthesia.

Cardiovascular Effects

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

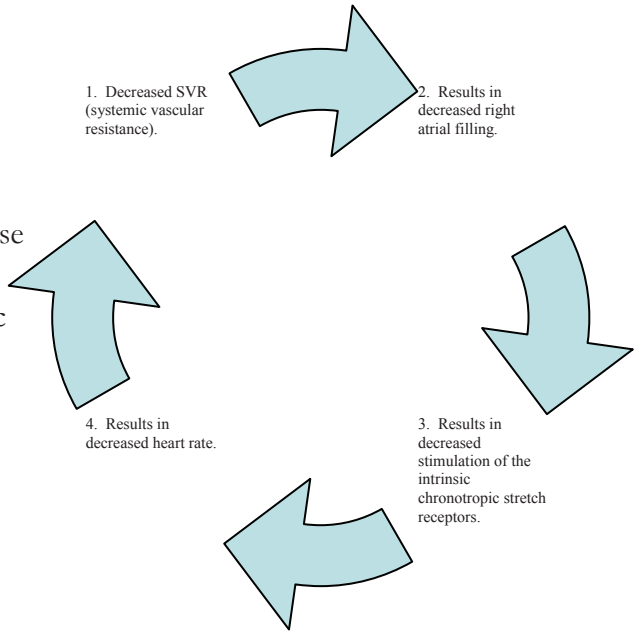
- Decrease in blood pressure
- Decrease in heart rate
- Decrease in cardiac contractility

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. Volume loading the patient with 10-20 ml/kg of fluid prior to 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. 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.

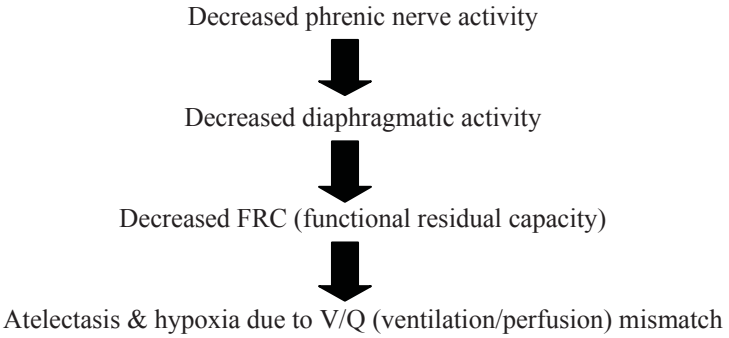
Respiratory Effects

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 block 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 predomination 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. 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. 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 prostrate 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.

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 loose 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. Local anesthetics vary in their potential to cause toxicity. The least to most toxic local anesthetic is as follows:

chloroprocaine< lidocaine< levobupivacaine < ropivacaine < bupivacaine

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.

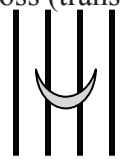
Symptoms include the following:

- 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 may last for weeks.

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. If using a cutting needle, place the bevel of the needle so that it is parallel to the longitudinal fibers. This will separate the fibers instead of cutting them.

Cutting across (transecting) fibers



Placing needle bevel parallel to longitudinal fibers

- Patients with the following characteristics are at increased risk:
    - ✓ Less than 50 years of age
    - ✓ Female
    - ✓ 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 help 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.

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

Common beverages and caffeine content.

- 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 90% of all postdural puncture headaches on the first treatment. A subsequent epidural blood patch will resolve symptoms in 90% 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 15-20 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. 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.
- 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 must occur within 8-12 hours of onset to avoid permanent injury.

Meningitis and Arachnoiditis

Meningitis is a rare complication. 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 identified.

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

Systemic Toxicity- has been covered earlier.

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. Local anesthetics associated with TNS include lidocaine, tetracaine, bupivacaine, mepivacaine, prilocaine, procaine, and ropivacaine. 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.

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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Spinal Anesthesia

Chapter Four

Spinal Anesthesia

Spinal anesthesia involves the use of small amounts of local anesthetic injected into the subarachnoid space to produce a reversible loss of sensation and motor function. The anesthesia provider places the needle below L2 in the adult patient to avoid trauma to the spinal cord. Spinal anesthesia provides excellent operating conditions for:

- ✓ surgical procedures below the umbilicus
- ✓ obstetric/gynecologic procedures of the uterus and perineum
- ✓ hernia repairs
- ✓ genitourinary procedures
- ✓ orthopedic procedures from the hip down.

In addition, it is an excellent technique to use in the elderly patient that may not tolerate a general anesthetic. It is important not to use a spinal anesthetic in patients who are hypovolemic or severely dehydrated. Patients receiving a spinal anesthetic should be preloaded with 1-1.5 liters of a crystalloid solution, such as ringers lactate, immediately prior to the block.

Advantages of Spinal Anesthesia

Several advantages of neuraxial blockade (including spinal anesthesia) were listed in the Introduction to Neuraxial Blockade section of this manual. There are additional advantages specific to spinal anesthesia.

- Easy to perform
- Reliable
- Provides excellent operating conditions for the surgeon
- Less costly than general anesthesia
- Normal gastrointestinal function returns faster with spinal anesthesia compared to general anesthesia
- Patient maintains a patent airway
- A decrease in pulmonary complications compared to general anesthesia
- Decreased incidence of deep vein thrombosis and pulmonary emboli formation compared to general anesthesia

Disadvantages of Spinal Anesthesia

Disadvantages include the following:

- Risk of failure even in skilled hands. Always be prepared to induce general anesthesia.

- Normal alteration in the patient's hemodynamics. It is essential to place the spinal block in the operating room, while monitoring the patient's ECG, blood pressure, and pulse oximetry. Resuscitation medications should be available.
- The operation could outlast the spinal anesthetic. Alternative plans (i.e. general anesthesia) should be prepared in advance.
- Risk of complications as outlined in the complications of neuraxial blockade chapter.

Contraindications

Please review Chapter 2 for contraindications.

Mechanism of Action

Local anesthetics administered in the subarachnoid space block sensory, autonomic, and motor impulses as the anterior and posterior nerve roots pass through the CSF. The site of action includes the spinal nerve roots and dorsal root ganglion.

Uptake & Elimination of Spinal Anesthetics

Four factors affect the uptake of local anesthetics in the subarachnoid space:

- Concentration of local anesthetic
- Surface area of neuronal tissue exposed
- Lipid content of the neuronal tissue
- Blood flow to the tissue

Local anesthetic concentration is highest at the site of injection. Spinal nerve roots lack an epineurium and are easily blocked. The surface area of the exposed nerves allow for absorption of the local anesthetic. As the local anesthetic travels away from the initial site of injection, its concentration decreases secondary to absorption into neural tissue and dilution by the CSF. Spinal cord tissue absorbs local anesthetics through the pia mater and the spaces of Virchow-Robin, which are extensions of the subarachnoid space. However, the site of action is not the spinal cord, but the spinal nerves and dorsal root ganglia.

Elimination occurs through vascular absorption in the subarachnoid and epidural space. Initial vascular uptake occurs through blood vessels in the pia mater and spinal cord. The rate of absorption is related to the vascular surface area that the local anesthetic comes into contact with. Lipid solubility of the local anesthetic solution enhances uptake into the tissue, further diluting the concentration. Local anesthetics also diffuse into the epidural space along a concentration gradient. Once in the epidural space, diffusion into the epidural vasculature occurs.

Factors Determining Distribution of Spinal Anesthetics

Several factors impact the distribution of local anesthetics within the subarachnoid space and subsequent height. Some factors play a major role while others play a minor/negligible role. These factors can be divided into 4 main categories:

- Characteristics of the local anesthetic medication
- Patient characteristics
- Technique of injection
- Characteristics of spinal fluid

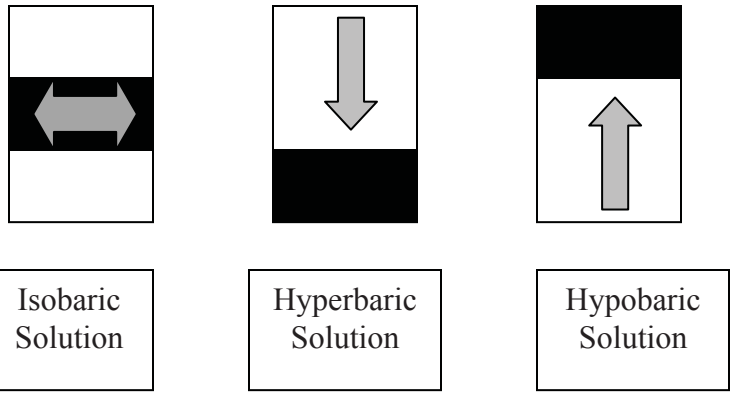
Characteristics of the Local Anesthetic Solution

Multiple characteristics of local anesthetic solution affect its spread within the subarachnoid space. These include density, dose, concentration, temperature, and volume.

Density- weight of 1 ml of solution in grams at a standard temperature.
Specific Gravity- density of a solution in a ratio, compared to the density of water.
Baracity- the ratio comparing the density of one solution to another.

- **Density/baracity**- the density or baracity of the local anesthetic exerts one of the greatest effects on subsequent height of the block. Local anesthetic movement within CSF is dependent on its specific gravity in relation to CSF, which at 37 degrees C is 1.003-1.008. A local anesthetic solution can be hyperbaric, hypobaric, or isobaric. Hyperbaric means that the solution is heavier than CSF. Dextrose is added to the local anesthetic solution to make it hyperbaric. Hypobaric means that the solution is lighter than CSF. This will allow it to move in a cephalad direction. Hypobaric solutions are created by adding sterile water to the solution. Isobaric solutions have the same specific gravity as CSF. Local anesthetic agents mixed in a 1:1 ratio with CSF create an isobaric solution.

Diagram of Gravities Influence on Baricity



Common Local Anesthetics and Specific Gravity

Local Anesthetic	Specific Gravity
Bupivacaine 0.5% in 8.25% Dextrose	1.0227-1.0278
Bupivacaine 0.5% plain	0.9990-1.0058
Lidocaine 2% plain	1.0004-1.0066
Lidocaine 5% in 7.25% Dextrose	1.0262-1.0333
Procaine 10% plain	1.0104
Procaine 2.5% in water	0.9983
Tetracaine 0.5% in water	0.9977-0.9997
Tetracaine 0.5% in D5W	1.0133-1.0203

Examples of baricities impact on the spread of local anesthetic solutions and patient position are described below.

- Head down position- a hyperbaric solution will spread cephalad; a hypobaric solution will spread caudad.
- Head up position- a hyperbaric solution will spread caudad; a hypobaric solution will spread cephalad.
- Lateral position- a hyperbaric solution will spread towards the dependent area; a hypobaric solution will spread to the non-dependent area.
- Any position with isobaric solution- will stay within the general area of injection.

Hyperbaric solutions move toward dependent areas. When the patient is supine, after injecting a hyperbaric solution of local anesthetic will move toward the T4-T8 area. The apex, following the normal curvature of the spine, is T4.

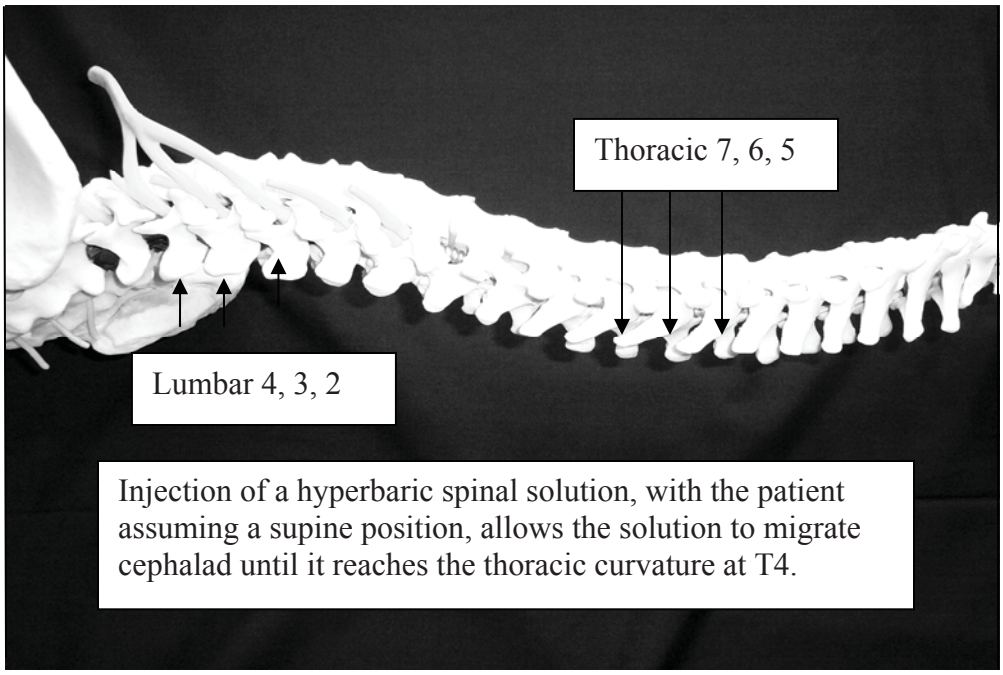
Additional characteristics of local anesthetic solutions include the following:

- **Dose**- the larger the dose, the higher the block.
- **Concentration**- the higher the concentration, the higher the block.
- **Temperature-** if the solution is cold it becomes viscous. This limits its spread within the CSF. The warmer the solution, the greater the spread. Temperature is a minor consideration.
- **Volume**- the greater the volume, the greater the spread.

Patient Characteristics

Patient characteristics include age, height, intra-abdominal pressure, anatomic configuration of the spinal cord, and patient position during and immediately after injection.

- **Age-** plays a minor role in block height. As we age there are anatomical changes in the subarachnoid area which increases block height.
- **Height-** plays a minor role. However, for the very short the dose of local anesthetic should be decreased, and for the very tall it may need to be increased.
- **Intra-abdominal pressure**- plays a role in relation to engorgement of epidural veins, decreasing CSF volume, resulting in a higher subarachnoid block. Conditions that increase intra-abdominal pressure include: pregnancy, obesity, ascites, large abdominal tumors, etc.
- **Anatomic configuration of the spinal cord**- natural lardosis and thoracic kyphosis influences spread of the local anesthetic solution. Medications injected above L3, with the patient in a supine position after injection, will spread cephalad reaching the thoracic curvature at T4. Abnormal anatomic changes that affect CSF can impact the level of blockade. Conditions such as severe kyphosis or kyphoscoliosis can result in decreased CSF volume and higher than expected blockade.



- **Patient position-** patient position during blockade can affect the spread of local anesthetic. This is a function of baricity and position of the patient. For example, a hyperbaric solution administered in the sitting position will result in a higher concentration of local anesthetic in the lower lumbar and sacral areas. A hyperbaric solution in the lateral position will result in a greater concentration of local anesthetic in the dependent portion of the patient. A hypobaric solution administered in the prone/jack knife position will result in blockade of the lower lumbar and sacral areas. Patient position is especially helpful after administering a hyperbaric solution. If the patient is left sitting up, the sacral and lower lumbar distribution will have a dense block. If the patient is supine, in a Trendelenburg position, hyperbaric solution will spread further reaching thoracic dermatomes.

Technique of Injection

Factors that influence the technique of injection include the site and direction of injection.

- **Site of injection**- the level of injection will influence spread. For example, a greater spread of local anesthetic will occur if injected at L2, as opposed to L5.
- **Direction of injection**- if the local anesthetic is injected in a caudad direction, the spread of local anesthetic will be limited compared to injection in a cephalad direction.
- It does not appear that rate of injection, barbatoge, coughing, or straining affects the height of block. The exception is the use of isobaric solutions.

Characteristics of Spinal Fluid

The volume and density of CSF influences subarachnoid block height.

- **CSF volume**- is inversely related to block height. Decreased volumes of CSF result in a higher block, whereas increased volumes of CSF decrease the level of blockade. CSF volume is influenced by patient characteristics (i.e. abnormal spinal anatomy).
- **CSF density**- has an impact on the spread of the local anesthetic. For example, if CSF is concentrated with a higher specific gravity, the local anesthetic may not spread as far as it normally would. Alternatively, dilute CSF, with a lower specific gravity, will result in a greater spread of the local anesthetic solution.

Factors that do not affect block height

- Vasoconstrictor use
- Coughing, staining, baring down, and barbotage
- Rate of injection (with the exception of isobaric)
- Gender
- Weight

Factors that affect block height but are out of the anesthesia provider's control

- Volume of CSF
- Density of CSF

Factors under the anesthesia provider's control

- Dose (volume/concentration)
- Site of injection
- Baricity of local anesthetics
- Position of the patient

Most important factors that determine block height

- Baricity of local anesthetic solution
- Position of patient during/immediately after injection
- Dose
- Site of injection

Local Anesthetics Used for Spinal Anesthesia

Several local anesthetics are used for spinal anesthesia. These include procaine, lidocaine, tetracaine, levobupivacaine, and bupivacaine. Local anesthetics are categorized by duration of action. Short acting spinal anesthetics are used for procedures that are < 90 minutes.

- Procaine
- Lidocaine

Long acting local anesthetics are used for procedures > 90 minutes.

- Tetracaine
- Bupivacaine
- Levobupivacaine

Local anesthetics administered for spinal anesthesia are preservative free. Preservative containing local anesthetics can be neurologically toxic and should be avoided.

Dosages of local anesthetic are generalized suggestions and may need to be adjusted according to individual patient characteristics.

Short Acting Spinal Anesthetics

Procaine

Historically, procaine was the second local anesthetic used for spinal anesthesia replacing cocaine. Procaine is an ester with a rapid onset (3-5 minutes) and a short duration of action (60 minutes). Procaine has several limitations:

- Short duration of action (<60 minutes)
- Higher frequency of nausea and vomiting
- Higher frequency of failed spinal anesthesia
- Despite its short duration of action it has a delayed time to full recovery

Procaine is increasing in popularity, since it has a lower frequency of TNS compared to lidocaine.

Medication	Preparation	Dose lower limbs	Dose lower abdomen	Dose upper abdomen	Duration plain	Duration epinephrine
Procaine	10% solution	75 mg	125 mg	200 mg	45 minutes	60 minutes

Lidocaine

In the past, this amide was popular for procedures less than 1.5 hours in duration. Like procaine, lidocaine has a rapid onset (3-5 minutes) and short duration of action (60-75 minutes). The most common preparation is a 5% solution in 7.5% dextrose. Less concentrated solutions have been

used in hopes of reducing the incidence of TNS. The old term for this syndrome was transient radicular irritation. TNS is discussed in detail in the Complications of Neuraxial Blockade chapter. The use of lidocaine has declined since this syndrome has been identified and described.

Medication	Preparation	Dose lower limbs	Dose lower Abdomen	Dose upper abdomen	Duration plain	Duration epinephrine
Lidocaine	5% solution in 7.5% dextrose*	25-50 mg	50-75 mg	75-100 mg	60-75 minutes	60-90 minutes

5% concentration no longer recommended due to risk of TNS….should be diluted to 2.5% or less or not used at all.

Long Acting Spinal Anesthetics

Three medications are available for long acting spinals. These include tetracaine, bupivacaine, and levobupivacaine.

Tetracaine

Tetracaine is an ester with a long and safe clinical record. It is available as niphanoid crystals (20 mg) or as a 1% solution (20 mg). Niphanoid crystals are mixed with 2 ml of preservative free sterile water. Next, mix the 1% solution with equal volumes of 10% dextrose, yielding a 0.5% tetracaine solution with 5% dextrose. Its onset is slow (5-10 minutes). Tetracaine is the longest acting spinal anesthetic. Duration of action is 2-3 hours for a plain solution. The addition of vasoconstrictors, such as epinephrine or phenylephrine (0.5 mg), increases the duration up to 5 hours for lower extremity surgical procedures. Epinephrine prolongs duration of blockade by 50%. The quality of motor blockade, when compared to bupivacaine, is more intense.

Medication	Preparation	Dose lower limbs	Dose lower abdomen	Dose upper abdomen	Duration plain	Duration epinephrine
Tetracaine	0.5% (1% solution in 10% glucose or as niphanoid crystals)	4-8 mg	10-12 mg	10-16 mg	90-120 minutes	120-240 minutes

Bupivacaine

Bupivacaine is an amide local anesthetic with a slow onset (5-10 minutes, longer with isobaric forms). It is a long acting spinal anesthetic appropriate for procedures that last 2-2.5 hours. It is comparable to tetracaine; however, tetracaine exhibits a more profound motor block and increased duration when vasoconstrictors are added. Available hyperbaric forms include concentrations of 0.5% and 0.75%, with dextrose 8.25%. Isobaric formulations are available in concentrations of

0.5% and 0.75%. When using isobaric solutions, the total mg dose is more important than the total volume of medication administered.

Medication	Preparation	Dose lower limbs	Dose lower abdomen	Dose upper abdomen	Duration plain	Duration epinephrine
Bupivacaine	0.75% & 0.5% hyperbaric solution in 8.25% dextrose and hypobaric solution	4-10 mg	12-14 mg	12-18 mg	90-120 minutes	100-150 minutes

Levobupivacaine

Bupivacaine is a stereoisomer containing a racemic solution of S and R isomers. A stereoisomer is a mirror image of the same compound. Each may exert different effects. As pharmacological advances continue, there will be more medications that are "pure" isomers, resulting in a greater degree of safety while limiting undesirable side effects. In the case of bupivacaine, the R isomer is more cardiotoxic than the S isomer. Levobupivacaine is the S isomer. Clinically, there is not a great advantage in using levobupivacaine for subarachnoid blocks. The risk of cardiotoxicity when using bupivacaine for a spinal anesthetic is non-existent. Clinically, levobupivacaine is dosed the same as bupivacaine.

Hypobaric, Isobaric, and Hyperbaric Anesthetic Solutions

A hypobaric solution for spinal anesthesia is less dense than CSF (less than 1.0069). To create hypobaric solutions with tetracaine mix 1% tetracaine, with sterile water (preservative free). This would make the baricity of the solution less than 0.9977. For anorectal procedures and hip repairs, a dose of 4-6 mg is generally adequate. Bupivacaine becomes hypobaric when warmed to 37 degrees C. Hypobaric solutions are not used often, but have their place in clinical anesthesia. Hypobaric solutions are useful for the patient with a fractured hip or extremity. Since it is painful for the patient to lie on the affected side, positioning them with the fracture up and administering a hypobaric solution will allow the patient to be more comfortable.

Isobaric solutions used for spinal anesthesia include bupivacaine, tetracaine, and levobupivacaine in 0.5% and 0.75% concentrations. Isobaric tetracaine is created by mixing 20 mg of niphanoid crystals with CSF.

Hyperbaric solutions are the most commonly administered spinal anesthetics. Control of the height is dependent on patient position during and immediately after injection. For a "saddle block" the patient should be kept sitting for 3-5 minutes to allow the medication to "settle down" to the lower lumbar and sacral nerves. If the patient is immediately positioned in a supine position after injection, the medication will move cephalad to the dependent area of the thoracolumbar curve. If

the patient is left in a lateral position for 5 minutes after injection, the level will be higher and denser in the dependent area compared to the non-dependent area.

Spinal Anesthetic Additives

Vasoconstrictors such as epinephrine (0.1-0.2 mg) and phenylephrine (0.5-2 mg) can be added to subarachnoid blocks to decrease vascular uptake and prolong duration of action. Epinephrine will prolong the duration of subarachnoid blockade when added to procaine, bupivacaine, tetracaine, and lidocaine. Phenylephrine has been found to increase duration for tetracaine and lidocaine, but not bupivacaine. Concerns about the administration of these agents in commonly administered doses (0.1-0.2 mg of epinephrine and 0.5-2 mg of phenylephrine) and the potential effects of vasoconstriction on the spinal cord are controversial, but largely unfounded. Epinephrine may have a weak spinal analgesic property secondary to the stimulation of α 2 adrenergic receptors.

Medication	Preparation	Dose lower limbs	Dose lower abdomen	Dose upper abdomen	Duration plain	Duration epinephrine
Procaine	10% solution	75 mg	125 mg	200 mg	45 minutes	60 minutes
Lidocaine	5% solution in 7.5% dextrose*	25-50 mg	50-75 mg	75-100 mg	60-75 minutes	60-90 minutes
Tetracaine	1% solution in 10% glucose or as niphanoid crystals	4-8 mg	10-12 mg	10-16 mg	90-120 minutes	120-240 minutes
Bupivacaine	0.75% & 0.5% hyperbaric solution in 8.25% dextrose and hypobaric solution	4-10 mg	12-14 mg	12-18 mg	90-120 minutes	100-150 minutes

Dosages of local anesthetic are generalized suggestions and may need to be adjusted according to individual patient characteristics.

Spinal Anesthesia Technique

It is difficult to teach a technique by describing it. Only through experience can one obtain a "feel" for the technique. We will, however, cover some important material that will be helpful when administering spinal anesthetics.

Technique

The technique of administering spinal anesthesia can be described as the "4 P's": preparation, position, projection, and puncture.

Preparation

Preparation of equipment/medications is the first step. It is important to think ahead.

- Discuss with the patient options for anesthesia. Explain risk and benefits. Inform the patient about the following: despite sedation the patient may remember portions of the surgical procedure but should not feel discomfort, the patient may feel pressure sensations but no pain, the patient will not be able to move their legs, and the approximate length of time that the block will last.
- Choose an appropriate local anesthetic. What local anesthetic should be used? Should it be a hypobaric, hyperbaric, or isobaric preparation? The duration of blockade should match the proposed length of the surgical procedure. Consider additives at this point. The addition of epinephrine may be considered to prolong and/or improve the quality of the block.
- Choose the appropriate spinal needle. Spinal needles are available in a variety of sizes (from 16-30 gauge), lengths, bevel types, and tip designs. Commonly, a 22 gauge needle is used in patients that are 50 years and older. A 25-27 gauge needle is used in patients that are less than 50 years of age. A smaller needle is used in the younger patient to decrease the incidence of post dural puncture headache. The removable stylet occludes the lumen and avoids tracking tissue into the subarachnoid space. Needles are cutting or blunt tiped. The Quincke needle is an example of a cutting needle, with the opening at the end of the needle.

Front View Side View

- Blunt tipped needles (pencil point) decrease the incidence of postdural puncture headaches compared to cutting needles. Whitacre and other pencil point needles, have a rounded tip with a side port. Sprotte needles have a long opening, allowing for excellent CSF flow.

Front View Side View

This may also lead to failed blocks since the opening may be partially within the subarachnoid space, leading to a partial dose of local anesthetic being administered.

- Prepackaged spinal kits are normally used and can be custom made.
- If a prepackaged spinal kit is not available, assemble the following equipment:
  - ✓ sterile towels
  - ✓ sterile gloves
  - ✓ sterile spinal needle
  - ✓ an introducer needle if using a small gauge needle (this can be a sterile 19 gauge disposable needle)
  - ✓ sterile filter needle to draw up medications
  - ✓ sterile 5 ml syringe for the spinal solution

- ✓ sterile 2 ml syringe with a small gauge needle to localize the skin prior initiation of the spinal anesthetic
- ✓ antiseptics for the skin (such as betadine, chlorhexidine, methyl alcohol)
- ✓ sterile gauze for skin cleansing and to wipe off excess antiseptic at needle puncture site
- ✓ single use preservative free local anesthetic ampoule. Local anesthetics from multi dose vials or those that contain preservatives should NEVER be used for spinal anesthesia. Ensure that the local anesthetic preparation is made specifically for spinal anesthesia.

- Prior to initiating a spinal block, carefully wash your hands.
- The patient should be attached to standard monitors including ECG, blood pressure, and pulse oximetry. Record an initial set of vital signs.
- Preload the patient with 1-1.5 liters of crystalloid intravenous solution.
- At any point during the administration of spinal anesthesia, if sterility is questioned or contamination of equipment occurs, stop, and start over with sterile equipment.

Positioning

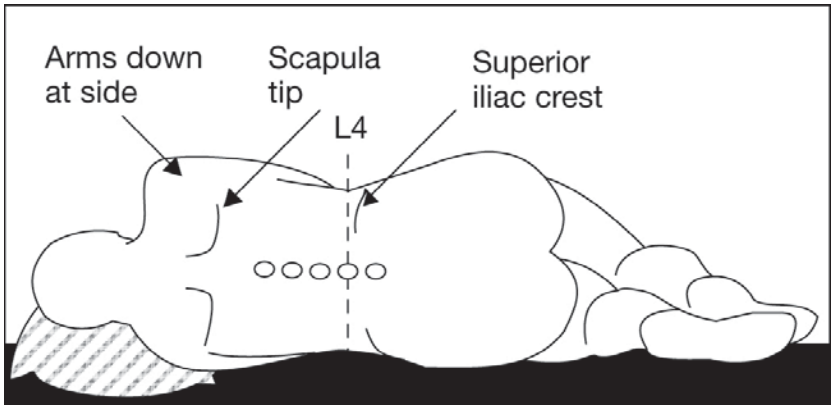
Proper positioning is essential for a successful block. Proper positioning can be difficult for several reasons.

1. Your assistant may not understand how the patient should be positioned or the rationale behind positioning.
2. The patient may not understand your instructions.
3. Sedation may make the patient unable to cooperate or follow directions.

There are three positions used for the administration of spinal anesthesia: lateral decubitus, sitting, and prone.

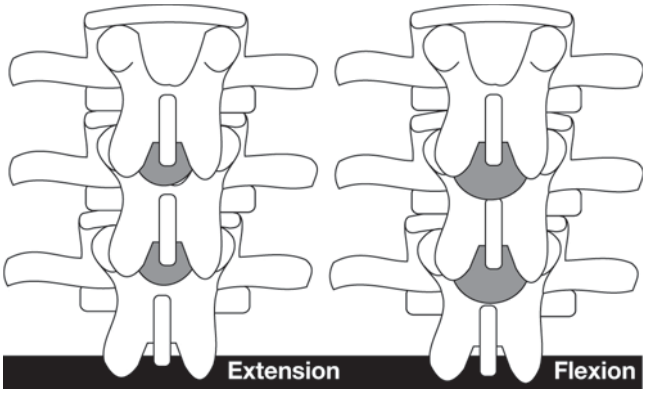
Lateral Decubitus

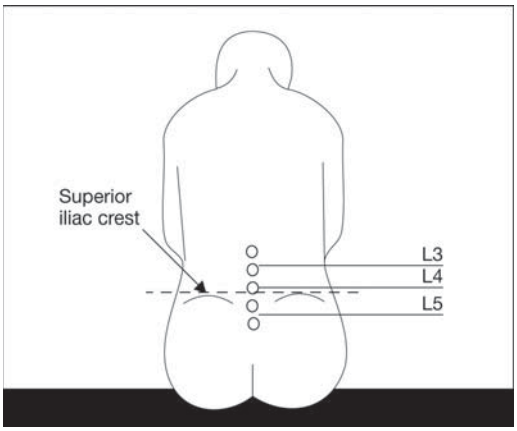
- Allows the anesthesia provider to administer more sedation- less dependence on an assistant for positioning. (Never over sedate a patient).
- The patient is positioned with their back parallel with the side of the OR table. Thighs are flexed up, and neck is flexed forward (fetal position).
- Patient should be positioned to take advantage of the baricity of the spinal local anesthetic.



Sitting

- Used for anesthesia of the lumbar and sacral levels (urological, perineal). Higher levels of anesthesia can be obtained if an appropriate dose of local anesthetic is administered, and the patient is quickly positioned to maximize the spread of local anesthetic.
- Identify anatomical landmarks. This may be a challenge in the obese or those with abnormal anatomical curvatures of the spine.
- Place the patients feet on a stool, have the patient sit up straight, head flexed, arms hugging a pillow, or on a table in front of them. Make sure the patient does not simply lean forward. A number of descriptions may help the patient understand how they should position themselves. For example, "please arch your back to resemble the letter C; or arch your back like a mad cat". This will maximize the "opening" of the vertebral interspaces.
- For a lower lumbar/sacral block (i.e. saddle block), leave the patient sitting for 5 minutes before assuming a supine position.





Prone

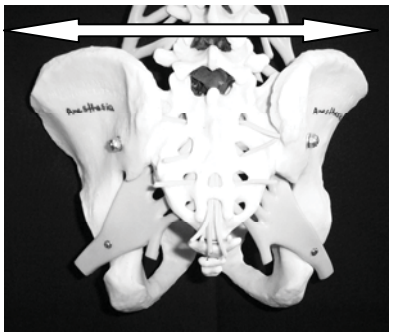
- The prone position is used when the patient will be in this position for the surgical procedure (i.e. rectal, perineal, lumbar procedures).
- Hypobaric local anesthetics are administered
- Patient positions self, lumbar lordosis should be minimized, a paramedian approach is often used.

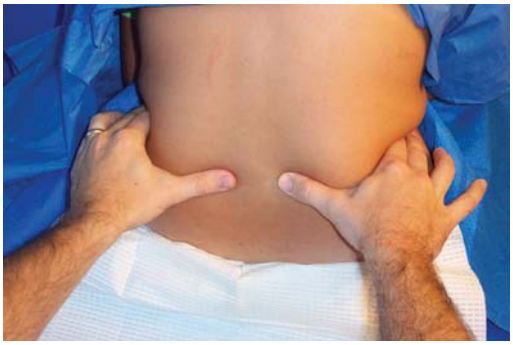
Projection and Puncture

There are two approaches to accessing the subarachnoid space: the paramedian and midline approach.

Midline Approach

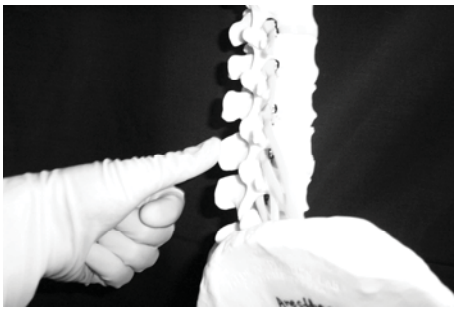
The midline approach affords the practitioner two advantages. Anatomic projection is only in 2 planes, making visualization of the intended trajectory and anatomical structures more apparent. The midline provides a relatively avascular plane. It is important to have the patient sitting up straight, not slumping to the side, to minimize lumbar lordosis, and maximize the space between the spinous processes. By proper positioning you should have access to L2-L3, L3-L4, L4-L5, and L5-S1. Identify the top of the iliac crest. Tuffier's line generally corresponds with the 4th lumbar vertebrae.

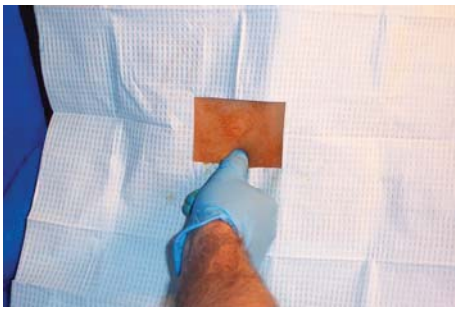




"Tuffier's" line is a line drawn across the iliac crest that crosses the body of L4 or L4-L5 interspace. This is a helpful landmark for the placement of spinal or epidural anesthetics.

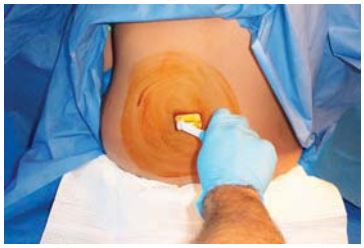
- Palpation in the midline should help to identify the interspinous ligament. The extent of the space is noted by palpating the cephalad and caudad spine. The midline is noted by moving your fingers from medial to lateral.



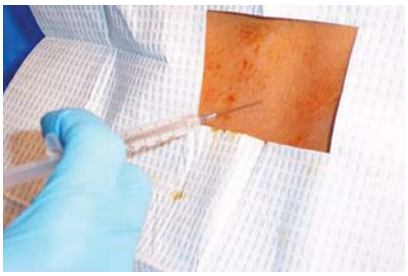


- Wash hands, put on sterile gloves, use sterile technique.

- Prepare the tray in a sterile fashion. An assistant may help with opening, in sterile fashion, specific items. Prepare the back with an antiseptic. Start at the area of intended injection and move out. This is done three times.

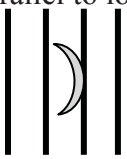


- Place a skin wheal of local anesthetic at the intended spinous interspace. Smaller gauge needles will require an introducer to stabilize the needle. Place the introducer firmly into the interspinous ligament.



- Anatomical structures that will be transversed include skin, subcutaneous fat, supraspinous ligament, interspinous ligament, ligamentum flavum, epidural space, and dura.
- Grasp the introducer with one hand and hold the spinal needle like a dart/pencil. Cutting needles should be inserted with the bevel parallel to the longitudinal fibers of the dura. This helps reduce cutting fibers and enhances tactile sensation as anatomical structures are crossed.

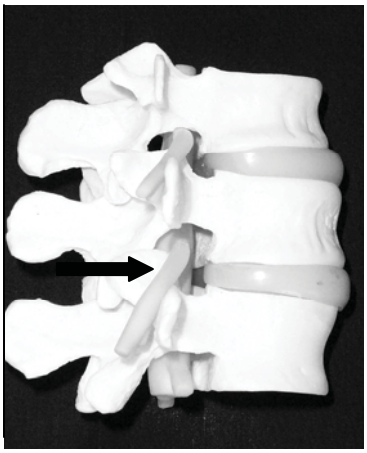
Placing needle bevel parallel to longitudinal fibers



- Control the needle carefully. Be prepared for unanticipated movement of the patient.
- As the ligamentum flavum and dura are transversed, a change in resistance is noted. Some will describe this as a "pop"; however, it may be a decrease in pressure or a loss of resistance.
- Once in the subarachnoid space, remove the stylet and CSF should appear. If CSF does not appear, rotate the needle 90 degrees until it appears. If no CSF appears then the stylet should be replaced. With smaller gauged needles it may take 20-30 seconds for CSF to appear. Assess the needle position. Is it at an appropriate depth? Is it midline or is its

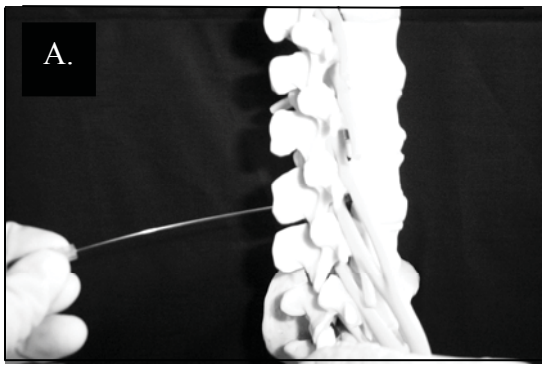
trajectory off the midline? Being off the midline is one of the most common reasons that CSF does not come back. If off the midline, remove the needle and start over.

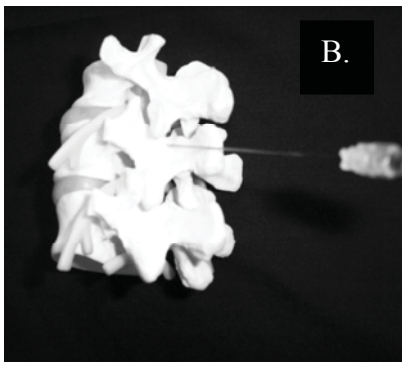
- If blood returns from the needle, wait to see if it clears. If it does not clear, reassess needle position. If the needle is midline, not lateral, it may be in an epidural vein. Advance the needle slightly further to transverse the dura. If the needle is not midline, remove it and start over.
- If the patient complains of a sharp pain in the hips or legs while inserting the needle, immediately remove the needle and reassess the approach. When the needle is not midline it is not uncommon to encounter a nerve root. Before starting again make sure that the pain has stopped.

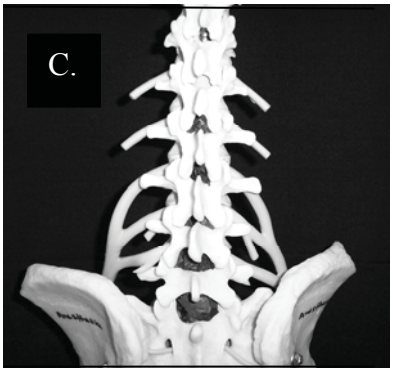


Pain/paresthesia may occur due to contact with a spinal nerve root.

- If bone is encountered, reassess the patient's position and ensure the needle is midline. If bone is contacted early, the needle may be contacting the spinous process. Move the needle slightly caudad (A). If bone is contacted late, the needle may be contacting the lamina of the vertebrae. Move the needle slightly cephalad (B). Moving down an interspace may increase the chance of success since the intervertebral spaces will be larger (C).







- After unsuccessful attempts, consider converting to a general anesthetic. The more attempts, the more trauma, increasing the risk of a spinal/epidural hematoma.
- Once CSF returns, steady the needle with the dorsum of the non dominant hand against the patients back. Attach the syringe with the intended spinal anesthetic. Gently aspirate some CSF into the syringe. If a hyperbaric technique is being used, a "swirling" in the solution will be noted due to the dextrose content. Aspiration with an isobaric technique will yield additional CSF fluid into the syringe. The cerebral spinal fluid should be clear. If blood is returned with aspiration, replace the stylet and start over.
- Inject the local anesthetic at a rate of 0.2 ml per second. After injection aspirate 0.2 ml of CSF to confirm that the needle remains in the subarachnoid space. If the patient complains of pain during injection, stop immediately. Redirect the needle away from the side of pain and into the midline.
- Place the patient in the appropriate position for the procedure and baricity of the spinal anesthetic solution.

Paramedian Approach

The advantage of the paramedian approach is a larger target. By placing the needle laterally, the anatomical limitation of the spinous process is avoided. The most common error when attempting this technique is being too far from the midline, which makes encountering the vertebral lamina more likely.

- Palpate the vertebral process and identify the caudad tip. Move 1 cm down and 1 cm laterally.
- Prepare the back with an antiseptic solution. Place a skin wheal of local anesthetic at the identified area of needle insertion. A longer needle is often required to infiltrate the tissue.
- Insert the introducer and/or spinal needle 10-15 degrees off the sagittal plane. At this point the most common error is inserting the needle too far cephalad, which results in encountering the lamina of the vertebral body. If bone is contacted, redirect the needle a little further caudad.

- It may be possible to feel the characteristic change in resistance or loss of resistance. With a lateral approach the needle is inserted further than with the midline approach.
- Once CSF is obtained, continue in the same manner as the midline approach.

Monitoring

After successful placement, the patient should be monitored continuously for block progression and complications. The patient's blood pressure should be taken every 3 minutes initially, more frequently if needed. The patient should be monitored for the following:

- Block progression- ensure that the block is adequate for the surgical procedure and it does not progress too high.
- Hypotension- treat aggressively, if blood pressure decreases by 20% or more from baseline
- Bradycardia- treat aggressively, it may progress to cardiac arrest
- Numbness of the arms and hands- may indicate that the block is too high
- Problems with breathing- may indicate that the block is too high
- Changes in the level of consciousness

An in-depth discussion of the complications of neuraxial complications have been discussed earlier.

Obstetric Care

Spinal anesthesia is generally preferred over a general anesthetic in the obstetric population, as long as not contraindicated. Spinal anesthesia in the obstetric population is covered in depth in the obstetric section of this manual.

Postoperative Care

Patient's recovering from a spinal anesthetic should receive the same vigilant monitoring as the patient recovering from a general anesthetic. In addition, the patient should be assessed for block regression. The patient with a spinal is more likely to experience hypotension in the postoperative period. Treatment includes a Trendelenburg position, additional intravenous fluids, oxygen, and vasopressors as needed. Urinary retention should be assessed in patients that do not have a urinary catheter. The patient should not be discharged from the recovery area until vital signs are stable and the spinal block is regressing. The patient should remain in bed until full sensory and motor function has returned. The first time a patient is ambulated, a nurse should assist the patient to ensure full function has returned.

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Epidural Anesthesia

Chapter Five

Epidural Anesthesia

Epidural anesthesia involves the use of local anesthetics injected into the epidural space to produce a reversible loss of sensation and motor function. Epidural anesthesia requires larger amounts of local anesthetic than a spinal anesthetic. Close attention to the total dose is required to avoid toxicity. Epidural anesthesia is versatile and can be administered by a single injection or through a catheter. The use of a catheter allows the anesthesia provider to add local anesthetics as surgery progresses, extending duration beyond the original dose. Epidural anesthesia can be combined with a general anesthetic or used as the sole anesthetic. In addition, the epidural catheter can be used for postoperative analgesia.

Epidural anesthesia provides excellent operating conditions for surgical procedures below the umbilicus. Procedures include:

- ✓ cesarean section
- ✓ procedures of the uterus, perineum
- ✓ hernia repairs
- ✓ genitourinary procedures
- ✓ lower extremity orthopedic procedures

In addition, it is an excellent option for the elderly patient who may not tolerate a general anesthetic. It is important not to use an epidural anesthetic in patients who are hypovolemic or severely dehydrated. Patients receiving an epidural anesthetic should be preloaded with .5-1 liter of crystalloid solution, such as ringers lactate, immediately prior to the block.

Advantages of Epidural Anesthesia

Several advantages of neuraxial blockade were listed in the Introduction to Neuraxial Blockade section of this manual. Additional advantages specific to epidural anesthesia include:

- Easy to perform (though it takes a bit more practice than spinal anesthesia)
- Reliable form of anesthesia
- Provides excellent operating conditions
- The ability to administer additional local anesthetics increasing duration
- The ability to use the epidural catheter for postoperative analgesia
- Return of gastrointestinal function generally occurs faster than with general anesthesia
- Patent airway
- Fewer pulmonary complications compared to general anesthesia
- Decreased incidence of deep vein thrombosis and pulmonary emboli formation compared to general anesthesia

Disadvantages of Epidural Anesthesia

There are several disadvantages to epidural anesthesia including:

- Risk of block failure. The rate of failure is slightly higher than with a spinal anesthetic. Always be prepared to induce general anesthesia if block failure occurs.
- Onset is slower than with spinal anesthesia. May not be a good technique if the surgeon is impatient or there is little time to properly perform the procedure.
- Normal alteration in the patient's blood pressure and potentially heart rate (generally slower onset with less alteration in blood pressure and heart rate than with a spinal anesthetic). It is essential to place the epidural block in the operating room/preoperative area with monitoring of an ECG, blood pressure, and pulse oximetry. Resuscitation medications/equipment should be available.
- Risk of complications as outlined in Introduction to Neuraxial Blockade chapter. There is an increase in the complication rate compared to spinal anesthesia.
- Continuous epidural catheters should not be used on the ward if the patient's vital signs are NOT closely monitored.
- Risk for infection, resulting in serious complications.

Contraindications

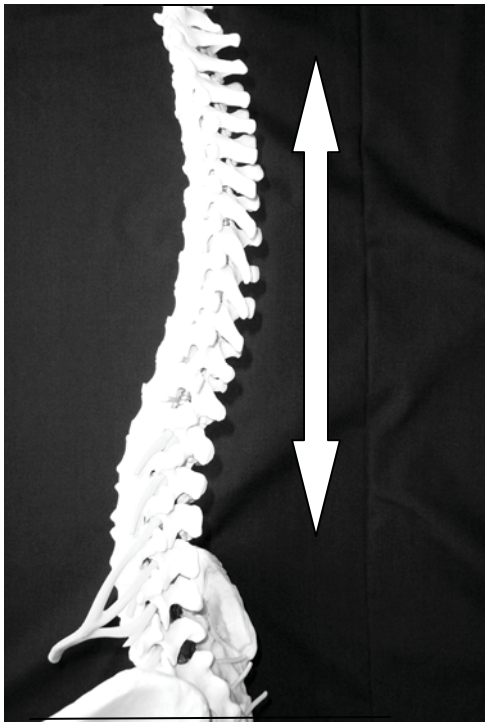
Please review chapter 2 for contraindications.

Site of Action for Local Anesthetics in the Epidural Space

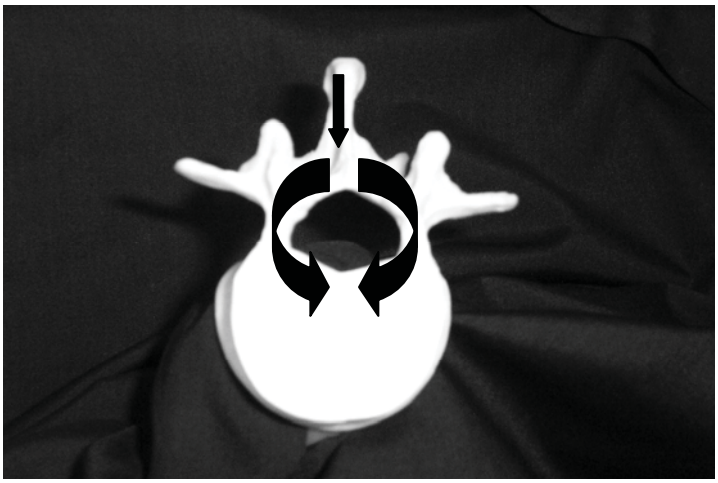
The majority of the local anesthetic administered is absorbed systemically by the rich venous plexus found within the epidural space. Dura surrounding spinal nerve/nerve roots are a modest barrier to the spread of local anesthetics. A small amount of local anesthetic will be absorbed into epidural fat. What remains will eventually reach its intended site of action, the spinal nerve and nerve roots.

Spread of Local Anesthetics within the Epidural Space

Local anesthetics administered in the epidural space move in a horizontal and longitudinal direction. Theoretically, if enough local anesthetic is injected, it could spread up to the foramen magnum and



Longitudinal Movement



Horizontal Movement

down to the sacral foramina. Clinically, the extent of longitudinal spread is volume dependent. Horizontal spread occurs through intervertebral foramina, entering the dural cuff. A small amount of local anesthetic may travel to the anterior epidural space. Diffusion into the CSF occurs at the dural cuff through arachnoid granules.

Distribution, Uptake & Elimination

It takes approximately 6-8 times the amount of local anesthetic in the epidural space to produce the same degree of blockade with a spinal anesthetic. This is due to the following factors:

- Larger mixed nerves are found in the epidural space.
- Local anesthetics must penetrate the arachnoid and dura mater.
- Local anesthetics are lipid soluble and will be absorbed into tissue and epidural fat.
- Epidural veins absorb a significant amount of local anesthetics. Peak blood concentrations occur 10-30 minutes after a bolus.

Local anesthetics are initially absorbed into epidural veins and diluted in the blood. The pulmonary system acts as a temporary buffer and protects other organs from the toxic effects of local anesthetics. The local anesthetic is redistributed to vessel rich organs, muscles, and fat. Long acting amide local anesthetics are bound to α-1 globulins, which have a high affinity for local anesthetics but become saturated quickly. Amide local anesthetics are metabolized in the liver and excreted by the kidneys. Ester local anesthetics are metabolized by plasma pseudocholinesterase. Rarely are there significant plasma levels

Factors Affecting Height

Factors that affect the height of epidural anesthesia are fewer and less predictable than spinal anesthesia and include the following:

- Volume of local anesthetic
- Age
- Height
- Gravity

Volume

Dosing an epidural can be variable. The rule of thumb is 1-2 ml of local anesthetic per dermatome segment. For example, if an epidural catheter is placed at L4-L5, and for surgical purposes a T4 sensory block is required, dose the patient with 12-24 ml of local anesthetic. There are 4 lumbar dermatomes (L1-L4) to block, as well as 8 thoracic dermatomes (T4-T12) to block, for a total of 12 dermatomes. Twelve (12) dermatomes times 1-2 ml = 12-24 ml of local anesthetic. This is quite a range of local anesthetic. Since the dose may be variable from patient to patient, it is important to dose the epidural in increments while continually assessing block progression. A segmental block for epidural analgesia would require a smaller dose. The volume of local anesthetic plays a crucial role in the block height.

Age

As patients age, less local anesthetic is required to achieve the same level of blockade as their younger counterpart. This is largely due to changes in the size and compliance of the epidural space.

Height

Height plays a role in epidural block height. The shorter the patient, the less anesthetic required to achieve the same level of anesthesia as a tall patient. For example, a patient who is 5'3 may require 1 ml per dermatome, whereas a patient who is 6'3" may require 2 ml per dermatome.

Gravity

Positioning the patient after injection of local anesthetic into the epidural space impacts its spread and height, but not to the degree that it does with spinal anesthesia. For example, positioning the patient in a lateral decubitus position will concentrate local anesthetic and extend block height in the dependent area compared to the non-dependent area. A sitting patient will have more local anesthetic delivered to the lower lumbar and sacral dermatomes. A Trendelenburg or reverse Trendelenburg position may help spread local anesthetic, or limit its spread.

Local Anesthetics used for Epidural Anesthesia

When choosing a local anesthetic for epidural anesthesia, consider the following:

- local anesthetic potency and duration
- surgical requirements and duration
- postoperative analgesia requirements

Seven local anesthetics can be used to produce epidural anesthesia. Only preservative free solutions should be used. Check the label to ensure the solution is "preservative free" and prepared specifically for epidural/caudal anesthesia/analgesia.

Short Acting:

- 2- chloroprocaine

Intermediate Acting:

- lidocaine
- mepivacaine

Long Acting:

- bupivacaine
- etidocaine
- ropivacaine
- levobupivacaine

Short Acting Local Anesthetics

2-Chloroprocaine

2-chloroprocaine is an ester local anesthetic. 2-chloroprocaine initially was associated with neurotoxicity (adhesive arachnoiditis) when large volumes were inadvertently administered in the subarachnoid space. This was attributed to bisulfate. In 1985, a reduced bisulfate solution was introduced. In 1987, a bisulfate free solution was produced. In 1996, a preservative free solution was introduced into clinical practice. Since the formulation of 2-chloroprocaine has been changed, there have been no reports of neurotoxicity. It is important to always check the contents of 2-chloroprocaine to ensure there is no bisulfite or preservative. Some formulations may still have bisulfate, or other preservatives which could cause problems if an inadvertent subarachnoid injection occurs. In addition, large doses of local anesthetics injected in the subarachnoid space may cause neurotoxicity. Back pain can occur after doses > 25 ml have been used. EDTA containing solutions are thought to "leach" calcium out of muscles. The preservative free formulations do not appear to cause back pain.

2-chloroprocaine is best suited for short surgical procedures. There are two concentrations available: 2% and 3%. The 2% concentration can be used for procedures that do not require muscle relaxation (it provides mild muscle relaxation); the 3% concentration provides for dense muscle

relaxation. Do not mix epidural opioids with 2-chloroprocaine. 2-chloroprocaine interferes with the analgesic effects of epidural opioids.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
2-chloroprocaine	2%	Fast 10-15 minutes	Analgesic	Mild to moderate	45-60 minutes	60-90 minutes
2-chloroprocaine	3%	Same	Dense	Dense		

Intermediate Acting Local Anesthetics

Lidocaine

Lidocaine is the prototypical amide local anesthetic. For epidural anesthesia, concentrations of 1.5 – 2% are commonly used. Epinephrine will prolong the duration of lidocaine by 50%. The addition of preservative free fentanyl (50-100 mcg) will accelerate the onset of analgesia and create a more potent/complete block.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Lidocaine	1.5%	Intermediate 15 minutes	Dense	Mild to moderate	80-120 minutes	120-180 minutes
Lidocaine	2%	Same	Dense	Dense		

Mepivacaine

Mepivacaine is similar to lidocaine. It is an amide local anesthetic that lasts about 15-30 minutes longer than lidocaine. Epinephrine will prolong duration by 50%.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Mepivacaine	1%	Intermediate 15 minutes	Analgesic	Minimal		
Mepivacaine	2%	Same	Dense	Dense	90-140 minutes	140-200 minutes

Long Acting Local Anesthetics

Bupivacaine

Bupivacaine is a long acting amide local anesthetic. For epidural anesthesia, the most common concentrations are 0.5-0.75%. Concentrations for analgesia range from 0.125-0.25%. Epinephrine (1:200:000) will prolong the duration of action of bupivacaine, but is not as reliable as with lidocaine and mepivacaine. Bupivacaine in concentrations of 0.75% should not be used for obstetric anesthesia. The FDA's recommendation in 1983 occurred after several cardiac arrests were reported

related to inadvertent intravenous injection. Bupivacaine (in addition to etidocaine) is more likely to impair the myocardium and conduction than other local anesthetics during inadvertent intravenous injection. Patients are difficult to successfully resuscitate due to bupivacaine's high protein binding and lipid solubility, which allow it to accumulate in the cardiac conduction system, resulting in refractory reentrant arrhythmias.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Bupivacaine	<0.25%	Slow	Dense	Minimal to moderate		
Bupivacaine	0.5-.75%	Same	Dense	Mild to dense	165-225 minutes	180-240 minutes

Levobupivacaine

Levobupivacaine is the S enantiomer of bupivacaine. Clinically it is used in the same concentrations and is indistinguishable from bupivacaine, except for one important fact; it is less cardiotoxic.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Levobupivacaine	<0.25%	Slow	Dense	Minimal to moderate		
Levobupivacaine	0.5-.75%	Same	Dense	Mild to dense	150-225 minutes	150-240 minutes

Ropivacaine

Ropivacaine is a long acting amide local anesthetic. It is a mepivacaine analogue. Ropivacaine is used in concentrations of 0.5-1% for anesthesia, and 0.1-0.3% for analgesia. Ropivacaine is similar to bupivacaine in onset, duration, and quality of blockade. When used for analgesia it provides excellent sensory blockade with minor motor blockade. It is less cardiotoxic than bupivacaine. Ropivacaine is unique among local anesthetics, since it exhibits a vasoconstrictive effect at clinically relevant doses.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Ropivacaine	0.1-0.2%	Slow	Analgesic	Minimal		
Ropivacaine	0.5%	Same	Dense	Mild to moderate		
Ropivacaine	0.75-1%	Same	Dense	Dense	140-180 minutes	150-200 minutes

Etidocaine

Etidocaine is a long acting amide local anesthetic. Its use clinically is infrequent due to intense motor blockade. Motor blockade is more intense than sensory. A 1% concentration is used for surgical anesthesia.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Etidocaine	1%	Slow	Dense	Dense	120-200 minutes	150-225 minutes

Epidural Additives

Epinephrine will increase the duration of action for all epidurally administered local anesthetics. Differences exist in the extent of increase among individual local anesthetics. The greatest increases are found with lidocaine, mepivacaine, and 2-chloroprocaine. It is less effective for bupivacaine, levobupivacaine, and etidocaine. Epinephrine is not added to ropivacaine due to its inherent vasoconstrictive effects. When epinephrine is compared to phenylephrine, it has been found that epinephrine is more effective in reducing peak blood levels of local anesthetics.

Sodium bicarbonate added to local anesthetics such as lidocaine, mepivacaine, and 2-chloroprocaine, appears to have several positive effects, including an increase in the concentration of free base, which enhances the rate of diffusion, speeding onset. Studies have found that the addition of sodium bicarbonate to 1.5% lidocaine produces a significantly faster onset of sensory blockade/anesthesia, and a more complete block. One (1) meq of bicarbonate is added to every 10 ml of local anesthetic (i.e. lidocaine, mepivacaine, 2-chloroprocaine). The addition of bicarbonate to bupivacaine is less popular since precipitation occurs above a pH of 6.8. If bicarbonate is added, it should be in a ratio of 0.1 ml of bicarbonate to every 10 ml of bupivacaine.

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Short Acting Local Anesthetics						
2-chloroprocaine	2%	Fast 10-15 minutes	Analgesic	Mild to moderate	45-60 minutes	60-90 minutes
2- chloroprocaine	3%	Same	Dense	Dense		
Intermediate Acting Local Anesthetics						
Lidocaine	1.5%	Intermediate 15 minutes	Dense	Mild to moderate	80-120 minutes	120-180 minutes
Lidocaine	2%	Same	Dense	Dense		
Mepivacaine	1%	Intermediate 15 minutes	Analgesic	Minimal		
Mepivacaine	2%	Same	Dense	Dense	90-140 minutes	140-200 minutes
Long Acting Local Anesthetics						
Bupivacaine	<0.25%	Slow	Dense	Minimal to moderate		
Bupivacaine	0.5-.75%	Same	Dense	Mild to dense	165-225 minutes	180-240 minutes
Levobupivacaine	<0.25%	Slow	Dense	Minimal to moderate		
Levobupivacaine	0.5-.75%	Same	Dense	Mild to dense	150-225 minutes	150-240 minutes
Ropivacaine	0.1-0.2%	Slow	Analgesic	Minimal		
Ropivacaine	0.5%	Same	Dense	Mild to moderate		
Ropivacaine	0.75-1%	Same	Dense	Dense	140-180 minutes	150-200 minutes
Etidocaine	1%	Slow	Dense	Dense	120-200 minutes	150-225 minutes

Epidural Anesthesia Technique

It is difficult to teach a technique by simply describing it. Only through experience can one obtain a "feel" for the technique. Important material will be covered that maybe helpful when administering epidural anesthetics.

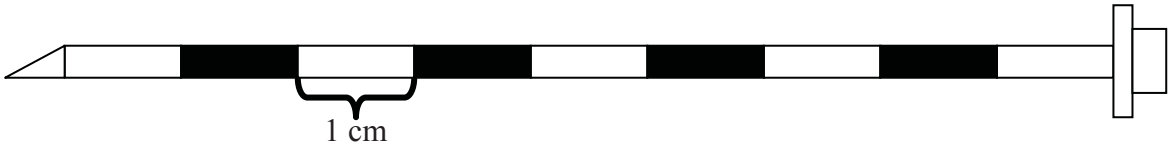
Equipment

Epidural Needles

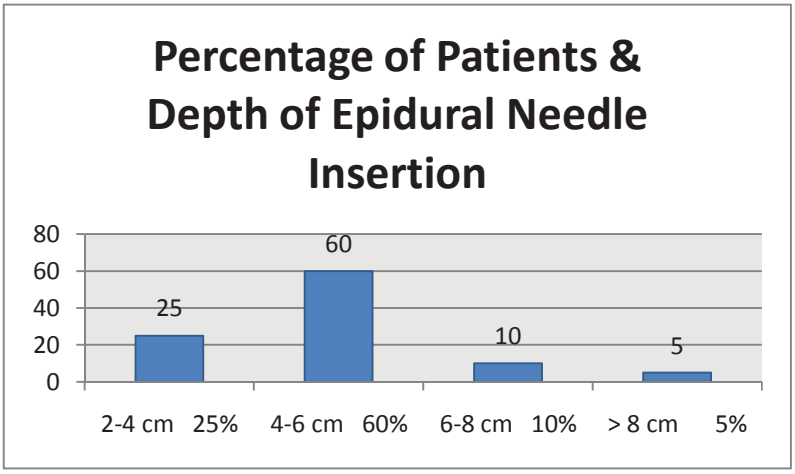
The standard epidural needle is 17-18 gauge, 9cm in length, with a blunt needle tip and gentle curve of 15-30 degrees. The Tuohy needle is an example of a blunt, curved needle. This design helps to push the dura away from the needle, reducing the risk of dural puncture and guiding the catheter

during insertion. Crawford needles are straight needles. There is an increased risk of dural puncture with straight needles. Additional modifications include wings at the hub to help with insertion, and introducer devices that act as a guide during the insertion of an epidural catheter.

The majority of epidural needles contain markings in 1 cm increments. These markings help the anesthesia provider estimate the depth of needle insertion. The average epidural needle will be 9 cm from the tip of the needle to the epidural hub.

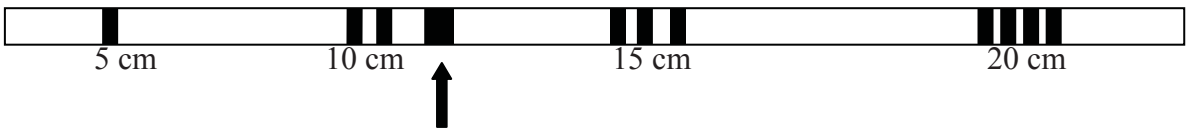


Patients will vary from 2-8 cm in skin to epidural space depth.



Epidural Catheters

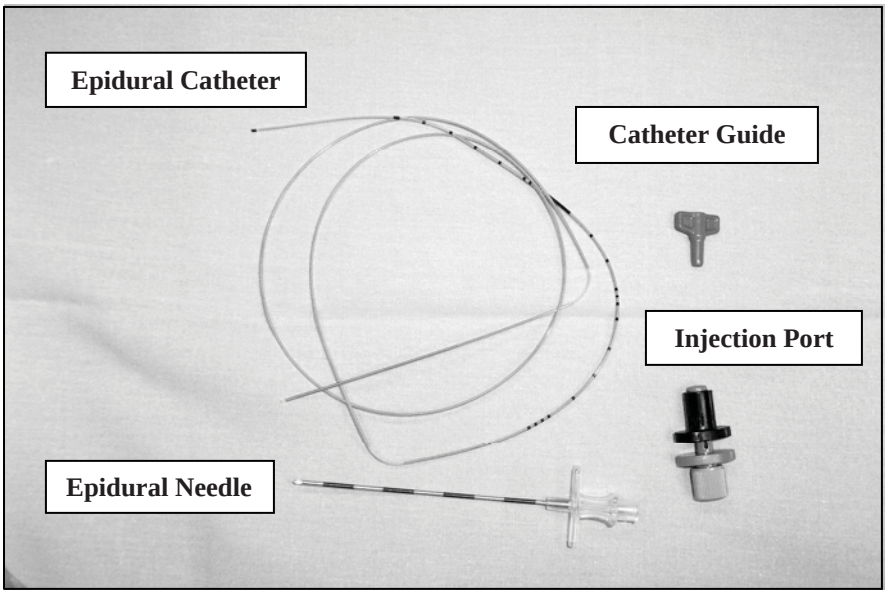
An epidural catheter increases flexibility in delivering anesthesia since a continuous infusion or intermittent bolus technique can be used. A 19 or 20 gauge catheter is placed through a 17 or 18 gauge epidural needle after identification of the epidural space. The curved tip of the Tuohy needle helps guide the catheter cephalad or caudad. Epidural catheters have distinctive markings to aid the anesthesia provider in determining the depth of insertion. The first single mark is located at 5 cm. Two marks are noted at 10 cm, three at 15 cm, and four at 20 cm.

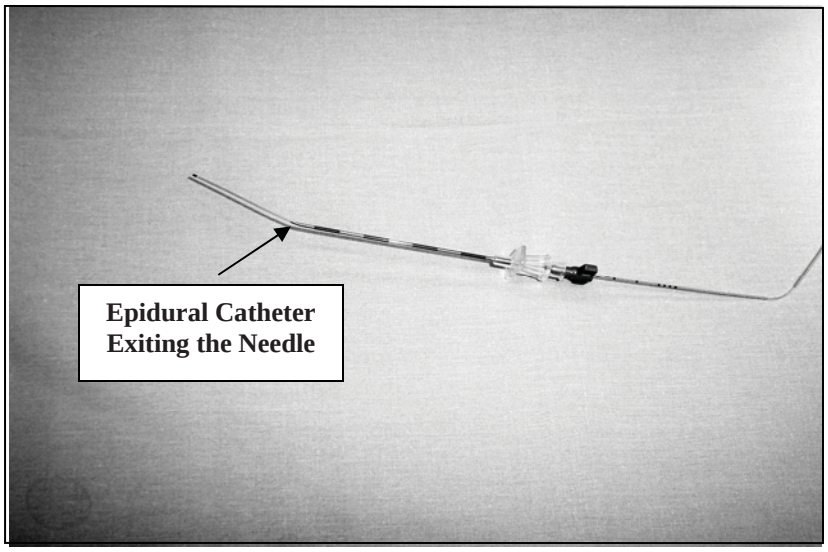


Most catheters will, in addition to the bold 5, 10, 15, and 20 cm markings, have single marks at 1 cm intervals. The single "thick" mark at the 11 cm (noted by the arrow above) indicates that the tip of

the epidural catheter is at the tip of the needle. Each cm after this mark will be the depth of insertion into the epidural space.

The epidural catheter is introduced 2-6 cm into the epidural space. Inserting the catheter the minimum distance may increase the risk becoming dislodged. On the other hand, inserting the catheter the maximal distance may lead to a unilateral block. The tip may exit the epidural space through the intervertebral foramina or enter the anterolateral recesses of the epidural space. Most anesthesia providers insert the epidural catheter to a depth of 3-4 cm. After the catheter has been placed, the needle is removed, and the epidural catheter is taped into place. Never pull the catheter out of the needle; this can lead to shearing. The catheter may have a single port or a closed end with several side ports. Some catheters have a stylet that aid in insertion. However, this may increase the risk of inadvertent dural puncture and/or intravascular insertion. Catheters with spiral wire reinforced walls decrease the risk of kinking and inadvertent intravascular injection.





Epidural Technique

The 4 p's for the administration of epidural anesthesia are preparation, position, projection, and puncture.

Preparation

- Prepare the patient. Discuss options, risks and benefits. Explain what to expect during an epidural anesthetic.
- Decide whether to use a single shot, continuous catheter, or intermittent bolus technique. For surgical procedures, a continuous catheter technique is often used. The Crawford needle is appropriate for a one shot technique, whereas a Tuohy needle is appropriate for epidural catheter insertion.
- Decide on the technique to identify the epidural space. Choices include loss of resistance and hanging drop technique.

Positioning

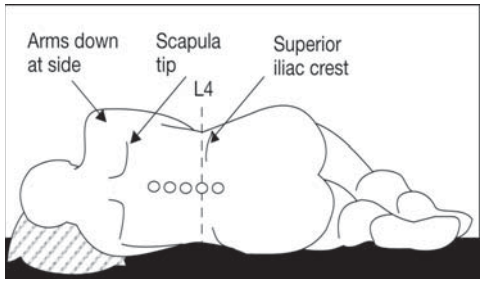
Proper positioning is essential for a successful block. Proper positioning can be difficult for several reasons.

1. Your assistant may not understand how the patient should be positioned or the rationale behind positioning.
2. The patient may not understand your instructions.
3. Sedation may make the patient unable to cooperate or follow directions.

There are three positions used for the administration of epidural anesthesia: lateral decubitus, sitting, and prone.

Lateral Decubitus

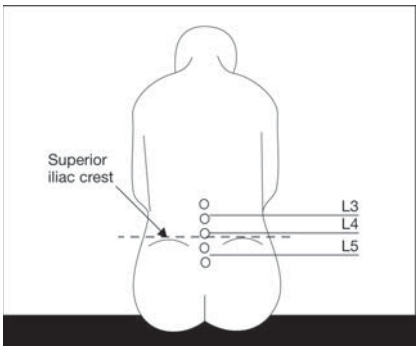
- Allows the anesthesia provider to administer more sedation- less dependence on an assistant for positioning. (Never over sedate a patient).
- The patient is positioned with their back parallel with the side of the OR table. Thighs are flexed up, and neck is flexed forward (fetal position).
- In children, a lateral decubitus position is often used for the caudal approach. This allows for the maintenance of a patent airway, since the caudal technique is often performed under general anesthesia in pediatric patients. Regional techniques are discouraged during general anesthesia in adult populations due to the risk of nerve injury.

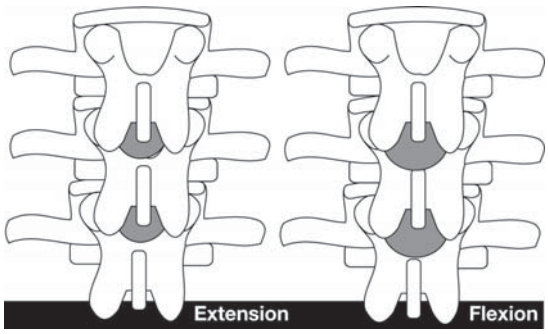


- Position changes are not as critical with epidural anesthesia. The onset of epidural anesthesia is faster in the dependent areas of the body.

Sitting

- Identify anatomical landmarks. This may be a challenge in the obese or in those with abnormal anatomical curvatures of the spine.
- Place the patients' feet on a stool. Have the patient sit up straight, head flexed, arms hugging a pillow, or on a table in front of them. Make sure the patient does not simply lean forward. A number of descriptions may help the patient understand the position they are to assume. For example, "please arch your back to resemble the letter C; or arch your back like a mad cat". This will maximize the "opening" of the vertebral interspaces.





Prone Position

- Used for caudal approach in adults.

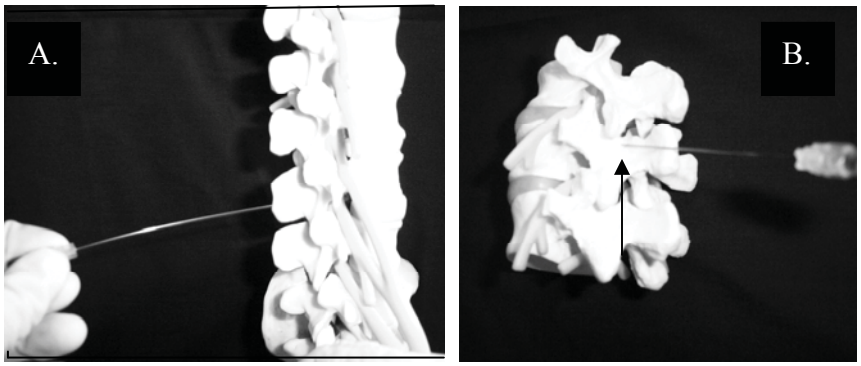
Projection and Puncture

- After a sterile prep, place a skin wheal at the predetermined site of insertion.
- Identify midline! If off the midline it will be difficult to locate the epidural space. If the needle is inserted further than normal, blood is returned in the needle, and/or the patient complains of a paresthesia, stop. Reassess landmarks and needle insertion point.
- Insert the epidural needle into the ligamentum flavum. Anatomical structures transversed include skin, sub cutaneous tissue, supraspinous ligament, and interspinous ligament. If the needle is not placed in the ligamentum flavum, the anesthesia provider may experience false positives with the loss of resistance technique.
- In the lumbar area, the depth of skin to ligamentum flavum is approximately 4 cm for most adults. Eighty percent of adults have a skin to ligamentum flavum depth of 3.5-6 cm. The average thickness of the ligamentum flavum is 5-6 mm. Controlling the needle is important to avoid a dural puncture. In the thoracic area, needle control is important to avoid dural puncture and risk of spinal cord injury.

- Loss of resistance technique: once the needle is placed into the ligamentum flavum, remove the stylet. Attach a glass syringe with 2-3 ml of preservative free normal saline and a small (0.25 ml) air bubble. The needle is held steady by the non-dominant hand. The dominant hand holds the syringe. Steady pressure is applied to the plunger to compress the air bubble. Slowly and steadily advance the needle until loss of resistance is noted.
- Hanging drop technique: place the needle into the ligamentum flavum. Next, apply a drop of preservative free normal saline to the hub of the needle. Apply slow, steady pressure to the needle until the hanging drop gets "sucked" in. The epidural space contains subatmospheric pressure.
- Once the epidural space has been identified, advance the needle 1-2 mm further. Some anesthesia providers do this to ensure the tip of the needle is not obstructed by tissue, hindering insertion of the catheter. On the other hand, this may increase the risk of inadvertent dural puncture. An alternative is to inject an additional 2-3 ml of preservative free normal saline, expanding the epidural space and pushing structures away.
- Insert the catheter 3-4 cm into the epidural space for surgical patients. Inserting the catheter further may lead to a unilateral block. For OB patients, insert the catheter 4-5 cm to prevent migration of the catheter out of the epidural space during labor and delivery.
- The dose and volume of local anesthetic for epidural anesthesia is large enough to cause systemic toxicity if injected into a blood vessel, and a high spinal if injected in the subarachnoid space. To help identify inadvertent venous cannulation or subarachnoid placement, a test dose should be performed.
- A test dose consists of 3 ml of 1.5% preservative free lidocaine with 1:200,000 epinephrine. Forty-five milligrams of lidocaine, if injected intrathecally, will result in a spinal anesthetic. Fifteen micrograms of epinephrine, if injected intravascularly, will result in a 20% or more increase in heart rate. Blood pressure may be elevated or remain the same. False positives may occur with epinephrine. For example, a laboring patient may have a contraction at the same time that the test dose was administered, resulting in a concurrent increase in heart rate or blood pressure. False negatives may occur. For example, a patient may be on a beta blocker which will block/blunt an increase in heart rate.
- Aspiration before each injection is helpful, but may not always detect intravascular or subarachnoid placement of a catheter.
- Incremental dosing of 5 ml every 5 minutes should be performed. This dose should be enough to cause symptoms of intravascular injection without seizures and/or cardiovascular collapse.
- Catheter migration may occur any time. This can lead to an intravascular or intrathecal injection. Aspirate before dosing and dose the epidural incrementally. Be cautious and have a high index of suspicion. Some clinicians will use a test dose with each subsequent injection, along with aspiration and incremental dosing.

Problems that may be Encountered when Administering Epidurals

- Bone is encountered by the needle. Reassess the direction of the needle, ensure the needle is mid line, and the patient is correctly positioned.



A. Needle contacting spinous process
B. Needle contacting lamina

- Inability to thread the catheter. Ensure the needle is in the epidural space by placing an additional 3 ml of preservative free normal saline. If there is a loss of resistance, then attempt to insert the catheter. Rotating the needle slightly may help. If the catheter still cannot be inserted, start over.
- Fluid returns through the needle. When using preservative free normal saline, a small amount may come back. If it does not stop and continues, then the needle may have transversed the dura. If this is suspected, place the epidural at another level and monitor for the development of a post dural puncture headache. If fluid stops, thread the catheter and administer a test dose to ensure that the needle/catheter did not cross the dura. If the test dose is negative, cautiously dose the epidural.
- Blood returns in the catheter or needle. The needle/catheter may have entered into an epidural vein. Remove the needle and/or catheter and start over. Make sure the needle is midline.
- Pain (paresthesia) upon insertion of the needle. Remove needle immediately and assess position. Commonly, insertion is not midline and the needle should be repositioned.
- Pain (paresthesia) upon insertion of the epidural catheter. It is not unusual to get a brief shock like symptom or sensation during catheter insertion. If it does not stop, remove the catheter. It may be in contact with a nerve root.
- Pain with injection. The direction of the catheter cannot be controlled during insertion. The tip may be against a nerve root. Pull the catheter back 1 cm and attempt to inject again. If pain continues, remove the catheter and start over.

Failed Epidural

Epidural anesthesia is more subjective than spinal anesthesia. There is not a clear cut end point, like CSF with a spinal. The anatomy of the epidural space lends to a less predictable spread of local anesthetic. There are several factors that may lead to a failed epidural block. These include false loss of resistance, misplaced local anesthetic, unilateral block, segmental sparing, and visceral pain.

- False loss of resistance. It is possible to insert a catheter in tissue other than the epidural space. Spinal ligaments may be soft, resulting in a false loss of resistance. Being off the midline, in the paraspinous muscle, may also result in a false sense of loss of resistance.
- Misplaced local anesthetic. Local anesthetics may be misplaced in other anatomical areas including the subarachnoid space, subdural space, and intravenously. These complications have been discussed earlier.
- Unilateral block. The catheter may have traveled out of the epidural space or pointed laterally. Pulling the catheter back 1-2 cm may move the catheter back into a midline position. Pulling back on the catheter may also move the catheter out of the epidural space.
- Segmental sparing may occur due to anatomical conditions (septa) within the epidural space. Additional local anesthetic may help alleviate this condition. Sacral sparing may occur due to the larger size of L5-S2. Elevating the head of the bed and adding local anesthetic will increase the concentration of local anesthetic in this area, creating a denser block.
- Visceral pain is not a failure of epidural anesthesia. Visceral afferent fibers travel with the vagus nerve and are difficult to block. Intravenous supplementation with analgesics and sedatives may be required to get the patient through "uncomfortable" portions of the surgical procedure. If unable to adequately treat discomfort, then general anesthesia should be induced.

Monitoring

After successful placement of an epidural anesthetic, the patient should be monitored continuously for block progression and complications. Heart rate, pulse oximetry, level of consciousness, and signs and symptoms of toxicity should be monitored continuously. Blood pressure should be taken every 3 minutes or more frequently if needed. The patient should be monitored for the following conditions:

- Block progression- ensure that the block is adequate for the surgical procedure and does not progress too high.
- Hypotension- treat aggressively, if > 20% of the baseline blood pressure
- Bradycardia- treat aggressively, since it may progress to cardiac arrest
- Numbness of the arms and hands- may indicate that the block is too high
- Problems with breathing- may indicate that the block is too high
- Changes in the level of consciousness

For an in-depth discussion of the complications of neuraxial complications, please refer to Chapter 3.

Obstetric Care

Neuraxial blockade is preferred over a general anesthetic in the obstetric population, if not contraindicated. Epidural anesthesia in the obstetric population is covered in depth in the obstetric section of this manual.

Postoperative Care

During recovery from an epidural anesthetic, the patient should receive the same vigilant care as the patient recovering from general anesthesia. In addition, the patient should be assessed for block regression. The patient with an epidural is more likely to experience hypotension in the postoperative period. Treatment includes a Trendelenburg position, additional intravenous fluids, oxygen, and vasopressors as needed. Assess the patient for urinary retention if they do not have an urinary catheter. Discharge should not occur until vital signs are stable and the epidural block is regressing. The patient should remain in bed until full sensory and motor function has returned. The first time a patient is ambulated a nurse should assist the patient, ensuring full function has returned.

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Peripheral Nerve Blocks

Chapter Six

Peripheral Nerve Blocks

Peripheral nerve blocks are safe and effective alternatives to general and neuraxial anesthesia. Knowledge of anatomy and pharmacology are essential for successful administration. The following peripheral nerve blocks will be covered: femoral/3 in 1, interscalene, axillary, elbow, wrist, digital, ankle, and intravenous regional anesthesia.

The ability to perform a variety of peripheral nerve blocks will enhance the anesthesia providers' flexibility and offer the patient additional options for their care.

Indications for Peripheral Nerve Block

A careful review of the patient's history will yield valuable information enabling the anesthesia provider to make an informed decision on the best options. For example, a peripheral nerve block may be used as the sole anesthetic (with or without sedation), as a supplement to general anesthesia, and/or postoperative analgesia. General considerations include:

- Suitability for the type of surgery being performed
- Surgeon's preferences
- Experience in performing the block
- Physiological and mental state of the patient

When obtaining informed consent, include options and risks/benefits. It is acceptable to present to the patient what may be the best choice based on co-morbidities. The final decision is the patients. Most patients' are accepting of the anesthesia provider's opinion if presented in a manner that can be clearly understood. Never try to scare a patient into a regional anesthetic. Be gentle and objective when presenting options. The following are general advantages:

- Improved patient satisfaction (especially in elderly)
- Less immunosuppression compared to general anesthesia
- Decreased incidence of nausea and vomiting
- A non general anesthesia option for patients with a history of malignant hyperthermia
- Alternative for patients who are hemodynamically unstable or too ill to tolerate general anesthesia
- Less post operative cognitive impairment, especially in the elderly

General risks include:

- Toxicity of local anesthetics
- Transient or chronic paresthesia

- Nerve damage. Never inject if the patient experiences pain or there is an increased resistance to injection. Never use sharp "cutting" needles. If only sharp needles are available, then blunt the needle using the sterile plastic sheath that comes with the needle. Maintain sterility. This is not "ideal" and will still place the patient at risk for injury, but may be the only option in environments where resources are limited.
- Intra-arterial injection, seizures, or cardiac arrest
- Block failure, the need to supplement or convert to general anesthesia
- Infection

Contraindications to Peripheral Nerve Blocks

Some contraindications for peripheral nerve blocks are relative, and others are absolute. Absolute contraindications include the following:

- Patient refusal
- Infection at the injection site. The insertion of a needle through infected tissue into healthy tissue may spread infection. In addition, local anesthetics do not work well in acidotic tissue.
- An allergy to local anesthetics. Ensure that it is a "true" allergy. Some patients may report symptoms such as dizziness, nausea, etc. during dental anesthesia. Ask the patient if they had trouble breathing, a rash, and other symptoms that would indicate a "true" allergy. If the patient had a true allergic reaction to a local anesthetic, identify which local anesthetic. Ester local anesthetics have a higher incidence of allergic reactions, related to their metabolism to PABA. Amide local anesthetics have a very low incidence of allergic reactions. There are no cross reactions between amides and esters. A true allergy is an absolute contraindication to a peripheral nerve block with the offending local anesthetic or others in the same class.
- The inability to guarantee sterile equipment to perform the block is an absolute contraindication. This could result in the introduction of infectious agents in otherwise healthy tissue.
- If the risk of local anesthetic toxicity is great (i.e. one would not want to perform bilateral axillary block; or repeat one). Do not exceed the maximum dose for local anesthetics.

Relative contraindications include the following:

- Pediatric, combative, and/or demented patients. For pediatric patients, most blocks are placed after general anesthesia. This practice is discouraged for adults due to the risk of intraneural injection. In addition, the patient is unconscious and unable to report subjective symptoms of impending toxicity associated with an intravascular injection. Demented/combative patients present the challenge of remaining still during the surgical procedure.
- Bleeding disorder (medication induced i.e. coumadin; or genetic i.e. hemophilia; or acquired i.e. DIC). Hematoma formation may increase the risk of ischemic nerve damage. In the

case of an extremity or end organ, it can lead to a tourniquet syndrome and ischemia (i.e. ankle, digits, etc.)

- Pre-existing peripheral nerve neuropathies may increase the risk for permanent nerve damage. Careful documentation of sensory and motor deficits should occur prior to the initiation of a peripheral nerve block.

Sedation during Peripheral Nerve Blocks

Light sedation may help relax the patient, increasing the likelihood of a successful block. It is important not to over sedate the patient. Over sedation may lead to the following:

- ✓ Unprotected airway
- ✓ Inability to communicate important signs and symptoms, such as paresthesia and those associated with intravascular injection.
- ✓ A confused, combative patient endangers themselves as well as the anesthesia provider.

If the sedation is inadequate, additional sedation may be required.

Maximum Local Anesthetic Doses

With peripheral nerve blocks that utilize small doses (i.e. digital blocks) the maximum dose of a local anesthetic is not a major concern. However, for other blocks it is an important consideration.

Local Anesthetic	Type	Onset	Duration	Maximum Dose Plain mg/kg	Maximum Dose with Epinephrine mg/kg
Mepivacaine	Amide	Fast	Moderate	4.5	7
Lidocaine	Amide	Fast	Moderate	4.5	7
Prilocaine	Amide	Fast	Moderate	8	-------
Bupivacaine	Amide	Moderate	Long	2.5	3
Ropivacaine	Amide	Moderate	Long	3	-------

In addition to maximum mg/kg doses, there are cumulative total dosages that should not be exceeded.

Local Anesthetic	Total Maximum Dose Plain	Total Maximum Dose with Epinephrine
Mepivacaine	300 mg	500 mg
Lidocaine	300 mg	500 mg
Prilocaine	500 mg	600 mg
Bupivacaine	175 mg	225 mg
Ropivacaine	200 mg	-------

Local Anesthetic Toxicity

Administering doses greater than what is recommended can result in the potentially devastating consequence of local anesthetic toxicity. In addition, local anesthetic toxicity can occur from accidental intravenous injection. Frequently aspirate for the presence of blood when administering peripheral nerve blocks. Absorption of local anesthetics into the vascular system occurs at different rates, depending on the anatomical location. The uptake of local anesthetic, from greatest to least, is as follows: IV> tracheal> intercostal> caudal> paracervical> epidural> brachial> sciatic> subcutaneous. Local anesthetics themselves vary in toxicity. From the least to most toxic local anesthetic: chloroprocaine< lidocaine < mepivacaine < levobupivacaine < ropivacaine < bupivacaine.

Steps to Reduce the Risk of Local Anesthetic Toxicity

Prevention is the best treatment.

- Choose the least toxic local anesthetic available.
- Carefully calculate local anesthetic doses on a mg/kg basis before starting any peripheral block.
- Never administer more than the maximum dose.
- Always check the concentration of local anesthetic. For example 10% lidocaine contains 100 mg per ml; 1% lidocaine contains 10 mg per ml. Simple mistakes should be avoided.
- Dilute the total dose of local anesthetic to the desired volume/concentration by mixing it with sterile, preservative free normal saline. Most peripheral nerve blocks are more dependent upon the volume of local anesthetic than total dose. Never add more local anesthetic to achieve the desired volume and risk administering a toxic dose.
- Epinephrine, in concentrations of 5-10 mcg/ml, is commonly used to decrease the absorption of local anesthetics. A 5 mcg/ml (1:200,000) dose of epinephrine will significantly reduce the peak blood levels of lidocaine, bupivacaine, etidocaine, and mepivacaine. Benefits of decreased absorption include increased neuronal uptake, enhanced quality of analgesia/anesthesia, prolonged duration of action, and decreased risk of toxicity.
- A concentration of 1:200,000 (5 mcg/ml) is commonly used for peripheral nerve blocks to reduce vascular absorption. To add epinephrine to a local anesthetic solution, use a 1mg/ml (1:1000) ampoule of epinephrine. Take the total volume of local anesthetic, divide it in half, and move the decimal point two places to the left. For example, 40 ml of 1% lidocaine- divide 40 by 2 and 20 is the result. Next, move the decimal point two places to the left. The result is 0.20. This is the amount of epinephrine added to the local anesthetic solution to yield a 1:200,000 concentration. To check the calculation, multiply 5 mcg/ml by 40 ml, which equals 200 mcg. It is important to always check the concentration of epinephrine and the total dose added to the local anesthetic.
- A second technique for adding epinephrine to local anesthetic preparations is detailed below:
  - ✓ 1:200,000 epinephrine concentration would equal 5 mcg/ml.

- ✓ Dilute epinephrine using a 10 ml syringe. Draw up 1 ml of 1:1000 epinephrine (1 mg per ml) and 9 ml of normal saline.
- ✓ Mix it by tilting the syringe back and forth.
- ✓ The concentration of epinephrine is now 100 mcg per ml.
- ✓ Add epinephrine to the local anesthetic solution (see table below).

1:200,000 Epinephrine Concentration	
Volume of Local Anesthetic	**Amount of Epinephrine Added to Local Anesthetic Solution**
20 ml	100 mcg of epinephrine
30 ml	150 mcg of epinephrine
40 ml	200 mcg of epinephrine
50 ml	250 mcg of epinephrine

- Always label the syringe of epinephrine. Once epinephrine is added to the local anesthetic, discard what remains. Epinephrine can be lethal and should be discarded to avoid inadvertent administration.
- Epinephrine containing local anesthetics should never be injected into end organs such as ears, nose, penis, fingers, or toes. Epinephrine may cause vasoconstriction and subsequent necrosis of tissue.
- Epinephrine should not be added to ropivacaine since it has inherent vasoconstrictive properties.
- The addition of epinephrine to local anesthetics may help identify an intravascular injection increasing heart rate and blood pressure.
- Aspirate for the presence of blood before and during a peripheral nerve block. Inject the local anesthetic slowly, at a rate of 10 ml/minute or less.
- Monitor the patient for signs and symptoms of intravenous injection. The patient should continually be monitored with an ECG, blood pressure, and pulse oximetry.
- Resuscitation medications/equipment should always be immediately available.

Local Anesthetic Toxicity Signs & Symptoms

When administering peripheral nerve blocks, it is important to converse with the patient. Ask, "How do you feel?" Be alert to signs and symptoms of intravascular injection. If there is any suspicion that local anesthetics are being injected intravascularly, stop immediately. Local anesthetic toxicity will primarily involve the central nervous and cardiovascular systems. Initial/early symptoms of central nervous system may include the following:

- Dizziness or feeling faint
- Confusion
- Abnormal taste in the mouth (sometimes described as "metallic")
- Numbness and tingling around the mouth
- Visual or hearing changes

- Ringing in the ears (tinnitus)
- Shivering, muscle twitching, and/or tremors

These symptoms can ultimately lead to the following:

- Tonic-clonic seizures
- Central nervous system depression
- Coma
- Respiratory depression/respiratory arrest

The onset of cardiovascular system toxicity will generally occur at higher blood concentrations than those associated with central nervous system symptoms (usually 4-7 times greater than the convulsant dose). The exception is bupivacaine. Local anesthetic toxicity of the cardiovascular system may exhibit the following symptoms:

- Tachycardia and increased blood pressure with epinephrine containing solutions
- Sinus arrest
- Sinus bradycardia
- Depressed contractility
- Ventricular fibrillation
- Cardiac arrest
- Hypotension

Treatment of Local Anesthetic Toxicity

If the patient experiences acute local anesthetic toxicity, it is important to remember the A (airway), B (breathing), and C's (circulation).

- A, B = (airway & breathing) maintain a patent airway, 100% oxygen, intubation and ventilation if patient becomes apneic.
- C = (circulation) hypotension should be treated with an open IV, Trendelenburg position, and vasopressors such as ephedrine (5-10 mg boluses) or phenylephrine (50-100 mcg boluses). If the patient is bradycardic, atropine should be administered in doses of 0.5 mg. For severe hypotension and bradycardia refractory to other treatments administer epinephrine. Start with boluses of 5-10 mcg and increase the dose as needed. Administer CPR as needed. Patients with bupivacaine induced ventricular fibrillation should be treated with CPR and defibrillation. CPR should be continued for at least 60 minutes. Bretyllium (5-10 mg/kg IV) may help convert the patient's ventricular fibrillation with cardioversion.
- C = convulsions/seizures should be treated with sodium pentothal in doses of 150-250 mg IV or diazepam 10-20 mg IV. Repeat as needed until convulsions/seizures stop.

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.

Recognition, vigilance, and prompt treatment are required for successful treatment of local anesthetic toxicity.

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Femoral Nerve Block

Chapter Seven

Femoral Nerve Block/3-in-1 Nerve Block

Femoral and/or 3-in-1 nerve blocks are used for surgical procedures on the front portion of the thigh down to the knee and postoperative analgesia. Both blocks are carried out in a similar manner. The differences will be discussed in the technique section.

Indications for Femoral Nerve Block/3-in-1 Nerve Block

Femoral Nerve Block

- Operations on the anterior thigh (i.e. lacerations, skin graft, muscle biopsy)
- Pin or plate insertion/removal (femur)
- Femur fractures
- Analgesia

3-in-1 Nerve Block

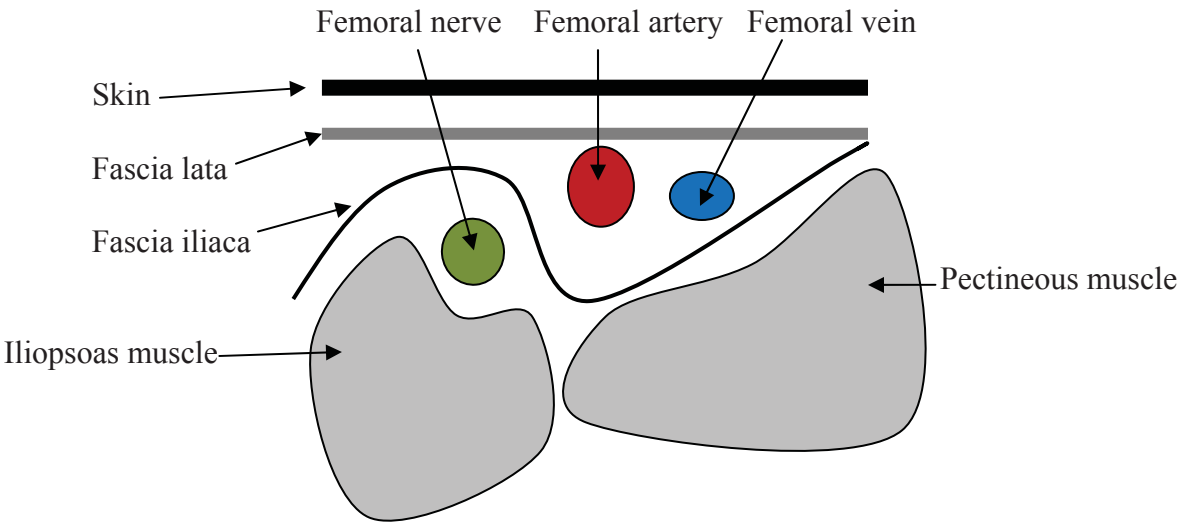
- Same indications as femoral nerve block
- Analgesia and anesthesia of the hip (dislocations, femoral neck fractures)
- Analgesia of the knee

Depending on the surgical procedure, femoral/3-in-1 nerve blocks may cover only part of the knee joint. The knee joint is innervated by the femoral, obturator, and sciatic nerve. Portions of the knee innervated by the sciatic nerve will not be covered. The hip joint is primarily innervated by the femoral, obturator, and lateral femoral cutaneous nerve. There is a small contribution from the sciatic nerve. Additional analgesia with intravenous opioids should be sufficient to cover the small sciatic contribution.

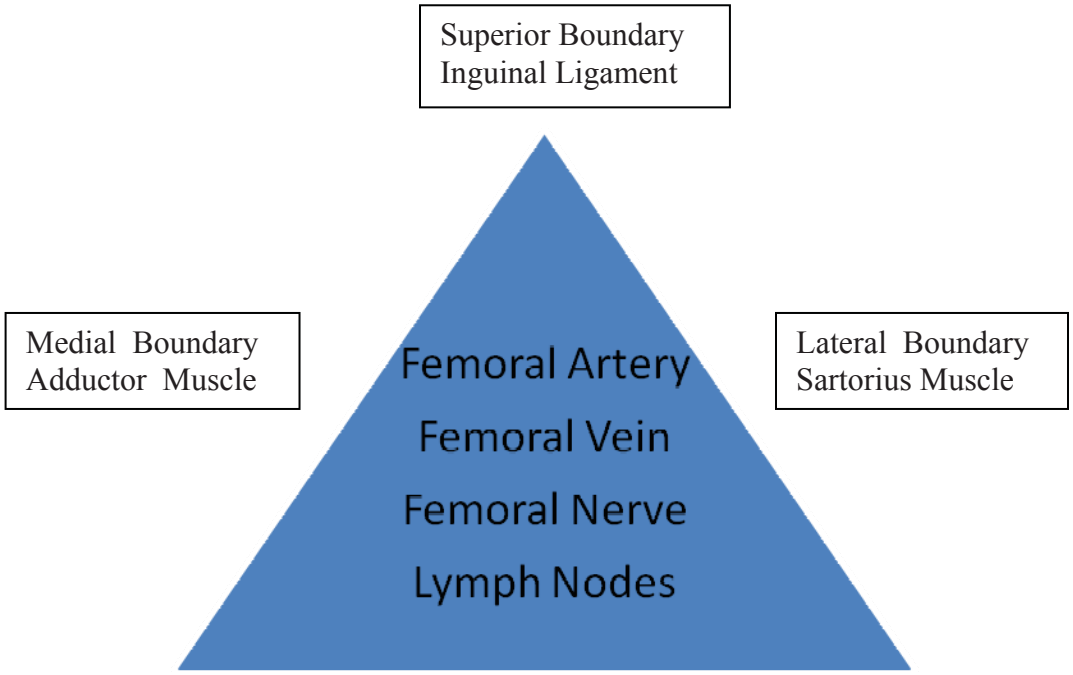
Anatomy

The femoral nerve is created by contributions from L2, L3, and L4 and is the largest branch of the lumbar plexus. The femoral nerve enters the thigh under the inguinal ligament, between the psoas and iliacus muscle, and is located below the fascia iliaca.

Anatomy



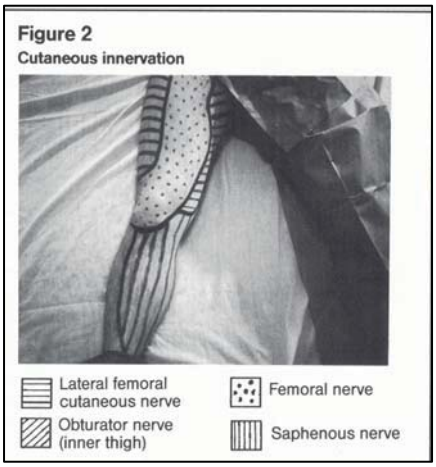
Below the inguinal ligament in the upper medial portion of the thigh is the femoral triangle. The femoral triangle contains the femoral nerve, artery, vein, and lymph nodes. The boundaries of the triangle include: superiorly – inguinal ligament, laterally- sartorius muscle, and medially- adductor muscle.



The lateral femoral cutaneous nerve is formed by contributions from L2 and L3. It is located more proximally than the femoral nerve, at the lateral border of the psoas muscle. The obturator nerve is formed from contributions of L2, L3, and L4. The obturator nerve is located at the medial border of the psoas muscle.

Innervations of the Femoral, Lateral Femoral Cutaneous, and Obturator Nerves

- Femoral Nerve- anterior and medial portion of the thigh and knee. Cutaneous innervation of the medial and lateral portion of the thigh. Periosteum of the femur.
- Lateral Femoral Cutaneous Nerve- sensory nerve to the lateral buttock, thigh, and knee joint.
- Obturator Nerve- sensory to the medial thigh, hip joint, and adductor muscle.



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Contraindications

- Burn or infection at injection site
- Inability to coagulate blood (congenital or acquired)
- Vascular graft of the femoral artery
- Neurological disease (relative contraindication)
- Patient refusal
- Local anesthetic allergy
- Inability to guarantee sterile equipment

Preparation

- Prepare the patient. Obtain a medical history, perform a brief physical exam, and review laboratory/other studies or tests. If elective, the patient should have fasted prior to surgery.

General anesthesia may be required if the block fails. Carefully explain the procedure, risks/benefits. Obtain consent from the patient to perform the procedure.

- Monitor the patient continuously with an ECG, blood pressure, and pulse oximetry.
- Intravenous access with a running IV.
- Emergency medications, airway/intubation equipment, and an oxygen source should be immediately available.
- Assemble local anesthetics, sterile equipment, and antiseptic agents.
- Consider light sedation. Over sedation may result in an uncooperative patient. In addition, it may mask signs and symptoms of intravenous injection of local anesthetics and/or intraneural injection.

Technique for Femoral Nerve Block/3-in-1 Nerve Block

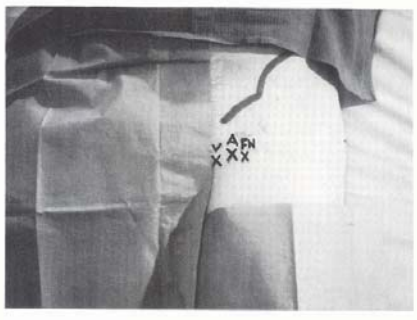
Techniques for both blocks are basically the same, with some minor alterations. There are several approaches to this block. Two techniques will be described here.

- Wash hands
- Position patient supine

Identify the landmarks.

- The femoral nerve is located just below the inguinal ligament. Locate the anterior superior iliac spine and the pubic tubercle. A line between these two structures is where the inguinal ligament is located.
- Next locate the pulsation of the femoral artery.
- The site for needle insertion is approximately 2 cm lateral to the pulsation. From medial to lateral the structures are femoral vein, femoral artery, and femoral nerve.

Figure 3
Landmarks



V—Femoral vein
A—Femoral artery
FN—Femoral nerve
Solid line—Inguinal ligament

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- Prepare the site with antiseptic
- Use sterile gloves; drape area with sterile towels
- Use a 21-23 gauge needle (should be blunted). A sharp "cutting" needle can transect a nerve.

Paresthesia Technique

- A small skin wheal of local anesthetic is placed at the identified injection site. The needle is inserted slowly in a perpendicular direction. The needle should be blunted to decrease the risk of neural trauma. As the needle is advanced, aspirate for blood. Once a paresthesia is noted in the distribution of the femoral nerve, withdraw the needle slightly and inject the local anesthetic. The patient should not feel pain during injection.
- If a depth of 4-5 cm has been reached with no paresthesia elicited, then withdraw the needle to the level of the skin and change the angle either slightly medially or laterally. Continue to seek a paresthesia.
- Always aspirate for blood during needle insertion before, during, and after injection to avoid an inadvertent intravascular injection.
- Paresthesia techniques carry a higher risk of nerve trauma than the two pop fascia iliaca technique.

Single Injection or "Two Pop" Technique (AKA Fascia Iliaca Block)

- Same landmarks as the femoral nerve block. First, identify the anterior superior iliac spine and draw a line to the outer portion of the pubic tubercle. Next, divide this line into thirds. Where the middle and lateral thirds meet move 2 cm down (below the line). This is the injection site.
- A small skin wheal of local anesthetic is placed at the identified injection site. A blunted needle should be used for this technique. If only "sharp" regular needles are available, then blunt the needle using the sterile plastic sheath that comes with the needle. Keep the needle sterile. The needle is inserted perpendicular. As the needle is advanced it is important to aspirate for blood.
- The needle should be held like a pencil. Short "jabs" will help to identify anatomical structures.
- As the needle is inserted there will be a slight increase in resistance, followed by a loss of resistance, or "pop". This indicates that the fascia lata has been crossed.
- Continue to insert the needle. There should be a second slight increase in resistance, followed by a loss of resistance, or "pop", which can be subtle and noted a few millimeters below the first "pop". This indicates that the fascia iliaca has been crossed. The femoral nerve is in this fascial plane.
- Stabilize the needle with the free hand so the needle does not move out of the fascial plane.

- Always aspirate during needle insertion before, during, and after injection, to ensure that inadvertent intravascular injection does not occur.
- If aspiration is negative, inject the local anesthetic.
- Consider redefining the fascial plane after injecting half of the intended dose of local anesthetic. The volume of local anesthetic can potentially "push" the needle out of the fascial plane.
- If the patient experiences pain or paresthesia with injection, withdraw the needle slightly. Continue with injection as long as there is no pain or paresthesia.
- Never point the needle up. This may cause the needle to go through the inguinal ligament and into the abdomen.

Differences between Femoral Nerve Block and 3-in-1 Nerve Block

There are two main differences.

1. Volume of local anesthetic. For femoral nerve blocks, the volume of local anesthetic is generally 20 ml or less. For 3-in-1 nerve blocks, the volume of local anesthetic is 25-30 ml. This allows the local anesthetic to spread further in the tissue plane resulting in blockade of the femoral, lateral femoral cutaneous, and obturator nerve.
2. Slight alteration in technique. Once the needle has been placed in the correct area, pressure should be applied 2-4 cm below the injection site. Next, administer the local anesthetic. Applying distal pressure helps spread the local anesthetic to the obturator and lateral femoral cutaneous nerve, in addition to the femoral nerve.

Local Anesthetics

A number of local anesthetics may be used for femoral and 3-in-1 nerve blocks. In general, the volume of local anesthetic for a femoral nerve block will range from 15-20 ml. For 3-in-1 nerve block, the volume ranges from 25-30 ml. The addition of epinephrine 1:200,000 or 5 mcg/ml will reduce absorption of the local anesthetic and prolong the duration of action. In addition, epinephrine may alert the anesthesia provider to an inadvertent intravascular injection before major complications occur. The anesthesia provider should always be aware of the maximum dose of local anesthetic for each patient. 1-2% lidocaine will have an onset of 10-20 minutes and last 2-5 hours for anesthesia and up to 8 hours for analgesia. The maximum dose of plain lidocaine is 4.5 mg/kg, or a total of 300 mg. The maximum dose of lidocaine with 1:200,000 epinephrine is 7 mg/kg, or a total of 500 mg. 0.25-0.5% bupivacaine will have an onset of 15-30 minutes and last 5-15 hours for anesthesia and up to 30 hours for analgesia. The maximum dose of plain bupivacaine is 2.5 mg/kg, or a total of 175 mg. The maximum dose of bupivacaine with 1:200,000 epinephrine is 3 mg/kg, or a total of 225 mg. If available, ropivacaine is a safer alternative to bupivacaine.

Complications

Vigilance during block placement is essential. Monitor the patient continuously with ECG, blood pressure, and pulse oximetry. Communicate with the patient during the block. Be prepared for potential complications. Complications include the following:

- Intravascular injection
- Local anesthetic toxicity (central nervous system and cardiovascular toxicity)
- Nerve trauma to the femoral nerve, resulting in transient or permanent injury
- Prolonged motor blockade of the muscles of the thigh
- Hematoma formation
- Block failure

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Brachial Plexus Anesthesia

Chapter Eight
Brachial Plexus Anesthesia

Four approaches to the brachial plexus include the interscalene, supraclavicular, infraclavicular, and axillary approach. The supraclavicular and infraclavicular approaches have a higher incidence of complications. For the purposes of this manual, the interscalene and axillary approaches will be covered. Nerve stimulator techniques can improve on the success rate and enhance patient safety. However, the specialized equipment is expensive and unavailable in most developing countries. For the purposes of this manual, other techniques will be discussed. The approach to brachial plexus anesthesia is largely dependent upon the surgical procedure. Each approach may "miss" a nerve distribution requiring supplementation. Peripheral nerve blocks at the elbow, wrist, and digits can supplement the missed nerve, as well as be used for minor surgical procedures of the hand and fingers.

Anatomical Consideration and Brachial Plexus Anesthesia

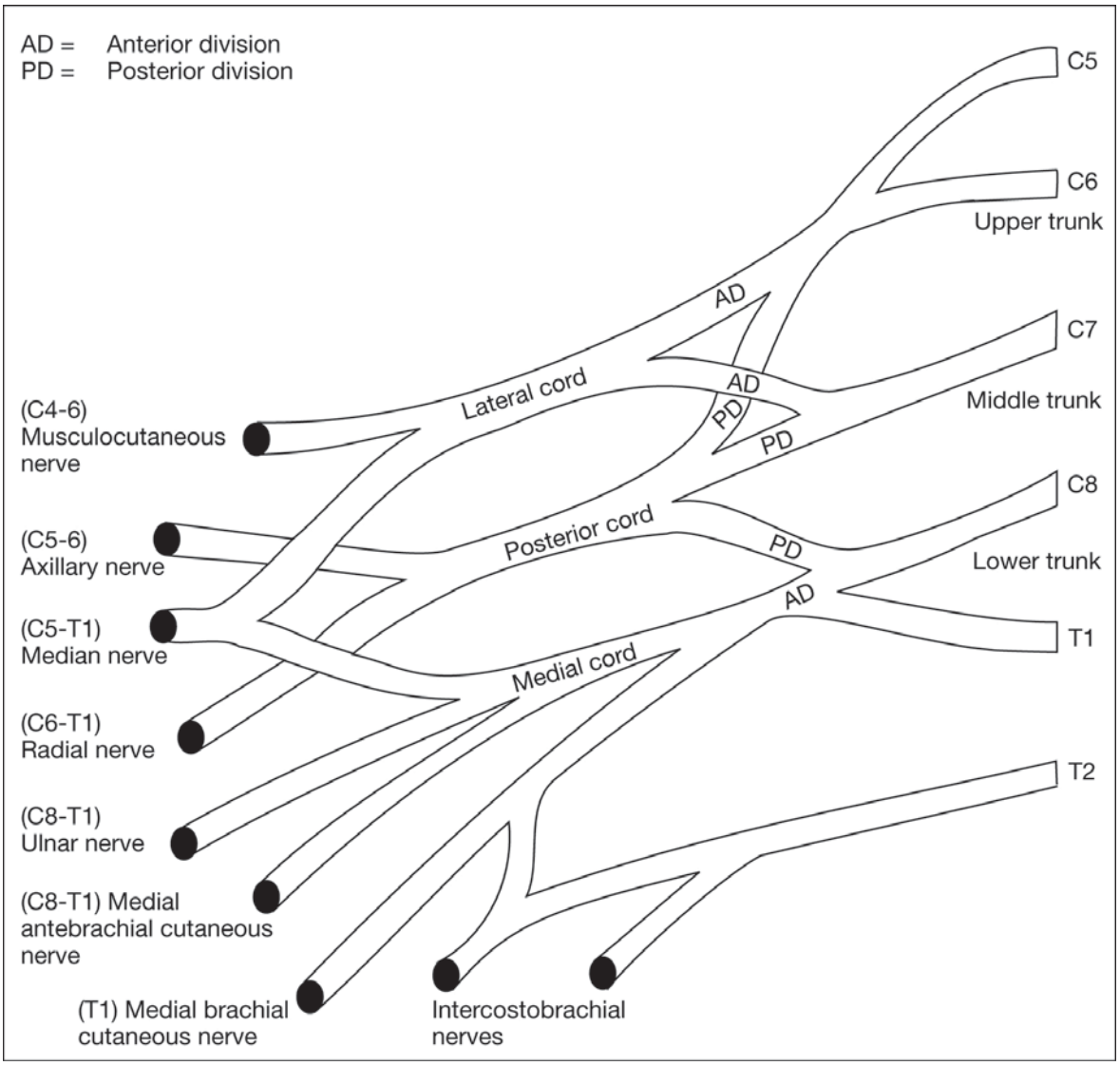
The brachial plexus is created by distributions from C5 to T1. Blockade of the brachial plexus can provide surgical anesthesia of the hands, upper/lower arm, and shoulder depending on the approach. A thorough knowledge of anatomy and its impact on the two techniques is important for success. Peripheral nerve blocks may be required to supplement brachial plexus anesthesia. For example, when a tourniquet is used, the medial brachial cutaneous (C8-T1) and intercostobrachial (T2) should be blocked to prevent tourniquet pain. The medial brachial cutaneous and intercostobrachial nerve innervate the medial and posterior portions of the proximal arm and are not located within the brachial plexus sheath. The medial brachial cutaneous nerve leaves the sheath just below the clavicle. For shoulder surgery, a portion of the anterior shoulder is innervated by the superficial cervical plexus (C1-C4). A field block, along the posterior border of the sternocleomastoid will effectively block the superficial cervical plexus.

Additional anatomical considerations will be discussed with each technique.

Anatomy

The anatomy of the brachial plexus is relatively complicated. The brachial plexus is primarily formed by ventral rami of C5-T1. C4 and T2 make minor contributions.

Brachial Plexus Anatomy



This is the most common anatomical configuration. Variation may occur among patients.

Contributions from C5-T1 come together, then separate to form trunks, divisions, cords, and main branches. At the level of the anterior and middle scalene muscles the trunks are already formed.

Upper Trunk C5-6
Middle Trunk C7
Lower Trunk C8-T1

Upper, middle, and lower trunks course over the lateral border of the first rib and under the clavicle. At this point, each trunk will separate into anterior and posterior divisions. As the brachial plexus emerges under the clavicle, the anterior and posterior divisions come together to form three cords. The lateral cord is lateral to the axillary artery; the posterior cord is located posterior to the axillary artery; and the medial cord is located medial to the axillary artery.

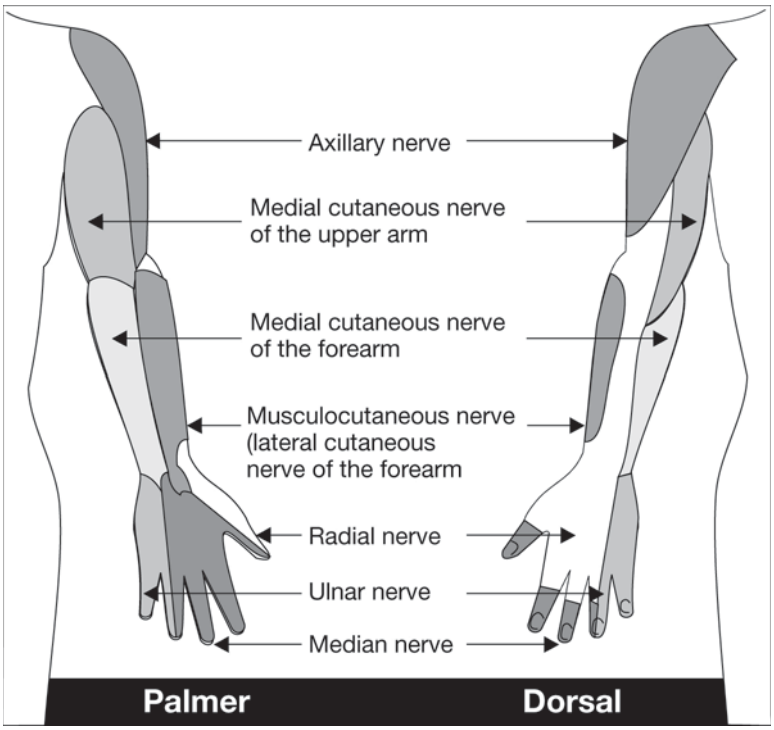
Lateral Cord: formed by anterior divisions of the upper and middle trunks.
Posterior Cord: formed by posterior divisions of all three trunks.
Medial Cord: formed by anterior division of the lower trunk.

At the lateral border of the pectoralis minor muscle, each cord divides into branches, terminating in individual nerves.

Lateral Cord: divides into the lateral branch of the median nerve and terminates in the musculocutaneous nerve.
Posterior Cord: divides into the axillary nerve and terminates in the radial nerve.
Medial Cord: divides into the medial branch of the median nerve and terminates in the ulnar nerve.

The brachial plexus is enveloped by a fascial sheath, formed by prevertebral and scalene fascia, extending from the intervertebral foramina to the upper arm. The formation of a sheath allows for the administration of brachial plexus anesthesia. Injection into the sheath, at any anatomical point, will allow for the spread of local anesthetics and subsequent blockade. Each approach to the brachial plexus impacts specific anatomical areas of the upper extremity. Choice of a specific technique should be made based on the surgical procedure.

Cutaneous Nerve Supply of the Arms



This is the most common anatomical configuration. Variation may occur among patients.

Major Motor Function of the Individual Nerves of the Brachial Plexus

Nerve	Major Motor Function
Axillary Nerve	Abduction of the shoulder
Musculocutaneous Nerve	Flexion of the elbow
Radial Nerve	Extension of the elbow, wrist, and finger
Median Nerve	Flexion of the wrist and finger
Ulnar Nerve	Flexion of the wrist and finger

Choosing a Technique for Brachial Plexus Blockade

The approach to brachial plexus anesthesia is based on anatomical knowledge and surgical procedure.

Interscalene Approach: excellent technique for surgical procedures on the shoulder, arm, and forearm. Intense block at C5-C7 and diminished blockade of C8-T1. Not a good technique for surgical procedures that involve the ulnar nerve distribution.
Axillary Approach: excellent technique for surgical procedures from the elbow to the hand. There is intense blockade of C7-T1. This approach is not adequate for the shoulder and upper arm (C5-6).

Interscalene Approach to the Brachial Plexus

Indications

The interscalene approach to the brachial plexus is a suitable technique for the following situations:

- Surgical procedures of the shoulder and upper arm
- Surgical procedures of the hand (will need to supplement the ulnar nerve distribution)
- Reduction of a dislocated shoulder, arm, and wrist fractures

Advantages & Disadvantages

Advantages of the interscalene approach include the following:

- Ability to perform surgical procedures of the shoulder and upper arm
- Avoiding complications associated with general anesthesia
- Muscle relaxation for the surgeon
- Postoperative analgesia
- Do not need to abduct the arm and flex at 90 degrees at the elbow

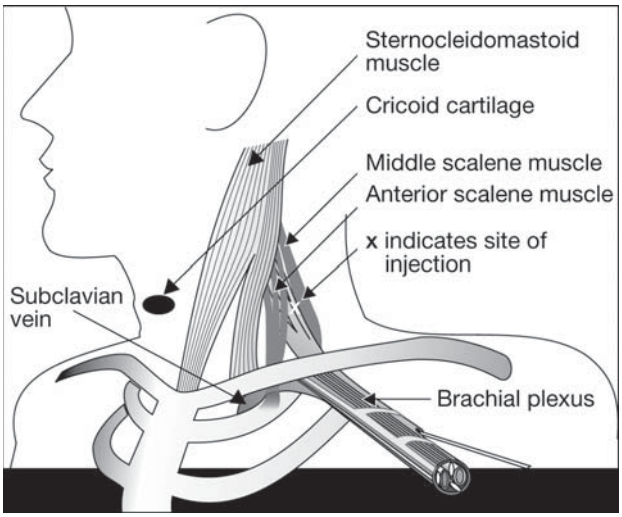
Disadvantages of the interscalene approach include the following:

- Moderate in complexity
- Need to supplement the ulnar nerve distribution for lower arm and hand surgery.
- Potentially serious complications can occur. Complications include inadvertent epidural/subarachnoid injection, vertebral artery injection, and pneumothorax.
- Common side effects may be distressing to the patient. These include blockade of the phrenic, recurrent laryngeal, vagus, and cervical sympathetic nerves.

Equipment

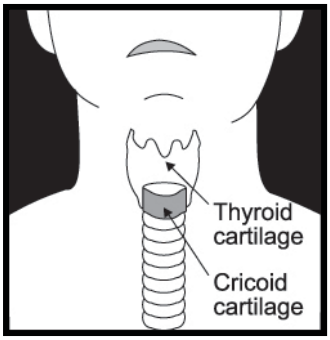
- Antiseptic solutions (i.e. betadine, hibiclens, and/or alcohol)
- 22 or 23 gauge, 4 cm, short beveled needle (for paresthesia technique)
- Sterile towels or drapes

Technique



The upper, middle, and lower trunks of the brachial plexus are found between the anterior and middle scalene muscles. This is called the interscalene groove. The trunks are named based on their vertical arrangement within the interscalene groove. The interscalene groove is found at the level of the cricoid cartilage. The external jugular vein will cross the area of the interscalene groove at the level of the cricoid cartilage. A common mistake, when first attempting this approach, is to confuse the groove between the sternocleomastoid and anterior scalene muscle. This groove is anterior to the interscalene groove.

Identification of the interscalene groove is carried out by placing the patient supine. Ask the patient to turn their head 30 degrees or less towards the non-operative extremity. Identify the cricoid cartilage. Identify the posterior border of the sternocleomastoid muscle. Slide your fingers posteriorly. The anterior scalene muscle will be noted just below the posterior edge of the sternocleomastoid muscle. Just posterior to the anterior scalene muscle, the middle scalene muscle is located. The interscalene groove is located between the anterior and middle scalene muscles. If difficulties are encountered identifying the interscalene groove, have the patient lift their head against light pressure, and/or take a deep breath. This will help the anatomical structures to "stand out" and be identified. Place a skin wheal, insert a 22 gauge, 4 cm, short bevel needle perpendicular to the skin. Advance the needle medially and caudally (towards the feet) at a 45 degree angle until a paresthesia is evoked in the arm below the elbow. The needle should not be inserted more than 2.5 cm.



Once the correct area is identified, a total of 30-40 ml of local anesthetic is injected. Frequent aspiration should occur during injection, ensuring an intravenous injection does not occur. Do not inject if the patient experiences pain or it is difficult to inject the local anesthetic. It is important not to exceed the maximum doses of local anesthetic. Digital pressure, applied proximally, may help with distal spread of the local anesthetic.

Local Anesthetics

Lidocaine, ropivacaine, and bupivacaine are commonly administered for the interscalene approach. Epinephrine will prolong the duration of action. An interscalene block, with lidocaine will have an onset of 5-15 minutes, an anesthetic duration of 3-6 hours, and provide analgesia for 5-8 hours.

Bupivacaine and ropivacaine will have an onset of 20-30 minutes, an anesthetic duration of 8-10 hours, and provide analgesia for 16-18 hours. Ropivacaine is a safer alternative to bupivacaine.

Complications

The complication rate for interscalene blocks is relatively high. The patient should be informed of what to expect and potential complications.

- The stellate ganglion, phrenic, and recurrent laryngeal nerve are near the brachial plexus. Patients with a history of chronic or acute pulmonary conditions should not have an interscalene block. The phrenic nerve is commonly blocked with this approach, which may result in respiratory failure. Horner's syndrome is common and may be distressing to the patient. Horner's syndrome manifests itself as miosis (contraction of the pupil), ptosis (drooping of the upper eyelid), and anhidrosis (diminished or absence of sweating). In addition, the patient may experience dyspnea and hoarseness.
- Intra-arterial injection may occur due to the proximity of the vertebral artery to the site of injection. Aspiration prior to and during injection is essential. A small dose of 1-3 ml will lead to seizures since the vertebral artery goes directly to the brain.
- Inadvertent venous injection will result in central nervous/cardiovascular system toxicity.
- Close proximity of the cervical neural foramina can result in an epidural, subarachnoid, or subdural injection.
- Pneumothorax can occur from inserting the needle too far laterally.

Axillary Approach to the Brachial Plexus

Indications

The axillary approach to the brachial plexus is suitable for the following:

- Surgical procedures of the lower arm and hand
- Reduction of lower arm, wrist, and hand fractures

Advantages & Disadvantages

Advantages of the axillary approach include the following:

- Ability to perform surgical procedures of the lower arm and hand
- Avoiding complications associated with general anesthesia
- Muscle relaxation for surgical purposes
- Postoperative analgesia
- Relatively low risk procedure
- Easy to master
- Arm must be abducted and flexed 90 degrees at the elbow

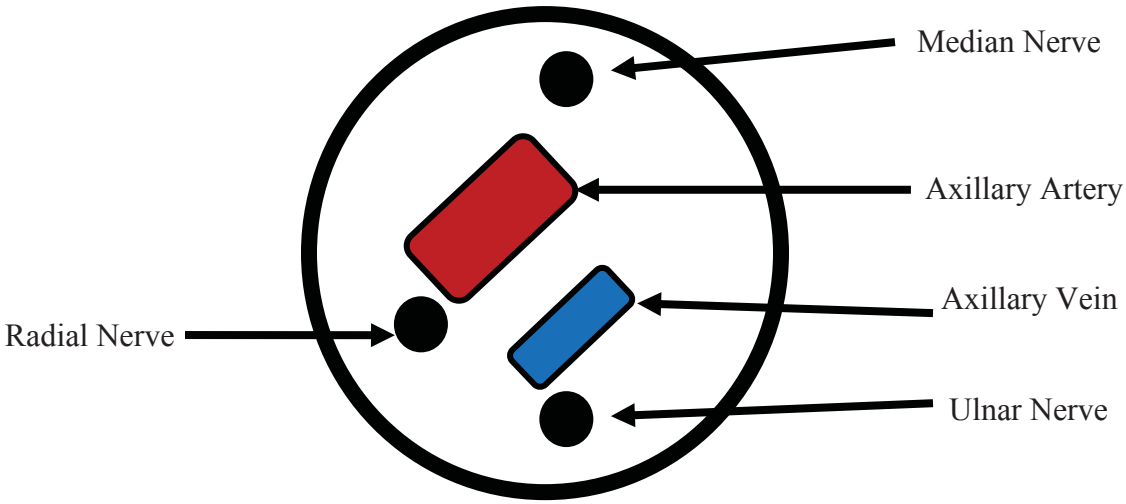
Disadvantages of the axillary approach include the following:

- May need to supplement "missed" nerve distributions
- Potentially serious complications (i.e. inadvertent intravascular injection and nerve injury)

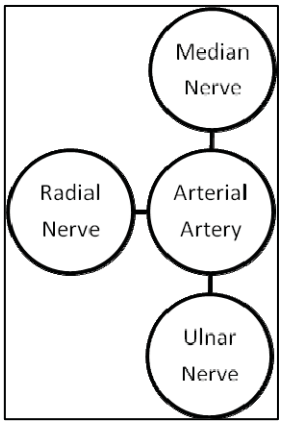
Anatomical Considerations

Beneath the clavicle, the subclavian artery becomes the axillary artery. The brachial plexus splits from upper, middle, and lower trunks into anterior and posterior divisions. The anterior and posterior divisions travel to the lateral border of the pectoralis minor and form the lateral, posterior, and medial cords. The cords split to form individual nerves that innervate the arm. The musculocutaneous nerve leaves the sheath prior to entering the axilla. The musculocutaneous nerve can be found within the coracobrachialis muscle, which is located immediately below the bicep. The musculocutaneous nerve should be blocked separately to ensure anesthesia. The brachial plexus sheath contains fascial sheaths and septa. This may result in patchy anesthesia by impeding spread of local anesthetic.

Knowledge of where each individual nerve is located within the sheath is important. The median nerve is superior to the axillary artery. The ulnar nerve is inferior to the axillary artery. The radial nerve is inferior and posterior to the axillary artery. The musculocutaneous nerve has already separated from the brachial plexus, traveling within the coracobrachialis muscle.



Schematic drawing of the contents of the brachial plexus sheath at the axilla



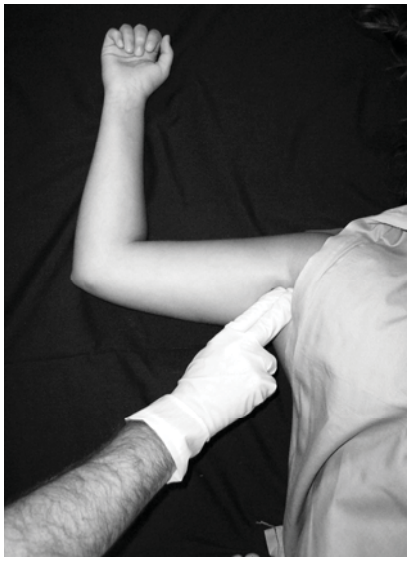
Another technique is to picture anatomical structures as a wheel. The "hub cap" is the arterial artery identified by palpation. The top is the median nerve, bottom is the ulnar nerve, while the side is the radial nerve.

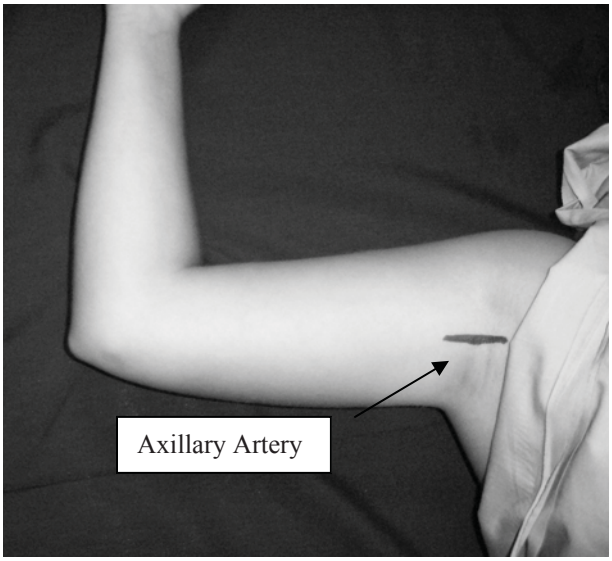
Equipment

- Antiseptic solutions such as betadine, hibiclens, and alcohol
- 22 or 25 gauge, 4 cm, short beveled needle (for paresthesia and transarterial techniques)
- Sterile towels or drapes

Technique

There are several approaches to the brachial plexus block at the axilla including transarterial and paresthesia.





Identification of the axillary artery is accomplished by palpation.

Transarterial Technique

1. Position patient supine with arm abducted and flexed 90 degrees at the elbow.
2. Palpate the axillary pulse as proximal in the axilla as possible.
3. Use a 22 or 25 gauge, 4 cm blunted needle. Advance it while aspirating.

4. Once blood is aspirated, either go "through" the artery or pull back out of the artery. Once aspiration is negative, inject a total of 35-40 ml of local anesthetic in a normal sized adult. Aspirate every 5 ml to ensure that the needle has not inadvertently entered an artery or vein. Monitor for signs and symptoms of intra-arterial injection including increased heart rate, "funny" metallic taste, faintness, seizures, etc. Some clinicians will inject half of the total local anesthetic dose posterior to the artery and the other half anterior to the artery. Ensure that the maximum dose of local anesthetic is not exceeded.
5. Distal pressure will help "push" the local anesthetic proximally.
6. Bring the arm down, hold pressure for up to 5 minutes. Ensure the patient does not have a hematoma forming.

Paresthesia Technique

1. Position patient supine with arm abducted and flexed 90 degrees at the elbow.
2. Palpate the axillary pulse as proximal in the axilla as possible.
3. Use a 22 or 25 gauge, 4 cm blunted needle. Advance it while aspirating.
4. Insert the needle towards the artery, but do not puncture it. If blood is aspirated, redirect the needle until a paresthesia is noted.
5. Knowledge of where each individual nerve is located within the sheath (as noted earlier in the schematic diagram of the brachial plexus sheath) is important.
6. Some clinicians choose any paresthesia of the arm or hand as an indication that they are in the sheath. Some will attempt to elicit a paresthesia in the operative distribution. Other clinicians will attempt to elicit a paresthesia in each distinct nerve distribution, injecting local anesthetic with each paresthesia.
7. Aspirate for blood prior to and during injection. It is important to ensure that the needle has not inadvertently entered a vessel. Do not continue to inject if the patient complains of pain or high injection pressures are required. Intra-neural injection can result in transient or permanent injury.

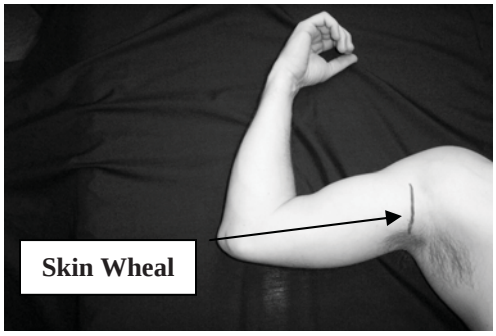
Musculocutaneous Block

Regardless of the technique used, it is essential to block the musculocutaneous nerve separately. The musculocutaneous nerve separates from the brachial plexus prior to its entrance into the axilla. The musculocutaneous nerve innervates the biceps and brachialis muscles, providing sensory innervation to the lateral forearm and wrist. Blockade of this nerve can be accomplished by redirecting the needle superiorly and proximally, piercing the belly of the coracobrachialis muscle, and inject 5-10 ml of local anesthetic.

Intercostobrachial and Medial Brachial Cutaneous Nerve Blocks

If a tourniquet is used, it is essential to block the intercostobrachial (T2) and medial brachial nerves (C8-T1). These two nerves are found superficially. Both provide superficial sensation of the medial

and posterior portions of the upper arm. A simple "skin wheal" of local anesthetic will reliably block these nerves.



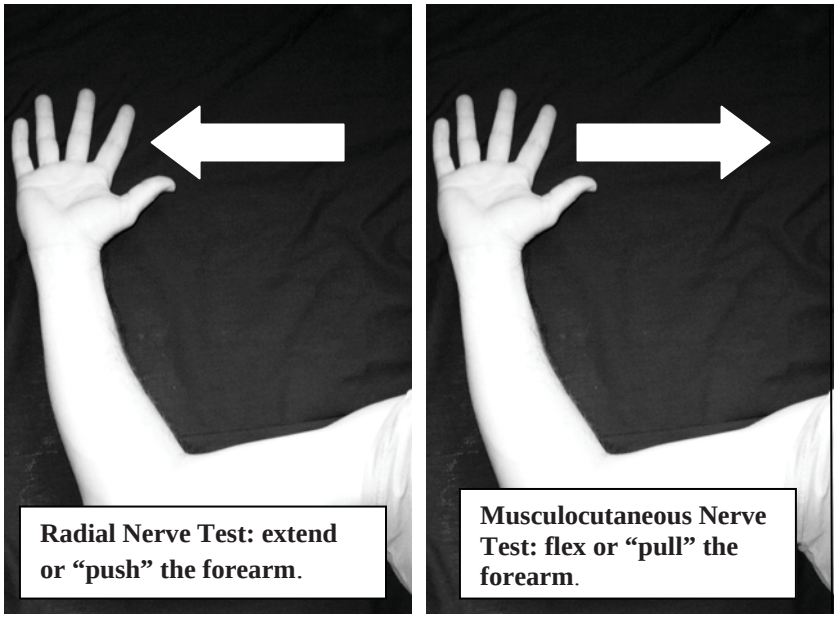
Intercostobrachial and medial brachial cutaneous nerve block

A Simple, Quick, and Easy Test an Axillary Block

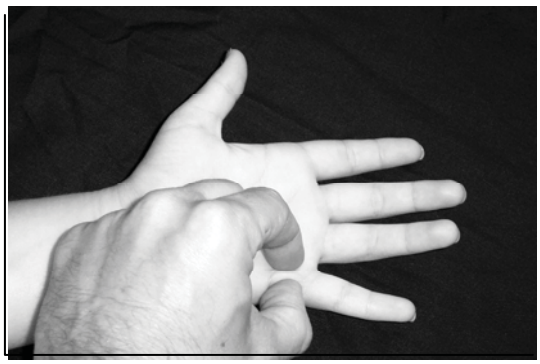
To quickly assess the block, perform the "push-pull-pinch-pinch" test. This test can be done in less than a minute. It can identify "missed" nerves allowing time to formulate an alternative plan of anesthesia (i.e. supplementation or general anesthesia).

- Ask the patient to extend or "push" their forearm against light resistance. This tests the radial nerve.
- Ask the patient to flex or "pull" the arm towards the nose against light resistance. This will test the musculocutaneous nerve.
- Pinch the thenar aspect of the hand (i.e. on the palmar surface of the hand). This will test the median nerve.
- Pinch the hypothenar aspect of the hand (i.e. small finger). This will test the ulnar nerve.

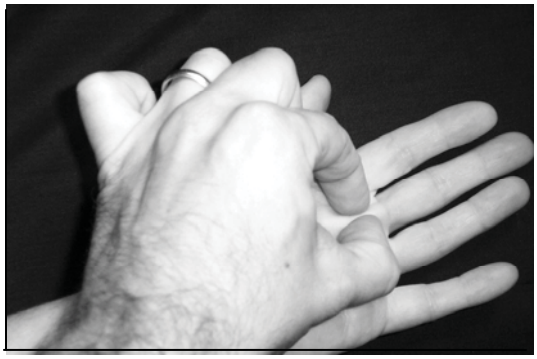
"Push and Pull"



"Pinch, Pinch"



Pinch the ulnar nerve distribution.



Pinch the median nerve distribution.

Local Anesthetics

Lidocaine, mepivacaine, or ropivacaine are commonly used for the axillary approach. Epinephrine will prolong the length of blockade with lidocaine and mepivacaine. Epinephrine is not added to ropivacaine due to its inherent vasoconstrictive properties. An axillary block with lidocaine/mepivacaine will have an onset of 5-15 minutes, an anesthetic duration of 2.5-4 hours, and provide analgesia for 3-6 hours. Ropivacaine will have a longer onset of 20-30 minutes, anesthetic duration of 6-8 hours, and provide analgesia for 8-12 hours. Many clinicians avoid bupivacaine secondary to the risk of cardiac arrest associated with inadvertent intravascular injection. It is important to calculate and not exceed the maximum dose of local anesthetic. The volume of local anesthetic varies from patient to patient depending on age and weight. For example, an adult can have 30-40 ml (smaller volumes if the patient weighs less than 70 kg), and teenagers who weigh 40-60 kg can have 25-30 ml.

Complications

- The incidence of complications is very low
- Intravascular injection is a potentially catastrophic complication. With careful and deliberate aspiration and monitoring, it can be avoided.
- Risk of nerve trauma secondary to eliciting repeated paresthesias'.
- Hematoma and infection are rare complications. Careful preparation of the site with antiseptic will decrease the risk of infection. Holding pressure at the site after penetrating the vessel will reduce the risk of hematoma.

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Peripheral Nerve Blocks at the Elbow, Wrist, and Digits

Chapter Nine

Peripheral Nerve Blocks at the Elbow, Wrist, and Digital Block

Peripheral nerve blocks at the elbow, wrist, or digits are easy to perform and useful in the clinical setting.

Indications

- Supplementing brachial plexus anesthetics that "missed" a nerve
- Postoperative pain relief after a Bier block
- Minor surgical procedures of the hand and fingers
- Closed reduction of the digits
- Analgesia for traumatic injuries of the hand or digits

Advantages & Disadvantages

Advantages include the following:

- Easy to administer
- Low incidence of block failure
- Safe technique when used appropriately
- Rapid onset
- Avoid complications associated with general anesthesia

Disadvantages include the following:

- Must have an intimate knowledge of anatomy
- Patient will have full motor control
- Muscles will not be relaxed
- Can not use an arm tourniquet due to tourniquet pain

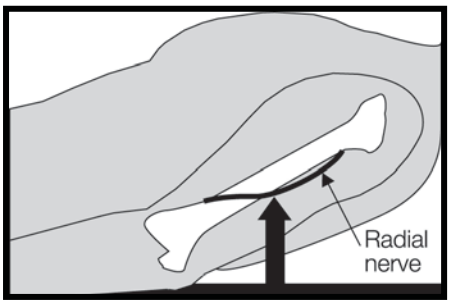
Complications

- Intraneural injection- never inject if the patient complains of pain/paresthesia or there is resistance to injection.
- Intravascular injection- always aspirate before, during, and after injection to ensure a vessel or artery has not been entered.
- Infection- cleanse site with antiseptic, maintain sterility

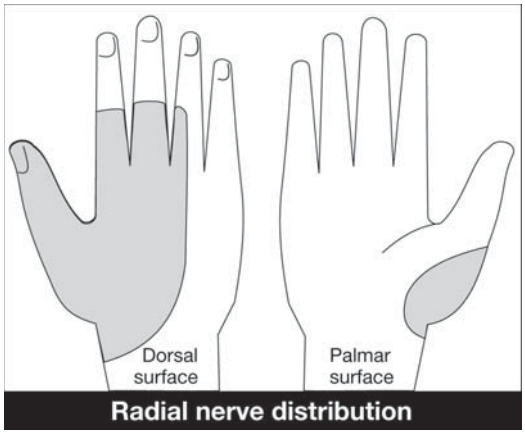
Peripheral Nerve Blocks at the Elbow and Wrist

When performing brachial plexus anesthesia techniques, there is always the risk that a nerve distribution may be "missed". If it is not in the surgical area then it is not a major concern. If it is, the ability to supplement the block is essential to a successful anesthetic. In addition, minor surgical procedures may be carried out by blocking individual nerves or a combination of nerves at the elbow and/or wrist. Peripheral nerve blocks require sterile equipment and antiseptic solution. Be careful not to contaminate the area of needle insertion.

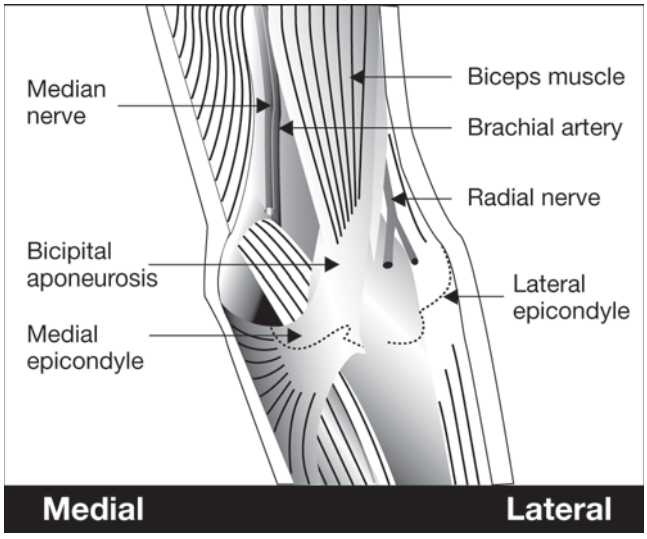
Radial Nerve



The posterior cord of the brachial plexus forms the radial nerve. The radial nerve separates from the brachial plexus sheath, traveling posterior to the humerus, innervating the triceps muscle. The radial nerve then travels to the lateral side of the elbow. Sensory branches, at this point, include the lateral cutaneous nerve of the arm and posterior cutaneous nerve of the forearm. (Refer to the illustration of the cutaneous innervation of the arm.) At the lateral epicondyle of the elbow, the radial nerve divides into superficial and deep branches. The deep branch innervates the postaxial extensor muscles of the forearm. The superficial branch supplies sensation to the dorsal wrist and lateral three and a half fingers.



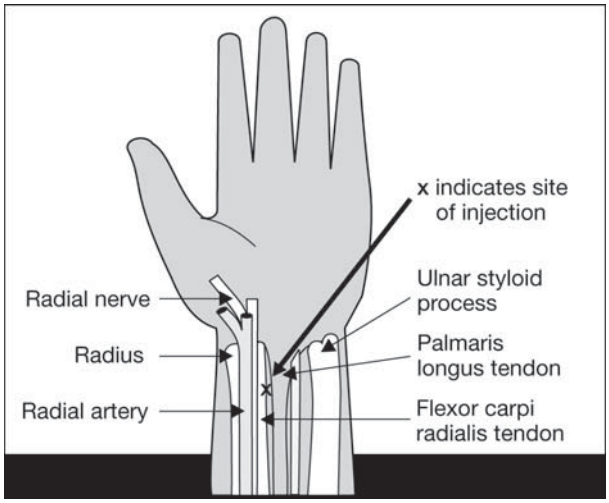
Blockade of the Radial Nerve at the Elbow

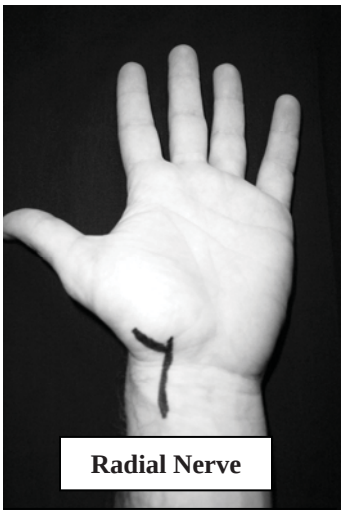


Identify the lateral aspect of the biceps tendon at the crease of the elbow. Insert a 22-27 gauge, 4 cm blunted needle parallel to the forearm. Direct the needle toward the lateral epicondyle. If a paresthesia is encountered, withdraw the needle slightly, and inject 5 ml of local anesthetic. Do not inject if the patient complains of a paresthesia. If no paresthesia is encountered, continue to insert the needle until bone is encountered. Withdraw the needle 1 cm and inject 5 ml of local anesthetic.

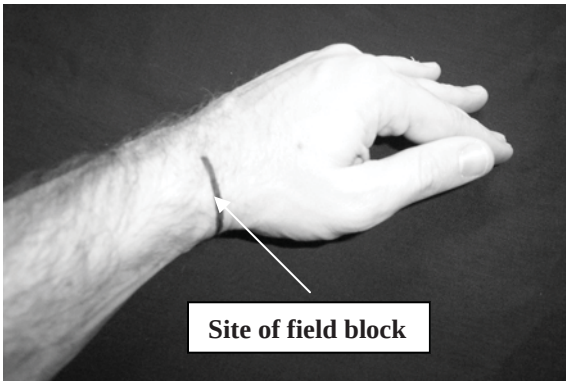
Blockade of the Radial Nerve at the Wrist

At the wrist, the radial nerve is located between the radial artery and flexor carpi radialis tendon. The administration of 2-5 ml of local anesthetic, deep to the flexor carpi radialis will block the sensory branches of the radial nerve which innervate the lateral side of the thumb. Proximal to the wrist, dorsal branches of the radial nerve provide sensation to the dorsal aspect of the lateral three and half fingers. This branch can be blocked by placing a superficial field block at the wrist.





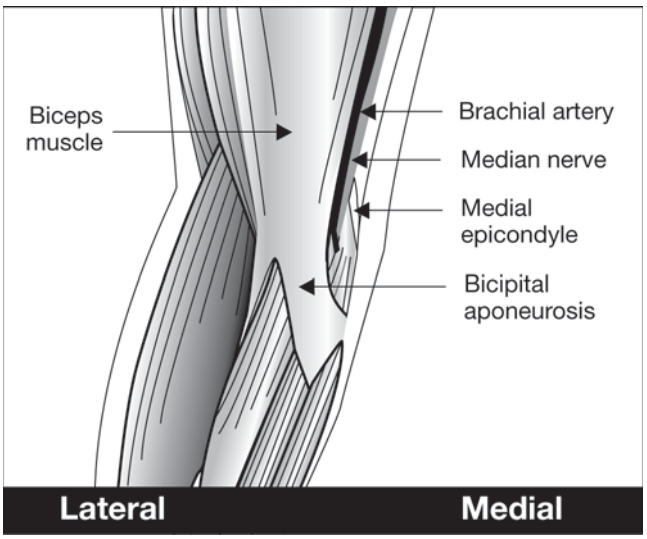
Field Block for the Superficial Radial Branches



Median Nerve

Lateral and medial cords of the brachial plexus contribute to the formation of the median nerve. The median nerve is medial to the brachial artery as it travels down the upper arm. At the elbow, the median nerve is located medial to the brachial artery near the insertion of the biceps tendon. It has several motor branches innervating portions of the wrist and finger flexors. The median nerve at the wrist is located beneath the palmaris longus tendon in the carpal tunnel.

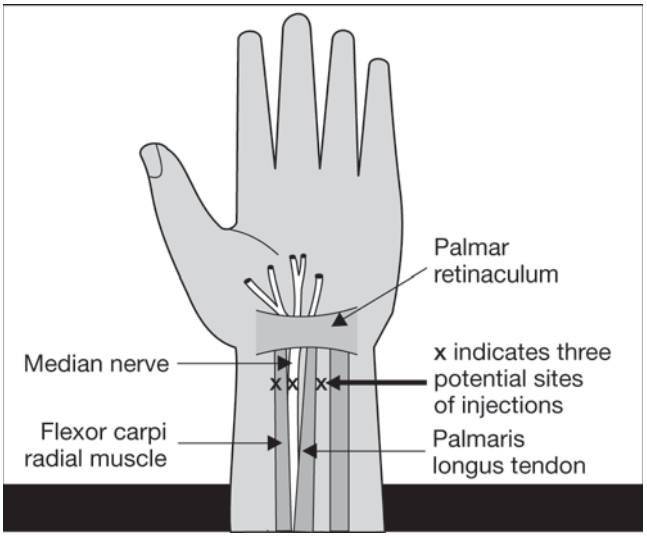
Blockade of the Median Nerve at the Elbow

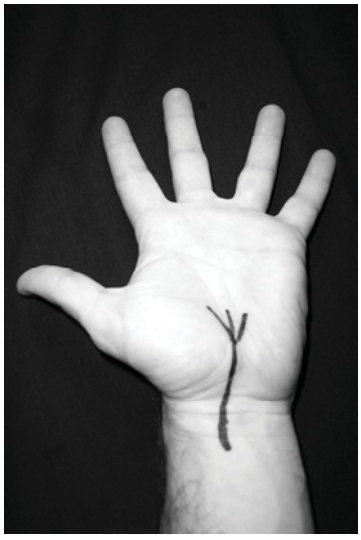


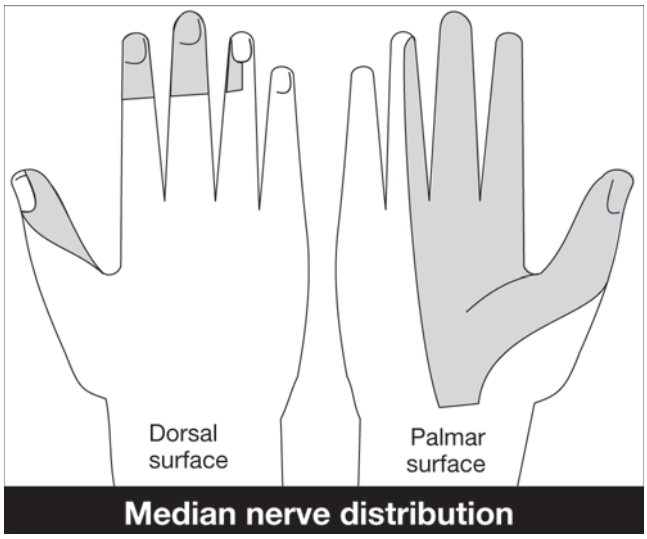
Identify the brachial artery in the antecubital space. It is generally located medially, at the biceps tendon insertion. Insert a 22-27 gauge, 4 cm blunted needle medial to the brachial artery. Direct the needle toward the medial epicondyle. If a paresthesia is encountered, withdraw the needle slightly and inject 3-5 ml of local anesthetic. If a paresthesia is not encountered, continue insertion of the needle until bone is contacted. At this point withdraw the needle 1 cm and inject 3-5 ml of local anesthetic.

Blockade of the Median Nerve at the Wrist

Identify the palmaris longus tendon by asking the patient to flex their wrist against resistance. Insert a 22-27 gauge blunted needle medial and deep to the palmaris longus. Alternatively, injection of local anesthetic into the flexor carpi radialis tendon or between the flexor carpi ulnaris and palmaris longus may carry a lower incidence of nerve trauma while being equally effective. If a paresthesia is encountered, withdraw the needle slightly and inject 3-5 ml of local anesthetic. If no paresthesia is encountered, infiltrate the area with 3-5 ml of local anesthetic.



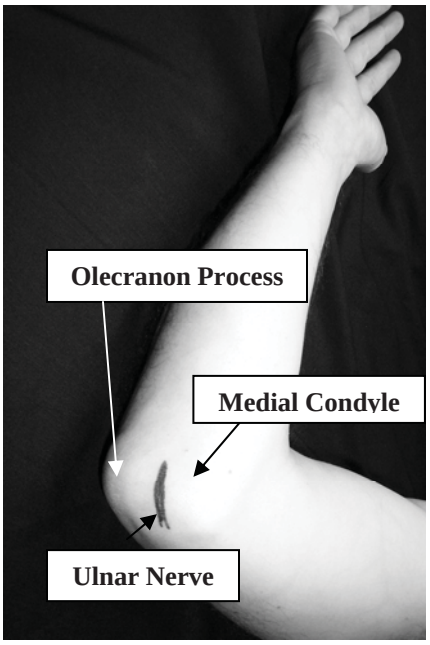




Ulnar Nerve

The medial cord of the brachial plexus contributes to the formation of the ulnar nerve. The ulnar nerve is located medial to the axillary and brachial arteries in the upper arm. At the elbow, the ulnar nerve can be palpated proximal to the medial epicondyle. The ulnar nerve, at the wrist, is located lateral to the flexor carpi ulnaris tendon and medial to the ulnar artery.

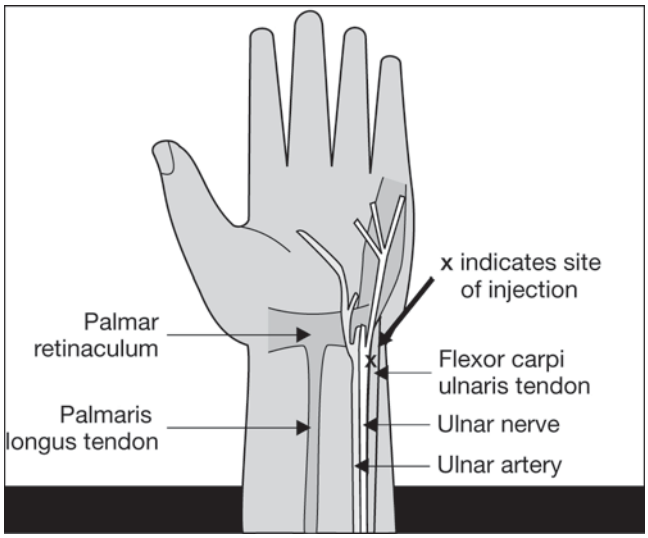
Blockade of the Ulnar Nerve at the Elbow

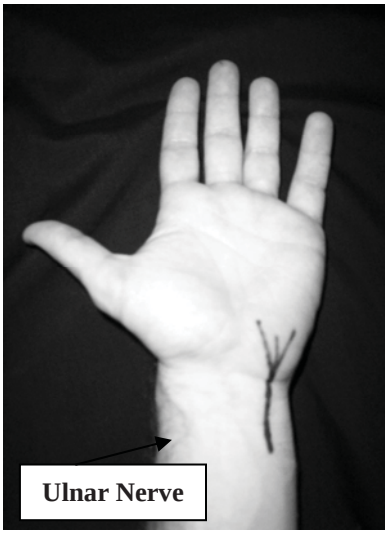


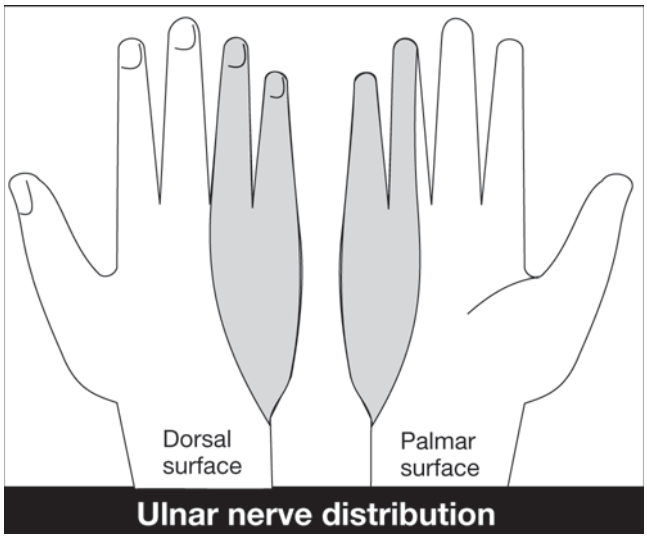
To anesthetize the ulnar nerve at the elbow, have the patient flex their arm 90 degrees. Identify the olecranon process and the medial condyle. The ulnar nerve can be palpated between these two structures. Insert a 22-27 gauge blunted needle slowly in this space. The ulnar nerve will be superficial. If a paresthesia is obtained, withdraw the needle slightly and inject 3-5 ml of local anesthetic. If no paresthesia, then superficially infiltrate the area with local anesthetic.

Blockade of the Ulnar Nerve at the Wrist

Locate the pulsation of the ulnar artery on the palmar surface of the wrist. Insert a 22-27 gauge blunted needle on the medial side of the arterial pulsation. The ulnar nerve is superficial. If a paresthesia is obtained, pull back slightly and inject 3-5 ml of local anesthetic. If there is not a paresthesia, infiltrate the area with local anesthetic.





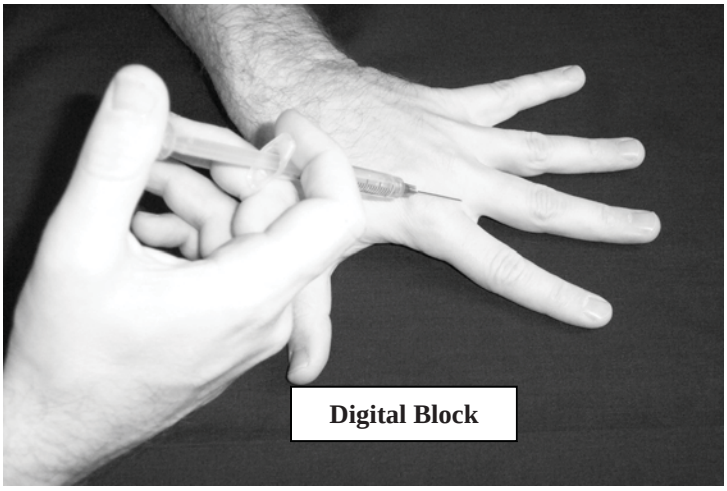


Digital Nerve Blocks

Each digit has two dorsal and two palmar branches of the digital nerve. Digital blocks are easy to administer and quite effective. There are some important considerations.

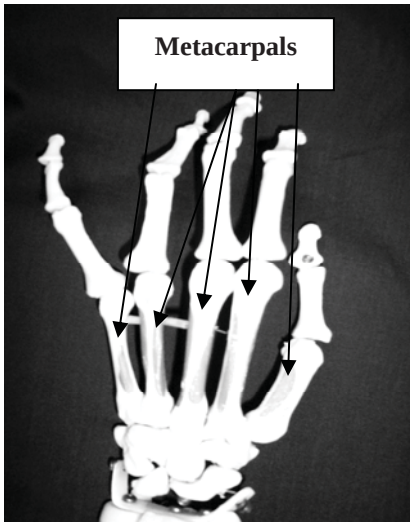
1. Never use epinephrine containing local anesthetics. Epinephrine can result in vasoconstriction, ischemia, and if severe loss of the digit.

2. Use the least amount of local anesthetic possible. Never administer more than 4 ml of total volume per digit. A volume exceeding this amount can result in a "tourniquet" effect, decreasing blood flow resulting in ischemia.



Metacarpal Block

A metacarpal block is an alternative to a digital nerve block. Some hand surgeons feel that it is a safer alternative to a digital nerve block due to concerns about the tourniquet effect exerted by local anesthetic volume. Never use epinephrine containing solutions. This can result in ischemia to the digit. A small skin wheal should be placed on the dorsum of the hand. Advance the needle while injecting local anesthetic parallel to the metacarpal bone. Do not go through the surface of the palm. The nerve is closer to the palmar surface than the dorsum. Most of the local anesthetic should be deposited in this region. A total of 3-5 ml of local anesthetic can be deposited. The same procedure should occur on the opposite side of the metacarpal.



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Bier Block (Intravenous Regional Anesthesia)

Chapter Ten
Bier Block (Intravenous Regional Anesthesia)

Note of Caution: The Bier block should not be used in settings where tourniquets may be unreliable. The risk of local anesthetic toxicity can be catastrophic. In settings where there are no reliable tourniquets, alternative anesthetic techniques should be used.

The basic idea behind the Bier block is to exsanguinate the extremity, apply an arterial tourniquet to isolate it from circulation, and inject local anesthetic into the extremities venous system, inducing anesthesia.

Indications

The Bier block is a suitable technique for the following:

- Surgical procedures involving the arm below the elbow (open procedures or closed reductions)
- Surgical procedures involving the leg below the knee (open procedures or closed reductions)
- Surgical procedures that will be completed within 40-60 minutes

Advantages & Disadvantages of the Bier block

Advantages include the following:

- Easy to administer
- Low incidence of block failure
- Safe technique when used appropriately
- Rapid onset and recovery
- Muscle relaxation for the surgeon

Disadvantages include the following:

- Should be used for only short procedures
- Patient may experience tourniquet pain after 20-30 minutes
- Sudden cardiovascular collapse or seizures may occur if local anesthetic is released into the circulation too early.

Contraindications

Patients with the following conditions:

- Reynaud's disease
- Homozygous sickle cell disease

- Crush injuries
- Young children

Equipment

- Double tourniquet or two single reliable sphygmomanometers with blood pressure cuffs. If using two single blood pressure cuffs, apply forceps that will not cause trauma to the tubing. Clamp the tubing to prevent the inadvertent release of air from the tourniquet. Prior to use test the tourniquet to ensure proper function.
- Eschmark bandage or an elastic rubber bandage to exsanguinate (remove blood) from the arm
- IV catheter (dorsum of the operative hand/foot is preferred)
- A running IV in the non operative arm to administer sedatives, analgesics, and emergency medications
- Resuscitation equipment should be available

Local Anesthetic Choice

Preservative free prilocaine and lidocaine are acceptable choices. Both have relatively low toxicity and a high therapeutic index. The concentration for both should be 0.5%. Never substitute other local anesthetics. The local anesthetic should NOT contain epinephrine, it should be plain. The recommended dose of lidocaine should not exceed 3 mg/kg. For a 70 kg adult this would be 50 ml of 0.5% plain lidocaine. The recommended dose for prilocaine should not exceed 6 mg/kg. The usual dose for an adult is 40 ml of 0.5% prilocaine. Lower extremity surgery may require larger volumes. For patients that weigh less than 70 kg, adjust the dose according to their weight. Do not increase the dose for larger patients. Never use higher doses/concentrations of prilocaine or lidocaine because of the risk of toxicity. One complication of prilocaine is methemoglobinemia (see chapter one). Prilocaine is metabolized to o-toluidine derivatives, which converts hemoglobin to methemoglobin. This generally occurs at high doses (>10 mg/kg) and should not occur with routine use at accepted doses.

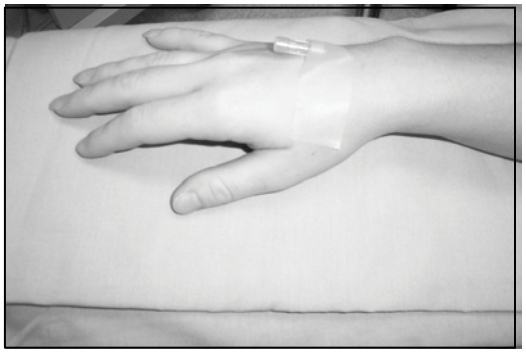
0.5 % Lidocaine Preservative Free
5 mg/ml
Suitable for infiltration and intravenous regional anesthesia.

XYZ Drug Company
Expiration Date: Month/Year

Read the label. Ensure it is the correct local anesthetic, concentration, and formulated for intravenous regional anesthesia.

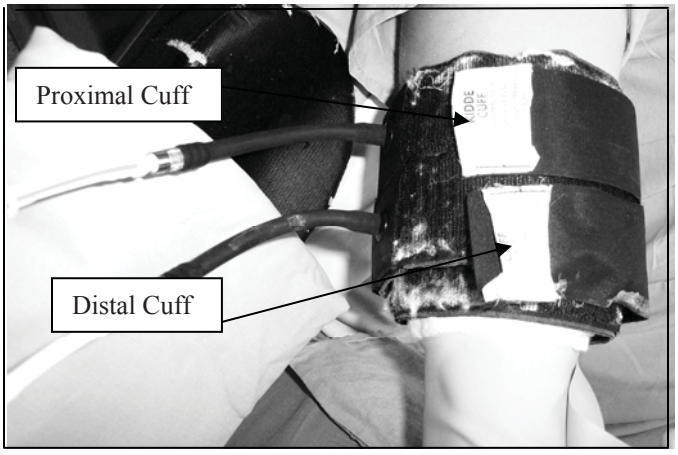
Technique

- Prepare the required materials
- Ensure that the 0.5% prilocaine or 0.5% lidocaine is preservative free, formulated for intravenous regional anesthesia, and does NOT contain epinephrine.
- Ensure proper tourniquet function
- Ensure that the patient has been fasting for an appropriate period of time.
- Attach routine monitors including ECG, blood pressure, and pulse oximetry.
- Place the IV catheter as distally possible on the operative limb. Place a running IV in the non operative arm.



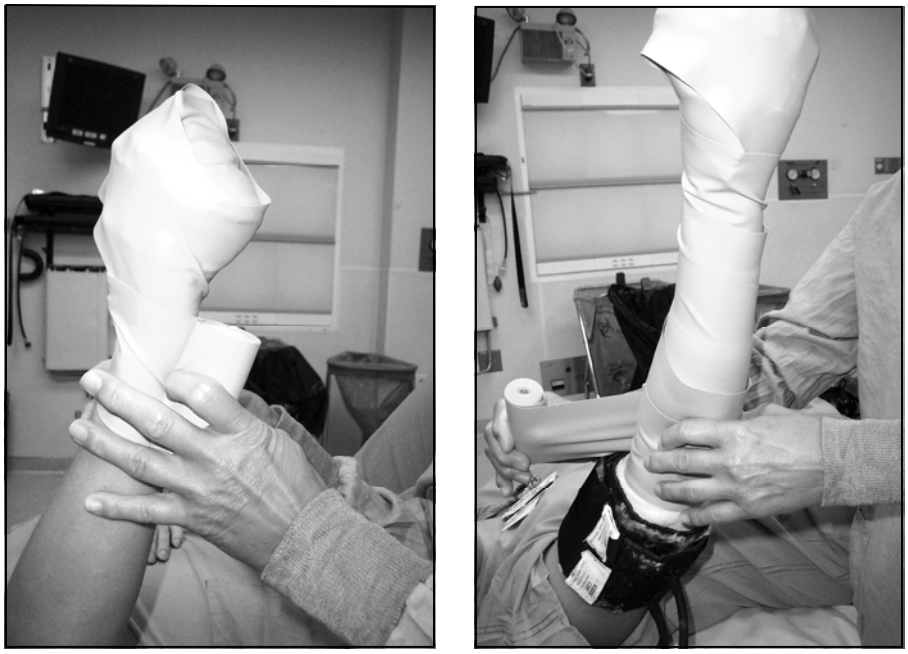
IV lock in the operative hand

- Double tourniquet placed on operative limb.



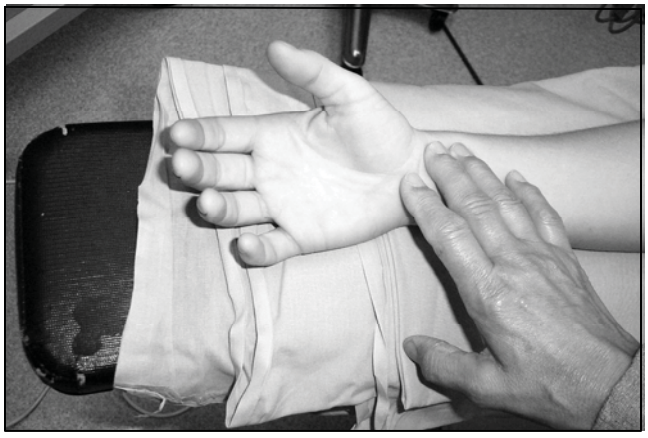
Double Tourniquet

- Have the patient hold the operative limb up. Exsanguinate the extremity with an Eschmark or rubber bandage. Exsanguination should occur from distal (hand/foot) to proximal (towards the tourniquet).



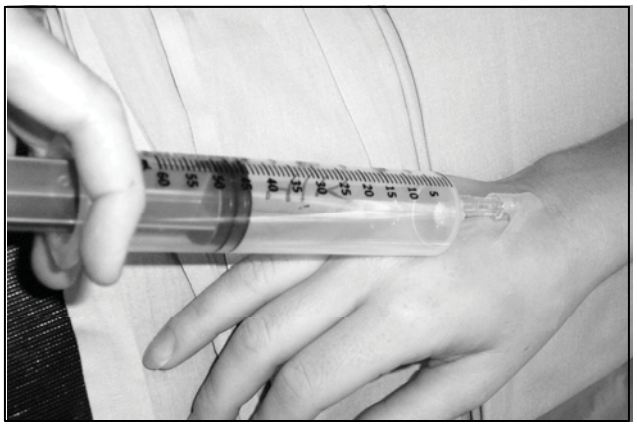
Exsanguation of the arm with an elastic bandage

- After exsanguination, the proximal tourniquet should be inflated to approximately 100 mmHg higher than the patient's systolic blood pressure.
- The Eschmark/rubber bandage should be removed. Confirm the absence of an arterial pulse (radial for arm/dorsalis pedis for leg).



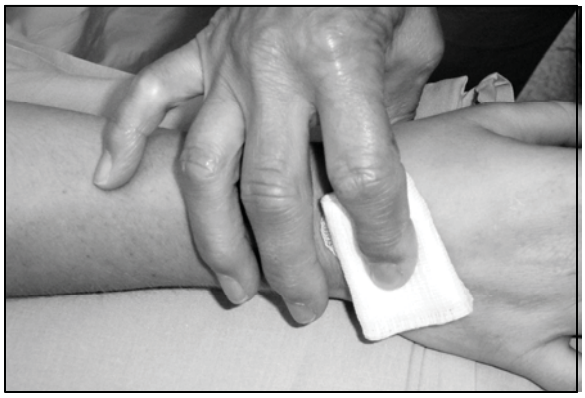
Confirming the absence of a radial pulse

- Inject the local anesthetic slowly (0.5% prilocaine or 0.5% lidocaine). Do not exceed the maximum dose.



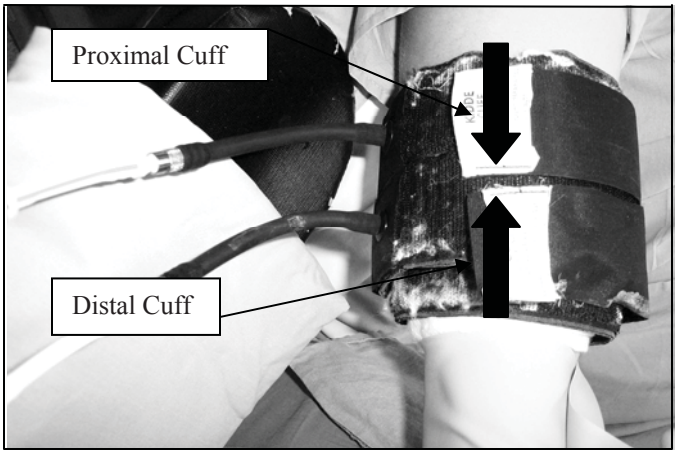
Injection of 0.5% lidocaine

- Once the injection is complete, remove the IV catheter and hold pressure at the site.



Removal of IV lock and pressure held on the site

- Now the OR staff can prep the arm. The onset of anesthesia will occur within 5 minutes.
- Inform the patient that the limb will feel numb or tingle. The limb may appear mottled. This is normal.
- When the patient complains of tourniquet pain, inflate the distal tourniquet. Once the distal tourniquet has been inflated deflate the proximal cuff.



First inflate the distal cuff. After successful inflation deflate the proximal cuff

- Analgesics should be administered for discomfort as needed
- Leave the tourniquet inflated for a minimum of 20-25 minutes. Releasing the tourniquet early may result in a large amount of local anesthetic being released, increasing the risk of toxicity.
- When releasing the tourniquet cyclic deflations/inflations in 10 second intervals will decrease peak levels of local anesthetic.
- Continue to monitor ECG, blood pressure, and pulse oximetry for 10 minutes after deflation.

Complications

- Tourniquet discomfort
- Rapid return of sensation after tourniquet release, resulting in subsequent pain
- Toxic reactions from malfunctioning tourniquets or deflating the tourniquet prior to 20-25 minutes.

Local Anesthetic Toxicity

- Signs and symptoms may include nausea, vomiting, dizziness, ringing of the ears (tinnitus), funny sensation around the mouth, loss of consciousness, and seizures.

Local Anesthetic Toxicity Management

Use the A, B, C's for the management of local anesthetic toxicity.

- A= airway. Maintain a patent airway, administer 100% oxygen.
- B= breathing. May need to assist the patient with positive pressure ventilation or intubation.
- C= circulation. Check for a pulse. If no pulse, initiate CPR.
- Seizures. Diazepam in doses of 5 mg, or alternatively sodium pentothal in doses of 50-200 mg will decrease or terminate seizures.

- Hypotension. Treat with ephedrine (typically 5 mg) IV, open up intravenous fluids, place the patient in a head down position (Trendelenburg). If hypotension is refractory to ephedrine, treat the patient with epinephrine (5-10 mcg). Repeat and escalate the dose as necessary.

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.

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Ankle Block

Chapter Eleven
Ankle Block

The ankle block is a common peripheral nerve block. It is useful for procedures of the foot and toes, as long as a tourniquet is not required above the ankle. It is a safe and effective technique.

Indications

The ankle block is suitable for the following:

- Orthopedic and podiatry surgical procedures of the distal foot.

Advantages & Disadvantages

Advantages include the following:

- Easy to administer
- Safe technique when used appropriately
- Rapid onset of anesthesia
- Avoids complications related to neuraxial or general anesthetic techniques

Disadvantages include the following:

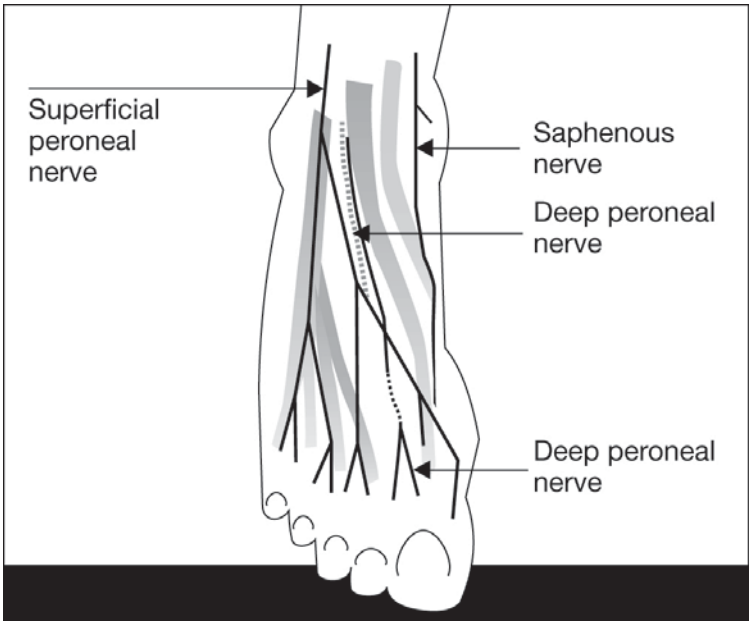
- Is a superficial block and purely sensory. The patient will still be able to move their toes and foot.
- Requires at least 3 separate injections
- Placing an ankle block is uncomfortable. The patient may require conscious sedation for analgesia and amnesia. It is important not to over sedate the patient and to maintain communication. Over sedation will result in an uncooperative patient who may move at critical times, placing the anesthesia provider and patient at risk for injury.
- It is possible to "miss" a nerve distribution. It is easy to remedy the situation by adding additional local anesthetic.

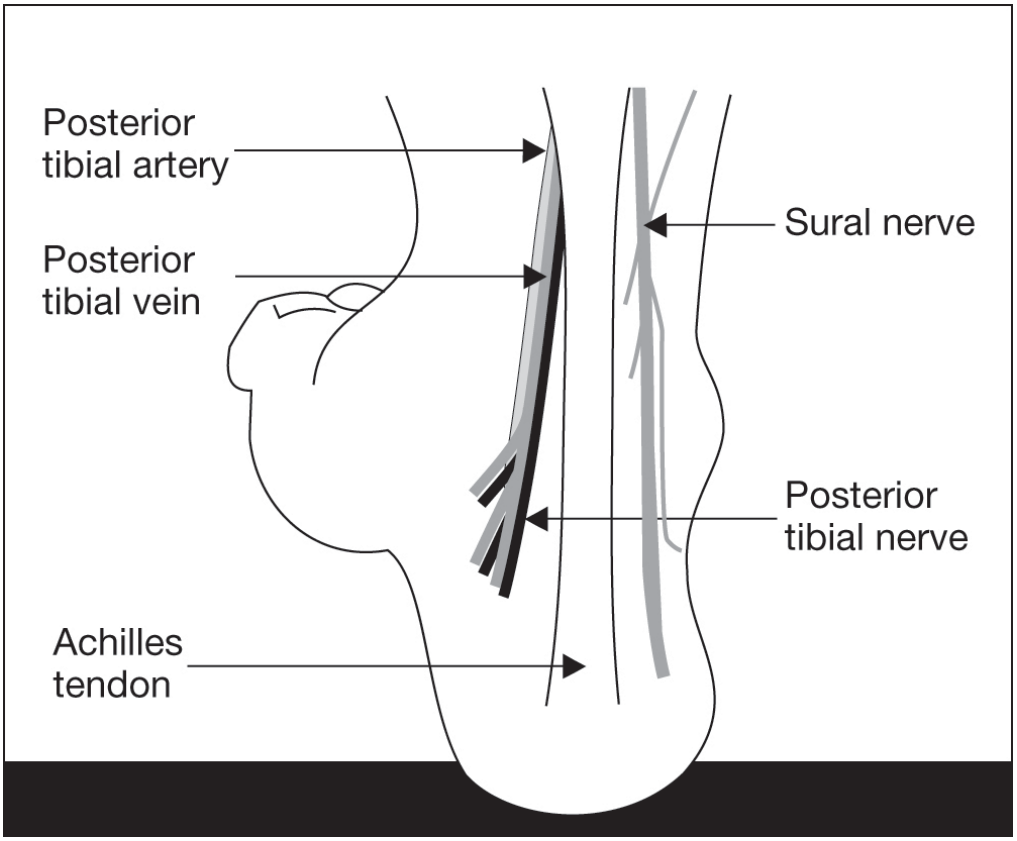
Contraindications

- Few contraindications exist. Should not be used when the foot is infected. Local anesthetic does not work in an acidotic environment. In addition, there is the risk of introducing infectious agents to healthy tissue.
- Young children
- Compromised circulation to the foot

Anatomy

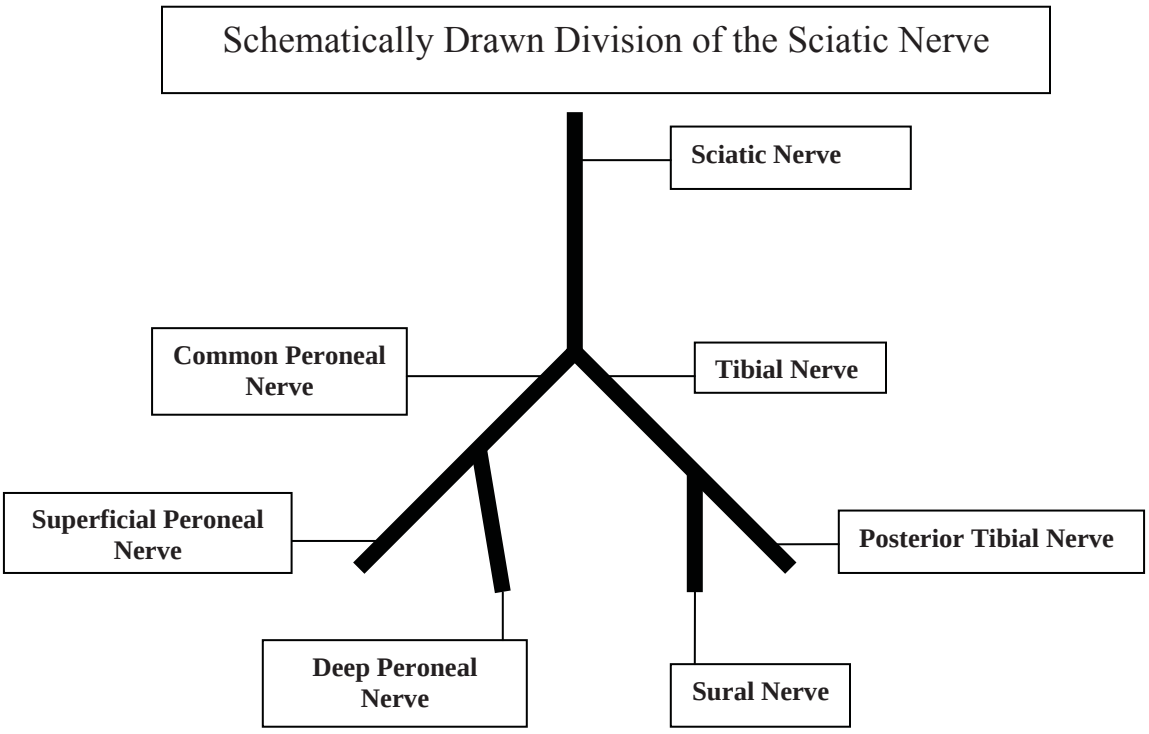
Nerve Distribution





The ankle block involves blockade of 5 nerves. Four of the five nerves are terminal branches of the sciatic nerve and include the following:

- Posterior tibial nerve
- Sural nerve
- Superficial peroneal nerve
- Deep peroneal nerve



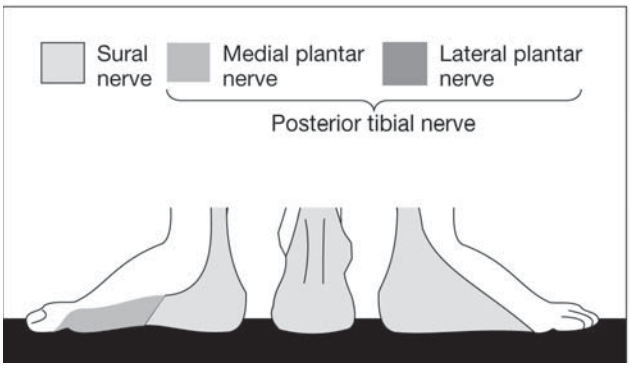
The sciatic nerve divides, forming two branches: common peroneal and tibial nerve. The common peroneal nerve descends laterally around the fibular head, dividing into superficial and deep peroneal nerves. The tibial nerve divides into the posterior tibial and sural nerve.

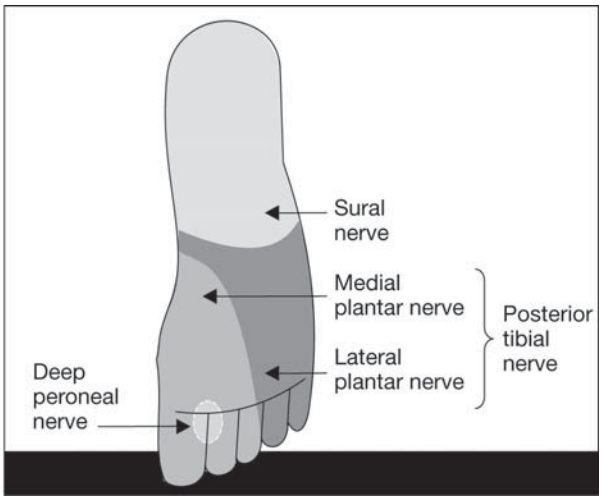
- Deep peroneal nerve- continues as an extension of the common peroneal nerve entering the ankle between the flexor hallucis longus tendons.
- Superficial peroneal nerve- continues as an extension of the common peroneal nerve entering the ankle lateral to the extensor digitorum longus.
- Posterior tibial nerve- continues as an extension of the tibial nerve entering the foot posterior to the medial malleolus where it branches into the lateral and medial plantar nerves. It is located behind the posterior tibial artery level adjacent to the medial malleolus.
- Sural nerve- continues as an extension of the tibial nerve entering the foot between the Achilles tendon and lateral malleolus.

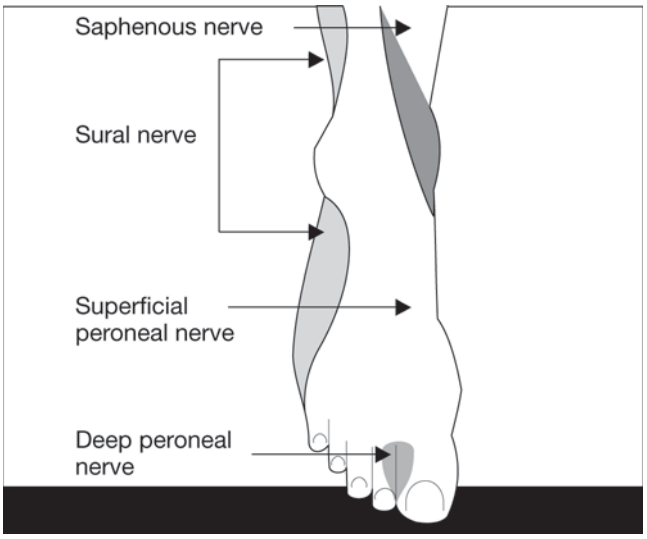
The saphenous nerve is a terminal branch of the femoral nerve.

- Saphenous nerve- located anterior to the medial malleolus.

Sensory Distribution







- Deep peroneal nerve- provides sensation to the medial half of the dorsal foot and between the first and second digits.
- Superficial peroneal nerve- provides sensation to the dorsum of the foot as well as all five toes.
- Posterior tibial nerve- provides sensation to the heel, medial, and lateral sole of the foot.
- Sural nerve- provides sensation to the lateral foot.
- Saphenous nerve- provides sensation to the anteromedial foot.

Equipment

- Betadine and alcohol wipes
- Sterile gloves
- Sterile 4x4 or 2x2's
- Sterile towels
- 2-3 10 cc syringes with local anesthetic
- 22-25 gauge, 4 cm blunted needle

Local Anesthetic Choice and Considerations

Choice of local anesthetic depends on the length of blockade. Longer acting local anesthetics are slower in onset. Some anesthesia providers mix 2% lidocaine with 0.5% bupivacaine to help speed onset and still have a moderate duration of action. NEVER USE EPINEPHRINE! This can cause vasoconstriction and ischemia.

Common Local Anesthetics Onset and Duration

Local Anesthetic	Onset	Duration
1.5% mepivicaine	15-20 minutes	2-3 hours
2% lidocaine	10-20 minutes	2-5 hours
0.5% ropivacaine	15-30 minutes	4-8 hours
0.75% ropivacaine	10-15 minutes	5-10 hours
0.5% bupivacaine	15-30 minutes	5-15 hours

Be careful with the total volume of local anesthetic. Since there are 5 nerves to block around the ankle, the volume of local anesthetic may increase the risk of a tourniquet effect, resulting in ischemia. This should be a consideration for patients with peripheral vascular disease, and diabetics. The provider may choose to block specific nerves required for the surgical procedure. If this is done, inform the patient that portions of his/her foot may have normal sensation. Blocks should not be performed in infected tissue. The site of infection should be removed from the injection site. Check with the surgeon to ensure there is not a risk of tracking the infection into healthy tissue. If this is a risk, choose an alternative form of anesthesia.

Preparing for the Ankle Block

- Assemble required equipment
- Intravenous access with a running IV should be initiated and maintained prior to an ankle block. This provides for the administration of analgesics, sedatives, and emergency medications.
- Attach routine monitors including ECG, blood pressure, and pulse oximetry.
- Ensure that local anesthetics do NOT contain epinephrine.

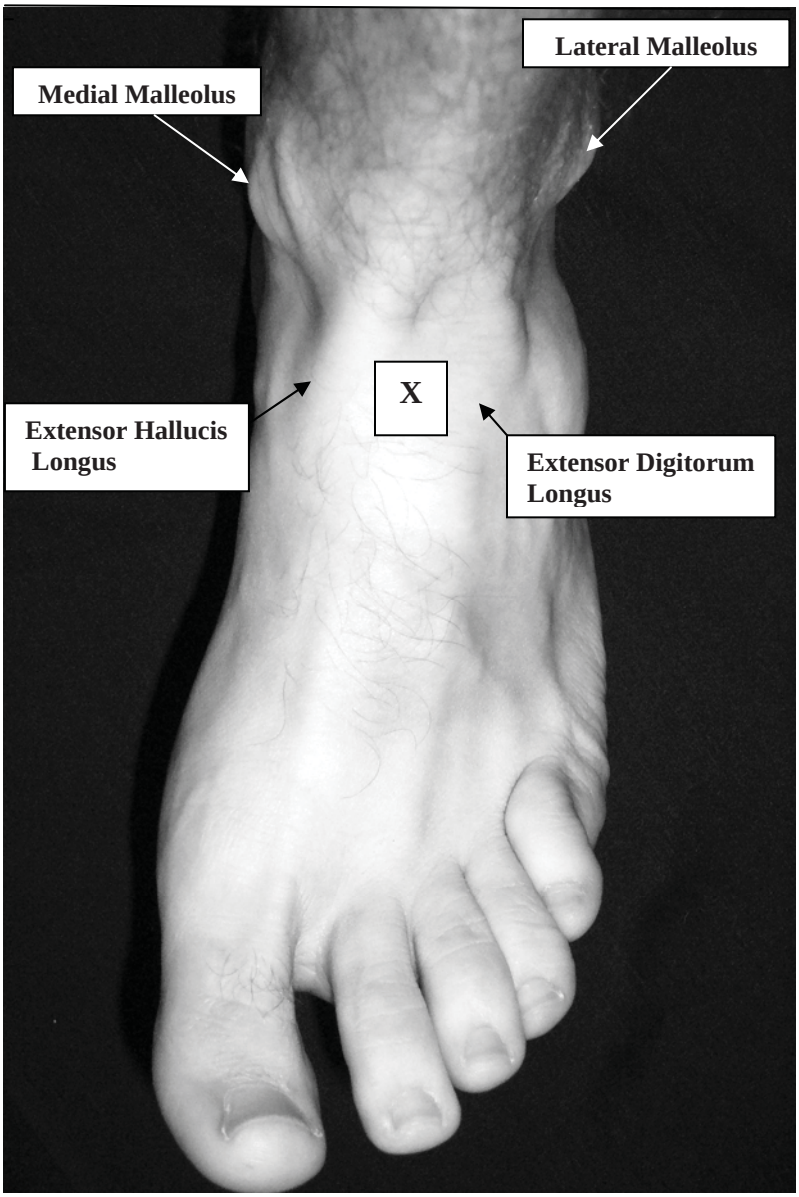
Performing the Ankle Block

Position the foot to access all five nerves. Placing blankets or pillows under the lower leg raises the foot off of the bed, improving access to all five nerves. Maintain sterile technique.

Blocking the Deep Peroneal, Superficial Peroneal, and Saphenous Nerve

Blockade of the deep peroneal, superficial peroneal and the saphenous nerves can be accomplished with one injection. The following steps should be followed:

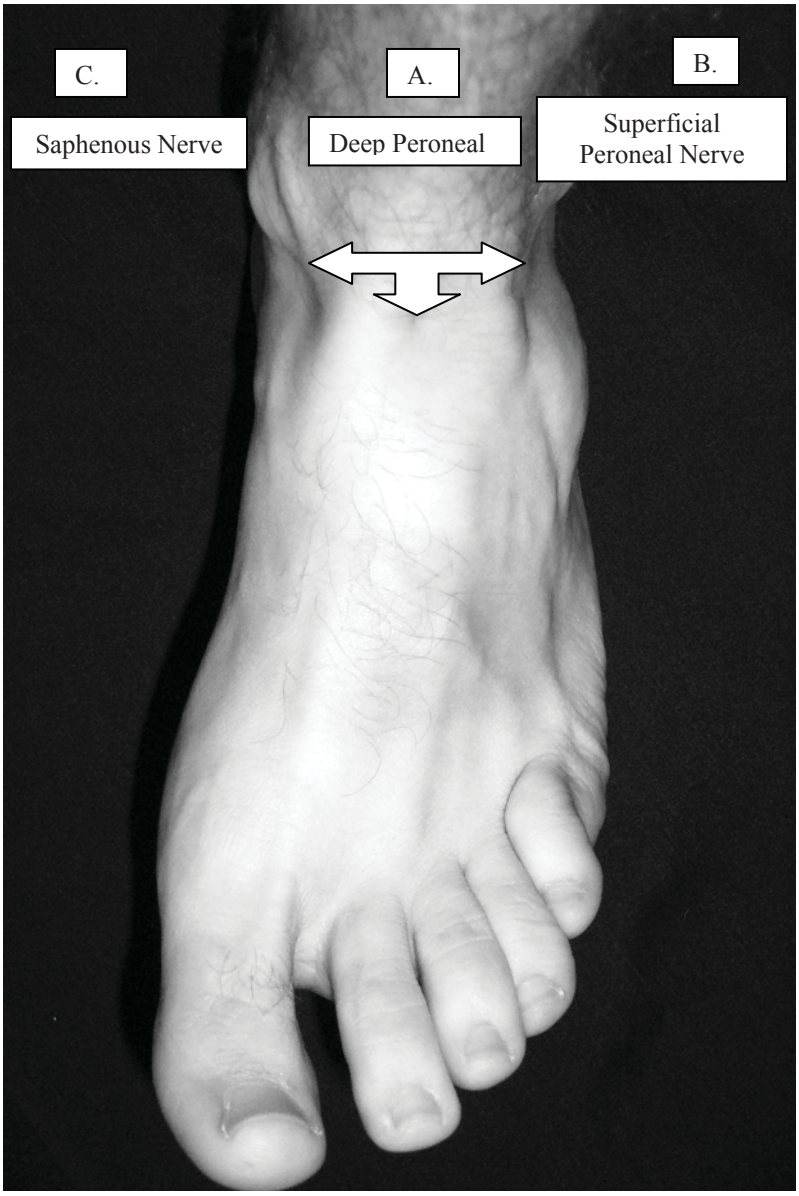
- Draw a line between the two malleoli
- Identify the extensor hallucis longus tendon by asking the patient to flex their toes, and palpate the anterior tibial artery, found between the extensor hallucis longus and the extensor digitorum longus muscle.



X = location of the deep peroneal nerve

- Place a skin wheal lateral to the artery

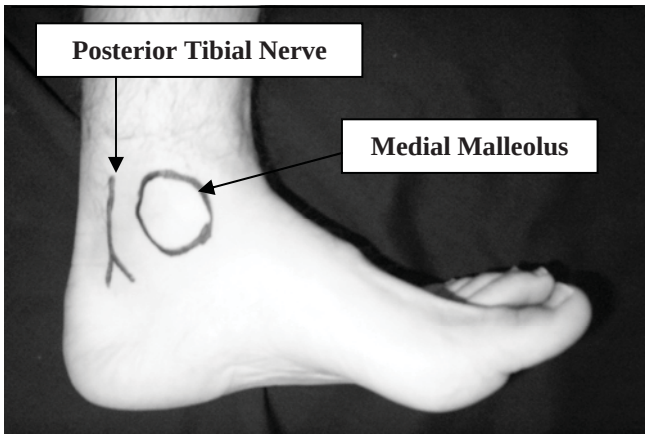
- Advance needle between these structures, perpendicular, aspirating for blood, and deposit 3-5 ml of local anesthetic deep to the extensor retinaculum. This will block the deep peroneal nerve.
- Bring the needle back, direct it superficially towards the lateral malleolus. Deposit 3-5 ml subcutaneously to block the superficial peroneal nerve.
- Now direct the needle superficially towards the medial malleolus. Deposit 3-5 ml subcutaneously to block the saphenous nerve.



A. **Deep Peroneal Nerve**- advance needle perpendicular and deep to the retinaculum.
B. **Superificial Peroneal Nerve**- direct needle superficially towards the lateral malleolus.
C. **Saphenous Nerve**- direct needle superficially towards the medial malleolus.

Blockade of the Posterior Tibial Nerve

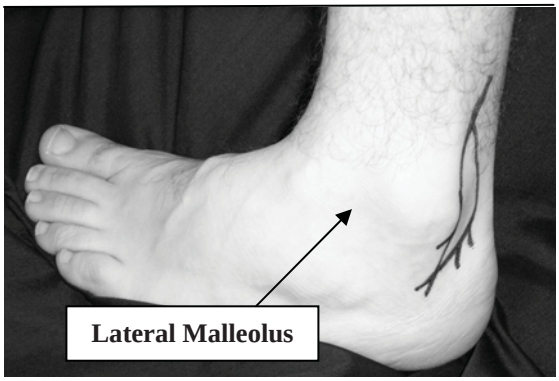
- Warn the patient to hold still in case a paresthesia is elicited! Movement may cause trauma to the nerve.
- Identify the posterior tibial artery by palpation
- Insert the needle posterolateral to the artery, at the level of the medial malleolus. Advance the needle slowly and carefully.
- If a paresthesia is elicited, withdraw the needle slightly and inject 3-5 ml of local anesthetic. Make sure that the patient does not experience a paresthesia with injection.
- Inject 7-10 ml of local anesthetic solution as you withdraw the needle. A paresthesia is not necessary for successful blockade of the posterior tibial nerve.



Blockade of the Sural Nerve

- Identify the lateral malleolus and Achilles tendon
- Insert the needle superficially, lateral to the tendon, in the direction of the lateral malleolus. Inject 5-10 ml of local anesthetic subcutaneously as the needle is withdrawn.

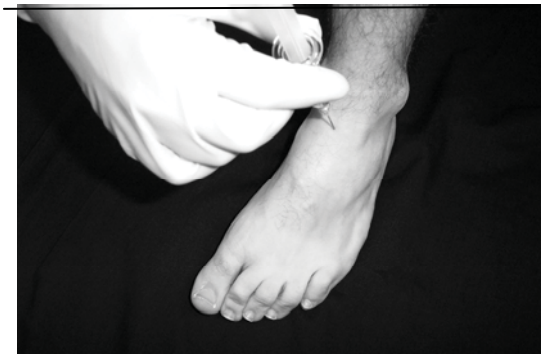
Sural Nerve



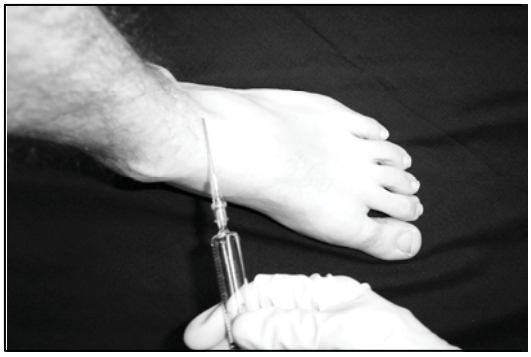
Complications

- Discomfort- conscious sedation for amnesia and analgesia
- Injury to the patients foot after discharge due to a "numb" foot- instruct patient to be aware of where their foot is and to be careful not to injure it
- Nerve injury or paresthesia- do not inject if patient complains of paresthesia or if resistance is met during injection. Do not repeat injections to anesthetized sites. Most paresthesia's are self limiting.
- Hematoma and vascular injury- avoid puncture of veins or arteries. Avoid multiple injection sites. Always aspirate. If a vessel or artery is punctured, hold pressure for 5 minutes, ensuring homeostasis.
- Infection- rare if sterile technique is used
- Intravascular injection- aspirate frequently during injection. The risk of local anesthetic toxicity is low.
- Block failure- may be supplemented by the surgeon or anesthesia.

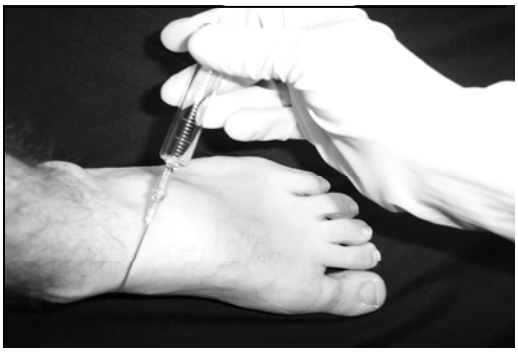
Placement of Ankle Block



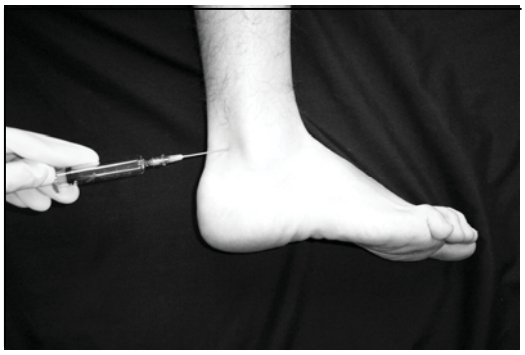
A. Deep Peroneal Nerve



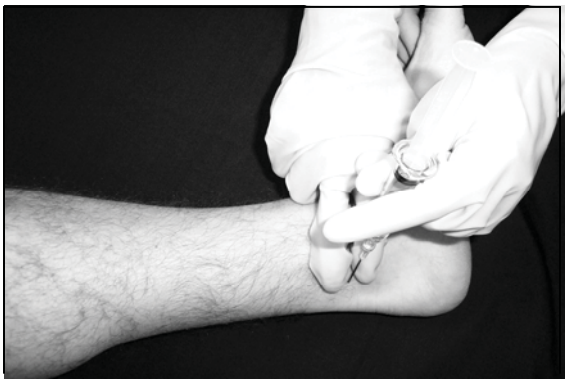
B. Superficial Peroneal Nerve



C. Saphenous Nerve

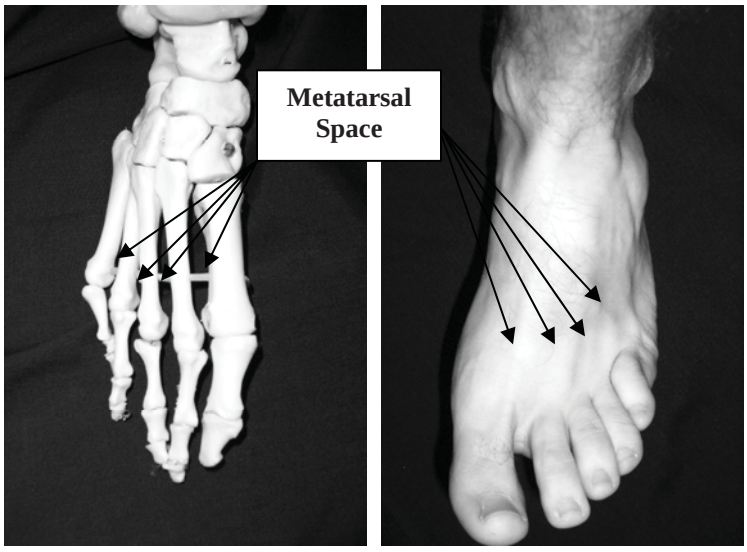


D. Sural Nerve



E. Posterior Tibial Nerve

Metatarsal Block of the Foot



A metatarsal block may supplement an ankle block if a nerve distribution has been missed. Never use epinephrine containing solutions. This can result in ischemia of the digits. Place a small skin wheal at the site of injection on the dorsum of the foot. Advance the needle while injecting local anesthetic parallel to the metatarsal bone. Do not go through the surface of the sole of the foot! The individual nerves are located closer to the sole of the foot than the dorsum. A total of 3-5 ml of local anesthetic solution may be deposited. The same procedure should occur on the other side of the metatarsal of the location that anesthesia is desired.

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Section II

Obstetric Anesthesia: Anatomy, Physiology & Anesthetic Implications

Chapter Twelve
Obstetric Anesthesia: Anatomy, Physiology & Anesthetic Implications

Obstetric anesthesia can be challenging. Despite being young and healthy, this population is at high risk for complications leading to mortality and morbidity. These risks are largely related to changes in anatomy and physiology, associated with the birthing process, or surgical intervention. Pregnant women have a high mortality rate in many countries throughout the world. Higher mortality rates occur in women who are greater than 35 years of age and those without prenatal care/or access to medical care. Direct causes of obstetric related deaths include: hemorrhage (25%); sepsis (15%); hypertensive disorders of pregnancy (12%), eclampsia in particular; complications of unsafe abortion (13%); and prolonged or obstructed labor (8%). Morbidity is more common where malnutrition is endemic, where girls marry young and begin childbearing before fully grown. Indirect causes of obstetric mortality (20%) include anemia, malaria, and HIV. Pulmonary embolism is a relatively rare cause of maternal mortality. Anesthesia accounts for up to 3% of the obstetric deaths in Western countries. Morbidity is more likely to occur during emergent compared to elective cesarean section. To reduce mortality and morbidity, the anesthesia provider should:

1. Have adequate knowledge of what to expect in this population
2. Use regional instead of general anesthesia, provided there are no contraindications.
3. Have adequate knowledge of anesthesia care for specific conditions and situations.

Obstetric Related Terms

Parturient- This term is applied to a woman in the process of giving birth.
Trimester- A three month period. Pregnancy is divided into three trimesters.
Gravid Uterus- An enlarging uterus that contains a fetus.

Pregnancy Related Physiological and Anatomical Changes

Numerous changes occur in a women's body during pregnancy. These changes affect almost every organ system. Changes early in pregnancy are largely due to increases in hormones (i.e. progesterone and estrogen) and increased metabolic demands of the fetus, placenta, and uterus. Later changes are due to the expanding uterus and growing fetus. All of these changes impact anesthetic care.

Cardiovascular System

Changes in the cardiovascular system are due to increased demands of both mother and fetus.

Blood Volume

Blood volume will increase progressively starting at 6-8 weeks. At the time of delivery, the average blood volume is increased by 1-1.5 liters. Estimated blood volume approaches 90 ml/kg. There is a greater increase in plasma volume compared to red blood cell mass, resulting in a relative anemia. The intake of supplemental iron and folic acid will help the mother maintain normal hemoglobin levels. White blood cell count and its components are increased during labor and delivery, sometimes markedly. However, in the absence of fever or other signs of infection/sepsis, it is reasonable to administer regional anesthetics. The clotting components of the patient's blood also increase to enhance clotting, reducing excessive bleeding during delivery. These components include fibrinogen, and factors VII, X, and XI. Increased blood volume meets the mother and fetus's metabolic demands, allowing the mother to tolerate blood loss during delivery. The average blood loss associated with vaginal delivery is 400-500 ml. With a cesarean section it is 800-1000 ml. Blood volume will return to normal within 2 weeks of delivery.

Cardiac Output

Cardiac output will increase up to 40% at term. Most of the increase occurs in the 1st and 2nd trimester. The exception is during labor, when cardiac output peaks secondary to increased heart rate and stroke volume (the volume of blood ejected from the heart with each contraction). To handle the increased blood volume, the myocardium and chambers enlarge. Cardiac output will return to normal 2 weeks after delivery.

Cardiac output can decrease after 28 weeks of pregnancy due to mechanical changes. When the patient assumes a supine position, the weight of the uterus can decrease the amount of blood returning to the heart by compressing the inferior vena cava. Lying supine can affect both the fetus and the mother, decreasing cardiac output by up to 24%. In 20% of obstetric patients, this can lead to supine hypotension syndrome. Symptoms include hypotension, sweating, nausea, vomiting, and a pale color. When the patient turns from a supine position to a lateral position, the symptoms go away. The uterus can also compress the aorta. Aortocaval compression can lead to fetal distress by causing hypotension, increased uterine venous pressure, and decreased perfusion of the uterus and placenta. Aortocaval compression can lead to fetal asphyxia. In addition, compression of the aorta increases the risk of phlebitis and blood clots. This is related to compression of the great vessels and hypercoagulable state of the mother. The implications for the anesthetist include placing a roll under the patient's right hip when supine. This will help protect both mother and baby from the effects of the gravid uterus.

Blood Pressure

Despite increases in the patients' cardiac output and blood volume, the patient's blood pressure does not normally increase from pre-pregnancy levels unless there is an abnormality such as pregnancy induced hypertension. A decrease in blood pressure occurs by about 8 weeks' gestation. By mid-pregnancy, the diastolic blood pressure and mean arterial pressure reach their lowest point (16-20

mmHg below pre-pregnancy values), returning to pre-pregnancy levels by term. The overall decrease in diastolic blood pressure and mean arterial pressure is 5-10 mmHg. Maintenance of vascular tone is largely dependent upon sympathetic outflow. Sympathetic blockade with a spinal or epidural anesthetic may result in significant hypotension. Fluid preloading is important.

Venous System

The venous system has an increased capacity for distension and dilation (up to 150%). This can reduce blood flow, delaying the absorption of subcutaneous or intramuscular medications. Distention of the vessels within the epidural space may increase the risk of vascular damage and bleeding during neuraxial blockade. This, along with hormonal changes, reduces the required amount of local anesthetics by 30%. Using the same dose in the pregnant patient as one normally would in the non-pregnant patient may result in a high neural block.

Summary of Anesthesia Implications due to Cardiovascular Changes

- White blood cells and their components may be markedly elevated during labor and delivery. In the absence of signs and symptoms of infection/sepsis/other contraindications it is reasonable to proceed with neuraxial blockade.
- Never lay the patient supine. Always place a wedge or roll under the right hip so the patient is "tipped" to the left. This maneuver will prevent a decrease in cardiac output, maternal hypotension, fetal distress, or asphyxia that can result from supine hypotension syndrome/aortocaval compression.
- The pregnant patient is dependent upon sympathetic outflow to maintain systolic blood pressure. Sympathectomy associated with regional anesthesia can result in severe hypotension. Always pre-load the patient with 1-2 liters of crystalloid fluids prior to neuraxial blockade.
- Vessel distention in the epidural space may increase the risk for vessel damage during neuraxial blockade. Vessel distention also decreases the intrathecal and epidural spaces. Decrease the dose of local anesthetics by 30% to avoid a high neuraxial block.
- Delayed absorption of subcutaneous and/or intramuscular medication.

Respiratory System

Several changes within the respiratory system have a direct impact on anesthesia care.

Lung Volumes

As the gravid uterus grows in size, it places pressure on the abdomen. This is compensated by an increase in the diameter of the chest. At term, the pregnant patient favors thoracic breathing over abdominal breathing. Of particular note is the change in functional residual capacity (FRC- the volume of air that is in the lungs at the end of a normal breath). FRC decreases 20% by term, returning to normal 48 hours after delivery. A decrease in FRC reduces the patient's reserve. In the

event of apnea, the patient can become hypoxic quickly. In addition, the pregnant patient will increase her tidal volume (normal volume with each breath) by 40%.

Respiratory Gas Exchange

Minute ventilation (amount of air breathed in one minute) increases by 50% by the second trimester. Respiratory rate will increase by 15% (2-3 breathes per minute). These changes speed the uptake of inhaled anesthetics. Alveolar ventilation (air that participates in gas exchange) will increase by 70% at term. Oxygen consumption increases by 20-50%. The patient undergoing surgical intervention should have supplemental oxygen during regional anesthesia. Pre-oxygenation with 100% O2 in preparation for a general anesthetic is important. The combination of a decreased FRC and increased oxygen consumption can result in hypoxia. During labor there is an additional increase in ventilation due to pain. Hyperventilation during labor can cause hypocarbia and respiratory alkalosis. If respiratory alkalosis is severe, it can decrease the release of oxygen to the mother and fetus. Periods of hyperventilation are often followed by periods of hypoventilation, compensating for respiratory alkalosis. During this time period the mother and fetus may become hypoxic.

Respiratory Tract Changes

As noted earlier, the pregnant patient has venous vascular engorgement. This results in a swollen respiratory tract and a diminished view during laryngoscopy. The obstetric population is more difficult to intubate compared to the non-pregnant population. A smaller than usual endotracheal tube may be required. Manipulation during laryngoscopy can result in bleeding, obscuring the view of the glottic opening.

Anesthesia Implications of Respiratory System Changes

- Patients undergoing regional anesthesia should have supplemental oxygen.
- Patients undergoing general anesthesia should be pre-oxygenated with 100% O2 prior to induction.
- Patients may desaturate despite pre-oxygenation due to increased oxygen consumption and decreased FRC.
- Be prepared for a difficult intubation. Swollen mucous membranes may decrease visualization. Ensure the patient is positioned optimally for laryngoscopy.
- Have smaller endotracheal tubes available for intubation. A smaller endotracheal tube may be required for intubation due to swollen tissue.
- Be very gentle during laryngoscopy as bleeding may obstruct the view.
- The patient will have an increased uptake of inhaled anesthetics secondary to decreased FRC and increased alveolar uptake.

Renal System

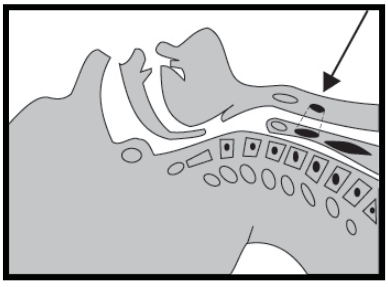
The pregnant patient's renal plasma flow and glomerular filtration rate will increase by 50-60% at term. This correlates with increased cardiac output and blood volume. Increases in renal plasma flow and glomerular filtration rate result in increased clearance of blood urea nitrogen and serum creatinine, which may be reduced by 40%. Increased glomerular filtration may result in minor spilling of glucose and protein as noted by urinalysis. Obstructive changes to the renal system can occur due to the enlarging uterus. This may result in an increased incidence of urinary tract infections and decreased blood flow to the kidneys.

Gastrointestinal System

Mechanical and hormonal alterations result in several changes within the gastrointestinal system. As the uterus enlarges, pressure is placed on the stomach resulting in an incompetent lower esophageal sphincter. In addition, progesterone will reduce the competence of the lower esophageal sphincter. Placental gastrin causes an increased secretion of gastric acid. These changes lead to reflux of gastric acid into the esophagus and delayed gastric emptying. Often the pH of these secretions is less than 2.5 and most patients will have fasting volumes of > 25 ml. Aspiration of gastric contents with a pH of less than 2.5 and volumes greater than 25 ml can result in Mendelson's Syndrome, which can be life threatening. The aspiration of solid material can result in atelectasis, pneumonia, or lung abscess. Food intake prior to labor may result in nausea, vomiting, and delayed stomach emptying. These changes place the pregnant patient at risk for aspiration during anesthesia.

Anesthesia Implications of Gastrointestinal System Changes

- Pregnant patients should be considered to have "full stomachs", regardless of fasting.
- If available, medications should be administered prior to anesthesia to reduce gastric acidity and volume. A non particulate antacid (i.e. sodium citrate) should be administered immediately prior to the anesthetic, reducing the acidity of stomach contents. To stimulate gastric emptying and increase lower esophageal sphincter tone, metoclopramide10 mg IVP should be administered 30-60 minutes prior to anesthesia. The use of histamine H2 blockers (i.e. famotidine 20 mg IVP) 30-60 minutes prior to surgical intervention may help to reduce acidity of stomach contents.
- Position the patient with a roll under the right hip. A slight reverse Trendelenburg position may be helpful in preventing passive reflux.



- Pre oxygenate the patient with 100% oxygen prior to general anesthesia.
- Cricoid pressure should be applied and held until the patient is intubated. Cricoid pressure should not be

released until it is confirmed that the endotracheal tube has been placed in the trachea.

- Do not routinely administer positive pressure ventilation, with a mask, prior to intubation. Positive pressure ventilation should occur if the patient's pulse oximetry reading starts to decline or a difficult airway is encountered. Unnecessary positive pressure ventilation prior to intubation may result in gastric distention, placing the patient at risk for aspiration.

Hepatic System

Overall function and blood flow to the liver are unchanged during pregnancy. There is a 25-30% decrease in pseudocholinesterase function at term. This should not produce a clinically significant prolongation of succinylcholine, mivacurium, or ester local anesthetics in the immediate delivery period. Pseudocholinesterase levels continue to drop the first 7 days post delivery. Up to 10% of post partum patients may be at risk for a prolonged block when using succinylcholine or mivacurium.

Anesthesia Implications of Hepatic System Changes

- Small risk of a prolonged neuromuscular block when using succinylcholine or mivacurium within 7 days post delivery.

Central Nervous System

Central nervous system induced alterations have a direct impact on anesthetics. Changes in hormones result in a decrease of up to 40% in minimal anesthetic concentration (MAC). By the 3rd day post delivery MAC levels return to normal. Hormonal changes and venous dilatation contribute to a 30% decrease in local anesthetic requirements for spinal and epidural anesthesia. Anatomical changes may create an epidural space that has positive pressure. Normally the epidural space has negative pressure, allowing for the "hanging drop" technique during identification of the epidural space. Positive pressure in the epidural space may make identification slightly more difficult and renders the "hanging drop" technique inaccurate.

Anesthesia Implications of Central Nervous System Changes

- Reduces the dose of inhaled anesthetics by up to 40%.
- Reduces the dose of local anesthetics for spinal and epidural anesthesia by up to 30%.
- Positive pressure in the epidural space may make it slightly more difficult to identify the epidural space.

Metabolism

The pregnant patient's metabolism is increased. Good nutrition is essential for both mother and baby. Increased metabolic demands place the pregnant patient at risk for hypoglycemia.

Uteroplacental Circulation

The circulation of blood to the uterus and placenta is essential for normal growth and development of the fetus. If normal circulation is compromised fetal growth can be restricted. If severe, fetal death can occur. Uterine blood flow continues to increase throughout pregnancy. At term uterine blood flow represents about 10% of the cardiac output (600-700 ml/minute). Up to 80% of the uterine blood flow goes to the placenta with the remaining 20% supplying the muscle of the uterus. Hypotension, uterine vasoconstriction, and uterine contractions are factors that decrease uterine blood flow. Hypotension can be due to assuming the supine position (aortocaval compression/supine hypotension syndrome), dehydration/hypovolemia, and sympathetic blockade associated with neuraxial blockade. In the past, alpha adrenergic medications such as phenylephrine and metaraminol were avoided in the treatment of hypotension since vasoconstriction decreases uteroplacental blood flow. Ephedrine (beta adrenergic agonist) was the vasopressor of choice. Subsequent studies have shown that small doses of alpha adrenergic agonists are just as effective as ephedrine and result in less fetal acidosis. Hypertensive disorders can decrease uterine blood flow through vasoconstriction. In addition, uterine contractions decrease uterine blood flow. If the uterus becomes hypertonic, uterine blood flow can be critically diminished.

The placenta is the fetus's life line. It is responsible for respiratory gas exchange, nutrition, and waste elimination. Maternal and fetal tissues form the placenta. Exchange of substances occur through diffusion (respiratory gases and medications); bulk flow (water); active transport (nutrition); pinocytosis (large molecules); and breaks in the membrane (Rh sensitization). Oxygen storage within the placenta is very small. Adaptive mechanisms such as redistribution of oxygen rich blood to essential organs, decreased oxygen consumption, and anaerobic metabolism can help the fetus survive up to 10 minutes during oxygen deprivation. Causes of oxygen deprivation include compromise of blood flow through the umbilical cord (cord compression, prolapse), abruption of the placenta, maternal hypoxemia, and maternal hypotension.

Anesthesia Implications for Uteroplacental Circulation

- Uteroplacental circulation is essential for the well being of the fetus. It is important to avoid complications that can jeopardize the fetus during surgical intervention.
- Avoid hypotension. Position, hydrate, and treat. Position the patient with a roll to prevent hypotension associated with the gravid uterus. Hydrate the patient prior to neuraxial anesthesia. Aggressively treat hypotension with alpha adrenergic (small doses of phenylephrine) and/or beta adrenergic (ephedrine) medications.

- Avoid hypoxemia. Use supplemental oxygen during neuraxial blockade. Pre-oxygenate the patient with 100% O2 prior to general anesthesia. Secure the airway as quickly as possible.

Anesthesia Medications effect on Uteroplacental Blood Flow/ Placental Transfer

Fetal effects of medications administered to the mother are dependent upon several factors including route of administration, timing of medication administration in regard to delivery and uterine contraction, dose, and maturity of the fetus. Medications can affect the fetus's heart rate, APGAR score, and responsiveness.

- Inhalational Agents- have a minimal effect on the fetus, if two factors are adhered to. Limited doses should be administered until delivery (less than 1 MAC). Delivery of the fetus should occur within 10 minutes of the anesthetic induction. When preparing for general anesthesia, have the patient prepped and draped, with the surgical team ready to "cut". Only then should the patient be induced with a general anesthetic. Once the airway is secured then the surgical team can proceed with delivery. All inhaled anesthetics can decrease uterine blood flow; however, in doses of < 1 MAC the effects are minimal. N2O has minimal effects on uterine blood flow when administered with a volatile anesthetic. Most clinicians will administer 50% N2O with a ½ MAC of a volatile anesthetic agent after intubation. After delivery of the fetus, N2O is turned off and the volatile anesthetic is increased to 1 MAC and titrated as needed.
- Intravenous Induction Agents- Thiopental, ketamine, and propofol are safe to use in standard induction doses. Fetal effects are generally minor. The effect on uterine blood flow varies. Thiopental and propofol may reduce uterine blood flow slightly. Ketamine in doses of < 1.5 mg/kg has minimal effects on uterine blood flow. Doses > 2 mg/kg may cause a hypertonic uterus, decreasing uterine blood flow. Benzodiazepines such as diazepam and midazolam cross the placenta readily and may depress the fetus. In addition, when used as an anesthetic induction agent they can decrease uterine blood flow and should be avoided.
- Opiates- All opiates cross the placenta. The effect on neonatal depression varies depending on opiate and route. Morphine is associated with respiratory depression in the neonate. Meperidine also results in respiratory depression, but its effects are less than morphine. Opioid agonist-antagonists such nalbuphine and butorphanol produce less respiratory depression than morphine and meperidine, but still may have depressant effects. Fentanyl is the opiate of choice. It has minimal depressant effects in doses < 1 mcg/kg. Neuraxial opioids have minimal effects and are safe to administer.
- Local Anesthetics- If the fetus is acidotic local anesthetics can be trapped. However, local anesthetics are not usually implicated in adverse outcomes in the neonate. Inadvertent intravenous injection of local anesthetics can result in decreased uterine blood flow. Spinal and epidural anesthesia does not decrease uterine blood flow as long as hypotension is avoided. Uterine blood flow actually increases with epidural anesthesia in the pre-eclamptic

patient. Dilute concentrations of epinephrine added to local anesthetics have minimal effects on uterine blood flow.

- Adjunct Medications- most medications (i.e. vasopressors, anti-emetics, etc.) will cross the placenta. They generally do not affect the neonate.

Labor

There are 3 stages of labor:

- Stage 1: onset of labor to complete cervical dilation. This stage includes the descent of the fetus and progressive cervical dilation. For primigravidas this generally lasts 8-12 hours; for multigravidas it lasts about 5-8 hours. There can be significant individual variation.
- Stage 2: full cervical dilation to the delivery of the baby. Contractions occur about every 1-2 minutes and last about 1-1.5 minutes. It can take from 15-120 minutes to deliver the baby.
- Stage 3: from the delivery of the baby until delivery of the placenta.

Anesthetic agents impact uterine activity and labor.

- ✓ All inhaled anesthetics depress uterine activity, relaxing the uterus in a dose dependent manner. High doses result in increased blood loss.
- ✓ N2O does not affect the uterus.
- ✓ Opioids do not affect labor.
- ✓ Ketamine, in doses of < 2 mg/kg, does not impact labor.
- ✓ Regional anesthetic techniques do not prolong labor or increase the rate of cesarean section when used for analgesia. If the parturient does not feel the urge to "push" during the 2nd stage, labor may be prolonged. Motor blockade related to labor analgesia may affect the ability to push.

Anesthesia Considerations for Non-obstetric Surgical Intervention during Pregnancy

Up to 2% of women will require surgical intervention during their pregnancy. The most common surgical procedures include appendectomy and cholecystectomy. The following considerations should be taken into account:

- Maternal mortality is not increased; fetal mortality may range from 5-35%.
- Only emergent/necessary surgical cases should be performed.
- If an elective surgical procedure, postpone until 6 weeks post delivery.
- Formation of fetal organs occurs between 15-56 days of gestation. There is no conclusive evidence that anesthetics cause problems during this time.
- Regional anesthesia is preferred over general anesthesia. Spinal anesthesia is preferred over epidural anesthesia.

- Increased risk during general anesthesia is related to technique. In the past, benzodiazepines and N2O were avoided. It was thought that they may contribute to teratogenic changes in the fetus. Currently there is no conclusive evidence that these medications cause teratogenicity. Exposure to all anesthetics should be kept to a minimum.
- Fetal monitoring should occur (when possible) for pregnant patients who are > 16 weeks.
- The patient should be monitored for pre-term labor.
- The patient is considered to have a full stomach.
- Displace the uterus with a roll under the patient's hip.
- Avoid hypoxemia (use a minimum of 50% O2).
- Avoid hypotension. Pre-hydrate the patient. Aggressively treat hypotension.
- Maintain adequate ventilation. Don't allow CO2 to accumulate or hyperventilate the patient.
- Extubate the patient when fully awake to avoid the risk of aspiration.
- Review the anesthetic implications associated with pregnancy.

Summary of Anesthetic Implications of Anatomical and Physiological Changes Associated with Pregnancy

Cardiovascular
The white blood cell count and its components may be markedly elevated during labor and delivery. In the absence of signs and symptoms of infection/sepsis or other contraindications it is reasonable to proceed with neuraxial blockade.
Never lay the patient supine. Always place a wedge or roll under the right hip so the patient is "tipped" to the left.
Sympathectomy associated with regional anesthesia can result in severe hypotension. It is important to pre-load the patient with 1-2 liters of crystalloid fluids.
Vessel distention in the epidural space can increase the risk of vessel damage during neuraxial blockade. Vessel distention also decreases the intrathecal and epidural spaces. Decrease the dose of local anesthetics by 30%.
Delayed absorption of subcutaneous/intramuscular medications.
Respiratory
Patients undergoing regional anesthesia should have supplemental oxygen administered.
Patients undergoing general anesthesia should be pre-oxygenated with 100% O2 prior to anesthesia induction.
Patients may desaturate quickly despite pre-oxygenation. This is due to increased oxygen consumption and a decrease in FRC.
Be prepared for a difficult intubation due to decreased visualization from swollen mucous membranes. Ensure that the patient is positioned for optimal viewing of the glottic opening prior

to anesthetic induction.
Have a smaller than usual endotracheal tube available for intubation in case the usual sized endotracheal tube is too large due to swelling.
Be very gentle during laryngoscopy so bleeding does not obstruct the view.
The patient will have a faster uptake of inhalational anesthetics due to a decreased FRC and increased alveolar uptake.
Gastrointestinal
All pregnant patients should be considered to have "full stomachs" regardless of fasting.
If available, medications should be administered prior to anesthesia to reduce gastric acidity and volume. A non particulate antacid such as sodium citrate should be administered immediately prior to the anesthetic to reduce the acidity of stomach contents. To increase gastric emptying and increase lower esophageal sphincter tone, the administration of metoclopramide, in a dose of 10 mg IVP should be administered 30-60 minutes prior to anesthesia. The use of a histamine (H2 blocker) such as famotidine 20 mg IVP 30-60 minutes prior to anesthesia may reduce the acidity of stomach contents.
Slight reverse Trendelenburg position may reduce passive reflux.
Cricoid pressure should be utilized and held until the patient is intubated and placement of the endotracheal tube in the trachea has been confirmed.
Do not routinely administer positive pressure ventilation with a mask prior to intubation unless the patient's pulse oximetry reading starts to decline or a difficult airway is encountered. Positive pressure ventilation may cause gastric distention increasing the risk of aspiration.
Hepatic
Small risk of prolonged neuromuscular blockade when using succinylcholine or mivacurium for surgical intervention within 7 days post delivery.
Central Nervous System
Reduce the dose of inhalation anesthetics by up to 40%.
Reduce the dose of local anesthetics by up to 30%.
Positive pressure in the epidural space may make it more difficult to identify the epidural space.

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Anesthesia Implications & Approaches for Cesarean Section

Chapter Thirteen

Anesthesia Implications & Approaches for Cesarean Section

One of the most common surgical procedures performed throughout the world is the cesarean section. The parturient is at increased risk for complications due to the unique anatomical and physiological changes that occur during pregnancy. It has been reported that up to 82% of all maternal anesthetic deaths occurred during a cesarean section. Anesthetic care differs significantly from the non-parturient. Additionally, the anesthesia provider is caring for two patients at the same time, the mother and her baby. Knowledge of anatomical and physiological changes associated with pregnancy, their impact on anesthesia care, and specific considerations for this special patient population can reduce mortality and morbidity. This chapter will cover the normal patient undergoing cesarean section in an elective or emergent situation. The next chapter will detail anesthetic care for specific medical/obstetric conditions.

Indications for Cesarean Section

Cesarean sections may be performed for a variety of reasons including:

- Fetal distress (non-reassuring fetal status)
- Umbilical cord prolapse
- Maternal hemorrhage or risk for hemorrhage (i.e. placenta previa, abruption of placenta, previous vaginal reconstruction)
- Impending maternal death with a viable infant
- Abnormal presentation (i.e. transverse or oblique position, breech)
- Pelvic to fetus disproportion (cephalo-pelvic disproportion or CPD)
- Dysfunction of uterine activity (i.e. failure of labor progression or fetal descent)
- Risk of, or suspected, uterine rupture (i.e. previous classical cesarean section, or uterine surgical intervention)

Common Concerns

- **Hypotension**:
    - Pregnant women are at risk for aortocaval compression when supine. The uterus should always be displaced using a hip roll such as a folded towel or liter bag of intravenous fluid.
    - Preloading the patient with non-dextrose containing crystalloid fluids prior to the administration of regional anesthesia reduces the incidence of hypotension.

- **Hemorrhage**:
  - Always maintain a functioning IV. If intravenous flow is inadequate, start another IV. The IV catheter should be at least an 18 gauge.
  - Start two IV's if the patient is at increased risk for hemorrhage.
- **Aspiration:**
  - All obstetric patients should be considered to have full stomachs.
  - Reduce risk of aspiration with pre-medications.
  - Use rapid sequence induction with cricoid pressure.
  - Do not routinely mask ventilate the patient prior to intubation.
  - Slight reverse Trendelenburg position may reduce passive reflux.
- **Hypoxemia:**
  - The obstetric patient has a 20% decrease in functional residual capacity (FRC) and increased O2 consumption. The patient is at increased risk for hypoxia.
  - 100% O2 should be administered to the patient undergoing general anesthesia.
  - Supplemental oxygen should be administered to patients undergoing regional/local infiltration techniques.
- **Difficult Intubation:**
  - Obstetric patients are at increased risk for difficult intubation.
  - Position the patient for optimal laryngoscopy in the "sniffing position".
  - Ensure availability of several laryngoscope blades, different sized endotracheal tubes, and functioning laryngoscope handle.
  - The pregnant patient generally requires a smaller endotracheal tube (6.0, 6.5 or 7.0mm ETT) due to the engorgement of the airway from pregnancy related increase in blood volume.
  - Be gentle during laryngoscopy to reduce the potential for bleeding which can obstruct the view.
- **Anesthetic Requirements:**
  - Reduce inhaled anesthetics by 40%.
  - Reduce the dose of local anesthetics placed during neuraxial blockade by 30%.

Preparation

It is important to always be prepared. The anesthesia provider should ensure that all the equipment that may be needed is available and working properly.

- Know what equipment is available
- Ensure the availability of an adequate supply of oxygen
- Ensure that the suction apparatus is functional (manual as a backup)
- A roll or wedge should be available (often a rolled up towel will suffice)

- Intubation equipment should be functioning with a variety of anesthesia masks, laryngoscope blades, endotracheal tubes, stylets, and oral airways.
- If available, alternative airways such as a laryngeal mask airway or combitube can be life saving in the event of difficult or failed intubation.
- A bag mask valve device should be available in case the anesthesia circuit fails
- Resuscitation equipment and medications should be readily available
- Monitoring equipment should be available and functional
- An assistant with experience in maintaining cricoid pressure should be available if a general anesthetic is required
- Sterile equipment for neuraxial blockade
- Sterile local anesthetic solutions
- Anesthetic medications for the induction of general anesthesia

Once a patient is identified as requiring an anesthetic for a cesarean section, it is important to perform an anesthetic evaluation as early as possible. In addition to normal preoperative evaluation, it is important to include:

- Maternal health history
- Anesthesia related history
- Vital sign assessment
- Laboratory values if available (i.e. complete blood count; PT, PTT, platelets for the patient with pregnancy induced hypertension).
- A thorough airway examination with the patient sitting (please refer to **Practical Guide to Anesthesia, volume 1**, chapter 8).
- Examination of the back for regional anesthesia.
- Documentation of oral intake. All parturient patients are considered to have a full stomach regardless of how long they have been fasting. Fasting prior to an elective cesarean section should be a minimum of 6 hours.
- Consent for anesthesia
- Functional IV
- Preoperative medications should focus on minimizing the risk of aspiration. Ranitidine 50 mg IVP or famotidine 20 mg IVP should be administered immediately to help decrease the acidity of stomach contents. A non particulate antacid should also be administered (i.e. 30 ml of sodium citrate) to reduce acidity of the stomach contents. Metoclopramide, 10 mg slow IVP, may help to reduce contents of the stomach. Metoclopramide should be administered approximately 30 minutes prior to the induction of anesthesia.
- May consider glycopyrrolate (0.1-0.2 mg IVP) or atropine (0.4 mg IVP) to reduce oral secretions
- Avoid the administration of sedative medications prior to cesarean section. This may depress the baby.

- Consideration of anesthetic technique. Neuraxial block may help reduce mortality and morbidity in this high risk population if not contraindicated. The anesthesia provider should always be prepared for a general anesthetic since regional blocks can fail or be inadequate. Cesarean section can also be performed under local infiltration.
- Pre-loading of the patient with non-glucose containing crystalloid solutions should occur prior to neuraxial blockade to reduce declines in blood pressure secondary to sympathetic blockade.
- Calculation of medications doses should be based on mg/kg basis.

Anesthetic Techniques

- Local infiltration
- Regional or neuraxial anesthesia (spinal or epidural)
- Endotracheal general anesthesia

Local Anesthetic Infiltration

This technique is not ideal. It is used in circumstances where the resources are minimal and in situations where the mother will not survive a regional or general anesthetic.

Advantages

- Alternative for a patient who may not survive a regional or general anesthetic.
- Addition of 1:200,000 epinephrine will reduce bleeding at the incision site, prolong duration and increase the density of blockade.
- Provides an alternative technique for emergencies in locations where the surgeon is the sole medically trained provider.
- Inexpensive technique

Disadvantages

- Not an ideal technique
- The surgeon must have experience in this technique since exposure is limited.
- Takes time to adequately anesthetize each layer of tissue. May lead to increased fetal mortality and morbidity.
- Maternal discomfort
- Risk for local anesthetic toxicity due to large volumes of local anesthetic required.

Technique

The patient should be thoroughly prepared for this technique. The patient will experience the sensation of touch and some discomfort. If the patient realizes that this is the only technique available to save her or her baby, she will be more cooperative. Knowledge of the maximum doses of local anesthetics is important to avoid toxicity (refer to Chapter 1 of this manual under local and topical local anesthetics). Generally, 0.5% lidocaine is used for local infiltration. The maximum dose of lidocaine, with 1:200,000 epinephrine, is 7 mg/kg with a maximum total of 500 mg (100ml of 0.5% lidocaine with 1:200,000 epinephrine). Most women in developing countries will not weigh 70kg. In these cases do not exceed 7 mg/kg of lidocaine with epinephrine. For those whose calculations on a mg/kg basis would exceed 500 mg of lidocaine, do **not** exceed the total maximum dose of 500 mg. The maximum safe dose should be calculated prior to local infiltration.

- For example, the patient weighs 50 kg and 0.5% lidocaine with 1:200,000 epinephrine is available.
- First, figure out the mg/ml of lidocaine. A 0.5% solution of lidocaine contains 5 mg/ml.
- Next, calculate 7 mg/kg by the patient's weight of 50 kg. 7 multiplied by 50 = 350 mg.
- Next, divide 5 mg into the total dose to calculate the total number of ml that can be safely administered. 5 divided into 350 = 70 ml.

If the solution is plain, epinephrine can be added to the local anesthetic to a concentration of 1:200,000 or 5 mcg per ml. The addition of 1:1000 (1mg/ml) epinephrine to the local anesthetic can be easily calculated using the following guide:

- Take the total volume of local anesthetic (i.e. 100 ml of 0.5% lidocaine), divide it in half, and move the decimal point two places to the left.
- 100 ml of 0.5% lidocaine divided by 2 = 50.
- Move the decimal point two places to the left. The result is 0.5.
- Add 0.5 ml (500 mcg) of epinephrine to the local anesthetic solution for a 1:200,000 concentration.
- To check this, simply multiply 5 mcg/ml by 100 ml which equals 500 mcg.
- **It is important to always check the concentration of epinephrine and double check the total dose added to the local anesthetic.**

A second technique for adding epinephrine to local anesthetic preparations is detailed below:

- 1:200,000 epinephrine concentration would be 5 mcg/ml.
- Dilute epinephrine using a 10 ml syringe. Draw up 1 ml of 1:1000 epinephrine (1 mg per ml) and 9 ml of normal saline.
- Mix it by tilting the syringe back and forth.
- The concentration of epinephrine is now 100 mcg per ml.
- Add epinephrine to the local anesthetic solution (see table below).

1:200,000 Epinephrine Concentration	
Volume of Local Anesthetic	**Amount of Epinephrine Added to Local Anesthetic Solution**
20 ml	100 mcg of epinephrine
30 ml	150 mcg of epinephrine
40 ml	200 mcg of epinephrine
50 ml	250 mcg of epinephrine

- Always label the syringe of epinephrine. Once epinephrine is added to the local anesthetic, discard what remains. Epinephrine can be lethal. It should be discarded to avoid inadvertent administration.

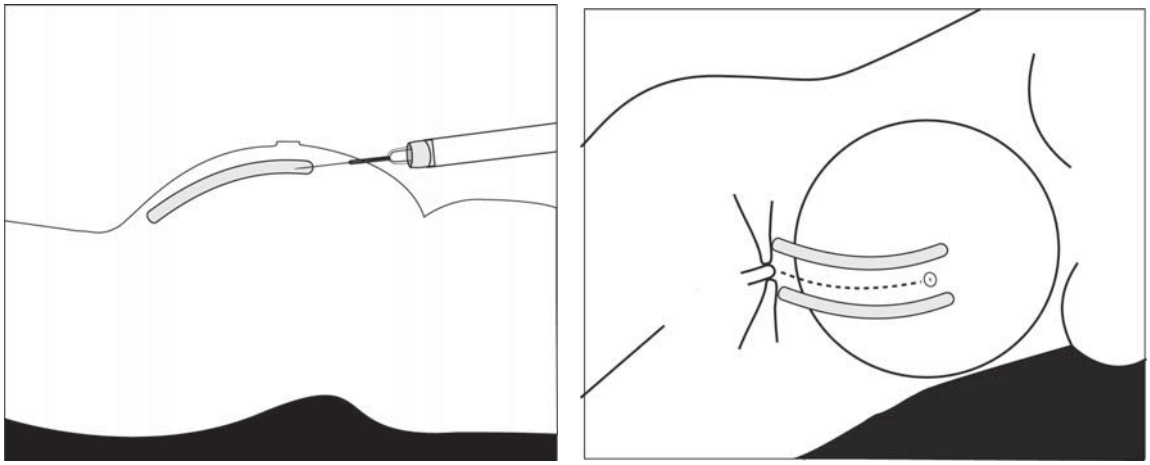
Lidocaine may be available in a 1% solution without epinephrine. The following is an example of how to calculate an appropriate dose and create a 0.5% solution for infiltration anesthesia.

- A 1% solution of lidocaine yields 10 mg/ml.
- The maximum dose of lidocaine is 7 mg/kg with epinephrine.
- The patient weighs 50 kg. 7 mg/kg x 50 = 350 mg.
- Divide 10 mg (1% solution) into the total dose to calculate the total number of ml. 10 mg divided into 350 mg = 35 ml.
- Next add an equal volume of sterile, preservative free normal saline. In this case 35 ml. The total volume should = 70 ml.
- The final step is to add epinephrine to reach a concentration of 1:200,000 or 5 mcg/ml. **It is important to always check the concentration of epinephrine, and double check the total dose added to the local anesthetic.**

If lidocaine is only available in a 2% solution without epinephrine, the following is an example of how to calculate the dose and create a 0.5% solution for use in infiltration anesthesia.

- 2% lidocaine (without epinephrine) is available. A 2% solution of lidocaine yields 20 mg/ml.
- To use the maximum dose of lidocaine is 7 mg/kg, with epinephrine.
- The patient weighs 50 kg. 7 mg/kg x 50 = 350 mg.
- Divide 20 mg (2% solution) into the total dose to calculate the total number of ml. 20 mg divided into 350 mg = 17.5 ml.
- Next add sterile, preservative free normal saline to yield a 0.5% solution. In this case 52.5 ml. The total volume now should = 70 ml. To check this multiply 5 mg x 70 = 350 mg.
- The final step is to add epinephrine to reach a concentration of 1:200,000 or 5 mcg/ml. **It is important to always check the concentration of epinephrine and double check the total dose added to the local anesthetic.**
- Administer supplemental oxygen
- Place a wedge under the patient's hip

- Always maintain sterile technique
- Consider intravenous administration of atropine (0.5 mg IVP) to reduce the incidence of bradycardia and nausea/vomiting due to vagal stimulation.
- Place a long wheal of local anesthetic 3-4 cm from the midline of the symphysis pubis (on either side) up to 5 cm above the umbilicus. Ensure that the needle is parallel to the skin. The abdominal wall is very thin and care must be taken to avoid injection through the peritoneum. Avoid injection into the uterus.



- As surgical exposure continues 10 ml of local anesthetic solution is administered under the linea alba to anesthetize the parietal peritoneum. Once the loose visceral peritoneum of the uterus is exposed, and the point of entry to the uterus is identified, an additional 5 ml of local anesthetic may be administered to reduce discomfort.
- Consider supplementation with ketamine in doses of 0.25-1 mg/kg IVP for patient discomfort. Ketamine in low doses will not usually depress the baby and should not cause an increase in the patient's blood pressure. Ketamine should not be administered to patients with hypertension. Ketamine may cause contraction of the uterus and should be avoided in situations of life threatening fetal distress.
- Once the baby is delivered, additional sedation and analgesics may be considered.
- Ergometrine or oxytocin is administered after delivery of the baby. These agents help the uterus to contract, reducing bleeding.
    - Ergometrine is supplied in 1 ml ampoules that contain 0.5 mg. The solution should be white/pink. If the solution is yellow/green it should not be used. The usual adult dose is 0.5 mg IM. Caution must be used if the patient has had the recent administration of a vasopressor since the combination of ergometrine and a vasopressor can produce intense vasoconstriction. Ergometrine is contraindicated in patients with hypertension.
    - Oxytocin generally is available in an ampoule that contains 10 units/1 ml. Oxytocin may be administered by intravenous drip, 10-20 units in 1000 ml of intravenous solution, infused at a rate of 2-3 ml per minute. The infusion rate may be increased

if the uterus is not contracting normally. Alternatively the infusion concentration may be increased.

- NEVER administer these medications prior to delivery since it can harm the baby. Do not draw up these medications until needed. Do not give the medication until it has been discussed with the surgeon.

Neuraxial Blockade

Neuraxial blockade for cesarean section is a common and safe technique when appropriately administered. Neuraxial blockade includes spinal and epidural blockade. For a detailed discussion of these techniques, please refer to the following sections of this manual: introduction to neuraxial blockade; neuraxial blockade anatomy, landmarks, physiologic effects & complications; spinal anesthesia; and epidural anesthesia.

Epidural anesthesia is more technically challenging than spinal anesthesia. It requires specialized equipment. Advantages of epidural anesthesia include the ability to prolong anesthesia by adding local anesthetic through the epidural catheter and a gradual onset of anesthesia. This limits sympathectomy and subsequent decrease in blood pressure when compared to spinal anesthesia.

Spinal anesthesia is technically easier to administer than epidural anesthesia. Onset is rapid. The patient is at risk for the development of hypotension due to sympathetic blockade. Spinal anesthesia cannot be prolonged beyond the initial dose. The equipment and medications required for spinal anesthesia are more likely to be available in most countries.

Neuraxial blockade offers several advantages over general anesthesia. The overall mortality rate is less with neuraxial blockade compared to general anesthesia. For routine cesarean sections, it is reasonable to choose regional over general anesthesia, unless there are specific obstetric/medical conditions that would contraindicate regional anesthesia. Advantages include: maintenance of the airway, decreased medication transfer to the fetus, reduced blood loss, and decreased expense. With experience, spinal anesthesia can be administered almost as quickly as general anesthesia. Epidural anesthesia takes more time and may not be suitable in emergent situations. Neuraxial anesthesia is contraindicated for some obstetric abnormalities including preeclampsia with coagulation abnormalities, hemorrhage, uncorrected hypovolemia, bleeding disorders, generalized sepsis, infection at the injection site, raised intra-cranial pressure, or uncontrolled hypertension. There are specific obstetric abnormalities that require clotting function to be tested prior to neuraxial blockade. These conditions will be discussed in the next chapter.

For either spinal or epidural anesthesia considerations, monitoring and potential complications are the same. In depth coverage of complications can be found in the regional anesthesia section of this manual.

Considerations for Neuraxial Blockade

- Discuss risks and benefits
- Obtain permission for the administration of neuraxial blockade.
- Administer aspiration prophylaxis
- Ensure that the IV is functional and preload the patient.
    - Spinal- 1-2 liters of fluid within 15-30 minutes of the spinal.
    - Epidural- 500-1000 ml prior to the initiation of the epidural.
- A roll for uterine displacement
- Ensure that medications and airway equipment are available for possible conversion to general anesthesia.
- Suction, oxygen, and resuscitation medications should be immediately available.
- Vasopressors such as ephedrine and phenylephrine should be drawn up and ready to administer. Atropine should be immediately available.
- Record a set of vital signs prior to neuraxial blockade.
- Sterility of all neuraxial equipment and medications should be ensured. Contamination can lead to infection or meningitis. Always double check that the appropriate preservative free, local anesthetic is being administered. Severe injury to the patient's spinal cord and nerves can be caused by the injection of a medication that does not belong in the epidural or subarachnoid space. Always double check local anesthetics for expiration date and sterility. Do not use if the local anesthetic is expired or sterility cannot be guaranteed.
- Reduce the dose of local anesthetic by 30% of the dose administered to a patient who is not pregnant.
- Always wear a hat, mask, and sterile gloves.
- Proper positioning improves the chance of successful neuraxial block.
- Administer the block

Technique

- Please refer to the neuraxial section of this manual

Monitoring

- Monitoring includes continuous ECG, pulse oximetry, and blood pressure.
- Fetal heart rate monitoring should be monitored up until regional anesthesia is initiated (if available).
- **Monitor the patient's vital signs frequently**. Check the patient's blood pressure every 1-2 minutes following neuraxial blockade until the baby is delivered. If the patient remains stable after the delivery, the blood pressure frequency can be decreased. Remember that spinal anesthesia may take up to 15 minutes to set up, epidural anesthesia may take longer.

- **Assess the block**. For cesarean block a level of T4-T6 should be adequate. Sympathectomy (loss of temperature sensation) can be used to initially test the block with an alcohol wipe. Warmth and vasodilatation should be noted in the feet and lower extremities. A "pin prick" sensation is more accurate than temperature or light touch. Do not use a needle or anything sharp that may break the skin. The stylet of the spinal or epidural needle will work well. First test it on the arm. Ask the patient when it feels as sharp as it did on the arm. Slowly work up from the lower limbs to the desired level (T4=nipple line to T6). The difference between temperature and sharp sensation may be as much as 2-8 dermatomes.
- **Monitor the patient for complications of neuraxial blockade**. Rapid and significant hypotension will accompany a sympathetic block, especially if the patient has not received an adequate fluid bolus. A decrease in the systolic blood pressure of > 20% must be treated. Ensure that the patient has a hip roll to avoid compression of the aorta and inferior vena cava. Increase the IV infusion and treat the patient with ephedrine (5-10 mg IVP) or phenylephrine (50-100 mcg IVP). **Nausea should be considered to be related to hypotension until proven otherwise!** This complaint should be a sign to immediately check the patient's blood pressure.
- **Monitor the patient's heart rate continuously**. A block above T4 may result in bradycardia due to blockade of the cardioaccelerator fibers (T1-T4). Treatment depends on the severity of bradycardia and includes the following: ephedrine (5-10 mg IVP) or atropine (0.5 mg IVP). Epinephrine (5-10 mcg IVP) may be used and titrated to effect if the patient is unresponsive to ephedrine or atropine.
- **Monitor the patient for shortness of breath**. The pregnant patient has decreased lung capacity and increased oxygen consumption. Neuraxial blockade will cause the patients diaphragm and intercostal muscles to be weak, decreasing the patient's lung capacity. The patient may also complain feeling funny when breathing due to blockade of the intercostal muscles. Monitor pulse oximetry and reassure the patient.
- **Monitor the patient for a high spinal**. The patient may experience numbness and tingling of the chest, fingers, arms, and neck. If the patient has problems speaking this indicates that the block is too high. Monitor the patient for respiratory distress and loss of consciousness. With a hyperbaric spinal solution you may try to elevate the head and neck. Administer 100% oxygen, treat blood pressure, and prepare for intubation. Support the patient's heart and respiratory function. At the same time, monitor the patient's vital signs and prepare for cardiac arrest. Generally the patient will recover quickly from a high spinal.
- **Monitor the patient for signs and symptoms of local anesthetic toxicity with epidural anesthesia**. It is important to use a test dose, aspirate the epidural catheter frequently, and ask the patient how they feel when dosing an epidural. Inadvertent intravascular injection can result in local anesthetic toxicity. Symptoms range from ringing in the ears, numbness of the mouth or tongue, dizziness, confusion, decreased level of consciousness, convulsions, and cardiac arrest.
- For a discussion of local anesthetics please refer to the spinal and epidural anesthesia sections of this manual. **It should be noted that 0.75% bupivacaine for EPIDURAL**

anesthesia should never be used in the obstetric population due to potential cardiotoxicity and cardiac arrest. The use of 0.75% SPINAL bupivacaine is approved and safe for the obstetric patient.

- If neuraxial block is "patchy" it may be necessary to supplement with small doses of ketamine (10-20 mg IVP) or 40-50% nitrous oxide until delivery. Nitrous oxide can result in an anesthetic rather than analgesic level due to decreased FRC. The patient who suddenly has a change in consciousness may be "too deep". The concentration of nitrous oxide should be decreased. Once the baby has been delivered, consider the administration of an analgesic such as fentanyl in a dose of 0.5-1 mcg/kg. If the block is inadequate to the point that the patient is in pain, alert the surgeon and induce general endotracheal anesthesia.
- Replace blood loss with crystalloid intravenous solutions (3 ml of crystalloid for 1 ml of blood loss). Alternatively, colloid solutions may be used (1 ml of colloid for 1 ml of blood loss).

General Anesthesia

General anesthesia should be reserved for emergent situations and patients that can't receive a regional anesthetic either due to the parturient's medical condition such as hemorrhage (i.e. valvular heart disease, clotting disorders, placenta previa, placenta accrete, abruptio placentae, or uterine rupture) or extreme fetal distress (i.e. umbilical cord prolaspe). General anesthesia is also used for the patient that refuses a regional anesthetic. The risk of pulmonary aspiration is 4 times higher and risk of failed intubation is almost 7 times higher for the parturient, increasing mortality and morbidity. In an attempt to reduce the incidence of a depressed baby, lower than usual concentrations of volatile anesthetics are administered until delivery, increasing the risk of awareness. After delivery the volatile anesthetic concentration may be increased.

Considerations for General Anesthesia

- Usually reserved for emergency situations. Quickly obtain a history and physical. Instruct the patient of risk and benefits. Obtain permission to proceed with the anesthetic. Reassure the patient that she will be asleep before the surgery begins but preparation of anesthesia and surgery will be conducted while the patient is still awake.
- Pre-operative medications should be administered (i.e. non-particulate antacids, metoclopramide 10 mg IVP, and a histamine 2 blocker such as ranitidine 50 mg IVP or famotidine 20 mg IVP).
- Ensure the IV is functional. If the patient is at risk for hemorrhage, ensure that the patient has two large bore IV's (18 gauge or larger). If a blood transfusion is anticipated, ensure that type and cross match is performed, if available.
- Prehydrate the patient
- Prepare anesthetic medications, suction, and resuscitation equipment.

- Ensure that resuscitation medications are immediately available.
- Vasopressors, including ephedrine and phenylephrine, should be drawn up and ready to administer. Atropine should also be immediately available.
- The patient's airway is a major concern for the anesthesia provider.
- Several different sized endotracheal tubes, stylets, airways, and laryngoscopy blades should be available for use.
- It is ideal to have airway alternatives such as a LMA, combitube, and emergency airway equipment to perform a cricothyroidotomy.
- An assistant should be available to apply cricoid pressure.

Technique

- Patient should be monitored by continuous ECG, continuous pulse oximetry, and blood pressure. If available use an oxygen monitor that measures the concentration of oxygen being administered and end-tidal carbon dioxide to measure exhaled carbon dioxide.
- Uterine displacement should be maintained with the use of a wedge or folded towel under the right hip.
- 100% O2 should be administered by mask to pre-oxygenate the patient.
- Position the patient in a "sniffing" position to maximize the view during laryngoscopy.
- The patient should be prepped and draped. The surgical team should be in place ready to "cut".
- Induce with general anesthesia.
    - Common induction agents for the parturient with a normal blood pressure include propofol 2 mg/kg IVP and thiopental 4 mg/kg.
    - The patient who has a low blood pressure, active hemorrhage, or severe asthma should be induced with ketamine in doses of 1-2 mg/kg.
- Apply cricoid pressure as the patient looses consciousness.
- Administer succinylcholine in a dose of 1.5 mg/kg (unless contraindicated). Non-depolarizing muscle relaxants may be used if succinylcholine is contraindicated; onset will be slower.
- Avoid mask ventilating the patient unless hypoxia occurs.
- Intubate when it is apparent that the patient is paralyzed.
- Cricoid pressure may be released once it is confirmed that the endotracheal tube is properly placed in the trachea.
- Surgery may start once the patient is intubated, ventilated, and the airway is secure.
- Inhaled anesthetics should be started at 0.5-0.75 MAC. 50% N2O may be used with 50% oxygen for the fetus that is not in distress. If the fetus is in distress, use 100% oxygen. The lower than normal dose of inhalational anesthetics should result in amnesia, should not

depress the fetus, and will not relax the uterus too much. All volatile anesthetics relax the uterus in a dose dependent manner.

- Avoid hyperventilation since this will cause vasoconstriction of uterine blood flow.
- Avoid hypotension which results in fetal distress. Treat decreases in blood pressure > 20-30% with intravenous fluids and Trendelenburg position. Medications such as ephedrine 5-10 mg IVP or phenylephrine 50-100 mcg IVP can be used. Repeat doses as necessary. For hypotension that is unresponsive to the above measures, epinephrine (5-10 mcg IVP) may be used. Repeat as necessary to effect.
- Once the baby is delivered, ergometrine or oxytocin is administered. These agents help the uterus contract, reducing bleeding. Ergometrine is supplied in 1 ml ampoules that contain 0.5 mg. The solution should be white/pink. If the solution is yellow/green it should not be used. The usual adult dose is 0.5 mg IM. Caution must be used if the patient has had the recent administration of a vasopressor, since the combination of ergometrine and a vasopressor can produce intense vasoconstriction. Ergometrine is contraindicated in patients with hypertension. Oxytocin generally is available in an ampoule that contains 10 units/1 ml. Oxytocin may be administered by intravenous drip with 10-20 units in 1000 ml of intravenous solution, infused at a rate of 2-3 ml per minute. The infusion rate may be increased if the uterus is not contracting normally. Alternatively, the infusion concentration may be increased. NEVER administer these medications prior to delivery since it can harm the baby. Do not draw up these medications until needed. Do not administer the medication until it has been discussed with the surgeon.
- The anesthetic may be deepened after delivery. Opioids should be administered for discomfort. Benzodiazepines may be administered for amnesia.
- If the uterus is not contracting, decrease the concentration or discontinue the use of the agent. Supplementing the anesthetic with ketamine in doses of 10-20 mg, benzodiazepines, and/or N2O in a concentration of 40-50% will be required for amnesia.
- Replace blood loss with crystalloid intravenous solutions (3 ml of crystalloid for 1 ml of blood loss). Alternatively, colloid solutions can be used (1 ml of colloid for 1 ml of blood loss).
- If possible use an oral gastric tube to suction out the stomach near the end of surgery. Avoid the nose due to increased risk of a bleeding.
- During emergence, ensure that the muscle relaxant has worn off. Reverse non depolarizing muscle relaxants.
- Extubate only when the patient exhibits a train of four, sustained tetanus, adequate ventilation, a 5 second head lift, and the ability to follow commands.

Monitoring

- Patient should be monitored by continuous ECG, pulse oximetry, and blood pressure. If available, use an oxygen monitor that measures the concentration of oxygen being administered and end-tidal carbon dioxide.
- The inability to intubate, ventilate, and oxygenate the parturient is a common cause of anesthetic related death. Confirmation of correct endotracheal tube placement is essential.

Confirmation of Correct Endotracheal Tube Placement
Trachea vs. Esophagus

Test	Result	How Reliable is it?
End tidal carbon dioxide testing	**Correct**: positive wave form **Incorrect position**: no waveform	Certain- is the best test
Esophageal detection device (i.e. 50 ml syringe with self inflating bulb)	**Correct**: air is easily aspirated **Incorrect:** the bulb does not aspirate air	Certain- unless the patient has a lot of air in the stomach.
Watch endotracheal tube go between vocal cords	**Correct:** easy view	Certain- unless visualization was poor.
Pulse oximetry	**Correct:** the reading easily comes up and reads within the normal range for the patient. **Incorrect:** the reading declines and continues to decline despite ventilation.	Certain
Listen with stethoscope	**Correct**: bilateral and equal breath sounds are noted. **Incorrect:** no breath sounds are noted/gurgling sound is noted over the stomach.	Probable- sounds can radiate and fool the anesthesia provider.
Ventilate the patient	**Correct:** easy to ventilate, chest rises. **Incorrect:** difficult to ventilate, stomach gurgles, chest does not rise.	Probable- the anesthesia provider can sometimes find it hard to distinguish between esophageal and tracheal placement of the endotracheal tube.
Observe the patient	**Correct**: the patient remains pink. **Incorrect:** the patient becomes cyanotic.	Certain/probable- by the time the patient becomes cyanotic the patient is very hypoxic.
Pushing on the patient's chest/condensation in the endotracheal tube	**Correct:** air comes back/condensation occurs. **Incorrect:** air does not come back/no condensation noted.	Probable- other techniques are more accurate.

- The anesthesia provider should use more than one of the above clinical tests to ensure that successful intubation and ventilation has occurred.
- Continually assess the patient's color, pulse oximetry, chest wall movement, end tidal carbon dioxide (if available). Use a pre-cordial stethoscope and monitor for changes in airway resistance.

- Do not hyperventilate. This will decrease CO2, causing uterine artery vasoconstriction, increasing fetal distress.
- Monitor the patient's blood pressure every 2-3 minutes until the baby is delivered. Rapidly treat hypotension. Hypotension will increase fetal distress.
- Once the baby is delivered, the blood pressure may be checked every 5 minutes if the patient is stable.
- Monitor the patient for inadequate anesthesia. The patient is paralyzed from the muscle relaxant and not able to move. Potential signs of light anesthesia in the paralyzed patient include tachycardia, hypertension, sweating, and dilation of the pupils.
- If non depolarizing muscle relaxants are used, make sure that the patient is fully reversed, able to sustain a 5 second head lift and follows command prior to extubation. If the patient is on magnesium the duration of the muscle relaxant may be prolonged.

Failed Intubation

The anesthesia provider should be prepared for a failed intubation. The risk of hypoxia and death is very real. In most circumstances the following steps should be followed:

- Continue cricoid pressure
- Remain calm
- Attempt to ventilate the patient. Inability to intubate is frustrating, the inability to ventilate is deadly!
- Consider additional attempts or have another experienced anesthesia provider attempt intubation. Optimize the intubating position of the patient.
    - Ensure that the patient's head is positioned in sniffing position
    - Try another blade, smaller endotracheal tube.
    - Ensure that cricoid pressure is properly applied and not obstructing view.
    - Do not persist with attempts if it is apparent that the patient cannot be intubated. This only creates more bleeding and swelling which may lead to difficulty in ventilation.
- If unsuccessful, continue to ventilate and allow the patient to awaken from the anesthetic.
- Once awake, proceed with a regional anesthetic (i.e. spinal, epidural, or local infiltration).
- If the cesarean section is an emergency (i.e. maternal hemorrhage or fetal distress) the anesthesia provider needs to communicate with the surgeon.
    - Can the patient be allowed to wake up and proceed with an alternative plan?
    - Is the situation extreme enough that the case must continue with mask ventilation, LMA, or combitube?
    - If unable to ventilate the patient at all, does the patient require an immediate airway by performing a cricothyroidotomy?
- If the case is continued using a mask or LMA, cricoid pressure must be maintained until the patient is awake.

Aspiration of Gastric Contents

The parturient is at risk for aspiration. Even a well conducted rapid sequence induction may result in this complication. Aspiration can occur during induction, emergence, or difficult intubation. Signs and symptoms include:

- Gastric contents in the endotracheal tube despite correct placement in the trachea.
- Wheezing, rales, or rhonchi (the most common site of aspiration is on the right side of the lung).
- Hypoxia (large particles of food can block lung passages).
- Signs of aspiration take a few hours to develop on chest x-ray.

Treatment is mainly supportive and includes:

- Suction the endotracheal tube. DO NOT lavage the endotracheal tube. This is not effective and may worsen the situation.
- Bronchoscopy (if available) may be required to remove large food particles.
- The patient may require postoperative ventilation.
- Cough, deep breath, and incentive spirometry for those who are not intubated. Supplemental oxygen may be required. Observe the patient for signs of respiratory distress.
- Corticosteriods may be considered. It is unknown if these agents are helpful.
- Broad spectrum antibiotics may be considered if solid food particles have been aspirated or bacterial pneumonia occurs.

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Valvular Heart Disease & Obstetric Related Conditions

Chapter Fourteen
Valvular Heart Disease and Obstetric Related Conditions

This chapter contains two distinct topics. Valvular heart disease and obstetric related conditions (i.e. hemorrhage, hypertension, and air embolism) both impact anesthetic management and increase mortality and morbidity. The purpose of this section is to briefly cover these conditions, specific anesthetic considerations, and implications.

Valvular Heart Disease

Patients with valvular heart disease are at increased risk for mortality and morbidity during labor and in the immediate post partum period. In women that do not have a history of valvular heart disease, pregnancy can result in subtle signs and symptoms of heart failure. Many women exhibit decreased exercise tolerance, peripheral edema, dyspnea, and fatigue. The following cardiovascular changes can exacerbate symptoms of valvular heart disease:

- ✓ An increase of 1-1.5 liters in blood volume
- ✓ An increase in stroke volume/heart rate which increases cardiac output by 40% at term.
- ✓ During labor, stroke volume will increase by 50%. Each uterine contraction results in 300-500 ml of blood being placed into the general circulation.
- ✓ Increase in systemic vascular resistance during labor.

It is important to identify patients with valvular heart disease during the preoperative evaluation, taking into account anesthetic considerations. Patients with moderate to severe valvular disease should be treated in an environment that is capable of the intensive care that these patients require, including invasive monitoring if available.

Mitral Stenosis

Mitral stenosis is the most common valvular lesion. Considerations for patients with mitral stenosis include:

- Mild mitral stenosis is generally well tolerated.
- The patient with moderate to severe mitral stenosis is at risk for pulmonary edema and congestive heart failure.
- Patients with moderate to severe valvular disease should be treated in an environment capable of the intensive care that these patients require, including invasive monitoring if available.

- Atrial fibrillation can occur. The onset of atrial fibrillation should be treated with a beta blocker, calcium channel blocker, or digoxin. Patients exhibiting symptoms of pulmonary edema related to atrial fibrillation should be immediately cardioverted, if available.
- Tachycardia should be avoided. Labor analgesia may be indicated. This can be accomplished by using epidural analgesia with slow titration of the block. Use the smallest amount of local anesthetic required for pain relief. Local anesthetics should be plain, since epinephrine may precipitate tachycardia.
- The patient does not tolerate abrupt changes in systemic vascular resistance. For cesarean section the patient would be better served by a general anesthetic.
- Decreases in blood pressure should be treated with small doses of phenylephrine (i.e. 50 mcg). Avoid medications that cause tachycardia.
- The anesthesia provider should be careful with intravenous fluids. Too much can cause pulmonary edema.
- The anesthesia provider should try to maintain a sinus rhythm and a slow heart rate.
- Avoid hypoxemia. This increases pulmonary vascular resistance. Use pulse oximetry and supplemental oxygen.
- Titrate medications on induction. Avoid large doses of thiopental, propofol, and inhalational anesthetics. This can result in excessive decreases in the systemic vascular resistance. On the other hand the anesthesia provider will want to avoid excessive stimulation that can cause tachycardia. Consider the use of a beta blocker to blunt the stimulation of laryngoscopy.

Aortic Stenosis

- Has a higher mortality rate than other valvular lesions.
- Patients with moderate to severe valvular disease should be treated in an environment that is capable of the intensive care that these patients require, including invasive monitoring if available.
- General anesthesia should be used for cesarean section.
- Use of invasive monitoring, if available
- Avoid tachycardia, hypovolemia, and hypotension.
- Treat hypotension with phenylephrine.

Mitral and Aortic Regurgitation

- Generally tolerate pregnancy well, except for severe lesions.
- Patients with severe valvular disease should be treated in an environment that is capable of the intensive care that these patients require, including invasive monitoring if available.
- Regional techniques work well to keep forward flow of blood, reducing regurgitation.
- Hypotension should be treated with ephedrine.

- Avoid bradycardia.

Conditions Associated with Pregnancy

Hypertension in Pregnancy

Hypertension in pregnancy refers to a blood pressure greater than 140/90 and includes a range of conditions:

- Chronic hypertension: predates pregnancy or develops within the first 20 weeks of pregnancy, lasting beyond 12 weeks postpartum.
- Gestational hypertension: blood pressure greater than 140/90 for the first time during pregnancy, resolving within a 12 week period in the postpartum period without proteinuria.
- Preeclampsia: blood pressure greater than 140/90 after 20 weeks of pregnancy with proteinuria. Severe preeclampsia is defined as a blood pressure > 160/105. It is associated with hypertension, proteinuria, and edema (including face and hands). Severe preeclampsia can be complicated by hemolysis, elevated liver enzymes, and low platelets (known as HELLP syndrome).
- Eclampsia: seizures associated with preeclampsia that cannot be attributed to another cause.
- Chronic hypertension with superimposed preeclampsia

Preeclampsia (including HELLP syndrome) and eclampsia are sometimes referred to as 'pregnancy induced hypertension'. This group of syndromes results in significant maternal and neonatal mortality and morbidity. The definitive treatment for pregnancy induced hypertension is delivery of the baby and placenta.

Pregnancy induced hypertension affects every major organ system. Symptoms are dependent upon the severity of the disease. The following organ systems may be affected:

- Cardiovascular
    - decreased intravascular volume
    - increased vascular resistance
    - hypertension
    - heart failure in severe cases
- Pulmonary
    - upper airway edema (greater than the normal parturient)
    - pulmonary edema
- Neurological
    - headaches
    - visual disturbances
    - seizures
    - stroke and cerebral edema

- Hepatic
    - elevated liver enzymes
    - possible hematoma and rupture of the liver
- Renal
    - spilling of protein (proteinuria)
    - retention of sodium
    - decreased glomerular filtration
    - potential for renal failure
- Hematological
    - low or abnormal platelets
    - prolonged PTT
    - hemolysis

Management of severe pregnancy induced hypertension should occur in a facility that can provide invasive monitoring and intensive care. The patient with mild pregnancy induced hypertension can often be managed with standard anesthetic practices. There are several considerations the anesthesia provider should be aware of.

- Control of hypertension. Caution must be used to ensure that maternal blood pressure is not decreased too rapidly/drastically or blood flow to the fetus will be affected.
- Decreased intravascular volume. Pre-loading with crystalloid should occur prior to regional anesthesia. This requires a careful balance between replacing the patients decreased intravascular volume prior to a regional anesthetic and avoiding too much IV fluid, leading to pulmonary edema.
- Laboratory values such as platelets and coagulation status should be checked prior to the initiation of neuraxial anesthesia.
    - Platelet count is an indicator of the severity of preeclampsia. Patients with a rapidly decreasing platelet count or a platelet count of less than 100,000 are not candidates for neuraxial blockade.
    - Electrolytes and liver function tests should be checked if there is known/suspected renal or liver involvement.
- An epidural is the anesthetic of choice, due to a slow onset of sympathetic blockade.
- Spinal anesthesia can be used, but vigilant care is required since the patient with a contracted intravascular volume is at increased risk for the development of hypotension.
- General anesthesia is the anesthetic of choice for patients with bleeding disorders, fetal distress, and uncorrected hypovolemia.
- Hypotension should be treated with small doses of ephedrine. Patients with pregnancy induced hypertension are more sensitive to the effects of ephedrine and may require smaller initial doses (i.e. 2.5 mg).
- Intubation

  - The anesthesia provider should be concerned about a possible difficult intubation, especially if the patient shows signs and symptoms of facial swelling and sudden voice changes.
  - Variety of smaller sized endotracheal tubes should be available.
  - Preparation for difficult intubation/alternative plans should be formulated prior to induction of general anesthesia.
  - Additional skilled help should be obtained prior to induction of general anesthesia.
- Measures should be taken to avoid increases in blood pressure prior to intubation.
  - Laryngoscopy can be very stimulating with significant increases in blood pressure.
  - Lidocaine 1 mg/kg or beta blockers such as esmolol 25-100 mg IVP, labetolol 10-20 mg IVP (do not use beta blockers in patients with asthma), or hydralazine 2.5-5 mg IVP, may be beneficial.
- **Ketamine should NOT be used in patients with pregnancy induced hypertension due to potential increases in blood pressure**.
- Magnesium is the treatment of choice for eclamptic patients for the prevention of seizures. Patients receiving magnesium should be monitored closely when neuromuscular blocking agents are administered. The combined use of muscle relaxant and magnesium may produce exaggerated or prolonged blockade. A reduced dose of muscle relaxant may be required.
- Extubation can be as stimulating as intubation. Consider the use of lidocaine or beta blockers to blunt the patient's blood pressure in response to this stimulus.
- Symptoms of pregnancy induced hypertension usually improve by 24-48 hours post delivery. However in severe cases, intensive monitoring may be required for several days. Anti-hypertensive treatment may be required for weeks.

Amniotic Fluid Embolism (AFE)

Amniotic fluid embolism is a rare event but has a high maternal mortality rate. There are no warning signs. This complication can occur at any time during labor and delivery and cesarean section. A break in the uteroplacental membrane allows the entry of amniotic fluid. The patient will experience an anaphylactic syndrome. Classically, the patient develops the following sequential signs and symptoms:

1. respiratory distress
2. cyanosis
3. cardiovascular collapse
4. hemorrhage
5. coma

Additional symptoms include: tachypnea, coughing, hypoxia, and cardiovascular collapse. The patient may experience acute pulmonary embolism, coagulation abnormalities, and uterine relaxation. The baby will experience fetal distress. The patient will require cardiopulmonary resuscitation, aggressive fluid and blood administration, and treatment of bleeding. Mortality is very

high (50-60%) even in the best of circumstances. Seventy-five percent of those who survive have neurological deficits.

Obstetrical Hemorrhage

Maternal hemorrhage is one of the most common causes of obstetric mortality and morbidity. Maternal hemorrhage can occur before delivery (antepartum), during delivery (peripartum), and after delivery (postpartum). Common causes of antepartum hemorrhage include abruptio placentae, placenta previa, and uterine rupture. Common causes of post partum hemorrhage include lacerations, retained placenta or placental fragments, and lack of appropriate uterine contraction, which may be attributed to a variety of conditions. General anesthetic considerations for hemorrhage include:

- General anesthesia is preferred. Vasodilatation associated with neuraxial block, combined with maternal hemorrhage, may create a situation in which it is difficult to control the patient's blood pressure.
- A minimum of 2 large bore IV's should be started (18 gauge or larger). If available a central line and arterial line should be considered.
- Type and cross match blood (if available)
- Laboratory values should include complete blood count, PT, PTT (if available).
- Emergency medications should be immediately available.
- Signs and symptoms of significant blood loss may not be apparent until the patient has lost 25-35% of her blood volume. The parturient has a normal increase in blood volume of approximately 40%.
- Extra help should be summoned at the first indication of hemorrhage. With massive blood loss, large amounts fluids (crystalloid and colloids) and blood products (if available) will need to be administered.

Antepartum Hemorrhage

Abruptio Placentae

Abruptio placentae or placental abruption is one of the most common causes of fetal death. Abruptio placentae is defined as premature separation of the placenta from the uterine wall. Abruption may occur suddenly, without warning. The patient will complain of painful, sustained uterine contractions. In 20% of the cases the bleeding is hidden within the uterus. Diagnosis is clinical, based on typical presentation and ruling out other causes of antepartum hemorrhage. Ultrasound only shows evidence in about half of the cases of confirmed abruption, but is important in ruling out placenta previa, another common cause of antepartum bleeding. Bleeding, with abruptio placentae, is not always apparent or visible if the placenta is implanted high in the uterus. The placenta, implanted low in the uterus, is associated with obvious bleeding that is dark and non-

clotting. In cases in which placenta previa has not been seen on ultrasound and other local vaginal pathology (i.e. tumors, infections, labor, etc.) have been excluded on physical exam, abruption is the most likely cause of antenatal bleeding.

The cause is unknown, although significant risk factors include:

- Maternal trauma
- Pre-eclampsia/eclampsia
- History of previous abruptio placentae
- Hypertension
- Advanced age
- Multiple births
- Drug or alcohol abuse
- Smoking
- African ethnic origin
- Premature rupture of membranes
- Short umbilical cord
- Abnormalities of the uterus

Complications include:

- Fetal demise
- Fetal distress
- Hemorrhagic shock (the uterus can hold up to 1,500 ml of blood)
- Coagulation abnormalities (disseminated intravascular coagulation or DIC is a condition in which the blood will not clot properly)
- Premature labor

Treatment varies depending on gestational age, severity of bleeding, and fetal/maternal condition. Whether opting for observation, attempted tocolysis, induction or cesarean section, these women must be closely observed as their status can change quickly. In cases of severe maternal hemorrhage or severe fetal distress with a baby near term, a general anesthetic is required for immediate cesarean section. Call for extra help. The patient is at risk for hemorrhage and potential development of DIC. If a coagulation abnormality develops, administer fresh frozen plasma 20 ml/kg (if available). Aprotinin, if available, may be helpful.

Placenta Previa

Placenta previa is a condition in which the placenta is abnormally implanted in the lower portion of the uterus, overlying the cervical os. This places the parturient at risk for hemorrhage. Risk factors for placenta previa include the following:

- Previous cesarean section and/or uterine surgery

- Advanced age

The patient with a placenta previa usually presents with painless, bright red vaginal bleeding in the 2nd or 3rd trimester. Diagnosis is usually confirmed by ultrasound with identification the placenta overlying or encroaching the cervical os. Most cases of placenta previa will require cesarean section, since the delivery of the placenta cannot precede delivery of the fetus. The patient who is stable, not currently bleeding, and well hydrated may have a regional anesthetic. General anesthesia is indicated for the patient who is actively bleeding or unstable. The patient is at risk for bleeding after delivery. The lower segment of the uterus lacks the ability of the upper segment to control hemorrhage by contracting.

The patient with a placenta previa and prior uterine surgery is at risk for placenta accrete, a condition in which the placenta attaches itself and grows into the uterus. The attached and implanted placenta cannot be removed; therefore a cesarean section hysterectomy will be required. Massive blood loss and the potential for the development of a coagulopathy should be anticipated. It is vital that extra help is obtained. Normal blood loss for cesarean section hysterectomy averages 3000 ml.

Uterine Rupture

Risk of uterine rupture increases with previous uterine surgery, uterine manipulation such as internal version, during difficult labors with strong contractions, and a weak uterine wall (e.g. obstructed labor or induced/augmented labor in women with multiple previous pregnancies). Signs of uterine rupture include abdominal pain, hemorrhage, hypotension, fetal distress, and loss of uterine tone (contractions stop). This is a life threatening situation secondary to hemorrhage. Immediate general anesthesia is required for rapid delivery of the fetus and to repair the uterus. A hysterectomy may be required. In addition to hemorrhage, the patient is at risk for the development of a coagulopathy.

Postpartum Hemorrhage

Postpartum hemorrhage occurs in up to 4% of parturient's in Western countries. This condition is defined as a blood loss greater than 500 ml after vaginal delivery or greater than 1000 ml after cesarean section. Risk factors for postpartum hemorrhage include:

- A "tired" uterus secondary to long labor or use of oxytocic medications.
- Infection of the uterus (chorioamnionitis)
- Current multiple pregnancy (i.e. twins)

Postpartum hemorrhage can be due to: (4 T's)

- Tissue (retained placenta)
- Tone (uterine atony whereby the uterus does not contract down)
- Trauma (lacerations and tears)
- Tocolytics (medications that prevent uterine contractions)

Anesthetic considerations include:

- Call for extra help!!!
- General anesthesia is indicated if the patient is actively hemorrhaging and surgical intervention is required.
    - Ketamine in doses of 1 mg/kg for induction may be helpful in situations where the patient is unstable due to hemorrhaging.
    - Ketamine can cause increases in uterine tone and maternal blood pressure.
- Insertion of a 2nd large bore IV (18 gauge or larger).
- Observe for a coagulopathy
    - Remove of few ml's of the patient's blood and place in a tube
    - Observe for signs of clotting
    - Absence of a blood clot after 8-10 minutes may be a simple bedside test that is indicative of a possible coagulopathy.
- Plan for potential hysterectomy if the bleeding cannot be stopped.
- Medications to stimulate uterine contraction should be available to administer once the surgeon requests them. Always check with the surgeon prior to the administration of these agents.
- All volatile anesthetics relax the uterus in a dose dependent manner.
- In cases where uterine relaxation is required (i.e. retained placenta) volatile anesthetics should be helpful. The use of nitroglycerin, in doses of 50-100 mcg IVP, has been shown to provide quick uterine relaxation (30-40 seconds) with a duration of 1 minute. The anesthesia provider should be prepared to treat hypotension.
- Prepare for blood transfusion if available

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Neonatal Resuscitation

Chapter Fifteen

Neonatal Resuscitation

The ideal situation for delivery of a newborn is to have dedicated staff members available for the initial assessment and resuscitation of the newborn. All neonates require at least a brief period of assessment. There may be circumstances in which you are called upon to render assistance. If you are involved in a cesarean section your first priority is with the mother. You may assist with the newborn if the mother is stable and you can safely attend to both patients. Vigilance is always necessary.

APGAR Scores

An APGAR score is used to assess all infants at 1 minute and 5 minutes of life. In depressed infants, APGAR scores should continue to be assessed at five minute intervals until the value reaches 7 or greater. Five areas are assessed and include: heart rate, respiratory effort, muscle tone, reflex irritability, and color. A score of 0, 1, or 2 is given for each of the 5 areas. The cumulative score at 5 minutes has some predictive value for the overall neurological outcome.

Area Assessed	0	1	2
Heart rate (beats per minute)	Absent	< 100	>100
Respiratory effort	Absent	Slow, irregular	Good effort; crying
Muscle tone	Flaccid	Some flexion	Active movement
Reflex irritability	No response	Grimace	Crying
Color	Blue or pale	Body pink; extremities blue	All pink

- 0-2 severe depression
- 3-4 moderate depression
- 5-7 mild depression
- 8-10 normal APGAR score

It is important to remember that you should never wait until the 1 minute APGAR to assess the infant and intervene as necessary. Infants should be assessed at the time of delivery and interventions should be implemented as needed. APGAR scores only reflect the infant's status at one moment in time. Effective resuscitation requires ongoing assessment and immediate intervention.

Neonates at Risk

The identification of "at risk" neonates will prepare you to care for the unstable infant. Equipment, supplies, and trained staff should be available for all deliveries.

- Fetal distress/meconium staining
- Depressed due to medications, especially if opioid medications or magnesium were administered within the last four hours
- Obstetric abnormalities (prolapsed cord, hemorrhage, preeclampsia, diabetes)
- Difficult delivery (forceps, abnormal presentation)
- Premature birth
- Small for gestational age

Essential Equipment for Neonatal Resuscitation

- Warm room
- Warm, dry clothes (dry linens, hats)
- Suction device with appropriate sized catheters (8 and 10 French catheters)
- Bag mask valve device
- Face masks that will fit a newborn or premature infant
- Oxygen source (there a growing body of evidence that bagging a neonate without oxygen is just effective as using oxygen)
- Oxygen tubing
- Laryngoscope with Miller blades size 00, 0, and 1. A size 0 can be used instead of a 00 in the very premature infant by not inserting the blade in too far. (Seldom do neonates need intubation).
- Stethoscope
- Endotracheal tubes size 2.5 (< 1000 grams in weight), 3 (2000 grams in weight), and 3.5 (3000 grams in weight) with stylet. Often a weight is not immediately available. Clinical judgment is important in the selection of the appropriate sized ETT. For a very premature infant start with a 2.5 sized ETT. For a term or near term infant start with a 3.5 sized ETT. For infants between these two parameters a 3.0 ETT should be used.
- Umbilical vein catheter 3.5- 5 French, stopcock, syringes, umbilical tape, normal saline, cleansing solution to prepare the umbilical stump prior to catheter insertion, sterile scalpel or blade, and sterile gloves.
- Seldom do neonates need medications however the following medications are helpful during some resuscitation circumstances.
- Resuscitation medications including epinephrine 0.1 mg/ml (1:10,000), naloxone (1 mg/ml solution), and 10% glucose (which may need to be given following delivery but often is not part of the initial resuscitation).

- Volume expanders should be available and include: normal saline 10 ml/kg over 5-10 minutes; ringers lactate and type O packed red blood cells are acceptable.

Care of the Neonate

The initial steps that follow are the most important steps in the care and resuscitation of the neonate after birth:

1. Initially the neonate will have their mouth suctioned first followed by suctioning of the nose.
2. Dry the baby well. The baby should have all fluids toweled off and be wrapped in a warm blanket. Remove the wet linens.
3. Provide warmth and stimulation. Keep the baby warm. Resuscitate the baby in a warm, draft free area, exposing only enough of the baby for adequate assessment (i.e. face and chest) if a radiant heater is not available. Cover the baby's head.
4. Position the infant in a neutral sniffing position. This may require a neck roll by rolling up dry linen behind the baby's neck.
5. Perform the initial assessment of respirations, heart rate, and color
6. Positive pressure ventilation when indicated

It has been found that 99% of neonates will respond to these measures and not require further resuscitation. Neonates that are not responsive should have resuscitation started within the first 30 seconds after birth.

Infants that are breathing well with heart rates over 100 do not usually require further intervention. If the neonate is breathing with a heart rate over 100 but is cyanotic, blow by oxygen should be administered. Rubbing the back or tapping the soles of the feet should stimulate infants who are gasping or apneic, or those whose heart rate is less than 100 beats per minute. If the infant does not respond to the stimulation with a few seconds, bag mask ventilation should be started. The respiratory rate should be 30-60 breaths per minute with positive pressure at 30-40 cm H2O for the first one to two breaths in normal term infants, then decrease to a pressure of 18-20 cm H2O. Continue positive pressure ventilation until the neonate has adequate spontaneous ventilations with a heart rate > 100 beats per minute. Bagging the infant should result in only a subtle rise and fall of the chest.

Indications for tracheal intubation include:

- If meconium is present and the infant is depressed (i.e. poor/absent respiratory effort, poor muscle tone, and a heart rate < 100) then the first line treatment is immediate suctioning of meconium with an endotracheal tube or catheter under direct visualization. Once suctioned the baby may be bag mask ventilated as needed.
- Continued bradycardia despite bag mask ventilation
- Difficulty in ventilating the neonate with a bag mask device

- During resuscitation with chest compressions
- Access for administering medications

For intubation:

- Position the neonate for optimal viewing
- Insert the endotracheal 2 cm beyond the vocal cords
- Listen for bilateral breath sounds. Absence of breath sounds may indicate an esophageal intubation.
- If breath sounds are heard only on the right side pull back the endotracheal tube slightly until equal breath sounds are auscultated.
- If intubation attempt fails continue masking with 100% O2 and then reattempt
- Each attempt should not take longer than 20-30 seconds or as tolerated by the infant
- Once intubated ventilate the neonate. Be careful of the pressure used to ventilate. Excessive positive pressure ventilation may result in a pneumothorax. Only use enough pressure to make the chest rise and fall slightly.

Chest compressions:

- Should be initiated if there is no heart rate or if after 30 seconds of stimulation/effective bag mask ventilation, the neonate does not have a heart rate > 60 beats per minute.
- There are two techniques for chest compressions. The first is to use the tips of two fingers at the middle to lower portion of the sternum. The alternative is to wrap your hands around the chest and use two overlapping thumbs over the middle portion of the sternum. The compression rate should be at 90 compressions per minute. The depth of compression should be 2 cm or 1/3rd the anterior-posterior diameter of the chest. The 2 second cycle should be the same for both intubated and non-intubated infant. Count/compress 1 and 2 and 3 and breathe, repeat. This results in 90 compressions and 30 breathes per minute.

Medications for Resuscitation

Medications are rarely required for resuscitation. Often stimulation and oxygenation are all that are required. If medications are required the best vascular access is the umbilical vein. The umbilical vein is the largest but thinnest walled vessel. A 3.5-5 French umbilical catheter, flushed with normal saline can be inserted so the tip is just below the level of the skin. Blood should come back into the catheter and be allowed to do so. Never inject air. Medications can be administered through the catheter. Never push the catheter in further. This is to prevent the injection of medications directly into the liver, which may result in liver damage. In the absence of vascular access epinephrine can be administered through the endotracheal tube.

Epinephrine- is used when the neonate has a heart rate < 60 beats per minute after 30 seconds of effective positive pressure ventilation followed by 30 seconds of positive pressure ventilation with

chest compressions. The dose is 0.01-0.03 mg/kg or 0.1-0.3 ml/kg of a 1:10,000 solution IV. If epinephrine is given by the ETT route the dose should be 0.3 ml/kg. Administer the medication into the ETT and continue to bag ventilate the infant. If there is no response epinephrine can be repeated every 3-5 minutes.

Naloxone- is used when the neonate is severely depressed due to maternal exposure to opioids within 4 hours prior to delivery. Recommended concentration is 1 mg/ml solution. The preferred route is IV. With the IM route the onset will be delayed. The recommended dose is 0.1 mg/kg. Use naloxone only when there is no history of maternal opioid abuse. Use of this medication in the infant whose mother abuses opioids can result in withdrawal and acute seizures. Monitor the infant closely. The duration of naloxone is shorter than the duration of most opioids. The infant may require further dosing.

Sodium Bicarbonate- is used only for prolonged resuscitation efforts when severe metabolic acidosis is suspected or documented. The dose of 4.2% sodium bicarbonate (0.5 meq/ml) is 2 meq/kg. This should be administered slowly at a rate of 1 meq/kg/minute. Faster rates of administration may lead to intracranial hemorrhage. The infant must be adequately ventilated at the time of dosing since sodium bicarbonate metabolizes into CO2 and H2O.

Volume expansion may be indicated if the neonate is hypovolemic as noted by capillary refill time of greater than 3 seconds. To assess capillary refill time, press firmly on the trunk for several seconds to blanch the skin. Once pressure is released count the number of seconds it takes for the skin to return to the normal color. If this takes longer than 3 seconds then volume expansion should be considered. Generally this is accomplished by the infusion of lactated ringers or normal saline in a dose of 10 ml/kg. Lactated ringers or whole blood may also be used.

10% Glucose- should only be administered if the neonate is hypoglycemic (<35 mg/dl). If the infant is stable and able to feed, consider early feeding instead of glucose. If the level is < 20 and/or the infant is unable to feed give an IV bolus of 2 ml/kg of 10% dextrose IVP. If a bolus is needed, it should be followed by an infusion of 6-8 mg/kg/min. Recheck the glucose level every 30 minutes until the glucose level is greater than 40.

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Section III

Trauma

Chapter Sixteen
Trauma

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Trauma is difficult even in the best circumstances. Trauma has been the ultimate challenge for anesthesia providers since general anesthesia became available in 1846. Prior to 1846 a "team" held the patient from moving while the surgeon preformed the operation, (e.g. leg amputation in 90 seconds). Anesthesia for trauma gives the surgeon the necessary time to repair damages and not be concerned with blood/fluid replacement, urine output, and monitoring of vital signs. Anesthesia is essential for the successful outcome of the traumatized patient. A chapter on basic approaches to anesthesia for trauma is warranted. Sophisticated equipment is nice to have but not necessary for most trauma cases. What is absolutely necessary is an anesthesia provider who is alert, vigilant during the entire case, knows how and when to intervene with less/no anesthetic, fluids/blood, and what changes in vital signs signify in relation to the type of surgical procedure.

Trauma is the leading cause of death throughout the world. Death from trauma usually occurs in one of three periods: 1) the time of the trauma; 2) the first several hours after the trauma; or 3) complications from the trauma, surgery, or anesthesia (i.e. coagulation disorders, sepsis, lung complications, and/or organ failure).

Type of Trauma Injury

Blunt Trauma usually is the result of a fall or vehicle accident. The sudden acceleration and stop cause vital organs to be damaged. Tearing of the vessel walls, compression, and disruption of an organs' functional capacity lead to a life-threatening situation. Blunt trauma injuries are not easily detected because the healthcare provider cannot "see" the internal damage, and initial vital signs seem reasonable. As time elapses and bleeding continues, the vital signs deteriorate, thus the need for frequent assessment of the patient. Suspicion of hemorrhage and respiratory impairment must always be top priorities.

Sometimes subtle blunt trauma can cause a contusion in an area where one least expects a problem until signs and symptoms of the problem develop. Capillary damage leads to hematoma formation in a closed compartment, (i.e. the cranium or muscle). The hematoma slowly expands, causing a rise in pressure in the closed space. The rise in pressure decreases the flow of blood into the space, and ischemia occurs. Releasing the pressure in the space is the only solution to remedy the ensuing ischemia and tissue death. A fasciotomy for a compartment syndrome in the leg or arm will relieve the pressure. In the brain, the development of a subdural hematoma may require an invasive procedure.

Penetrating trauma is usually identified by the telltale hole left in the skin. Stab wound, bullets, and other items cut through the skin and any organ, tissue, blood vessel, nerve, etc., in an inward path. It is difficult to accurately know what structures or organs may be involved. Blood loss is always expected and can quickly extinguish life. When patients have a single penetrating wound or multiple trauma with no blood pressure or heart rate detected by usual monitors, (i.e. arm blood pressure and finger on the pulse), there is not time for the usual workup. The patient must be taken to the operating room immediately; the wound explored to find and stop the bleeding site, and fluid/blood resuscitation done. Time is of essence. Patients die in radiology from failure of the trauma team to act immediately.

Sometimes a penetrating injury does not have a rapid onset of hypotension and tachycardia because the object of penetration is still present. Occasionally someone is stabbed, hit with an axe, or a flying object imbeds itself in the body. The object cuts a vessel in an inward path but the vessel does not bleed until the object is removed. Take these patients to the operating room with the object in place and remove only when everything is ready for a surgical repair.

Basic Rules of Trauma Evaluation

1. **Full stomach** – All trauma patients are considered to have a full stomach. Assume the patient ate a full dinner just minutes before the trauma.

2. **Cervical spine injury** – Must be ruled out in every case of multiple trauma, especially falls and vehicle accidents.

3. **Decreasing level of consciousness** – Any patient who develops a decreasing level of consciousness during the assessment and watching period is assumed to have an internal head injury until proven otherwise.

4. **Airway patency and respiratory depression**– Must be continually evaluated, preoperatively and postoperatively. Swelling from injury in the lung and airway can be slow and continue for a long time.

5. **Hypovolemia**– Hypotension may not be present because of compensatory mechanisms, but always keep hypovolemia in mind as a possibility in any trauma victim.

6. **Hypothermia**– The trauma patient begins to lose body temperature from the moment of injury. Take all measures possible to maintain body temperature near normal and outcomes in trauma will be improved.

Evaluating the Trauma Patient

In the majority of multiple trauma patients, time is of essence and evaluation must be done quickly (i.e. two to five minutes). The following are basic considerations during an evaluation, but some of them can be eliminated in the near-death patient. Basic monitoring at the site of evaluation should include a means to measure blood pressure, measure heart rate, evaluate respiratory status, and, if possible, a pulse oximeter to measure oxyhemoglobin saturation. It is best when there is a designated trauma team. Sometimes the "team" is an anesthesia provider and a surgeon. When time allows, a plan of attack should be devised, even if it means taking the patient to the operating room immediately to stop the bleeding. In a larger hospital each trauma team member is assigned a specific task in the initial evaluation of the patient, making the evaluation more efficient, effective, and rapid.

Essentials in the Initial Evaluation

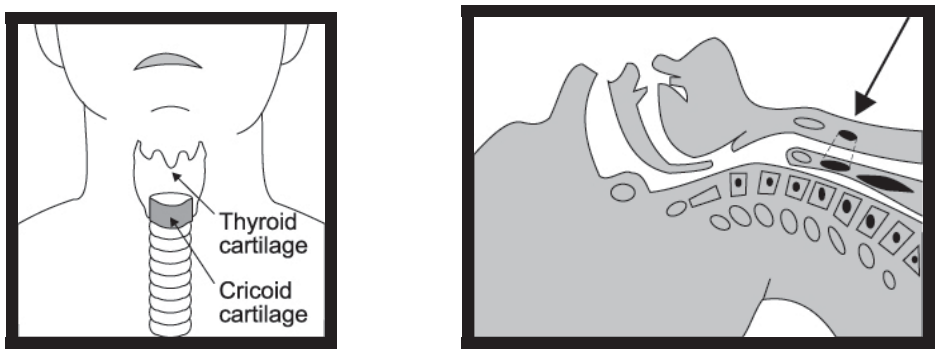
What is written here will take longer to read then it will to perform. Make these basic elements second nature so they become a "checklist" for evaluation of any trauma victim. The ABC's of trauma have proven helpful in remembering what must be looked at and the order of priority.

A = Airway

If the patient does not have a patent airway then establish one. Any evidence in the unconscious patient of respiratory depression must be corrected by intervention. Be sure to clear the airway of secretions, blood, vomitus, and other material. In some cases a nasal or oral airway will suffice but in most cases an endotracheal tube will be required.

<u>Warning</u>: Do not put nasal airways, nasotracheal tubes, or stomach tubes in the nose of patients with facial fractures. These tubes can end up inside the cranial cavity and/or cause meningitis.

When placing the endotracheal tube it may be helpful to use cricoid pressure, but remember the precautions needed to be taken in patients with a possible cervical fracture.



Downward pressure is applied to the cricoid cartilage.

Endotracheal intubation - Be quick, be careful not to over-extend the head, and insert a cuffed endotracheal tube rapidly. Manual inline stabilization of the head and neck should be performed by an assistant to prevent movement in patients with suspected cervical neck injuries. A rapid sequence induction should be considered since all patients should be treated as though they have a full stomach. The method of intubation must be based on the awake status of the patient, stability of vital signs, and the ability to intubate the patient successfully.

Some experts warn against using succinylcholine and ketamine in head injury patients and suggest the substitution of sodium thiopental and a non-depolarizing muscle relaxant. Your assessment and availability of medications must determine the approach to the needed endotracheal intubation. It is a matter of risk versus benefit.

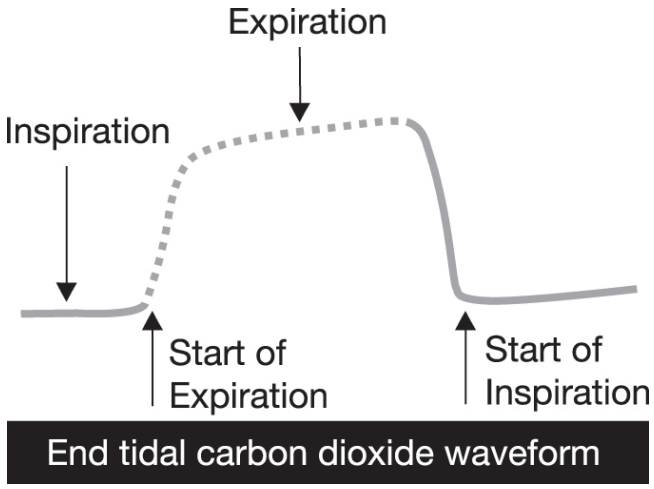
After successful placement of the endotracheal tube, it is essential to determine if the endotracheal tube is in the correct position. Use a combination of observation and measuring instruments (if available). Ask the following questions:

- Observations:
  - Did you see the endotracheal tube pass through the vocal cords?
  - Do both sides of the chest rise when you inflate the lungs?
  - Is the patient becoming cyanotic?

- Measuring Instruments:
  - Can you hear bilateral breath sounds with your stethoscope?
  - When you place the stethoscope over the stomach can you hear air entering during lung inflation?
  - Can you hear breath sounds during inflation of the lungs through your esophageal stethoscope?
  - Does the pulse oximeter show deteriorating oxygenation?

Oxygenation	Oximetry Reading
Normal	95-100%
Mild hypoxia	91-94%
Moderate hypoxia	86-90%
Severe hypoxia	Less than 85%

- Does the end-tidal carbon dioxide analyzer show the presence of carbon dioxide on 4 to 5 successful breaths?



- Does the esophageal detection device easily aspirate air from the trachea or the self inflating bulb fails to reinflate indicating an esophageal intubation? These devices can be created with common materials. A 50-60 ml syringe can be attached to rubber tubing with a right angled endotracheal tube connector. If aspiration returns air, with no resistance, then it is likely that the endotracheal tube is placed in the trachea. If there is resistance to aspiration then the endotracheal tube may be in the esophagus. A second device is similar except it utilizes a self inflating bulb. The same principles apply. The esophageal detection device is not fool proof but can aid the anesthesia provider in identification of endotracheal tube placement.

The establishment of an open, patent airway is essential. If unable to establish an airway by conventional intubation techniques alternative methods such as a combitube or laryngeal mask airway should be utilized. If there are healthcare providers with training in the establishment of an airway through emergency cricothyroidotomy or tracheostomy and an airway cannot be established by other means then these are viable options.

B = Breathing

When the trauma patient arrives at the hospital he/she should be given oxygen (if available) by mask.

Oxygen Delivery Device	Oxygen Flow Rate	Approximate O2 % Delivered
Simple Face Mask	6-10 liters per minute	35-60%
Face Mask with O2 Reservoir	6 liters per minute	60%
Face Mask with O2 Reservoir	7 liters per minute	70%
Face Mask with O2 Reservoir	8 liters per minute	80%
Face Mask with O2 Reservoir	9 liters per minute	90%
Face Mask with O2 Reservoir	10-15 liters per minute	95-100%

Respiration can be evaluated by asking the following questions:

1. **LOOK**
    a. Is the chest rising equally?
    b. Is the patient struggling to breathe?
    c. Is the patient using accessory muscles to breathe?
    d. Does the patient look cyanotic? If so, measure the oxyhemoglobin level with a pulse oximeter, if available.
    e. Does the patient have a flail chest?
    f. Is the trachea midline or deviated to one side?
    g. Are there obvious penetrating wounds to the chest?

2. **LISTEN**
    a. Patient arrives <u>without</u> an endotracheal tube in place:
        i. Are the breath sounds equal and bilateral?
        ii. Are the breath sounds diminished?
    b. Patient arrives <u>with</u> an endotracheal tube in place:
        i. Are the breath sounds equal and bilateral?
        ii. Is carbon dioxide present (if an end-tidal carbon dioxide monitor is available) on exhalation?
        iii. Can you hear air enter the stomach when the patient is ventilated?

3. **FEEL**
    a. Do you feel any crackling of the skin over the chest or throat? Air can get trapped under the skin and cause subcutaneous emphysema.
    b. Does the trachea feel like it is midline or deviated to one side?
    c. Can you feel the chest rise?
    d. Can you feel a broken rib?
    e. Does gentle chest percussion sound normal?

By using LOOK, LISTEN, and FEEL one can discover life-threatening condition requiring immediate treatment.

Simple Pneumothorax:

- Cause: Air accumulating in the pleural space between the chest wall and the lungs.
- Signs and Symptoms:
  - Respiratory distress
  - Hypoxia
  - Diminished breath sounds on the affected side.
  - Hyper resonance with percussion
  - A collapsed lung on chest x-ray (if available)
- Therapy:
  - Cover the wound and seal it on three sides. One side is left unsealed to function as a one-way valve so no additional air will be "sucked" into the chest during inspiration.
  - Insert a chest tube, when possible, into the fourth or fifth intercostal space just anterior to the midaxillary line.

Tension Pneumothorax:

- Cause: Air accumulating in the pleural space in the chest cavity between the lung and the chest wall, and no exit is possible. Each breath increases the pressure in the cavity and deflates the lung.
- Signs and Symptoms:
  - Rapid respiratory rate, hypotension, and tachycardia.
  - Lung collapse on the affected side. Breath sounds are diminished or absent.
  - Tracheal deviation to the opposite side
  - Hyper resonance with percussion on the affected side
  - Distended neck veins.
- Therapy:
  - Insert a 3 to 6 centimeter long 14-gauge intravenous catheter into the second intercostal space at the midcavicular line.
  - Insert a chest tube, when possible, into the fourth or fifth intercostal space just anterior to the midaxillary line.

Hemothorax:

- Cause: Bleeding into the pleural cavity.
- Signs and Symptoms:
  - Decreased breath sounds
  - Dullness with percussion over the affected side
  - Mediastinal shift to the unaffected side
  - Cardiovascular instability

- Therapy:
    - Pleural drainage
    - If initial volume is greater than 1500ml or bleeding continues at a rate greater than 200ml/hr, then an emergency thoracotomy is required to stop the bleeding.

Flail Chest:

- Cause: Two or more rib fractures. Patients will typically have associated pulmonary contusions that affect oxygenation and ventilation.
- Signs and Symptoms:
    - Paradoxical respiration. The section of the chest wall that is affected will move out during inspiration and in during expiration.
    - Hypoxia
    - Respiratory distress
- Therapy
    - Intercostal blocks
    - Thoracic epidural block
    - Oxygen administration
    - Sometimes endotracheal intubation and ventilation

Tracheal/Bronchial Rupture:

- Cause: Break in the integrity of the trachea or bronchus. Air accumulates in the mediastinum and/or around the heart. Identification of airway anatomy such as the cricothyroid membrane may be difficult due to associated trauma.
- Signs and Symptoms:
    - Subcutaneous emphysema in the neck
- Therapy:
    - Immediate intubation of the trachea or bronchus beyond the rupture.
    - Immediate thoracotomy to repair the rupture
    - Prognosis is poor

C = Circulation

Two priorities of circulation to be considered in the trauma patient are to 1) stop the bleeding and 2) replace the lost blood.

Stop the Bleeding

- ✓ Apply pressure to the sites of obvious hemorrhage.
- ✓ Bleeding of the extremities should be treated with direct pressure, pressure dressings, and packing the wound. Tourniquets may be used but must be applied carefully and for short periods of time to avoid injuring the tissue distal to and under the tourniquet.

- ✓ Penetrating objects should be left in place. The surgeon should remove the object in the operating room when appropriate.
- ✓ Chest trauma bleeding is often from intercostal arteries. Re-expansion of the lung can often reduce or even stop this bleeding.
- ✓ Bleeding from abdominal trauma may be decreased or stopped by the accumulation of the blood within the closed space of the abdomen. Do not be deceived. Bleeding will start immediately when the surgeon removes the accumulated blood. Prior to surgical intervention the anesthesia provider should ensure that a minimum of two large bore IVs are in place.

Hemorrhage

Loss of blood results in hemorrhagic shock, a condition that leads to poor or absent tissue perfusion. Protective mechanisms of the body compensate for the loss of blood by increasing sympathetic nervous system output. Heart rate is increased, blood vessels to less-vital organs constrict, and blood is shunted to the heart, brain, kidneys, and liver. A price is paid for shunting blood. Hypoxic changes in poorly perfused tissues cause acidosis and can have a negative affect during resuscitation when acidotic fluid re-enters the circulation. Because of the constriction of blood vessels and conservation of fluid, urine output decreases, a sign the patient is hypovolemic (urine output less than 0.5 ml/kg/hr).

Estimating blood loss in the trauma patient is an educated guess, but there are some basic tenets that have proved useful. First you will need to know or estimate the patients' weight. Next, for an adult, multiply the weight in kilograms by 70 ml/kg. If the patient weighs 70 kg then the pre-trauma blood volume would have been 4900 ml. Use this baseline figure to determine the percentage of blood loss. Most patients do not show signs of hemorrhagic shock until they lose great than 25% of their blood volume. Do not underestimate the blood loss. Blood loss can be deceptive. For instance, a pelvic fracture can hide from 1500ml to 2500ml and a femoral fracture 500ml to 2000ml.

Signs, Symptoms of Hemorrhagic Shock

- Rapid heart rate – Take the pulse and/or use an electrocardiogram/pulse oximeter (if available).
- Decreased blood pressure – Use a manual blood pressure. Feel the peripheral pulse for rate as well as quality. In general the lowest palpable systolic blood pressure for the carotid artery is 60 mmHg; femoral artery is 70 mmHg; and radial artery is 80 mmHg.
- Poor tissue perfusion
    - Check capillary refill by applying pressure to the end of the patient's fingernail until the nail bed whitens. Release the pressure. Rapid pink coloring in the nail bed should return within two seconds.
    - Pull the lower eyelid down slightly. If the area is pale then circulation to the head is compromised.

- Low urine output – Insert a Foley catheter (if available). A low urine output is defined as less than 0.5 ml/kg/hr in adults and less than 1 ml/kg/hr.
- Changes in respiratory rate – Not a dependable sign of hemorrhagic shock. Sometimes will increase to greater than 20 respirations per minute, in the adult, during severe hemorrhagic shock.
- Changes in facial/extremity color – May turn a pale or ashen color, and may be cool to touch. Difficult to access in some races.
- Change in level of consciousness – Patient may present from being alert to being confused, sleepy, or even unconscious.

A few observations that might be helpful in the assessment of blood loss:

- Every trauma victim will compensate in some measure to the loss of blood. Base line health, daily medications, age, gender, and other factors will determine the extent of compensation.
- Diastolic pressure will change before systolic pressure because of the release of epinephrine and nor-epinephrine by the sympathetic nervous system. Initially, the diastolic pressure will be normal, as blood loss continues the diastolic pressure will increase but eventually decrease.
- Elderly patients, especially those with pre-existing cardiac disease, will be more sensitive to blood loss.
- Young, healthy patients usually show the greatest resistance to massive blood loss. Many trauma teams have seen a young, healthy, 18 year old patient who is talking coherently but has no palpable femoral or radial pulses (only a carotid pulse), and an undetectable blood pressure with the manual blood pressure cuff.

Hemorrhage Resuscitation

Intravenous catheters:

1. Size: At least 14 gauge or 16 gauge
2. Sites:
    a. Antecubital vein at the elbow
    b. Femoral vein
    c. External jugular vein
    d. Sometimes a cut down on a peripheral vein (cut the tip off a venous line and insert the venous line directly into the vein).
    e. Central line, i.e. subclavian, if done frequently by the anesthesia provider or surgeon.
3. Cautions:
    a. Do not insert into a femoral vein if the lower extremity has a fracture or an intra-abdominal hemorrhage is expected.
    b. Avoid insertion of an intravenous catheter into an extremity with a fracture.

c. Central lines are excellent but have serious complications if not done by someone with experience. Central lines usually take time to insert and secure.

Fluids:

1. Crystalloid
    a. Normal saline or lactated ringers. Lactated ringers contain a buffer for acidosis which is helpful in the trauma victim. Large volumes over several days may result in alkalosis.
    b. Do not use D5W
    c. Over two-thirds of crystalloid fluids quickly leave the intravascular system.
    d. Initial dose is 20-30ml/kg followed by 3ml crystalloid for every 1ml of estimated blood loss.
2. Colloids
    a. Includes: plasma, albumin, Dextran 70, hetastarch, and haemacoel. There is a small risk of an allergic reaction with the administration of colloids (i.e. albumin, hetastarch).
    b. Stays in the intravascular space for about 4-8 hours
    c. Initial dose is 10-20 ml/kg followed by 1ml colloid for every 1ml of estimated blood loss.
    d. Maximum dose of Dextran 70 is 1500ml/24 hours
    e. Hetastarch maximum total dose is 1000ml.

Blood:

1. If blood is available, send a blood sample for type and cross of at least 8 units of packed red blood cells and 4 units of fresh frozen plasma.
2. In extreme emergencies give the patient O negative or O positive blood. Rule of thumb: if more than 6 units of O blood are administered, then continue with O blood for future blood transfusions over the next six weeks. Do not return to the patients' original blood type for transfusions until at least six weeks after the last O blood transfusion. This procedure will decrease the possibility of blood incompatibility reactions.
3. Practical considerations for massive transfusions:
    a. Definition:
        i. Transfusion of greater than 10 units of blood
        ii. Transfusion of greater than 150% of the patient's estimated blood volume (15 units [500ml each] in a 70kg patient).
    b. Realistic issues:
        i. On the internal medicine ward, the patient is given one unit of blood over a 4 to 8 hour period and is constantly monitored. You may be transfusing up to 30 units of blood per hour, and you cannot tell which unit caused the blood transfusion reaction if one occurs during the surgery.
        ii. When hives are seen during a rapid transfusion, it is best to give diphenhydramine (25-50 mg IVP) and continue the transfusion.

iii. Monitor the urine output. If it turns pink or red then suspect a hemolytic blood reaction.
iv. The anesthetic itself may mask many of the symptoms of blood transfusion reactions.

c. How do you transfuse blood rapidly?
   i. Insert at least two large bore catheters, e.g. 14ga or 16ga.
   ii. Use pressure infusion bags (if available) over the blood units (or have someone squeeze them). Make sure that there is no air in the infusion bag or line. If air is infused into the patient, a massive air embolism with subsequent mortality will occur. You must monitor the infusion closely when using pressure bags to infuse blood or other fluids.
   iii. Use the leg tourniquet or Dual Cuff Auxiliary Block tourniquet, if available, to pressurize the infusion bags.
   iv. Use commercial blood infusion machines when available.

4. Complications:
   a. Hives – Treat with diphenhydramine and continue the transfusion.
   b. Hemolysis – A major blood reaction which can cause death. This usually shows up in the recovery room. Treatment consists of the following steps:
      i. Stop the infusion immediately.
      ii. 100% oxygen by mask.
      iii. Treat hypotension and/or bronchospasm with 0.5ml to 1ml of 1:10.000 epinephrine. Titrate boluses of epinephrine to effect.
      iv. Consider cortisteroids and bronchodilators.
      v. Administer a diuretic (i.e. furosemide) and monitor urine output closely.
      vi. Treat hypotension with a 20-30ml/kg fluid bolus and consider vasopressors.
   c. Acidosis – Usually the result of inadequate volume replacement.
   d. Citrate Toxicity – Secondary to the rapid transfusion of large volumes of blood.
   e. Hypocalcemia – Signs and symptoms include hypotension, bradycardia, and arrhythmias. If you suspect hypocalcemia, you can give 500mg to1000mg calcium chloride.
   f. Hypothermia – Warm patient with all means available (i.e. warm the OR, warm blankets, warm fluids).
   g. Coagulation disorders:
      i. Usually starts about the 5-6th unit of blood.
      ii. Factors for coagulation are depleted or diluted and not present in bank blood.
      iii. Treatment:
         1. Best therapy is fresh, warm blood.
         2. Use fresh frozen plasma (if available) at a dose of 15mg/kg (if you can measure the prothrombin time).

3. Use factor VII/fibrinogen (if available) concentrate (if you can measure the partial thrombin time).
4. Depletion of platelets – Administer platelet concentrates, if available, if the patient demonstrates signs of bleeding and if you can measure a platelet count (less than 50,000). Usually one platelet concentrate will increase the total platelet count by 5,000 to 10,000. Usual dose of platelet concentrate is 1 per 10kg.

iv. Disseminated intravascular coagulation (DIC) occurs in massive blood transfusions when the normal clotting factors are consumed. Treatment of this condition is difficult and requires the support of a well-stocked blood bank. Recommendations for treatment include the following:
   1. Fresh frozen plasma, 1 pack per 15kg.
   2. Fresh blood, if available.
   3. Crycoprecipate, if fibrinogen is low, partial prothrombin time is prolonged, or thrombin time is prolonged.
   4. Platelets if the platelet count is less than 50,000. One platelet concentrate per 10kg.

5. Warming blood and fluids
   a. Use a commercial warmer when available. They are safer and have fewer complications.
   b. During massive transfusions when no commercial warmer is available you can put blood units and fluid bags in a pan of tepid water. **WARNING**: if you use this technique you must be very careful to test each pan of water with your elbow or hand to make sure the water is not too hot. Hot water (42 degrees C or greater) will hemolyze the red blood cells.

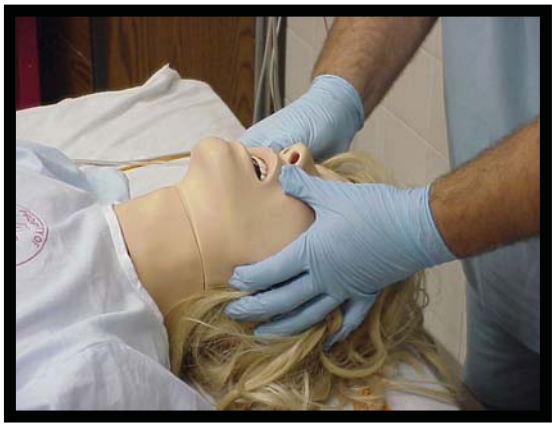
Remember, rapid blood and fluid transfusions are the key to successful resuscitation. Patients with massive trauma do not survive if blood is not available. Colloids and crystalloids can buy time for these patients, but eventually blood transfusion will be necessary. Perhaps fluid transfusion will allow you time to transport the patient to a hospital with a blood bank, or have the blood transported to you.

Indicators of adequate fluid resuscitation include a urine output of 0.5 ml/kg/hour for adults and 1 ml/kg/hr in children, maintenance of a mean arterial blood pressure of > 60-70 mmHg, a decrease in heart rate, and normal pulse pressure.

D = Disability or Disorders of the Central Nervous System

Cervical Spine Injuries occur in about 2% of trauma victims, but this percentage increases to over 10% if there is an associated severe head injury. Patients who are alert and not complaining of neck pain are unlikely to have a cervical spine injury.

The signs and symptoms of cervical spine injury include neck pain, an array of neurological signs and symptoms, and loss of consciousness. The intubation technique for patients with a cervical spine injury must emphasize keeping the head maintained in the neutral position during laryngoscopy. Maintain inline stabilization of the head and neck. Do not tilt the head back when intubating the patient. The preferred maneuver to open the mouth is the jaw thrust.



Jaw Thrust – The rescuer grasps the angles of the patient's lower jaw and lifts with both hands. The jaw thrust can be done with the head tilt, as pictured above, or it can be done alone without the head tilt. The jaw thrust without head tilt is the technique of choice for a patient with a suspected neck injury since it causes the least movement of the cervical spine. (Courtesy: Department of Nurse Anesthesia, Virginia Commonwealth University. Richmond, VA)

Immobilize the head, but do not place in traction. An assistant can hold the head in the neutral position. Once the endotracheal intubation is accomplished, a cervical collar (if available), sand bags, or intravenous bags can be used to immobilize the head during surgery. Assessment of the central nervous system: The awake, verbal response, painful response, unresponsive (AVPU) system has proven useful in rapidly and accurately assessing the CNS. Ask yourself the following questions while doing the AVPU assessment:

- A – Is the patient awake?
- V – Does the patient verbally respond to your voice?
- P – Does the patient respond to painful stimuli? (sternal rub or ear pinch)
- U – Does the patient have total lack of response?

Do not forget to evaluate the four extremities for a spinal cord injury.

E = Exposure

Take off the patient's clothes when evaluating real and potential injuries. Keep in mind that exposure can lead to rapid heat loss. Cover the patient immediately after completing the initial assessment.

Secondary Examination

Once the patient has been resuscitated and is stable, either before or after surgery a more thorough examination will be necessary. These basic suggestions have proven helpful in this phase of the trauma assessment:

- Short history:
  - The patient is the best source of information, but family and a witness of the trauma may prove vital.
  - The use of the acronym AMPLE may be helpful in the evaluation:
    - A = allergies.
    - M = medications (especially tetanus shot).
    - P = previous medical history.
    - L = last time of oral intake.
    - E = events leading up to the injury.
- Head and neck evaluation:
  - Record a baseline Glasgow Coma Scale score.

Test	Response & Score	
Eye opening	Spontaneous	4
	To request	3
	To pain	2
	None	1
Verbal Response	Oriented	5
	Confused	4
	Inappropriate	3
	Sounds	2
	None	1
Motor Response	Follows verbal request	6
	Localizes pain	5
	Flexion & withdrawal	4
	Abnormal flexion	3
	Extension	2
	None	1

A score of 3-15 is possible. A score of 3-8 generally indicates that the patient is in a coma.

  - Basal skull fracture
    - Blood exiting the nose or ears. Bruising around the orbits of the eyes.
    - Periorbital or subconjunctival bleeding.
  - Evaluate the neck for injuries
- Thorax: Fractures, subcutaneous emphysema, tracheobronchial trauma, aortic dissection, rupture of the diaphragm.
- Extrusion of the bowels: Cover with warm saline soaked pads until surgery.
- Genital exam:
  - Men: Blood at the meatus or scrotal swelling may indicate urethral injury.
  - Women: May discover a pelvic fracture or breach of the vagina during a vaginal examination.

- Extremities: Note obvious deformities, Check peripheral pulses. Assess sensation.
- Spine:
    - Urinary retention, flaccidly of the extremities, poor anal sphincter tone, priapism, diaphragmatic breathing, persistent bradycardia, and/or hypotension.

Basic Equipment in the Trauma Receiving Area

1. Oral and nasal airways
2. Laryngoscope
3. Endotracheal tubes
4. Emergency airway equipment (if possible). This may include LMA, combitube, bag mask valve device, or cricothyrotomy).
5. Usual anesthesia medications
6. Fluids for intravenous administration
7. Large bore intravenous catheters (14ga and 16ga)
8. If available:
    a. Pulse oximeter
    b. Arterial and CVP monitor
    c. Pressure bags for blood transfusion
    d. Warm blankets

Anesthesia for Trauma

Pre-check

- If an endotracheal tube is in place, confirm placement is correct.
- Talk to the patient, awake or unconsciousness?
- Consider rapid sequence induction.
- Be prepared for an emergency tracheotomy.
- Stabilize the neck if a cervical spine injury is suspected.
- Check the intravenous catheters to make sure they are functional.

Induction

- Apply basic monitors:
    1. Manual blood pressure cuff
    2. Chest and/or esophageal/precordial stethoscope
- Rapid-Sequence Induction of Anesthesia
    1. The patient is given four or five large breaths of 100% oxygen by anesthesia mask.

2. Firm pressure is applied by an assistant with thumb and finger over the patient's cricoid cartilage in the anterior neck, compressing the esophagus between the vertebral column and the cricoid ring to minimize the risk of passive regurgitation of gastric contents into the posterior pharynx. Precautions need to be taken if the patient has a possible cervical neck fracture.
3. The chosen anesthetic agent and muscle relaxant are administered.
    - Suggested doses for regimens for rapid-sequence induction:
        - Ketamine (1-2 mg/kg) immediately followed by succinylcholine (1-2 mg/kg).
        - Etomidate (0.2 – 0.4 mg/kg) immediately followed by succinylcholine (1-2 mg/kg).
        - Sodium thiopental (1-4 mg/kg) immediately followed by succinylcholine (1-2 mg/kg).
        - A non-depolarizing muscle relaxant should be used if the patient has a contraindication to succinylcholine. Since non-depolarizing muscle relaxants are slower during onset, the patient will require gentle ventilation while maintaining cricoid pressure.
        - Scopolamine in a dose of 0.4mg may be considered for amnesia.
4. When the patient is unresponsive and has lost lid reflex, the larynx is visualized and the endotracheal tube is passed into the larynx. The cuff is inflated.
5. The presence of bilateral equal breath sounds is confirmed. Only after this fifth step is completed should the assistant release cricoid pressure.

- Cascade anesthesia technique - Individualize the induction based on the vital signs of the patient.

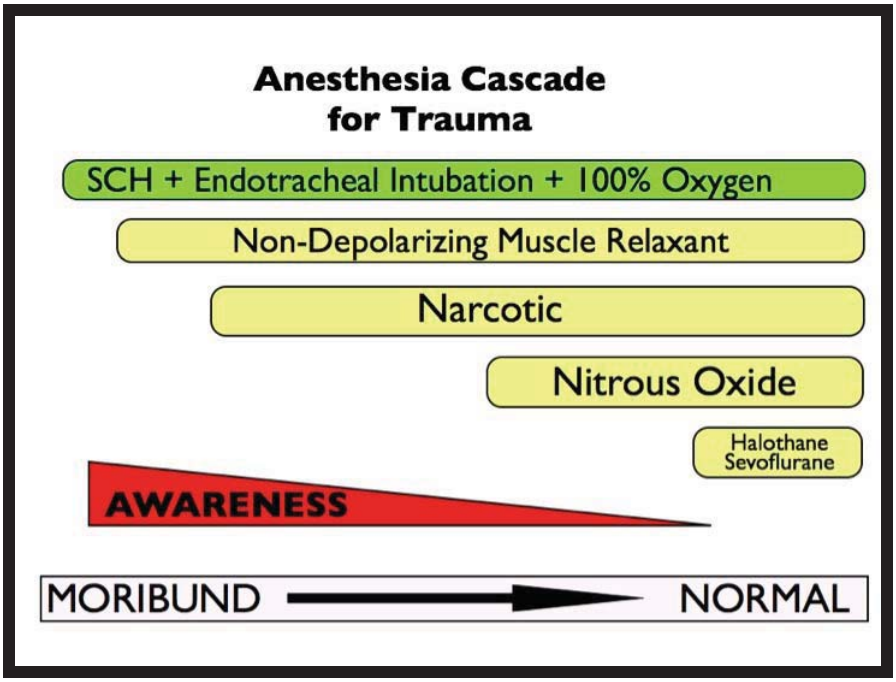


Diagram courtesy of Dr. Petty.

If the patient is moribund (that is, non-responsive to anything), the blood pressure cannot be detected by conventional means, and there is a high heart rate, then the patient is given oxygen, a fast acting muscle relaxant (succinylcholine), rapidly intubated, and the surgery started. Once the bleeding is stopped, the blood pressure usually comes up, the heart rate down, and the patient is on the way to recovery. Then add a narcotic for pain relief, eventually nitrous oxide (reversed easily), and finally a very low amount of halothane or sevoflurane. Each drug added during the resuscitation is given in low doses at first to make sure the patient can withstand the change in physiology induced by the drug. Then gradually increase the dose, and add the next drug on the cascade.

1. If the patient is unconscious and has no detectable blood pressure and a rapid thready pulse, administer 100% oxygen, give succinylcholine, intubate, give a non-depolarizing muscle relaxant, and ventilate with 100% oxygen.
2. If the patient is conscious but has no detectable blood pressure and a rapid thready pulse, while you administer 100% oxygen tell the patient he/she may have awareness during part of the anesthetic, and you will give them pain medications as soon as you can restore their vital signs. Then you can give ketamine (2mg/kg), followed by succinylcholine, intubation, a non-depolarizing muscle relaxant, and ventilate with 100% oxygen. Ketamine (1mg/kg) may be repeated every 30 minutes.
3. Combating awareness during general anesthesia - In many cases awareness must be accepted as a necessary element of the anesthetic technique. Take time to let the patient know they may be awake long enough for you to stabilize their vital signs. Then you will give them pain medications and anesthesia. Talk to the patient during the induction and throughout the case. Tell them they are getting fluid and blood to stabilize their vital signs. Let them know when you are giving them drugs for "sleep" and "pain." Be sure and ask them after the surgery what they remembered, and assure them it was absolutely necessary to do what you did to save their lives. They will almost universally thank you for your efforts.

Maintenance of Anesthesia

- Once the bleeding is stabilized and you have caught up with the blood loss, you can start adding anesthetic drugs, i.e. fentanyl for pain, midazolam or scopolamine (if not already administered) for blocking recent memory.
- As physiological conditions continue to stabilize, you can try adding a little nitrous oxide (since it can easily be removed) and a very small portion of halothane or sevoflurane.
- Each time you add a new drug, be sure to monitor the patients' vital signs carefully before you add another drug.

- The entire anesthetic must be carefully formulated to make sure the patient is safe and is given the best chance for survival.
- To stress again: If general anesthesia cannot be induced from the beginning of anesthesia, it is vital to stay in verbal contact with the patient, even when you have no idea if they can or cannot comprehend what is being said. Tell them they were paralyzed while awake in order to do surgery to save their life. Assure them that as soon as possible you will give them drugs to stop the terrible pain and fright they are suffering. Some of your patients will have recall. Multiple postoperative visits are necessary to assure the patient you did what you had to do to save their life.
- Start with small doses of any anesthetic drug. The drug may "relax" the sympathetic tone, cause cardiac depression, or interfere with some other mechanism the patient is using to counteract the loss of blood volume. Large initial doses of any anesthetic drug may cause abrupt, devastating, changes in vital signs.
- Ketamine should be avoided, when possible, in patients with head injuries
- Caution should be used when giving succinylcholine to trauma patients after initial resuscitation. This is due to a potentially life threatening release of potassium.
- Non-depolarizing muscle relaxants are invaluable to the surgeon and for respiratory control.
- Hyperventilate patients with head injuries to reduce intracranial pressure.
- Many anesthesia providers avoid nitrous oxide in trauma patients because they do not have it available or they do not feel there is a need for nitrous oxide in anesthesia. When using the "cascade" method of anesthesia described, nitrous oxide provides some additional pain relief, decreased awareness, and can be removed quickly.
- Monitor the urine:
    - Output – Try to maintain a rate greater than 1ml/kg/hr. Give mannitol to increase output when necessary.
    - Color – Tea color means myoglobin is present from damaged muscle. Red color usually means damage to the kidneys or bladder. May consider the administration of mannitol.
- Avoid any insertions in the nose in patients with basilar skull fractures, i.e. nasogastric tube, nasal endotracheal tube, nasal airway, temperature probes.
- Communicate with the surgeon(s) throughout the case, and keep an eye on the surgical field so you know what is going on.
- Check frequently for tension pneumothorax in patients with chest trauma. Therapy – insert a 14ga needle in the second intercostal space at the midcavicular line until a chest tube can be placed.
- Pericardial tamponade can occur rapidly or insidiously. The blood around the heart must be removed by pericardiocentesis or via a thoracotomy
- Fat embolism is difficult to detect during anesthesia for trauma. If the patient has a chest injury and a fractured femur, it is hard to differentiate how much each one contributes to the respiratory failure. If you have an isolated fracture, i.e. femur, without lung damage,

then a continual downward spiral in respiratory function can be assumed to be fat emboli. Supportive care is given as there is no definitive treatment for fat embolism.

Emergence and Postoperative Care

1. Many patients with multiple traumas will require ventilation after surgery. Continue narcotics, muscle relaxants, and 100% oxygen.
2. If you plan to extubate the patient in the operating room or the recovery room, give the airway the same intense care you did for the intubation. Do not extubate until the patient can protect their own airway.
3. When reasonably possible, an anesthesia provider should be available for consultation and sometimes, give direct postoperative care.
4.

Summary

Anesthesia for trauma is not easy. Most of the time the anesthesia provider is resuscitating the patient with fluids and blood. Knowledge of the basic problems, techniques, and possible complications will prepare you for the trauma victim. Many hospitals may not have the best equipment, a blood bank, or drugs. Our primary responsibility is to provide resuscitation and anesthesia, but we can assist in all aspects of patient care when possible. Doing the best you can do with the equipment and drugs available will result in the best outcomes for trauma victims in your hospital.

An excellent resource for basic trauma care is available at http://www.primarytraumacare.org . A thirty-nine page manual is available in English, Chinese, Spanish, French, Indonesian, Mongolian, Farsi, and Vietnamese.

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Appendices to Basic Guide to Anesthesia for Developing Countries Volume 2

Appendix A : Local Anesthetics

Amides	Esters
Bupivacaine	Chloroprocaine
Levobupivacaine	Cocaine
Etidocaine	Procaine
Lidocaine	Tetracaine
Mepivacaine	
Prilocaine	
Ropivacaine	

Local Anesthetic	pKa
AMIDES	
Bupivacaine and Levo-Bupivacaine	8.1
Ropivacaine	8.1
Lidocaine	7.8
Prilocaine	7.8
Etidocaine	7.7
Mepivacaine	7.6
ESTERS	
Chloroprocaine	9.0
Procaine	8.9
Cocaine	8.7
Tetracaine	8.2

Local Anesthetic	Potency and Lipid Solubility/Duration of Action
AMIDES	
Bupivacaine/Levo-Bupivacaine	4/4
Etidocaine	4/4
Ropivacaine	4/4
Mepivacaine	2/2
Lidocaine	2/2
Prilocaine	2/2
ESTERS	
Tetracaine	4/3
Cocaine	2/2
Procaine	1/1
Chloroprocaine	1/1

1= Least; 4= Most

Site of Injection

The site of injection greatly impacts the blood levels of local anesthetic. Areas of high vascularity will result in greater uptake and higher blood concentrations. The uptake of local anesthetic, from greatest to least, is as follows:

IV> tracheal> intercostal> caudal> paracervical> epidural> brachial> sciatic> subcutaneous

Duration of Action

Duration of action is dependant on individual local anesthetic characteristics. Local anesthetics are classified as follows:

- Short acting: procaine and chloroprocaine
- Moderate acting: lidocaine, mepivacaine, prilocaine
- Long acting: tetracaine, bupivacaine, etidocaine, ropivacaine, levobupivacaine

Medication Interactions with Local Anesthetics

Ester Local Anesthetics	Succinylcholine- may potentiate the effects since both are dependant on pseudocholinesterase for metabolism.
Ester Local Anesthetics	Cholinesterase inhibitors such as neostigmine and pyridostigmine can lead to a decrease in the metabolism of ester local anesthetics.
Local Anesthetics in General	Opioids and alpha adrenergic agonists potentiate the analgesic effects of local anesthetics.
Local Anesthetics in General	Potentiate the effects of non-depolarizing muscle relaxant blockade.
Chloroprocaine (epidural)	May interfere with the analgesic effects of subarachnoid opioids.
Lidocaine	Cimetidine and propranolol decrease hepatic blood flow and lidocaine clearance. This acts to increase the risk of systemic toxicity.

Plain local anesthetics (max doses based on 70 kg man)

Local Anesthetic	Type	Concentration %	Max dose	Max dose mg/kg	Duration
Lidocaine	Amide	0.5-1.0	300	4.5	30-60 minutes moderate duration
Mepivacaine	Amide	0.5-1.0	300	4.5	45-90 minutes moderate duration
Bupivacaine	Amide	0.25-0.5	175	2.5	120-240 minutes long duration
Ropivacaine	Amide	0.1-1	200	3	120-360 minutes long duration

Local anesthetics with epinephrine (1:200,000) for infiltration:

Local Anesthetic	Type	Concentration %	Max dose	Max dose mg/kg	Duration
Lidocaine	Amide	0.5-1.0	500	7	120-360 minutes moderate duration
Mepivacaine	Amide	0.5-1.0	500	7	120-360 minutes moderate duration
Bupivacaine	Amide	0.25-0.5	225	3	180-420 minutes long duration

Summary of Common Local Anesthetics

Local Anesthetic	Type	Onset of Action	Duration	Clinical Use
Procaine	Ester	Slow	Short	Spinal
Bupivacaine	Amide	Moderate	Long	Peripheral Nerve Blocks Infiltration Spinal Epidural
Ropivacaine	Amide	Moderate	Long	Peripheral Nerve Blocks Epidural
Chloroprocaine	Ester	Fast	Short	Peripheral Nerve Blocks Epidural
Etidocaine	Amide	Fast	Long	Peripheral Nerve Blocks Infiltration Epidural
Lidocaine	Amide	Fast	Moderate	Peripheral Nerve Blocks Infiltration Spinal Epidural Bier Block
Mepivacaine	Amide	Fast	Moderate	Peripheral Nerve Blocks Infiltration
Prilocaine	Amide	Fast	Moderate	Peripheral Nerve Blocks Infiltration Bier Block

Adding Epinephrine to Local Anesthetic Solutions

In general, a concentration of 1:200,000 (5 mcg/ml) is used for peripheral nerve blocks to reduce vascular absorption. When adding epinephrine to a plain solution use a 1mg/ml ampule of epinephrine. Take the total volume of local anesthetic, divide it in half, and move the decimal point two places to the left. For example, to add epinephrine to 40 ml of 1% lidocaine simply divide 40 by 2 = 20. Next move the decimal point two places to the left which results in 0.20. This is the amount of epinephrine added to the local anesthetic solution to yield a 1:200,000 concentration. To check this multiply 5 mcg/ml by 40 ml = 200 mcg. It is important to always check the concentration of epinephrine and double check the total dose added to the local anesthetic.

A second technique for adding epinephrine to local anesthetic preparations is detailed below:

- 1:200,000 epinephrine concentration would be 5 mcg/ml.
- Dilute epinephrine using a 10 ml syringe. Draw up 1 ml of 1:1000 epinephrine (1 mg per ml) and 9 ml of normal saline.
- Mix it by tilting the syringe back and forth.
- The concentration of epinephrine is now 100 mcg per ml.
- Add epinephrine to the local anesthetic solution (see table below).

1:200,000 Epinephrine Concentration	
Volume of Local Anesthetic	**Amount of Epinephrine Added to Local Anesthetic Solution**
20 ml	100 mcg of epinephrine
30 ml	150 mcg of epinephrine
40 ml	200 mcg of epinephrine
50 ml	250 mcg of epinephrine

- Always label the syringe of epinephrine. Once the epinephrine is added to the local anesthetic discard what remains. Epinephrine can be lethal if inadvertent administration occurs.

Appendix B: Neuraxial Blockade Indications and Contraindications

General considerations:

- Suitability for the type of surgery being performed
- Surgeon's preferences
- Experience in performing neuraxial blockade
- Physiological state of the patient
- Patient mentally prepared to accept neuraxial blockade and the loss of motor function
- No contraindications to neuraxial blockade

General risks:

- Toxicity of local anesthetics (with epidural techniques)
- Transient or chronic paresthesia
- Nerve damage
- Intra-arterial injection and seizures or cardiac arrest (with epidural techniques)
- Block failure and the need to supplement or convert to general anesthesia

Contraindications for Neuraxial Blockade

Absolute Contraindications:

- Patient refusal
- Infection at the site of injection
- Coagulopathy (acquired, induced, genetic)
- Severe hypovolemia. Hypovolemia should be corrected prior to spinal anesthesia. A spinal anesthetic in a severely hypovolemic patient may lead to cardiac arrest.
- Increased intra-cranial pressure (i.e. brain tumor or recent head injury with associated increases in intra-cranial pressure)
- Severe aortic stenosis
- Severe mitral stenosis
- Ischemic hypertrophic sub aortic stenosis
- Severe uncorrected anemia
- Inability to guarantee sterility of medication or equipment
- An allergy to local anesthetics. Ensure that it is a "true" allergy. Some patients may report symptoms such as dizziness, nausea, etc during dental anesthesia. Ask the patient if they had trouble breathing, a rash, and other symptoms that would indicate a "true" allergy. If the patient had a true allergic reaction to a local anesthetic, identify which local anesthetic. Ester local anesthetics have a higher incidence of allergic reactions, related to their metabolism to PABA. Amide local anesthetics have a very low incidence of allergic reactions. There are no

cross reactions between amides and esters. A true allergy is an absolute contraindication to neuraxial blockade with the offending local anesthetic or others in the same class.

Relative Contraindications:

- Sepsis (may spread infection to subarachnoid/epidural space)
- Uncooperative patient (dementia, psychosis, emotional instability)
- Preexisting neurological deficits (hard to differentiate worsening symptoms related to neuraxial blockade versus natural progression)
- Demyelinating lesions (i.e. MS may be exacerbated by the stress of surgery and temperature changes however this may be hard to differentiate the true cause)
- Stenotic valvular heart lesions
- Severe spinal deformity

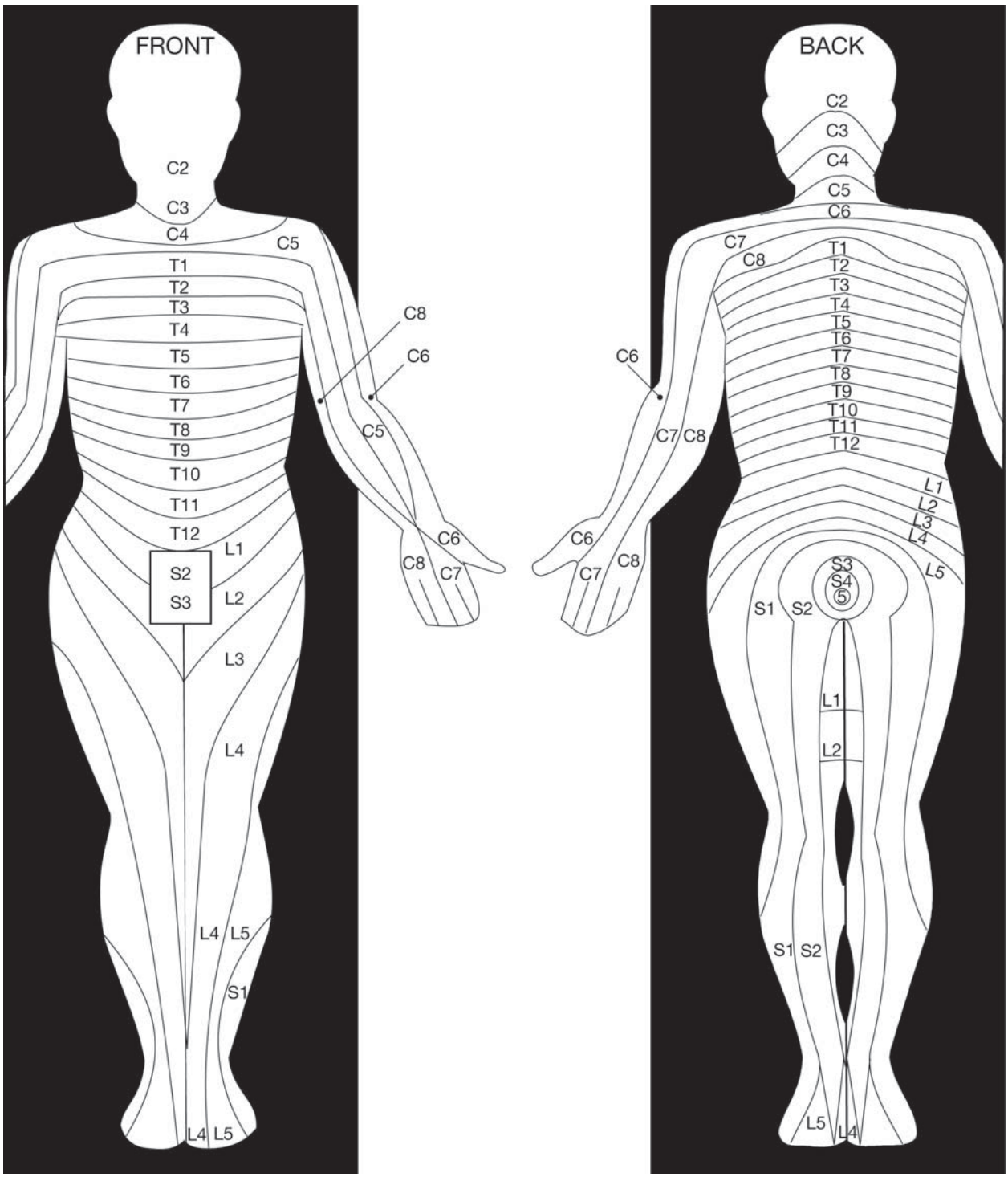
Controversial:

- Prior back surgery
- Inability to communicate with the patient
- Complicated surgeries that may involve a prolonged amount of time to perform, major blood loss, and maneuvers that may compromise respiration.

Appendix C: Current Recommendations for Spinal/Epidural Anesthesia and Anticoagulants

Classification	Medications	Recommendations	Laboratory
Antiplatelet's	Aspirin/NSAIDS	None	None
	Ticlopidine	DC 14 days before	None
	Clopidogrel	DC 7 days before	None
	Abciximab Eptifibatide Tirofiban	Avoid	None
Anticoagulants	Warfarin	DC 4-5 days before Monitor patient for 24 hours post spinal, epidural or removal of catheter	PT/INR prior to needle placement or catheter removal; INR <1.5
Heparin	Subq heparin	Delay until after block	>4 days check plt count
	IV heparin	Delay until 1 hour after block; remove catheter 2-4 hours after last dose.	Measure PTT
LMWH	Ardeparin Dalteparin Enoxaparin Tinzaparin Danaparoid	*Preop: block 10-12 hrs after last dose; high dose delay 24 hrs (enoxaparin). *Postop: Twice daily dose delay 1st dose for 24 hrs; 2 hr delay after catheter removal. Once daily dose 1st dose 6-8 hrs post op; remove catheter 10-12 hr after last dose and wait 2 hrs till next dose (enoxaparin).	None
Herbal Preparations	Garlic Ginkgo Ginseng Ginger Feverfew Vitamin E	DC 5-7 days before surgery	None
New Anticoagulants	Bivalirudin Lepirudin	Unknown; assess risk	None
	Fondaparinux	Extreme caution; atraumatic needle placement; no catheters	None

Appendix D: Dermatome Levels



This is the most common anatomical configuration. Variation may occur among patients.

Appendix E: Neuraxial Blockade Dermatome Levels and Systemic Effects

Operative Site	Level
Intraabdominal Procedures (other than lower abdominal)	T4
Lower Intraabdominal Procedures	T6
Lower extremities with a tourniquet Testicular and ovarian surgical procedures	T8
Hip surgery Vaginal or uterine surgical procedures Bladder and prostate surgical procedures	T10
Lower extremity surgery without a tourniquet	T12

Surface Anatomical Area	Dermatome Level	Systemic Effects
Fifth finger (digit)	C8	Blockade of all cardioaccelerator fibers (T1-T4)
Inner aspect of arm and forearm	T1-T-2	Some degree of cardioaccelerator fiber blockade
Apex of axilla	T3	Possible cardioaccelerator fiber blockade
Nipple	T4-T5	Possible cardioaccelerator fiber blockade
Bottom of xiphoid process	T7	Possible splanchnic blockade (T5-L1)
Umbilicus	T10	Sympathetic nervous system blockade
Inguinal ligament area	T12	Sympathetic nervous system blockade is limited to the legs
Lateral foot	S1	

Appendix F: Spinal Anesthesia

Advantages of Spinal Anesthesia

- Easy to perform
- Reliable form of anesthesia providing excellent operating conditions
- Generally is less costly than general anesthesia
- Normal return of gastrointestinal function generally occurs faster than with general anesthesia
- Patient maintains a patent airway
- Less pulmonary complications when compared to general anesthesia
- Decreased incidence of deep vein thrombosis and pulmonary emboli formation when compared to general anesthesia

Disadvantages of Spinal Anesthesia

- Risk of block failure can occur even in very skilled hands, though it is rare. This point stresses the need to always be prepared to induce general anesthesia if block failure occurs.
- Normal alteration in the patient's blood pressure and potentially heart rate. It is essential to place the spinal block in the operating room with monitoring of an ECG, blood pressure, and pulse oximetry. Resuscitation medications should always be available.
- Risk that the operation could last longer than the duration of the local anesthetic. Alternative plans such as general anesthesia should be prepared for in advance.
- Risks of complications outlined in the complications of neuraxial blockade that are specific to these techniques

Baracity of Local Anesthetics used for Spinal Anesthesia

Local Anesthetic	Specific Gravity
Bupivacaine 0.5% in 8.25% Dextrose	1.0227-1.0278
Bupivacaine 0.5% plain	0.9990-1.0058
Lidocaine 2% plain	1.0004-1.0066
Lidocaine 5% in 7.25% Dextrose	1.0262-1.0333
Procaine 10% plain	1.0104
Procaine 2.5% in water	0.9983
Tetracaine 0.5% in water	0.9977-0.9997
Tetracaine 0.5% in D5W	1.0133-1.0203

Spinal Anesthetic Dose and Duration

Medication	Preparation	Dose lower limbs	Dose lower abdomen	Dose upper abdomen	Duration plain	Duration epinephrine
Procaine	10% solution	75 mg	125 mg	200 mg	45 minutes	60 minutes
Lidocaine	5% solution in 7.5% dextrose*	25-50 mg	50-75 mg	75-100 mg	60-75 minutes	60-90 minutes
Tetracaine	0.5% (1% solution in 10% glucose or as niphanoid crystals)	4-8 mg	10-12 mg	10-16 mg	90-120 minutes	120-240 minutes
Bupivacaine	0.75% & 0.5% hyperbaric solution in 8.25% dextrose and hypobaric solution	4-10 mg	12-14 mg	12-18 mg	90-120 minutes	100-150 minutes

Dosages of local anesthetic are generalized suggestions and may need to be adjusted according to individual patient characteristics.

Appendix G: Epidural Anesthesia

Short Acting:

- 2- Chloroprocaine

Intermediate Acting:

- lidocaine
- mepivacaine

Long Acting

- bupivacaine
- etidocaine
- ropivacaine
- levobupivacaine

Caffeine Content of Common Beverages

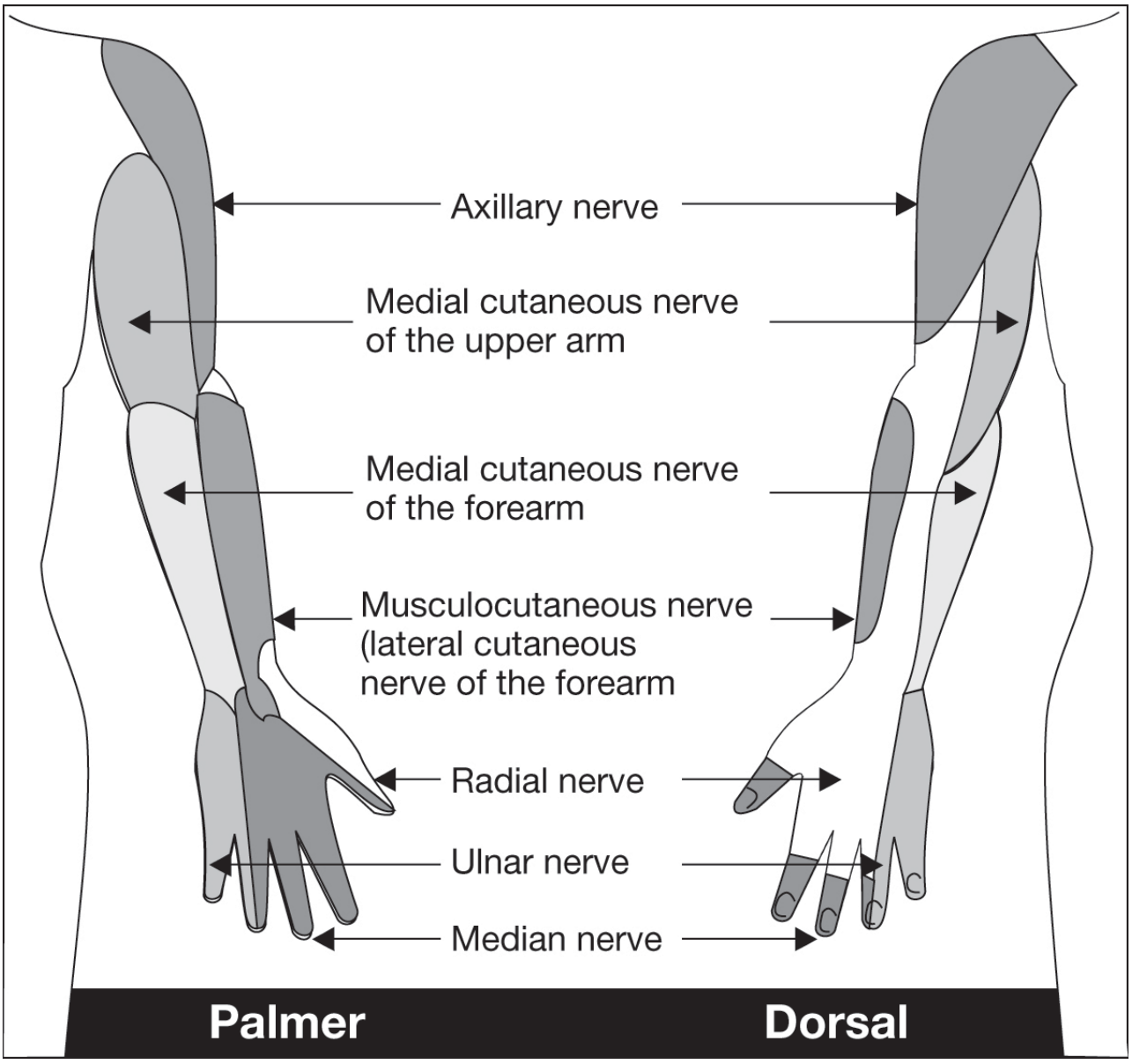
Beverage	Caffeine Content
Regular Coke™	34 mg/12 oz
Coffee (brewed)	80-135 mg/ 7 oz
Coffee (instant)	65-100 mg/ 7 oz
Tea (black)	70 mg/ 6 oz
Tea (green)	35 mg/ 6 oz

Epidural Local Anesthetics

Agent	Concentration	Onset	Sensory Block	Motor Block	Plain Solution	1:200,000 Epinephrine
Short Acting Local Anesthetics						
2-chloroprocaine	2%	Fast 10-15 minutes	Analgesic	Mild to moderate	45-60 minutes	60-90 minutes
2- chloroprocaine	3%	Same	Dense	Dense		
Intermediate Acting Local Anesthetics						
Lidocaine	1.5%	Intermediate 15 minutes	Dense	Mild to moderate	80-120 minutes	120-180 minutes
Lidocaine	2%	Same	Dense	Dense		
Mepivacaine	1%	Intermediate 15 minutes	Analgesic	Minimal		
Mepivacaine	2%	Same	Dense	Dense	90-140 minutes	140-200 minutes
Long Acting Local Anesthetics						
Bupivacaine	<0.25%	Slow	Dense	Minimal to moderate		
Bupivacaine	0.5-.75%	Same	Dense	Mild to dense	165-225 minutes	180-240 minutes
Levobupivacaine	<0.25%	Slow	Dense	Minimal to moderate		
Levobupivacaine	0.5-.75%	Same	Dense	Mild to dense	150-225 minutes	150-240 minutes
Ropivacaine	0.1-0.2%	Slow	Analgesic	Minimal		
Ropivacaine	0.5%	Same	Dense	Mild to moderate		
Ropivacaine	0.75-1%	Same	Dense	Dense	140-180 minutes	150-200 minutes
Etidocaine	1%	Slow	Dense	Dense	120-200 minutes	150-225 minutes

Appendix H: Brachial Plexus Anesthesia

Cutaneous Nerve Supply of the Arms



This is the most common anatomical configuration. Variation may occur among patients.

Major Motor Function of the Individual Nerves of the Brachial Plexus

Nerve	Major Motor Function
Axillary Nerve	Abduction of the shoulder
Musculocutaneous Nerve	Flexion of the elbow
Radial Nerve	Extension of the elbow, wrist, and finger
Median Nerve	Flexion of the wrist and finger
Ulnar Nerve	Flexion of the wrist and finger

Choosing the Correct Approach to Brachial Plexus Anesthesia

Interscalene Approach: excellent technique for surgical procedures on the shoulder, arm, and forearm. Intense block at C5-C7 and diminished blockade of C8-T1. Not a good technique for surgical procedures that involve the ulnar nerve distributions.
Axillary Approach: excellent technique for surgical procedures from the elbow to the hand. There is intense blockade of C7-T1. This approach is not adequate for the shoulder and upper arm (C5-6).

A Simple, Quick, and Easy Test of your Axillary Block

To quickly assess an axillary block perform the "push-pull-pinch-pinch" test. This test can be done in less than a minute. It can help identify "missed" nerves and allow time to formulate a plan (i.e. supplementation or general anesthesia or give the block more time to set up).

- Ask the patient to extend or "push" their forearm against light resistance. This tests the radial nerve.
- Ask the patient to flex or "pull" the arm towards the nose against light resistance. This will test the musculocutaneous nerve.
- Pinch the thenar aspect of the hand (i.e. on the palmar surface of the hand) which will test the median nerve.
- Pinch the hypothenar aspect of the hand (i.e. small finger) which will test the ulnar nerve.

Axillary Block Transarterial Technique by Wayne Smith, MD

Patient Selection

1. Able to expose axilla
2. For procedures of the lower humerus, forearm, and hand
3. For patients who cannot tolerate a general anesthetic

Contraindications

Anticoagulated, infection at site of injection

Local Anesthetics

Choices: Lidocaine 1% with epinephrine 1:200,000 (5 mcg per ml) or epinephrine 1:400,000 (2.5 mcg per ml). The maximum dose for lidocaine with epinephrine is 7 mg/kg or a total of 500 mg. Bupivacaine 0.25% with epinephrine 1:200,000 or 1:400,000. The maximum dose for bupivacaine with epinephrine is 3 mg/kg or a total of 225 mg. (**Extreme caution with bupivacaine-intravascular injection may result in cardiac arrest)**

Duration: Lidocaine with epinephrine 4-6 hours; bupivacaine with epinephrine 5-24 hours.

Supplies

23 gauge or smaller butterfly needle, 40 ml of local anesthetic divided in 10 or 20 ml syringes, gauze for pressure on the artery, diazepam, ketamine, atropine, and resuscitation equipment.

Preparation

Have supplies available, medications drawn up, lightly sedate the patient, and prep the axilla.

Technique

Palpate the artery. Advance the needle towards the artery slowly while aspirating the syringe. Once blood is noted advance the needle till blood stops. The needle is on the other side of the artery. Stabilize the needle. Aspirate one more time, if no blood then inject 5 ml of local anesthetic. Aspirate again and if no blood inject another 5 ml. Repeat this process until a total of 25 ml have been placed behind the artery. Continuously monitor the patient for intravascular injection (ringing in ears, tachycardia, metal taste in mouth, dizziness). Slowly withdraw the needle, while aspirating, until the blood stops. Stabilize the needle and inject 5 ml. Aspirate and if no blood than inject another 5 ml for a total of 10 ml. Local anesthetic deposited around the artery should block the radial, ulnar, and median nerve. A skin wheal should be placed in the axilla if a tourniquet is used to block the intercostal brachial and medial brachial cutaneous nerve to prevent tourniquet pain. The remaining 5 ml of local anesthetic is used to anesthetize the musculocutaneous nerve. This may be accomplished in the axillary area by injecting 5 ml of local anesthetic in the belly of the coracobrachialis muscle (located just below the bicep and above the tricep).

Appendix I: Ankle Block

Common Local Anesthetics Onset and Duration

Local Anesthetic	Onset	Duration
1.5% mepivacaine	15-20 minutes	2-3 hours
2% lidocaine	10-20 minutes	2-5 hours
0.5% ropivacaine	15-30 minutes	4-8 hours
0.75% ropivacaine	10-15 minutes	5-10 hours
0.5% bupivacaine	15-30 minutes	5-15 hours

Never use epinephrine in an ankle block.

Appendix J: Obstetric Anesthesia

Physiological Changes Associated with Pregnancy

Cardiovascular
The white blood cell count and its components may be markedly elevated during labor and delivery. In the absence of signs and symptoms of infection/sepsis or other contraindications it is reasonable to proceed with neuraxial blockade.
Never lay the patient supine. Always place a wedge or roll under the right hip so the patient is "tipped" to the left.
Sympathectomy associated with regional anesthesia can result in severe hypotension. It is important to pre-load the patient with 1-2 liters of crystalloid fluids.
Vessel distention in the epidural space can increase the risk of vessel damage during neuraxial blockade. Vessel distention also decreases the intrathecal and epidural spaces. Decrease the dose of local anesthetics by 30%.
Delayed absorption of subcutaneous/intramuscular medications.
Respiratory
Patients undergoing regional anesthesia should have supplemental oxygen administered.
Patients undergoing general anesthesia should be pre-oxygenated with 100% O2 prior to anesthesia induction.
Patients may desaturate quickly despite pre-oxygenation. This is due to increased oxygen consumption and a decrease in FRC.
Be prepared for a difficult intubation due to decreased visualization from swollen mucous membranes. Ensure that the patient is positioned for optimal viewing of the glottic opening prior to anesthetic induction.
Have a smaller than usual endotracheal tube available for intubation in case the usual sized endotracheal tube is too large due to swelling.
Be very gentle during laryngoscopy so bleeding does not obstruct the view.
The patient will have a faster uptake of inhalational anesthetics due to a decreased FRC and increased alveolar uptake.
Gastrointestinal
All pregnant patients should be considered to have "full stomachs" regardless of fasting.
If available, medications should be administered prior to anesthesia to reduce gastric acidity and volume. A non particulate antacid such as sodium citrate should be administered immediately prior to the anesthetic to reduce the acidity of stomach contents. To increase gastric emptying and increase lower esophageal sphincter tone, the administration of metoclopramide, in a dose of 10 mg IVP should be administered 30-60 minutes prior to anesthesia. The use of a histamine (H2 blocker) such as famotidine 20 mg IVP 30-60 minutes prior to anesthesia may reduce the acidity of stomach contents.
Slight reverse Trendelenburg position may reduce passive reflux.
Cricoid pressure should be utilized and held until the patient is intubated and placement of the endotracheal tube in the trachea has been confirmed.
Do not routinely administer positive pressure ventilation with a mask prior to intubation unless the patient's pulse oximetry reading starts to decline or a difficult airway is encountered. Positive pressure ventilation may cause gastric distention increasing the risk of aspiration.
Hepatic
Small risk of prolonged neuromuscular blockade when using succinylcholine or mivacurium for surgical intervention within 7 days post delivery.
Central Nervous System
Reduce the dose of inhalation anesthetics by up to 40%.
Reduce the dose of local anesthetics by up to 30%.
Positive pressure in the epidural space may make it more difficult to identify the epidural space.

Anesthesia Considerations for Non-obstetric Surgical Intervention during Pregnancy

Up to 2% of women will require surgical intervention during their pregnancy. The most common surgical procedures include appendectomy and cholecystectomy. The following considerations should be taken into account:

- Maternal mortality is not increased; fetal mortality may range from 5-35%.
- Only emergent/necessary surgical cases should be performed.
- If an elective surgical procedure, postpone until 6 weeks post delivery.
- Formation of fetal organs occurs between 15-56 days of gestation. There is no conclusive evidence that anesthetics cause problems during this time.
- Regional anesthesia is preferred over general anesthesia. Spinal anesthesia is preferred over epidural anesthesia.
- Increased risk during general anesthesia is related to technique. In the past, benzodiazepines and N2O were avoided. It was thought that they may contribute to teratogenic changes in the fetus. Currently there is no conclusive evidence that these medications cause teratogenicity. Exposure to all anesthetics should be kept to a minimum.
- Fetal monitoring should occur (when possible) for pregnant patients who are > 16 weeks.
- The patient should be monitored for pre-term labor.
- The patient is considered to have a full stomach.
- Displace the uterus with a roll under the patient's hip.
- Avoid hypoxemia (use a minimum of 50% O2).
- Avoid hypotension. Pre-hydrate the patient. Aggressively treat hypotension.
- Maintain adequate ventilation. Don't allow CO2 to accumulate or hyperventilate the patient.
- Extubate the patient when fully awake to avoid the risk of aspiration.
- Review the anesthetic implications associated with pregnancy.

Anesthetic Concerns for Cesarean Section

- **Hypotension**:
  - Pregnant women are at risk for aortocaval compression when supine. The uterus should always be displaced using a hip roll such as a folded towel or liter bag of intravenous fluid.
  - Preload the patient with non-dextrose containing crystalloid fluids prior to the administration of regional anesthesia reduces the incidence of hypotension.

- **Hemorrhage**:
  - Always maintain a functioning IV. If intravenous flow is inadequate, start another IV. The IV catheter should be at least an 18 gauge.
  - Start two IV's if the patient is at increased risk for hemorrhage.
- **Aspiration:**
  - All obstetric patients should be considered to have full stomachs.
  - Reduce risk of aspiration with pre-medications.
  - Use rapid sequence induction with cricoid pressure.
  - Do not routinely mask ventilate the patient prior to intubation.
  - Slight reverse Trendelenburg position may reduce passive reflux.
- **Hypoxemia:**
  - The obstetric patient has a 20% decrease in functional residual capacity (FRC) and increased O2 consumption. The patient is at increased risk for the hypoxia.
  - 100% O2 should be administered to the patient undergoing general anesthesia.
  - Supplemental oxygen should be administered to patients undergoing regional/local infiltration techniques.
- **Difficult Intubation:**
  - Obstetric patients are at increased risk for difficult intubation.
  - Position the patient for optimal laryngoscopy in the "sniffing position".
  - Ensure availability of several laryngoscope blades, different sized endotracheal tubes, and functioning laryngoscope handle.
  - The pregnant patient generally requires a smaller endotracheal tube (6.0, 6.5 or 7.0mm ET) due to the engorgement of the airway from pregnancy related increase in blood volume.
  - Use great care during laryngoscopy to reduce the potential for bleeding which can obstruct the view.
- **Anesthetic Requirements:**
  - Reduce inhaled anesthetics by 40%.
  - Reduce the dose of local anesthetics placed during neuraxial blockade by 30%.

Preparing for a Cesarean Section

It is important to always be prepared. To avoid problems with equipment the anesthesia provider should ensure that all the equipment that may be needed is available and working properly. The anesthesia provider should be aware of the following:

- Know what equipment is available
- Ensure the availability of an adequate supply of oxygen
- Ensure that the suction apparatus is functional (manual as a backup)
- A roll or wedge should be available (often a rolled up towel will suffice)
- Intubation equipment should be functioning with a variety of anesthesia masks, laryngoscope blades, endotracheal tubes, stylets, and oral airways.
- If available alternative airways such as a laryngeal mask airway or combitube can be life saving in the event of difficult or failed intubation.
- A bag mask valve device should be available in case the anesthesia circuit fails
- Resuscitation equipment and medications should be readily available
- Monitoring equipment should be available and functional
- An assistant with experience in maintaining cricoid pressure should be available if a general anesthetic is required
- Sterile equipment for neuraxial blockade
- Sterile local anesthetic solutions
- Anesthetic medications for the induction of general anesthesia

Confirmation of Correct Endotracheal Tube Placement

Trachea vs. Esophagus

Test	Result	How Reliable is it?
End tidal carbon dioxide testing	**Correct**: positive wave form **Incorrect position**: no waveform	Certain- is the best test
Esophageal detection device (i.e. 50 ml syringe with self inflating bulb)	**Correct**: air is easily aspirated **Incorrect:** the bulb does not aspirate air	Certain- unless the patient has a lot of air in the stomach.
Watch endotracheal tube go between vocal cords	**Correct:** easy view	Certain- unless visualization was poor.
Pulse oximetry	**Correct:** the reading easily comes up and reads within the normal range for the patient. **Incorrect:** the reading declines and continues to decline despite ventilation.	Certain
Listen with stethoscope	**Correct**: bilateral and equal breath sounds are noted. **Incorrect:** no breath sounds are noted/gurgling sound is noted over the stomach.	Probable- sounds can radiate and fool the anesthesia provider.
Ventilate the patient	**Correct:** easy to ventilate, chest rises. **Incorrect:** difficult to ventilate, stomach gurgles, chest does not rise.	Probable- the anesthesia provider can sometimes find it hard to distinguish between esophageal and tracheal placement of the endotracheal tube.
Observe the patient	**Correct**: the patient remains pink. **Incorrect:** the patient becomes cyanotic.	Certain/probable- by the time the patient becomes cyanotic the patient is very hypoxic.
Pushing on the patient's chest/condensation in the endotracheal tube	**Correct:** air comes back/condensation occurs. **Incorrect:** air does not come back/no condensation noted.	Probable- other techniques are more accurate.

Glossary of Common Terms

-A-

Acetylcholinesterase (Pseudocholinesterase)- An enzyme found in red blood cells and nerve endings. It is responsible for metabolism of acetylcholine. Succinylcholine has a short duration of action related to this enzyme. Patients with abnormally low levels of this enzyme may have a prolonged neuromuscular block related to the administration of succinylcholine.

Acid- A substance which releases H+ ions.

Action Potential- A change in the polarity of a nerve membrane which is important for the transmission of nerve impulses. The rapid change from negative to positive results in depolarization. This is due to sodium ions moving into the nerve membrane and potassium ions moving out. The reverse occurs during repolorization. Local anesthetics prevent an action potential from occurring.

Adrenalin- A term for epinephrine used by the British.

Adverse Reaction- A reaction to a medication that is not desired.

Afferent- Going towards the center. For example, afferent sensory information is sent to the central nervous system.

Agonist- A medication that binds to a specific receptor resulting in stimulation. For example, opioids stimulate mu receptors resulting in analgesia.

Allergin- A substance or medication that results in an allergic reaction.

Allergic Reaction- Signs and symptoms resulting from exposure to an allergen. Signs and symptoms may range from minor itching/rash to a life threatening anaphylactic reaction. An anaphylactic reaction must be treated rapidly to prevent cardiovascular collapse.

Alveoli- The portion of the lung that oxygen is delivered to the blood and carbon dioxide is exchanged.

Ambu-Bag- Also know as a bag-mask device. Contains a mask, self inflating bellows, a one way valve to prevent re-breathing, and oxygen tubing for an oxygen source. Often used for the resuscitation of patients outside the operating room. An ambu-bag should be available in the operating room in the event that the anesthesia circuit fails.

Amniotic Fluid Embolism- A rupture in the membrane that allows amniotic fluid to enter the mothers circulation. This will present suddenly as shock and cardiovascular collapse. Cardiovascular resuscitation will be required. This condition carries a high mortality rate.

Anemia- Abnormally low hemoglobin levels which impact the oxygen carrying capacity of the patient's blood. May be caused by excessive blood loss, diminished red blood cell production, and/or red blood cell destruction. These causes may be related to disease processes, hemorrhage, and malnutrition.

Antacid- A medication that reduces the acidity of stomach contents making the contents more alkaline. An example of an antacid would include sodium citrate.

Antagonist- A medication that binds to a specific receptor resulting in the blockade of stimulation. For example, naloxone will block the mu receptors resulting in the reversal of the respiratory depression associated with opioids.

Anticoagulant- Any medication or herbal product that affects coagulation. These medications have serious implications for surgery, peripheral nerve blocks, and are contraindicated for neuraxial blockade. Anticoagulants include antiplatelet agents, oral anticoagulants, heparin, low molecular weight heparin, and newer anticoagulants. Herbal preparations that may interfere with coagulation include garlic, ginkgo, ginseng, ginger, feverfew, and vitamin E.

Antiemetic- A medication used to prevent nausea and vomiting.

Antihistamine- A medication that is an antagonist to histamine. There are two types of histamine receptors. H-1 receptors are found in the intestines, blood vessels, and lungs. An allergic reaction will stimulate these receptors resulting in wheezing or bronchospasm. Diphenhydramine is a specific antagonist to H-1 receptors. H-2 receptors are found in the stomach. Cimetidine is a specific antagonist to H-2 receptors.

Antisialagogue- A medication that reduces salivation. Atropine and glycopyrrolate are traditional "drying" agents used prior to anesthesia. An antisialagogue is important to administer prior to a ketamine anesthetic.

Anxiety- A feeling of being uneasy or scared. The patient may experience tachycardia, sweating, increased blood pressure, and tremors. Patients often experience anxiety prior to surgery. Anti-anxiety agents such as diazepam or midazolam help to decrease anxiety.

Aortic Stenosis- A narrowing of the aortic valve which obstructs the outflow of blood from the left ventricle. The goal of an anesthetic in these patients include: maintaining the heart rate but avoiding tachycardia, maintaining a normal sinus rhythm, increasing/maintaining preload, maintaining afterload and blood pressure. This valvular condition increases the risk of mortality during anesthesia. Spinal anesthesia should be avoided.

Aortocaval Syndrome or Compression- Caused by the enlarging uterus of the pregnant patient. The pressure of the uterus may cause a decreased flow by compressing the aorta and vena cava. This results in a decrease in blood pressure, dizziness, and loss of consciousness. Left uterine displacement with a roll helps decrease the risk of this condition.

APGAR Score- A system of evaluating the newborn at 1 and 5 minutes after birth. This system measures the newborns heart rate, respiratory effort, muscle tone, reflex irritability, and color.

Area Assessed	0	1	2
Heart rate (beats per minute)	Absent	< 100	>100
Respiratory effort	Absent	Slow, irregular	Good effort; crying
Muscle tone	Flaccid	Some flexion	Active movement
Reflex irritability	No response	Grimace	Crying
Color	Blue or pale	Body pink; extremities blue	All pink

0-2 severe depression
3-4 moderate depression
5-7 mild depression
8-10 normal APGAR score

Apnea- Absence of breathing.

Arachnoiditis- inflammation of the arachnoid membrane which is located between the dura and pia mater. Rarely can occur after spinal anesthesia.

Aromatic- A group of molecules that are rings and have double bonds.

Arrhythmia- Any change in the patient's heart rhythm from normal sinus rhythm.

Aspiration- Any liquid or solid matter that enters the trachea and bronchioles. It is one of the most feared complications during anesthetic induction in patients that require emergent surgery and have recently eaten; pregnant patients; diabetic patients; obese patients; and any medical condition that may increase gastric contents or weaken sphincter control. Aspiration of stomach contents can result in life threatening bronchospasm and pneumonia. Medications to help reduce the risk of aspiration include: metoclopramide, to help empty the stomach; an antacid and histamine 2 antagonists, to reduce the acidity of contents. The application of cricoid pressure may reduce the incidence of aspiration.

Asystole- The heart stops contracting. The electrocardiogram will show a straight line. This must be rapidly treated by finding the cause. Common causes of asystole include: lack of oxygen, a abnormally high or low potassium, abnormally low body temperature, low blood pH, or a medication overdose. Additionally CPR, epinephrine, and atropine should be used during resuscitation.

Atelectasis- A collapse of portions of the patients alveoli (where oxygen exchange occurs) due to bronchial obstruction. This may impair the patients ability to remain oxygenated.

-B-

Barbiturate- A medication that results in generalized depression of consciousness and other vital bodily functions. An example of a barbiturate is sodium thiopental. Barbiturates should not be used in any patient who is hemorrhaging, hypovolemic or has a history of porphyria.

Base- A substance that is capable of binding to H+ ions. Local anesthetics are weak bases.

Benzene Ring- The most common aromatic molecule containing six carbon and six hydrogen atoms.

Benzocaine- An ester local anesthetic that is used as a topical anesthetic. The most commonly implicated agent for causing methemoglobinemia.

Benzodiazepine- A medication that reduces anxiety and can result in the inability to recall events. Examples of a benzodiazepine include diazepam and midazolam. Benzodiazepines can be used to treat seizures caused by local anesthetic toxicity.

Bier Block- Also known as intravenous regional anesthesia. Lidocaine is injected into a limb that has been exsanguinated through an intravenous catheter. A reliable double tourniquet must be used. If unreliable equipment is used this technique can be dangerous.

Bleeding Disorder- Any condition that results in the inability to clot blood normally. It may be the result of excessive blood loss, medication, herbs, or a disease process.

Bleeding Time- A laboratory test that determines how well the patient's platelets work. The platelets are responsible for forming the initial clot. Bleeding times are not very accurate.

Block- A generic term used to describe the injection of local anesthetic to prevent nerve conduction.

Blood Coagulation- The formation of the primary fibrin clot.

Blood Type- Individuals have a predetermined blood type. Patients that require blood transfusions should have type specific blood administered. If the blood is incompatible then a transfusion reaction can occur. This can result in a syndrome that resembles an allergic reaction.

Brachial Plexus Block- A regional anesthetic block that provides anesthesia to the upper arm, forearm, and hands by blocking the brachial plexus. Several approaches can be taken including the interscalene and axillary approach.

Bronchospasm- A narrowing or contraction of the patient's bronchioles. This may be a natural response to irritating inhaled anesthetics and airway manipulation, including intubation. A bronchospasm impairs oxygen exchange. It is often noted by an increase in the amount of pressure it takes to ventilate the patient and wheezing. It is treated by deepening the anesthetic, positive pressure ventilation, and bronchodilating medications. Severe cases may require epinephrine.

Bronchus- After the trachea the airway will divide into a right and left bronchus. From the right and left bronchus the airway will divide into several smaller bronchi. The right bronchus is straighter than the left. It is easy to insert an endotracheal too far and ventilate only the right side of the lungs. This is why it is important to listen to bilateral breath sounds after intubation. In addition, if the patient aspirates the right side of the lung is the most likely site to accumulate the contents.

-C-

Capnograph- A device used to monitor the expired carbon dioxide during anesthesia.

Carbon Dioxide- A gas product produced by the body and exhaled during respiration.

Cardiac Tamponade- An accumulation of fluid that compresses the heart. Common signs and symptoms of cardiac tamponade include: decreased blood pressure, decreased pulse pressure, tachycardia, and engorgement of the jugular veins.

Catecholamine- A term used to describe norepinephrine, epinephrine, and dopamine. These substances are naturally produced by the body as well as pharmacologically produced.

Cauda Equina Syndrome- A syndrome that can occur after the administration of local anesthetics. Generally it is caused by the accidental injection of toxic or contaminated agents. Signs and symptoms include: urinary and bowel dysfunction, sensory and motor deficits, and paralysis.

Cerebrospinal Fluid- Fluid that surrounds the brain and is found within the subarachnoid space. It is normally clear. Cerebrospinal fluid is returned in the spinal needle when this area is entered.

Chloroprocaine- A short acting ester local anesthetic.

Cholinesterase- An enzyme that metabolizes succinylcholine as well as other medications. An abnormally low level of this enzyme may result in a prolonged block for patients that have received succinylcholine.

Chronic Obstructive Pulmonary Disease- A chronic lung disease that results in abnormal gas exchange. May be chronic bronchitis or emphysema. For the patient with chronic bronchitis there is an irreversible narrowing of the bronchioles which decrease the amount of air that reaches the alveoli. For the patient with emphysema there is an irreversible destruction of the alveoli with reduces the amount of air exchange that occurs at the alveoli.

Cocaine- An ester local anesthetic. The first local anesthetic that was discovered and used for regional anesthesia. It has a high incidence of addiction and toxicity. It is rarely used clinically for anesthetic purposes.

Colloid- An intravenous infusion that helps expand the intravascular space. Colloids remain in the intravascular space longer than crystalloid solutions. Examples include hetastarch, dextran and albumin.

Conduction Anesthesia- A generic term to describe the injection of local anesthetics to block action potential and sensation. This term covers all forms of regional anesthesia.

Conscious Sedation- The use of intravenous medication to relax the patient, cause amnesia, and provide comfort. The patient is able to respond to stimulation, maintain spontaneous respiration, cardiovascular function, and airway.

Consent- When the patient gives permission to the anesthesia provider to administer an anesthetic. Prior to consent the patient should be informed of what the anesthesia provider will be doing, what to expect, complications, and alternative forms of anesthesia.
Creatinine- A laboratory value that indicates kidney function. Elevated levels may indicate kidney dysfunction (i.e. renal failure or insufficiency).

Crystalloid- An intravenous solution used to replace fluid loss. Examples include normal saline and lactated ringers.

-D-

Denitrogenation- The pre-oxygenation of a patient prior to the induction of anesthesia with 100% oxygen for at least 3 minutes is important to remove nitrogen. This allows for a reservoir of oxygen during an anesthetic induction.

Diastolic Pressure- A measurement of the patient's arterial pressure when the ventricles of the heart are at rest and filling with blood. This is the "bottom" number of the blood pressure.

Differential Block- When a local anesthetic is injected near a nerve it will affect various nerve fiber types at different rates. This is a function of local anesthetic concentration and nerve fiber type. For example with spinal anesthesia there are different levels for sympathetic, sensory, and motor blockade.

Disseminated Intravascular Coagulation (DIC)- An abnormal clotting disorder that results in hemorrhaging. It can occur in trauma patients and obstetric patients. This results in abnormal clotting and bleeding.

Dissociation Constant (pKa)- The pH at which 50% of a medication is ionized and 50% is non-ionized. The dissociation constant plays an important role in the onset of local anesthetics after a regional anesthetic block.

Drug Interaction- When one medication affects another medication. This can be in the medications rate of metabolism, ability to work, excretion, or tissue binding. For example the addition of an opioid to 2-chloroprocaine will result in the opioid being ineffective.

Dural Puncture- When a needle crosses the dura. This can be done on purpose, as with a spinal anesthetic technique, or accidentally, as when performing an epidural anesthetic.

Dyspnea- Any condition in which the patient feels that they can not breathe adequately. This may be noted by an increase in the work of breathing and inability for the patient to "catch" their breath.

Dystocia- An abnormal or difficult labor and delivery of an infant.

-E-

Eclampsia- Seizures that can not be attributed to other causes in a pregnant woman with preeclampsia.

Efferent- To move away from the center. Efferent motor nerves will transmit signals from the central nervous system to the muscle.

Electrocardiogram (EKG/ECG)- A monitor that records the electrical activity of the heart. This monitor helps diagnose changes in the patient's rhythm.

Elimination (medication)- the removal of a medication that ends its action. The medication may undergo a change through metabolism or be removed from the body unchanged.

Embolism- A rapid obstruction of a blood vessel. This may be caused by a blood clot, amniotic fluid, foreign objects, and/or air. A large pulmonary embolism may result in sudden cardiovascular collapse.

Emesis- Any stomach contents that are forcefully ejected (vomiting).

Emphysema- A form of chronic obstructive pulmonary disease.

Endobronchial Intubation- May be the result of mistakenly inserting an endotracheal tube too deep. Lung sounds are generally present only on the right side of the lungs. Alternatively, some surgical procedures require endobronchial intubation for one lung ventilation.

End Tidal Carbon Dioxide- The measurement of expired carbon dioxide by capnography. Helps detect successful endotracheal intubation and the adequacy of ventilation.

Ephedrine- A medication that has central and peripheral effects. It helps the heart contract stronger and vasoconstricts blood vessels. It is a common vasopressor that is used during anesthesia.

Epidural Anesthesia- A regional anesthetic technique that involves the injection of local anesthetics into the epidural space. This allows the local anesthetic to block spinal nerve trunks through direct contact and diffusion across the dura. It differs from spinal anesthesia in respect to anatomical site; slower onset; more local anesthetic volume/dose required to produce a block; may be less dependable (i.e. sacral sparing; one sided blockade); and may demonstrate a more identifiable differential block.

Epidural Needle- A specially designed needle that allows for the identification of the epidural placement as well as the insertion of an epidural catheter. There are several types.

Epidural Blood Patch- A technique to treat a postdural puncture headache, due to the leakage of cerebral spinal fluid, after conservative measures have failed. The epidural site, at the level of the original dural puncture, is accessed and the patient's blood is injected to reduce signs and symptoms.

Epinephrine- A substance produced by the body as well as a potent medication. Epinephrine, in small amounts, can be used as a vasoconstrictor with the administration of local anesthetics. Epinephrine is also used for the resuscitation of patients. Larger doses of epinephrine increases cardiac contractions and vasoconstricts blood vessels.

Expiration- The elastic recoil of the lung and chest wall resulting in the elimination of air from the lungs.

-F-

Fasciculation- Random, uncoordinated contraction of muscles. Associated with the administration of succinylcholine.

Fibrinolysis- The breakdown of a fibrin clot.

Field Block- A regional anesthetic technique in which the clinician injects local anesthetic around the surgical site. It can be used as a primary technique or in combination with other regional anesthetic techniques.

Flail Chest- Often the result of traumatic rib fractures. The portion of the chest wall that is affected will move out during inspiration and move in during expiration (paradoxical respiration). Treatment may include mechanical ventilation.

-G-

Ganglion- A concentration of nerve cells outside of the central nervous system. Nerve fibers that enter the ganglion are known as preganglionic nerve fibers. Nerve fibers that leave the ganglion are known as postganglionic fibers. A large collection of ganglia that form a network are called a plexus.

Gas Cylinder- A metal container that holds compressed gas. It is important to know the contents of gas cylinders to avoid the administration of gases other than oxygen. To help prevent catastrophic mistakes in delivering the incorrect gas to patients an international color code has been applied to gas cylinders to indicate the contents. It is important to check if your particular country has a system in place. In the absence of a reliable system it is important to ensure the correct gas is available prior to administration. This may be accomplished by sniffing the gas, checking with the supplier, and checking the contents with gas/oxygen analyzers if available.

International Color Codes

Gas	Symbol	Color
Oxygen	O2	White
Carbon Dioxide	CO2	Gray
Nitrous Oxide	N2O	Blue
Nitrogen	N2	Black
Air		White & Black

Glottis- A term to describe the opening to the larynx between the vocal cords.

-H-

Hallucination- A patient may experience a dream like state that includes things that are not real but experienced by sight, hearing, or feeling. Often noted with ketamine anesthesia.

Hematocrit- A laboratory test that indicates the volume of mature red blood cells. Used to determine blood loss, the need for blood replacement, and oxygen carrying capacity.
Hemoglobin- The specific portion of the red blood cell that carries oxygen. Used to help determine blood loss, the need for blood replacement, and oxygen carrying capacity.

Hemolysis- The destruction of hemoglobin.

Hemophilia- A genetic bleeding disorder that usually occurs in males. Regional anesthetic techniques should be avoided in patients with bleeding disorders. Surgery can result in hemorrhage.

Hemoptysis- Sputum that contains blood with origins in the trachea, bronchi, or lungs.

Hemorrhage- Excessive or uncontrollable bleeding.

Heparin- A medication that is capable of preventing clotting.

Hetastarch- A colloid fluid that is used as a volume expander. It is used to replace blood loss. Large doses may result in coagulation problems. There is a small risk of allergic reactions.

Histamine- A substance found throughout the body. There are two types of histamine. Histamine 1 (H1) receptors are found in the intestines, blood vessels, and lungs. An allergic reaction will stimulate these receptors resulting in wheezing or bronchospasm. Diphenhydramine is a specific antagonist to H-1 receptors. Histamine 2 (H-2) receptors are found in the stomach. Cimetidine is a specific antagonist to H-2 receptors.

Horner Syndrome- A side effect of interscalene block. Symptoms include miosis, ptosis, nasal congestion, and facial flushing. This side effect can be distressing to the patient.

Hyperbaric Solution- A spinal anesthetic solution that has a specific gravity that is greater than cerebral spinal fluid.

Hypercapnia- Abnormally high carbon dioxide level.

Hypertension- Abnormally high arterial blood pressure. Defined in adults as greater than 180 for systolic pressure and 100 for diastolic pressure.

Hyperventilation- An increase in respiratory rate and/or volume that decreases carbon dioxide levels to lower than normal levels.

Hypobaric Solution- A spinal anesthetic solution that has a specific gravity less than cerebral spinal fluid.

Hypocapnia- Abnormally low carbon dioxide level.

Hypotension- An arterial blood pressure that is lower than normal. Significant hypotension may result in decreased tissue perfusion and potential cardiovascular/neurological impairment. Hypotension can be treated during anesthesia by decreasing the amount of anesthetic that a patient

is receiving, increasing fluid administration, a head down position, and the administration of vasopressors.

Hypothermia- A lower than normal body temperature. Patients should be protected from hypothermia by keeping them covered.

Hypoventilation- A decrease in respiratory rate and/or volume that increases carbon dioxide levels to higher than normal levels.

Hypovolemia- A decrease in intravenous fluid volumes that can lead to hypotension and decreased tissue perfusion.

Hypovolemic Shock- A condition in which hemorrhage leads to tissue hypoxia, metabolic acidosis, and eventual cellular death. It is important to restore the patient's intravascular volume in treating this condition. Signs and symptoms and may include hypotension, tachycardia, cool and clammy skin, and increased respiratory rate.

Hypoxia- A decrease in oxygen delivered to tissue. Hypoxia can be caused by anemia related to a decrease in oxygen transport to tissue; from a lack of oxygen delivery to the patient; toxicity that does not allow oxygen to be used by tissue; and severe hypotension which does not allow the oxygen to be delivered to the tissue in enough quantity.

-I-

Informed Consent- The explanation and acceptance of the risks and benefits associated with any procedure/anesthetic.

Intraspinous Ligament- The ligament found on the dorsal side of the vertebral column. It connects the spinous processes together. It is located between the ligamentum flavum and supraspinous ligament.

Interscalene Block- An approach to the brachial plexus that takes advantage of the interscalene groove at the level of the cricoid cartilage. It is a suitable approach for surgical procedures of the shoulder, upper arm, lower arm, and hand.

Insensible Water Loss- Body fluids that are lost from the skin due to evaporation and ventilation.

Intrathecal Injection- Any medication administered into the subarachnoid space (i.e. spinal block).

Intubation- The placement of an endotracheal tube through the nose or mouth into the trachea.

Ion- A molecule that contains a charge that may be positive or negative.

Isobaric Solution- A spinal anesthetic solution that has the same specific gravity as cerebral spinal fluid.

-J-

Joule- A measurement of energy. The energy used in defibrillation is measured in joules.

-K-L-

Labor- The process of dilatation of the cervix and eventual delivery of a newborn and placenta. There are three stages. In the first stage uterine contractions result in the dilation of the cervix. In the second stage the newborn is delivered. In the third stage the placenta is delivered.

Laryngoscopy- The use of a laryngoscope to view the glottic opening to place an endotracheal tube.

Laryngospasm- A contraction of the muscle of the larynx resulting in the inability/increased difficulty in the ventilation. This complication occurs when an endotracheal tube is not in place in the glottis. It is often related to light anesthesia and can be life threatening. Avoiding the manipulation of the airway when the patient is "light" will decrease this complication. Treatment includes the use of positive pressure to overcome the obstruction and the use of succinylcholine/non depolarizing muscle relaxant. Use a non depolarizing muscle relaxant if succinylcholine is contraindicated.

Larynx- The portion of the airway that contains cartilage rings. It is the space between the pharynx and trachea. In the adult the larynx is found between the 4^{th} and 6^{th} cervical vertebrae.

Lidocaine- An amide local anesthetic.

Ligamentum Flavum- The last ligament to be crossed prior to the epidural space. It is located between the interspinous ligament and epidural space.

Lignocaine- A British term for the local anesthetic lidocaine.

Local Anesthetic- A medication that produces a temporary loss of sensation. Local anesthetics prevent action potentials from occurring. The two types of local anesthetics are amides and esters.

Amides	Esters
Bupivacaine	Chloroprocaine
Levobupivacaine	Cocaine
Etidocaine	Procaine
Lidocaine	Tetracaine
Mepivacaine	
Prilocaine	
Ropivacaine	

Lumbar Puncture- A regional anesthetic technique that administers a local anesthetic into the subarachnoid space. The needle must cross several anatomical structures including the skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, and dura. A lumbar puncture may also be performed to collect cerebral spinal fluid for diagnostic purposes.

-M-

Malignant Hyperthermia- A genetic condition caused by volatile anesthetics and succinylcholine. It results in a high fever, tachycardia, arrhythmias, hypoxia, increased carbon dioxide production, and muscle stiffness. It must be treated rapidly with dantrolene sodium along with symptomatic treatment. Death can ensue rapidly without appropriate treatment.

Mean Arterial Pressure- This is the average blood pressure. To derive this number the formula is as follows: Mean Arterial Blood Pressure = diastolic blood pressure plus 1/3rd of the pulse pressure (systolic minus diastolic pressure).

Mediastinum- The anatomical area that is located between the lung, sternum, and vertebral column. This area contains many vital structures including the heart, esophagus, and trachea.

Mepivacaine- An amide local anesthetic.

Metabolic Acidosis- A condition that results in a decrease in normal body pH. This can be caused by such conditions as diarrhea and kidney dysfunction.

Metabolic Alkalosis- A condition that results in an increase in normal body pH. This can be caused by such conditions as vomiting and endocrine abnormalities.

Mitral Regurgitation- A condition in which the mitral valve does not close resulting in blood being ejected back into the left atrium. Goals of anesthesia include maintaining or slightly increasing heart rate, maintaining preload, reducing afterload and blood pressure.

Mitral Stenosis- A condition in which there is a narrowing of the mitral valve. This results in an increased pressure in the pulmonary artery and right ventricle. Pulmonary edema can occur. Careful control of the heart rate is important. Tachycardia should be avoided.

Morbidity- An unintended effect/complication of a procedure. For example, a post dural puncture headache is a morbidity associated with spinal anesthesia.

Mortality- The possibility of death or actual death.

Myelin Sheath- This is an anatomical material that contains lipids and covers nerve fibers. The myelin sheath helps to facilitate the transmission of nerve impulses.

Myocardial Infarction- A condition that is the result of a decrease in oxygen supply to the heart muscle. This may be caused by any obstruction or decrease of blood flow to the heart muscle. Death can occur from myocardial infarction.

-N-

Nerve Fiber- A term to describe axons which contain both sensory and motor fibers. Nerve fibers vary in size as well myelination. They are classified based on anatomy and function. Local anesthetics will block nerve fibers at varying rates.

Fiber Type	Function	Diameter (mm)	Speed of Conduction	Local Anesthetic Sensitivity*	Myelination
Aα	Motor	12-20	Fast	1	Yes
Aα	Proprioception	12-20	Fast	2	Yes
Aα	Proprioception	12-30	Fast	2	Yes
Aβ	Touch Pressure/Proprioception	5-12	Medium	2	Yes
Aγ	Motor	3-6	Medium-Slow	2	Yes
Aδ	Pain Cold Temperature Touch	2-5	Medium-Slow	3	Yes
B	Preganglionic autonomic fibers	<3	Medium-Slow	4	Some
C (dorsal root)	Pain Warm and Cold Touch	0.4-1.2	Slow	4	No
C (sympathetic)	Postganglionic sympathetic fibers	0.3-1.3	Slow	4	No

Normocapnia- A normal level of carbon dioxide.

Normothermia- A normal temperature.

-O-

Opiate- A group of medications that are used to treat pain. Examples of opiates include fentanyl, morphine and meperidine.

Oximeter- A monitoring device that uses two different wavelengths of light to estimate oxygen saturation of blood. This monitor, along with the capnograph, greatly reduces the mortality and morbidity associated with anesthesia.

-P-

Para-Aminobenzoic Acid (PABA)- A breakdown product of some ester local anesthetics that is a known allergin.

Partial Thromboplastic Time (PTT)- A test that measures the ability to clot blood. The PTT may be affected by a number of medications, liver dysfunction, and other conditions.

pH- Is the measurement of hydrogen ion content. As the pH of a solution increases the hyrdrogen ion concentration will decrease. The pH of a local anesthetic has direct clinical implications concerning onset.

Pharmacodynamics- What a medication does in the body (i.e. the effects, toxicity).
Pharmacokinetics- The absorption, distribution, and elimination of a medication.

Pharynx- The area between the mouth and larynx. This can be divided into the nasopharynx, which is area in the mouth that contain the nasal passages; the oropharynx which includes the back of the mouth down to the epiglottis; and the laryngopharynx, which includes the epiglottis to the larynx.

Phenylephrine- A medication that is used as a vasopressor during anesthesia. Phenylephrine will increase blood pressure by contracting blood vessels. It is very potent and care must be taken not to administer an overdose.

Physical Status- Is a classification system that is used for anesthesia to address the overall health of the patient.

ASA Class	Examples of Systemic Conditions
I	Healthy patient
II	Mild to moderate disease such as controlled hypertension, controlled diabetes, and/or obesity.
III	Severe disease such as angina, COPD, and prior myocardial infarction.
IV	A severe disease that is a constant threat to life such as congestive heart failure, renal failure, and liver disease.
V	A patient not expected to live more than 24 hours and includes a ruptured aneurysm.

Plasma Expander- A solution that is used to replace blood loss. Examples include albumin, hetastarch and dextran. Blood loss is replaced at a 1:1 ratio.

Platelet- A component of the blood that is responsible for creating an initial blood clot.

Platelet Concentrate- A collection of platelets. It is used to treat patients that have a low number of platelets and in situations of severe hemorrhage. It will temporarily help to reduce bleeding in these situations.

Pneumothorax- A collection of air in the mediastinum that usually is the result a puncture to the lung. An open pneumothorax is a wound that communicates through the chest wall. As the patient breathes in, more air will accumulate in the chest. A closed pneumothorax does not have a wound that communicates with the outside. It may be the result of fractured ribs. A closed pneumothorax may quickly turn into a tension pneumothorax.

Postive End Expiratory Pressure (PEEP)- A term that describes an elevated pressure in the lungs at the end of expiration. PEEP helps to keep small airways from collapsing.

Preeclampsia- A condition that can occur during pregnancy that may lead to increased mortality and morbidity. The patient will exhibit hypertension, swelling, and protein in the urine.

Preservative- A chemical that is added to a solution to help with stability and/or prevent the growth of infectious agents. Only preservative free solutions should be used for neuraxial anesthesia.

Prilocaine- An amide local anesthetic.

Procaine- One of the first ester local anesthetics.

Prolapse- A portion of an organ that falls down out of anatomical position. For example an prolapse of the umbilical cord means that the umbilical cord has "fallen down" below the fetus.

Prothrombin Time (PT)- A test that measures the ability to clot blood. The PT may be affected by a number of medications, liver dysfunction, and other conditions.

Pulmonary Edema- The presence of fluid in the lung tissue. This is usually the result of cardiac dysfunction.

Pulse Pressure- The difference between the diastolic and systolic blood pressure.

-Q-R-

Respiratory Acidosis- A rise in the carbon dioxide level of a patient that is above normal (35-45 mmHg), not in response to metabolic alkalosis.

Respiratory Alkalosis- A decline in the carbon dioxide level of a patient that is below normal (35-45 mmHg), not in response to metabolic acidosis.

Respiratory Failure- The failure of a patient to be able to breathe adequately due to physiological conditions or medication.

-S-

Shock- Any condition when the body is unable to meet basic metabolic demands. This leads to tissue hypoxia, metabolic acidosis and eventual cellular death. Shock may be termed hypotensive, cardiogenic, neurogenic, and septic. It is important to treat the cause. Signs and symptoms and may include hypotension, tachycardia, cool and clammy skin, and increased respiratory rate.

Sodium Channel- The site in a nerve membrane that allows sodium to go in. This is the site of action for local anesthetics.

Spinal Anesthesia- The administration of a local anesthetic into the cerebral spinal fluid resulting in anesthesia. Spinal anesthesia must be administered below the termination of the spinal cord. In most adults this would be below the second lumbar space.

Spinal Headache- This is a complication of spinal anesthesia or accidental dural puncture. Cerebral spinal fluid will leak out of the dura resulting in a severe headache related to an upright

position. Conservative treatment includes bed rest, caffeine, analgesics, and increased fluid intake. If conservative measures do not work then an epidural blood patch may be necessary.

Spinal Needle- A specially designed needle that is used for the administration of local anesthetics into the subarachnoid space.

Stridor- A high pitched noise noted during inspiration. This is generally caused by an obstruction of the larynx. This may be caused by a foreign object or more commonly swelling related to intubation.

Supraspinous Ligament- The ligament found on the dorsal side of the vertebral column. It connects the tip of the spinous processes together. It is located between the subcutaneous tissue and interspinous ligament.

Synapse- This is the space located between two neurons. Impulses move from one nerve cell to another through the synapse.

Systolic Blood Pressure- Measurement of the arterial pressure when blood is ejected from the ventricles. This is the "top" number of the blood pressure.

-T-

Tachypnea- A faster than normal respiratory rate.

Tension Pneumothorax- A collection of air in the mediastinum that usually is the result a puncture to the lung. A closed pneumothorax does not have a wound that communicates with the outside. It may be the result of fractured ribs. A closed pneumothorax may quickly turn into a tension pneumothorax.

Tetracaine- An ester local anesthetic.

Thrombocytopenia- A reduced number of platelets. This condition may lead to an increased risk of bleeding.

Topical Anesthesia- The application of local anesthetic to the skin or mucous membrane that results in the loss of sensation.

Total Spinal- A complication of spinal anesthesia. The local anesthetic that is injected into the subarachnoid space travels high enough to effect cardiac and respiratory function. This complication must be rapidly treated to support the patients airway, breathing, and circulation.

Tourniquet- A device that applies pressure around a limb to stop or prevent bleeding. A double tourniquet is used for Bier block. The tourniquet must be tested to ensure proper function prior to the initiation of a Bier block.

Transfusion Filter- A filter that is added to a transfusion to prevent any debris from enter the circulation of the patient.

Transfusion Reaction- A potentially life threatening reaction related to the administration of incompatible blood to a patient. This can result in shortness of breath, hives, chills, chest pain, problems with coagulation, and cardiovascular collapse.

Tuohy Needle- A special needle designed with a curved tip for the insertion of an epidural catheter.

U-V-W-X-Y-Z

Ultrasound- A device that uses sound waves to evaluate anatomical structures. Ultrasound is used often to evaluate the fetus in the uterus.

Uterine Contraction- Contraction of the uterine smooth muscle during labor to help in the delivery of the newborn.

Von Willebrand Disease- An inherited disorder that results in bleeding. This condition is a contraindication to regional anesthetic techniques.

Considerations

Considerations is intended to supplement textbooks for students who are learning basic anesthesia and intensive care.

Considerations is primarily concerned with the fundamentals of physiology and pharmacology. A few other areas of interest are also presented.

(Editor's note: Dr. Fell has had extensive experience teaching anesthesia in Africa and Asia. He has developed a very practical handout. Dr. Fell has graciously offered to allow the addition of **Considerations** as a supplement in the hope that it will benefit the reader with additional, practical information.)

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Simple Steps to Reduce Mortality & Morbidity
Always be vigilant & prepared!

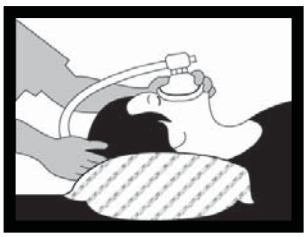
1. Perform a thorough preoperative assessment.
2. Ensure that the patient has not eaten in the last eight hours.

3. Place the patient on a operating room table that can tip.
4. Ensure that the anesthesia machine is functional and cylinders have an adequate supply of oxygen.

5. The ability to suction should be immediately available.

6. Ensure that the airway is kept open and clear.

7. Be prepared to control the patient's ventilation.



8. Ensure you have adequate intravenous access.

9. Check an initial set of vital signs including blood pressure, pulse, and pulse oximetry if available.

10. Ensure that you have someone who can administer and maintain cricoid pressure.

The ABCDs

The fundamental skills of anesthesia and intensive care are the ability to control:

Airway:
Unobstructed air passages allow the patient to breathe.

Breathing:
Oxygenation and ventilation of the alveoli add oxygen and remove carbon dioxide from the blood circulating through the lungs.

Circulation:
Adequate blood volume, blood pressure and blood flow deliver oxygen to the body's cells and remove carbon dioxide from the body's cells.

Drugs:
Multiple types of drugs help with the ABCs or provide anesthesia and pain relief.

Anesthetic Principles

1) Preparation.
The anesthetist must: know anatomy, pharmacology and physiology; have practiced the type of anesthesia contemplated; have done a proper history and physical examination; know the laboratory values; have communicated with the surgeon; have all necessary equipment and drugs immediately available; and have a specific plan for the anesthetic.
2) Vigilance.
The anesthetist must be alert during all phases of the anesthesia. Observation of the patient and surgical field, continuous monitoring of heart rate and respiration and intermittent determination of blood pressure are minimum requirements.
3) Skepticism.
The anesthetist must be aware that his/her evaluation of the patient and the information received from the patient, the family, the surgeon or the laboratory may be inaccurate or misleading.
4) Versatility.
The anesthetist must be able to modify the initial anesthetic plan depending on changing circumstances.
5) Routines are traps.
If the anesthetist treats everyone in the same way, some patients will be harmed. Just two examples among many are: Not all C-sections should have spinal anesthesia. Not all patients can tolerate atropine before induction.
6) Expediency is the enemy of the patient.
The social or family demands on the anesthetist or surgeon should not influence how or when a patient needing emergency care is treated.
7) When in doubt about the patient's breathing or level of oxygenation:
Ventilate and oxygenate the patient! No harm will be done.
8) In addition to the ABCDs, there are many things to consider during an anesthetic. Some are:
a) The provision of appropriate and safe analgesia, amnesia and muscle relaxation.
b) The components of blood pressure and minute ventilation and how drugs and surgery effect them.
c) The third space.
d) The urine output.
e) The functional residual capacity.
9) Avoid hubris
10) The patient is not a sack of potatoes.
Treat the patient as you would like to be treated.

Necessary Routines

If the following routines are followed for every patient there will be far fewer anesthetic complications.

1) Visit, properly evaluate and prepare the patient for the anesthesia. Make sure he/she is NPO.
2) Start an intravenous infusion.
3) Check the anesthesia machine, suction device, airway equipment, oxygen supply and drugs.

4) Take the vital signs before beginning the anesthesia.
5) Have an assistant during the induction.
6) Monitor and keep a record of the vital signs during surgery.
7) Continuously stay with the patient until you transfer the patient's care to the post-operative nurse.

CARDIOVASCULAR CONSIDERATIONS

(I) BLOOD PRESSURE

A) Changes in systemic and/or pulmonary arterial blood pressure may cause differences in:
1) Mental state
2) Level of physical activity
3) Skin: color, temperature and presence of edema fluid
4) Respiratory rate, depth, and rhythm
5) Presence or change in sputum
6) Appearance of neck veins
7) Body temperature
8) Pulse rate, fullness and rhythm
9) Urine output
10) Oxygenation and acid base status of the fetus if placental blood flow changes.
11) Chest sounds
12) Liver and abdominal size
13) Central venous pressure (C.V.P.)

B) Equations regarding blood pressure and cardiac output:
1) Blood pressure = Cardiac output X Peripheral resistance
2) Cardiac output = Stroke volume X Heart rate
3) Blood pressure = Stroke volume X Heart rate X Peripheral resistance
   a) The equations state that blood pressure is the value obtained when cardiac output (which is the same as stroke volume X heart rate) is multiplied by peripheral resistance.
   b) If stroke volume and/or heart rate and/or peripheral resistance change, their relative changes will determine if blood pressure rises, decreases, or remains the same.

C) Cardiac output is the amount of blood pumped by either the right or left ventricle in one minute. Cardiac output = Stroke volume X Heart rate.
1) In the average adult, each ventricle will normally pump five liters of blood per minute.
2) A normal ventricle will pump whatever amount of blood (venous return) is presented to it up to about 10 or 15 liters of blood per minute. The central venous pressure (C.V.P.) and left atrial pressures will be normal.
3) A diseased heart may only be able to pump 1-2 liters of blood per minute. The central venous pressure (C.V.P.) and left atrial pressures will probably be high.

D) Stroke volume is the amount of blood pumped by one ventricle in one beat. Stroke volume is about 70-80 ml/beat in a healthy resting adult.
1) Stroke volume is determined primarily by how much blood, also known as venous return, is presented to the heart.
   a) If the heart is unable to pump the blood presented to it, the venous return can be reduced by fluid restriction or diuretics or, in very rare circumstances, phlebotomy.
   b) Stroke volume is often decreased during pregnancy when the large uterus compresses the inferior vena cava and interferes with venous return. This is known as the supine hypotensive syndrome. The treatment is to push the uterus to the left.
2) Stroke volume is also determined by the heart muscle contracting well enough to empty the ventricles. (Myocardial contractility)
   a) A diseased heart may not be able to empty one or both the ventricles, have low stroke volumes, and be in heart failure.
      i) Right heart failure causes high systemic venous and capillary pressures.
      ii) Left heart failure causes high pulmonary venous and capillary pressures.
   b) Depending upon the circumstances, drugs such as digitalis, ephedrine, isoproterenol, and epinephrine may be used to increase heart muscle strength.
   c) Most anesthetic drugs including halothane, thiopentone and diazepam will decrease the strength of the myocardium. Stroke volume will decrease.

E) Heart rate is the number of heart beats per minute.
1) If there is a low venous return, the normal heart will usually increase its rate.
2) In some types of heart disease, patients are unable to increase their cardiac outputs because their heart rates cannot increase. These patients have limited activities.
3) Infants are very dependent on heart rate for their cardiac output. A slow heart rate is a dangerous sign in infants. During pediatric anesthesia, bradycardia is usually due to vagal stimulation, a high dose of halothane or because the patient is hypoxic.
4) Some patients with heart disease have very rapid heart rates. There is little time for blood to fill the ventricles. Stroke volume and cardiac output will be low. The usual treatment is digitalis.
5) Some drugs (atropine, ketamine, ether, flaxedil, etc.) will cause tachycardia, and perhaps increased blood pressure. These drug-induced changes may make it difficult to use the heart rate and blood pressure as signs of blood volume status or anesthetic depth.

F) Systemic peripheral resistance is the resistance in the systemic arteries and arterioles that the left ventricle muscle must overcome when pumping blood to the organs.
1) The amount of contraction of the smooth muscles in the arterial and arteriolar walls determines the diameter of the blood vessels and thus how much resistance there is.
2) If resistance is high because of small diameter blood vessels, it is more difficult for the left ventricle muscle to pump the blood.
a) Sudden high resistance, which usually causes high blood pressure, is common with pain, especially post-operative pain. Opiates are the best treatment.
b) Long term hypertension of uncertain etiology is associated with increased peripheral resistance and a decrease in blood volume. The hypertension is treated with oral drugs, which result in relaxation of the smooth muscles in the arteriolar walls.
c) Eclamptic patients have; hypertension, high peripheral resistance and low blood volume.
3) If resistance is low, when the arteries and/or veins are dilated, the blood will remain in them and there will be a decreased venous return. This will result in a decreased stroke volume because less blood returns to the heart. Heart rate will usually increase to compensate for the decrease in cardiac output.
a) Low resistance may occur in neurogenic shock, septic shock and spinal anesthesia. The treatment is blood, Ringers lactate, 0.9% saline and/or raising the legs.
b) Ephedrine may be used to treat the decreases in peripheral resistance and heart rate that occur during spinal anesthesia.

G) Pulmonary peripheral resistance is low. The right ventricle has the same stroke volume and heart rate as the left ventricle but does not work as hard to pump the same amount of blood per minute. Pulmonary artery blood pressure is much lower than the systemic artery blood pressure.

H) The autonomic nervous system is made up of two parts:
1) The sympathetic nervous system releases norepinephrine and epinephrine which:
a) Increase heart rate.
b) Increase the strength of heart muscle.
c) Constrict the muscles of systemic arterioles and veins.
2) The parasympathetic nervous system releases acetylcholine (Ach) which:
a) Decreases heart rate.
b) Relaxes the muscles of some arterioles.

I) **The balances among:**
1) The sympathetic nervous system
2) The parasympathetic nervous system
3) The amount of blood that is presented to the heart (venous return)
4) The ability of the heart to pump

Determine:
5) The heart rate ➔
6) The stroke volume ➔ Determine: ➔ **BLOOD PRESSURE**
7) The peripheral resistance ➔

(II) BASICS OF THE AUTONOMIC NERVOUS SYSTEM

A) The brain, spinal cord and peripheral nerves are one organ with billions of interconnecting nerve cells.
   1) Almost instantaneous electrical signals are always being sent back and forth among the brain, the spinal cord, peripheral nerves and the organs.
   2) The constant motor, sensory and autonomic electrical signals help maintain the body during rest, physical activity and in times of stress.

B) The spinal cord has 31 bilateral segments:
   1) Motor nerve impulses go from the brain and spinal cord to the muscles.
   2) Sensory nerve impulses go from the organs to the spinal cord and brain.
      a) Pain, touch, pressure, temperature, and proprioception have their own nerves.
      b) Nerves for odor, taste, vision and hearing go directly to the brain.
      c) Blood pressure, pulse rate, and carbon dioxide levels are also sensed.
   3) In addition, the brain via spinal cord segments T_1 to L_3, sends sympathetic nerve signals to the organs -- the Sympathetic Nervous System.
   4) In addition, the brain via the vagus nerve and S_{2-4} segments of the spinal cord, sends parasympathetic nerve signals to the organs--the Parasympathetic Nervous System.

C) Together, the sympathetic and parasympathetic nervous systems are known as the Autonomic Nervous System.
   1) The term autonomic means automatic or independent. We have no conscious control over either of the sympathetic or parasympathetic nervous systems.
   2) Both the sympathetic and parasympathetic nervous systems are always stimulating the body's organs.
      a) One nervous system is able to temporarily dominate the other, depending on the body's requirements.

D) When the brain is stimulated by fright, hypotension, hypoxia, pain etc., it will increase its signals through the spinal cord and sympathetic nerves to the organs.
   1) The sympathetic nerves almost instantaneously increase the release of norepinephrine (also known as noradrenaline) in the organs.
      a) The heart will beat stronger and faster.
      b) Most of the arterioles and venules will constrict.
      c) The bronchioles will dilate.
      d) Minute ventilation will increase.
      e) The adrenal gland will release more norepinephrine and epinephrine into the blood to maintain the effect of the increased norepinephrine released at the organs by the sympathetic nerves.
   2) Ketamine and ether stimulate the sympathetic nervous system to release more norepinephrine and epinephrine. That is why blood pressure, pulse and respiration are maintained at normal or high levels when the drugs are used properly.

E) While resting, or when the brain is stimulated by peritoneal traction, or when the sympathetic system is blocked, the brain will increase its signals through the spinal cord and parasympathetic nerves to the organs.
   1) The parasympathetic nerves will increase the release of acetylcholine (Ach) in the organs.
      a) The heart will beat weaker and slower.
      b) The arterioles and venules will relax.
      c) The bronchioles will constrict.
      d) The minute ventilation will decrease.

(III) SHOCK

A) Shock is present when organs are functioning poorly because of a low oxygen supply to the organs' cells. The low oxygen supply may be a result of inadequate hemoglobin in the blood or, much more commonly, because of low blood flow to the organs.
   1) Low blood flow is caused by an inadequately pumping heart, a low blood volume or a combination of the two causes.
   2) Hypovolemic shock and septic shock are two of the most common forms of shock and are discussed here. Other types of shock are cardiogenic, neurogenic and anaphylactic shock.

B) Hypovolemic shock is a situation in which there is a low venous return.
   1) Symptoms:
      a) Thirst
      b) Feeling of cold
      c) Fatigue
   2) Signs:
      a) Tachycardia and weak pulse
      b) Decreased blood pressure
      c) Mental changes of lethargy, confusion or restlessness
      d) Cold extremities and decreased body temperature
      e) Tachypnea
      f) Decreased or absent urine output
   3) Hypovolemic shock may result from external losses of blood, plasma, water and/or electrolytes.
      a) Open fractures, surgery, placenta previa, abortions, lacerations and hemoptysis, etc. cause external losses of blood.
      b) Burns cause external losses of plasma, water and electrolytes.
      c) Vomiting, diarrhea, bowel fistulas, ileostomy tubes, nasogastric suction etc. cause external losses of blood, plasma, water and electrolytes.
   4) Hypovolemic shock may also be the result of internal losses of blood, plasma, water and electrolytes that are not available to the circulatory system.
      a) Closed fractures, ruptured spleen, ectopic pregnancy, abruptio placenta, and bleeding into the pleural or peritoneal cavities may cause internal losses of blood.
      b) Peritonitis, pancreatitis, crush injuries, sepsis, and surgical trauma may cause loss of water, plasma, and electrolytes into the fluid spaces between cells. These losses are known as interstitial or third space losses. In addition to the interstitial fluid space, the other major fluid spaces are the intravascular and intracellular fluid spaces.
      c) Spinal anesthesia, by blocking the sympathetic nervous system, will cause systemic arterioles and veins to dilate and, therefore, more blood will remain in them. This will decrease both venous return and stroke volume that will usually decrease blood pressure and may result in shock.
   5) When hypovolemia and/or decreased venous return are present, the heart will attempt to maintain cardiac output by increasing heart rate to compensate for the decrease in stroke volume. (Cardiac output = Stroke volume X Heart rate)
   6) Treatment of hypovolemic shock varies with the situation but basically consists of:
      a) Careful monitoring and recording of changes in blood pressure, heart rate, respiration, temperature, mental state, urine output, laboratory values and C.V.P.
      b) Treatment of the basic disease with surgery, fracture stabilization, antibiotics, etc.
      c) Replacement of intravascular volume with blood, Ringers lactate and/or 0.9% saline.
      d) Oxygen.

C) Septic shock is caused by the toxic effects of a widespread bacterial infection. The effects on the blood vessels are increased capillary permeability and vasodilatation. This results in large losses of water and electrolytes into the third space and decreased venous return.
   1) The symptoms and signs of septic shock are similar to hypovolemic shock. In addition:
      a) Fever and chills may be present.
      b) Kidney and lung failure often occur.
      c) Clotting abnormalities are frequent.

d) Large third space losses are common.

2) Common causes of septic shock are:
   a) Peritonitis.
   b) Septic abortions.
   c) Biliary and urinary tract obstructions with secondary infections.
   d) Infected indwelling urinary, endotracheal and intravenous catheters.
3) Treatment:
   a) Rapid and thorough treatment of the basic cause of the infection with antibiotics and/or surgery is imperative, as the death rate is very high even with the best of care.
   b) Meticulous attention to oxygenation, ventilation, i.v. fluid management, nutrition and urine output is absolutely necessary for survival.
   c) Metabolic acidosis is always present in septic shock. The only effective treatment is to provide a normal cellular oxygen supply by improving capillary blood flow.
   d) If the patient does not respond to optimal fluid management, an i.v. drip of dopamine, or possibly adrenaline, for maintenance of blood pressure may be of benefit.

(IV) PRE-ECLAMPSIA AND ECLAMPSIA

A) Pre-eclampsia is systemic vasoconstriction causing hypertension greater than 140/90 mmHg, proteinuria and edema after 20 weeks of pregnancy.
   1) It occurs in about 4% of pregnancies and usually resolves within 48 hours of delivery.
   2) There may be headaches, visual disturbances and epigastric pain.
   3) Mild pre-eclampsia can be treated with bed rest and oral anti-hypertensives such as labetolol or alpha methyldopa.
   4) The management of severe pre-eclampsia is to deliver the baby and placenta and treat the symptoms.
      a) The mother's condition must be stabilized so that anesthesia and surgery can be performed safely. This will involve controlling the blood pressure, ensuring adequate intravascular filling, checking that the coagulation is normal, and prevention of seizures.
      b) When seizures occur the condition is known as eclampsia.
   5) The mother's blood pressure, urine output, conscious state and tendon reflexes must be frequently checked.

Fluid resuscitation in eclampsia

A) Because of the persistent and severe vasoconstriction, pre-eclampsia causes a reduced intravascular volume. The mother will need intravenous fluid replacement.
   1) The mother may have mild to severe dehydration with a low urine output.
   2) Intravenous fluid replacement should be guided by monitoring the blood pressure, pulse and urine output with an indwelling urinary catheter.
   3) The patient should have at least 1ml/kg of urine output each hour. It is important not to give too much intravenous fluid as the patient may develop pulmonary edema due to leaky capillaries.
      a) Most patients will need 1 liter of 0.9% saline or Ringer's lactate rapidly followed by 1 liter over the next hour. If the urine output is still less than 30 mls/hr the patient may need another 500mls over half an hour until urine output is normal.
      b) If the urine output is still low, then the patient may need a diuretic such as furosemide.

Magnesium sulfate for blood pressure and seizure control in eclampsia

A) Hyperreflexia, headache, visual changes and high blood pressure all indicate that the patient may soon have a seizure.
   1) Giving an antiepileptic drug such as diazepam or phenytoin may prevent seizures.
   2) However, magnesium sulphate is the best drug.
      a) Magnesium sulphate will cause vasodilatation to lower blood pressure and depress the central nervous system to prevent seizures.

B) Magnesium sulphate is given as an intravenous bolus of 2 to 4 g over 15 minutes, then as an intravenous infusion of 1 to 3g/hr.
   1) An alternative regimen is to give 10gram of magnesium sulphate intramuscularly followed by 5gram intramuscularly every 4 hours until 24 hours post delivery.

2) If magnesium sulfate does not adequately control the hypertension, intravenous hydralazine or labetolol should be added.
   a) Hydralazine can be given as bolus injections of 5 to 10 mg every 15 minutes or as an infusion of 2 to 4mg/hour.

C) The patient must have frequent observation of their tendon reflexes, respiratory rate and heart rate. If depression of reflexes occurs, stop the infusion until the reflexes return.

D) Magnesium sulfate will also increase the patient's sensitivity to depolarizing and non-depolarizing muscle relaxants. The anesthetist may need to reduce the dose of muscle relaxants by about 30% of the predicted dose if the patient needs a general anesthetic.

Choice of anesthetic in eclampsia

A) The choice of anesthetic technique for cesarean section will depend on the health of the mother, health of the fetus and the technical ability of the anesthetist.

B) General anesthesia may avoid the hypotension that can occur with spinal anesthesia and is safer when thrombocytopenia is present.
   1) Pre-eclamptic patients may be very difficult to intubate because of severe airway edema. The anesthetist must assess the pre-eclamptic patient's airway with extreme care and always be prepared for a difficult or impossible intubation.
   2) The pre-eclamptic patient will probably have exaggerated cardiovascular responses of hypertension and tachycardia to Ketamine, intubation and extubation.

C) Spinal anesthesia
   1) Pre-eclamptic or eclamptic patients <u>rarely</u> have coagulation abnormalities.
      a) The platelet count should ideally be above 100,000.
      b) If a platelet count is not available a good estimate of clotting ability can be made by observing needle puncture sites. They should not bleed excessively.
      c) A spinal needle puncture in a patient with clotting abnormalities may cause a hematoma in the epidural space resulting in permanent paralysis.
   2) The decreased peripheral resistance from spinal anesthesia should not cause a severe drop in blood pressure if the patient's blood pressure is controlled and they have had adequate fluid resuscitation.

Post delivery care in eclampsia

A) The anesthetist must be aware that the patient remains at risk from pre-eclampsia for up to 48 hours after delivery. More than 50% of convulsions and pulmonary complications occur in the postpartum period.

(V) HEART FAILURE

A) In right heart failure, there is a decreased right ventricular output because the ventricle is unable to pump the blood coming to it. This results in increased systemic venous and systemic capillary pressures. Central venous pressure (C.V.P.) will be increased.
   1) Symptoms:
      a) Fatigue and dyspnea
      b) Awareness of fullness of neck or abdomen
   2) Signs:
      a) Distention and large pulsation in the external jugular vein
      b) Enlarged and tender liver
      c) Edema of legs and/or sacrum
      d) Ascites
      e) Tachycardia
   3) Disease states causing right ventricular failure include:
      a) Left ventricular failure causing high pulmonary venous, pulmonary capillary, and pulmonary artery pressures.
      b) Lung disease, such as chronic bronchitis or TB, which will cause high pulmonary artery pressure by destroying much of the lung and its vessels.
      c) Pulmonary emboli causing high pulmonary artery pressure.
      d) Congenital or acquired heart disease
      e) Administration of too many i.v. fluids
   4) Treatment of right ventricular failure:
      a) Rest
      b)Oxygen
      c) Improve myocardial contractility with digitalis
      d) Decrease blood volume with sodium restriction, diuretics or phlebotomy
      e) Control of arrhythmias

B) In left heart failure, there is a decreased left ventricular output because the ventricle is unable to pump the blood coming to it. This results in increased pulmonary venous and pulmonary capillary pressures that may result in pulmonary edema.
   1) Symptoms:
      a) Dyspnea on exertion
      b) Fatigue
      c) Dyspnea while sleeping at night
   2) Signs:
      a) Sitting position is favored
      b) Rales
      c) Cough with blood tinged sputum
      d) Pleural effusion
      e) Tachycardia
      f) Cyanosis
   3) Disease states causing left ventricular failure include:
      a) Hypertension
      b) Coronary artery disease
      c) Aortic and/or mitral valve disease
      d) Administration of too many i.v. fluids
   4) Treatment:
      a) Similar to that for right ventricular failure.
      b) If a patient has acute pulmonary edema with rapid progression of the above signs and symptoms, treatment with intravenous diuretics and digitalis will be necessary.
      c) Hypertension must be controlled.

(V) BASICS OF INTRAVENOUS FLUID MANAGEMENT

A) Normal adult body fluid spaces.
- 1) 60% of the body's total weight is fluid. (42 liters in a 70 kg person)
- 2) Intracellular fluid space:
  - a) 40% of the body's weight is fluid in the cells. (28 liters in 70 kg)
  - b) The cell membranes actively keep high levels of salt from entering the cells.
- 3) Extracellular fluid space
  - a) 20% of the body's weight is fluid outside of the cells. (14 liters in 70 kg)
    - i) 5% of the body's weight is fluid in the plasma. (3.5 liters in 70 kg)
    - ii) 15% of the body's weight is fluid in the spaces between cells. This space is known as the interstitial space or the third space. (10.5 liters in 70 kg)
  - b) Water and salt pass freely between the plasma and the third space.

B) Normal blood volumes:
- 1) A normal adult has a blood volume of about 65 ml/kg body weight.
- 2) A normal newborn has about 85 ml/kg; an infant 80 ml/kg; a child 75 ml/kg.

C) Clinical determination of adequate blood volume.
- 1) Knowledge of the patient's history and physical status before the illness is very useful.
- 2) Careful monitoring of the patient's mental state, blood pressure, heart rate, temperature, breath sounds and urine output (by using a bladder catheter) is imperative.
- 3) If there is an adequate urine output without using diuretics three things are known:
  - a) There is enough blood pressure and blood flow to the kidney.
  - b) There is enough fluid of the correct composition in the plasma.
  - c) The kidneys are functioning.
- 4) A good urine output (0.5-1 ml/kg/hour) usually means that there is adequate blood flow to all other organs.

D) Distribution of fluids given intravenously:
- 1) The amount of protein and sodium that an intravenous fluid contains determines in which of the body's fluid spaces it will remain.
- 2) Blood, which has the normal amounts of protein and sodium in plasma, remains in the intravascular space and increases blood volume and venous return.
- 3) 0.9% saline and Ringers lactate, which have no protein but have normal amounts of sodium, disperse to the intravascular and interstitial spaces. To increase blood volume and venous return as much as blood does, about three times as much 0.9% saline or Ringers lactate must be given.
- 4) 5% dextrose and water, which has no protein and no sodium, disperses to all the body fluid spaces (cells, interstitial, plasma) and increases blood volume and venous return very little. It is useless for the treatment of hypovolemia or in replacement therapy.

E) Replacement intravenous fluid therapy:
- 1) Third space losses from burns, bowel obstructions, peritonitis, diarrhea etc. should be treated before surgery to prevent severe hypotension or cardiac arrest with induction of anesthesia.
  - a) Use either 0.9% saline or Ringers Lactate along with maintenance fluids.
  - b) Fluid replacement should be guided by monitoring the urine output with an indwelling urinary catheter.
  - c) Urine output should be 0.5-1.0 ml/Kg body weight/hour.
  - d) The patient's urine output, mental status, lung sounds, blood pressure, and pulse must be carefully observed and recorded.
- 2) General guidelines for using 0.9% saline or Ringers lactate for third space and other losses during surgery:
  - a) Minimal tissue trauma--4 ml/kg/h. Hernia repair, etc.
  - b) Moderate trauma--6 ml/kg/h. Elective intra-abdominal surgery.
  - c) Severe trauma--8 ml/kg/h. Higher rates may be necessary to maintain urine output.
- 3) Blood should only be given for specific indications.
  - a) The appropriate hemoglobin level depends on the patient's disease, physical condition, status of hydration and previous and anticipated blood losses.
  - b) Many patients who lose blood only need 0.9% saline or Ringers lactate.

F) Maintenance intravenous fluid therapy for NPO pediatric and adult patients is 5% dextrose in 0.45% saline. (Maintenance in the pre and post op period NOT intraop.)
1) Alternating bottles of 5% dextrose and 0.9% saline is appropriate for adults.
2) Some anesthetists use 5% dextrose in 0.25 % saline as the intravenous maintenance fluid for children who weigh less than 10 kilograms.
3) Formulas for 24-hour maintenance requirements:

First 10 kg	4 ml/kg/hour	100 ml/kg/24 hours
Second 10 kg	2 ml/kg/hour	50 ml/kg/24 hours
Remainder-including adults	1 ml/kg/hour	20 ml/kg/24 hours

4) Give preoperative maintenance fluids if the patient will be NPO for longer than 12 hours.

G) Pediatric intravenous replacement fluid therapies for third space losses, surgical trauma and blood losses are the same as the adult.
1) Because the blood volumes and fluid spaces are so small, it is easy to give too much or too little intravenous fluid or blood.
2) Hyperglycemia is a normal response to the stress of surgery. High blood sugar levels are common during pediatric and adult surgery.
a) Intravenous fluids containing 5% or 10% dextrose are usually not needed during surgery.
b) If possible, during a long surgery, check the blood sugar levels in pediatric patients.
c) Newborns, infants and some children may need 5% or10% dextrose before and after surgery.

(VII) FLUID MANAGEMENT OF PERIOPERATIVE PATIENTS

A) **A previously healthy 50-year-old man** has had a bowel obstruction for 10 days. He has been vomiting and has all the signs of severe dehydration. He is afebrile and weighs 60 kg. He needs surgical release of the obstruction. Hb 13gms%.
1) This is not an acute emergency. Surgery is safer after the patient is rehydrated.
2) Place an i.v. catheter and a bladder catheter.
3) Monitor blood pressure, pulse, mental status, lung sounds and urine output carefully.
4) Give 0.9% saline or Ringers Lactate until there is adequate urine output. Warm the fluids if possible. Do not use diuretics.
5) Determine the plasma electrolytes if possible. If not, use potassium cautiously only after the patient begins to urinate.
6) Surgery can proceed when the patient is stable and urinating.
7) Do not forget to give maintenance fluids in addition to replacement fluids before, during and after surgery.
8) Careful fluid and nutritional management after surgery are critical for the patient's survival.

B) **A healthy 3-year-old girl** is scheduled for elective hernia repair.
1) Stop all food (including milk) six hours before surgery.
2) Allow only water until two hours before surgery.
3) Allow her to drink water as soon as she wishes after surgery.

C) **A 26-year-old woman** in septic shock from an abortion needs an immediate hysterectomy.
1) Delay the surgery only long enough to insert two big i.v. catheters and a bladder catheter.
2) Think carefully about the anesthetic drugs.
3) These patients are severely acidotic. The only effective treatments are surgery, antibiotics, and adequate blood volume and flow to the cells.
4) Huge volumes of 0.9% saline and blood may be necessary. Warm them if possible.
5) Do not use diuretics. Do not worry about maintenance fluids or potassium.
6) To prevent death from kidney failure the patient must urinate during surgery or soon after. Measure the urine every hour.

D) **A 65-year-old man** has been given enemas for two days before an elective colon resection. He weighs 50 kg.
1) He needs maintenance and replacement fluids either orally or i.v. during the two days.
2) He should come to surgery with a normal urine output and without a fluid deficit.
3) He needs a bladder catheter during surgery.
4) Plan on about 6-8 ml/kg/hour or more of 0.9% saline during surgery.

E) **A 5-year-old girl** needs surgery for a fractured humerus. She is stable until 30 minutes into the operation when her blood pressure rapidly declines and her pulse increases. These changes persist after deep anesthesia and hypoxia are ruled out.
   1) Trauma patients often have injuries other than the obvious one.
   2) She should have been carefully examined for other injuries before surgery.
   3) Even with careful examination, a ruptured spleen can be missed.
   4) Use 0.9% saline until blood can be obtained.
   5) Place a bladder catheter and measure the urine output every hour.

F) **A 17-year-old woman** who had been in labor for 18 hours needs a C-section. She is healthy and has had no known complications of her pregnancy. Except for a few sips of water, she has been NPO. She is fully alert with a blood pressure of 90/60 and a pulse of 100/minute. An intravenous solution of 5% dextrose is started. She is given a spinal anesthesia, becomes hypotensive with a very rapid heart rate and has a baby with an Apgar of 1. The mother dies of hypotension in spite of being given large amounts of 5% dextrose.
   1) She died because of a reduction in her stroke volume -- she was dehydrated, the pregnant uterus may have obstructed her inferior vena cava, she lost blood during the surgery and spinal anesthesia blocked the sympathetic nervous system.
   2) She and the baby would have done well if the anesthetist had given 1-2 liters of 0.9% saline or Ringers lactate before giving the anesthesia, had continued the infusion during the surgery, had displaced the uterus to the left and had used ephedrine if hypotension had occurred.
   3) 5% dextrose is useless when trying to increase stroke volume.

Usual effects on the cardiovascular and respiratory systems

	Heart Rate	Stroke Volume	Peripheral Resistance	Blood Pressure	Minute Ventilation	Functional Residual Capacity
Atropine	↑	-	-	-	-	-
Blood	-	↑	-	↑	-	-
Curare	-	-	↓	↓	↓	-
Diazepam	-	↓	↓	↓	↓	-
Diuretics	-	↓	-	↓	-	-
Dopamine	↑	↑	dose	↑	-	-
Ephedrine	↑	↑	↑	↑	↑	-
Epinephrine	↑	↑	↑	↑	↑	-
Ether	↑	↑	↑	↑	↑	-
↑ Ether dose	↓	↓	↓	↓	↓	-
Fentanyl	↓	-	-	↓	↓	-
Flaxedil	↑	-	-	-	↓	-
Halothane	- ↓	↓	- or↓	↓	↓	↓
I.V. 0.9% Saline	-	↑	-	↑	-	-
I.V. 0.5% D/W	-	-	-	-	-	-
Ketamine	↑	↑	↑	↑	-	-
↑Ketamine dose	↓	↓	↓	↓	↓	-
Lidocaine	-	-	-	-	-	-
Morphine	-	-	-	-	↓	-
Neostigmine	↓	-	-	-	-	-
Neosynephrine	-	-	↑	↑	-	-
Pethidine	↓	-	-	↓	↓	-
Succinylcholine	-	-	-	-	↓	-
Thiopentone	-	↓	↓	↓	↓	-

Eclampsia	↑	↓	↑	↑	-	↓
↑CO_2 in blood	↑	↑	↑	↑	↑	-
↓CO_2 in blood	-	-	-	-	↓	-
Heart failure	↑	↓	-	varies	-	↓
One-year-old	↑	fixed	-	-	↑	↓
Pain	↑	↑	↑	↑	↑	-
Parasym. N.S.	↓	-	↓	↓	-	-
Pos. Pres. Vent.	-	↓	-	↓	-	-
Shock: hypovol.	↑	↓	↑	↓	↑	-
Shock: septic	↑	↓	↓	↓	↑	-
Spinal anesthes.	↓	↓	↓	↓	-	↓
High spinal anes	↓	↓	↓	↓	↓	↓
Sup. Hypoten. syndrome	↑	↓	-	↓	-	↓
Surgery trauma	↑	↓	↑	↓	-	varies
Sympath. N.S.	↑	↑	↑	↑	↑	-
Term pregnancy	↑	-	↓	↓	↑	↓
	Heart Rate	Stroke Volume	Peripheral Resistance	Blood Pressure	Minute Ventilation	Functional Residual Capacity

RESPIRATORY CONSIDERATIONS

(I) GENERAL CONSIDERATIONS

A) External respiration is the absorption of oxygen (O_2) through the lungs into the blood and the elimination of carbon dioxide (CO_2) from the blood through the lungs.

B) Internal respiration is the exchange of O_2 and CO_2 between the blood and the billions of cells in the body. O_2 is taken up by the cells and CO_2 is given off by them into the blood.
1) While resting, the cells in the average adult use 250 ml of oxygen and produce 300 ml of carbon dioxide every minute.
2) When necessary, cells use 20 times as much O_2 and produce 20 times as much CO_2.

C) Oxygen and food are used by the body's cells to produce:
1) Energy; used by the cells for movement, heat and cell function.
2) Carbon dioxide; dissolves into the blood and is exhaled by the lungs.
3) Water; passes into the blood and is made into urine by the kidneys.

D) The metabolism of the cells is inefficient when low levels of O_2 are available to the cells.
1) The inefficiency decreases the production of energy, carbon dioxide and water.
2) The inefficiency results in high levels of metabolic acids.
3) The low levels of energy and increased blood acid levels adversely effect all the cells.
a) The brain, heart, kidneys, muscles and liver function poorly.
4) The only effective treatment is to supply the cells with normal amounts of oxygen.
a) Depending on the situation, a combination of airway control, ventilation, O_2, surgery, antibiotics, intravenous fluids, blood, or lung and heart drugs will be necessary.
b) Sodium bicarbonate and steroids are not helpful because they do not treat the basic problem.

E) The percentages of the gasses in air: (% of a gas in air = gas pressure/760 x 100)

Nitrogen – 78%	O_2 - 21%;	Argon -0.9%	CO_2 - 0.03%.

F) The total pressure of the gases in air at sea level is 760 mmHg.

Nitrogen – 592 mmHg	O_2 - 159 mmHg	Argon – 7 mmHg	CO_2 - 0.3 mmHg

G) Normal pressures (mmHg at sea level) of oxygen and carbon dioxide in air and blood:

	Oxygen	Carbon dioxide
Inhaled gas (air)	159	0.3
Exhaled gas	120	27
Arterial blood	100	40
Venous blood	40	47

(II) BASIC ANATOMY

A) Nasal cavities and turbinates. Air is warmed, humidified and filtered here.
B) Pharynx. Food and air share this space.
C) Larynx. Vocal cords are here. The vocal cords are the narrowest part of the adult airway.
D) Trachea. All the cartilages are semicircular except for the cricoid cartilage, which is circular. The cricoid cartilage is the narrowest part of the pediatric airway.
E) Bronchi. Larger air passages. The right mainstem bronchus is more vertical than the left.
F) Bronchioles. There are thousands of these small air passages. Well-developed muscles in their walls can constrict and cause bronchospasm and wheezing.
G) Terminal tubes. There are about a million.

H) Pulmonary alveoli.
1) There are about 300 million of these microscopic air sacs.
2) O_2 and CO_2 are exchanged between the air and the blood across the very thin alveolar-capillary membrane that separates the alveoli from the pulmonary capillaries.
3) The thin membrane is about 40 times the surface area of the body.
4) The alveoli can collapse or fill up with blood, fluid or pus. The collapse will prevent oxygen and carbon dioxide from being exchanged easily across the membrane.

I) Pleura. A very thin and elastic membrane:
1) The visceral pleura covers the outside of the lungs.
2) The parietal pleura lines the inside of the thorax.
3) The two pleurae slide easily on one another.
4) The pleural space is the potential space between the two pleurae.
5) Air, blood, or pus can easily separate the two pleurae and cause pneumothorax, hemothorax or pyothorax.

(III) RESPIRATORY TERMS

A) Respiratory rate. The number of breaths in one minute.
1) A normal resting adult breathes about 12-15 times per minute.
2) Infants and children breathe much faster.

B) Tidal volume. The volume of gas exhaled in one breath.
1) A normal resting adult exhales about 400-500 ml. of gas per breath.

C) Minute ventilation. The volume of gas exhaled in one minute.
1) The volume of gas breathed out/minute = respiratory rate X tidal volume.
2) Minute ventilation is about 5-6 liters in the normal adult.

D) Vital capacity. The maximum volume of gas that can be forcibly breathed out after a maximum inhalation.
1) Adults have a vital capacity of about 4-6 liters.

E) Functional residual capacity or FRC. The volume of gas remaining in the lungs after a normal resting exhalation.
1) The normal adult FRC is about 1500-2500 ml.
2) The oxygen in the FRC allows pulmonary capillary blood to be oxygenated between inspirations.
3) Pediatric patients, pregnant women at term, and patients with distended abdomens have relatively small FRCs. They quickly become hypoxic if not well ventilated and oxygenated.
4) Depending on the level, spinal anesthesia decreases FRC.
5) General anesthesia decreases FRC about 20%.
6) During laryngospasm or apnea, the FRC will rapidly have its oxygen removed by the blood flowing through the pulmonary capillaries and the patient will become hypoxic.
a) It is very important that the FRC have as much oxygen as possible when a patient is intubated or extubated because hypoxia can easily occur at these times.

(IV) INHALATION

A) For gas to flow in to the lungs a negative pressure must be created in them by the muscles of respiration, which increase the size of the thoracic cavity:
1) The ribs are moved up and out by the intercostal muscles.
2) The neck muscles lift the upper ribs when inhalation is difficult.
3) The diaphragm contracts and becomes flat.
a) The diaphragm is the primary muscle of respiration.
b) Bilateral phrenic nerves control the diaphragm and arise at C-3,4,5.

(V) EXHALATION

A) For gas to flow out of the lungs a positive pressure must be created in them. This is done by:
   1) Recoil of the elastic tissues in the lung and chest wall.
   2) Relaxation of the diaphragm and intercostal muscles. The diaphragm moves up.
   3) Forceful contraction of the abdominal muscles during exercise, asthma, chronic bronchitis or cough.

(VI) COUGH

A) A cough is produced by:
   1) Taking a deep breath which flattens the diaphragm.
   2) Closing the vocal cords.
   3) Suddenly and forcibly contracting the abdominal muscles which push the abdominal contents against the flattened diaphragm.
   4) Partially opening the vocal cords which allows the gas to escape with a high velocity.

B) Most patients have an ineffective cough with:
   1) Post-operative or post-traumatic pain.
   2) Spinal anesthesia.
   3) Abdominal distention.
   4) CNS depression from general anesthesia, trauma or drugs.
   5) Pleural effusion, chronic bronchitis, or chronic obstructive lung disease.

(VII) SECRETIONS

A) Large amounts of secretions are normally made by the cells that line the trachea, bronchi and bronchioles.
B) The cells have microscopic, moving, external hair-like projections called cilia.
C) The cilia move the secretions to the larynx where they are coughed or swallowed.
D) The purpose of these secretions is to trap organisms and foreign material in the airway, thereby preventing infection.
E) Anesthetic drugs, opiates, bronchitis and tobacco smoke depress activity of the cilia.
F) Atropine decreases the amount of secretions.

(VIII) RESPIRATORY STIMULATION

A) The main control of how much we breathe depends upon how much carbon dioxide (CO_2) we have in our arterial blood. Varying levels of arterial CO_2 in the brain stimulates or depresses the phrenic nerve and diaphragm to increase or decrease the minute ventilation.
   1) Arterial CO_2 is regulated within a very narrow range.
   2) As a result of normal metabolism, we are always producing CO_2 in our cells. The CO_2 diffuses into the venous blood. The lungs remove most of the CO_2. We unconsciously adjust our minute ventilation to keep arterial CO_2 levels normal.
   3) Either too high or too low levels of arterial CO_2 may seriously interfere with cellular function and disturb the cardiovascular, pulmonary, renal and central nervous systems.

B) Decreased minute ventilation increases the amount of arterial CO_2 (hypoventilation).
   1) In the normal person, an increase in arterial CO_2 causes the brain to send more signals to the respiratory muscles which stimulates them to contract more often. Minute ventilation increases. Arterial CO_2 will then decrease because more CO_2 is removed from the lungs.
   2) Symptoms and signs of high arterial CO_2 levels include headache, hypertension, tachycardia, arrhythmia, tachypnea, deep respirations, and skin flushing.
   3) In spite of a conscious patient's attempts to increase his/her minute ventilation, high arterial CO_2 levels may persist in asthma, chronic lung disease and airway obstruction.
   4) Higher than normal arterial CO_2 levels result in an increased intracranial pressure.
   5) Anesthetic drugs, opiates and sedatives depress the brain's response to CO_2 and allow arterial CO_2 levels to rise.
      a) If the patient is breathing spontaneously:

i) As the anesthetic, opiate or sedative dose increases, the more the minute ventilation will be depressed and the more the arterial CO_2 will increase.
ii) Halothane, thiopentone, diazepam, morphine, pethidine, pentazocine etc. will depress the brain's response to CO_2 and allow increases in the arterial CO_2.
iii) Ketamine and ether given in appropriate doses will not result in high arterial CO_2 levels. However, high doses of these drugs will result in high levels of CO_2.

6) Higher than desired arterial CO_2 levels are managed by ventilating the patient.

C) Increased minute ventilation decreases the amount of arterial CO_2 (hyperventilation).
1) A decrease of arterial CO_2 causes the brain to send fewer signals to the respiratory muscles which causes them to contract less often. Minute ventilation decreases. Arterial CO_2 will increase because less CO_2 is removed from the lungs.
2) Six reasons that patients do not breathe well at the end of a general anesthetic are:
a) Hyperventilation.
b) Anesthetic overdose.
c) Residual muscle relaxants.
d) Hypothermia.
e) Electrolyte imbalance.
f) Acid-base problems

D) A low level of oxygen in the blood is less stimulating to respiration than high levels of carbon dioxide. Low levels of oxygen in the blood occur with:
1) Airway obstruction.
2) Hypoventilation.
3) Anesthesia. For a safe margin, do not use less than 40% oxygen during an anesthetic.
4) Fluid filled or collapsed alveoli that prevent oxygen from reaching pulmonary capillaries.

E) Metabolic acidosis is a big stimulus to respiration.

(IX) CO_2 and ANESTHETIC CIRCUITS

A) All anesthetic circuits are designed to deliver oxygen and anesthetic gases. They also eliminate the patients exhaled CO_2. Except for the circle system, an anesthetic circuit that allows the patient to rebreathe the gases in the circuit will cause an increase in arterial CO_2.
1) Make sure that the soda lime in the circle system is working -- the soda lime absorbs the patient's exhaled CO_2.
2) Make sure that the non-rebreathing valve on the EMO circuit does not stick open and that the magnet on the bellows is correctly placed for the valve being used. The magnet is on for the Ambu-E valve.
3) The Magill circuit requires at least a 6 liter flow and the bag to be filled when using spontaneous ventilation.
4) The Magill circuit should not be used for controlled ventilation unless very large flows are used and the circuit is purged often.
5) The Ayres-T system must have a minimum flow of gas that is equal to three times the patient's minute ventilation to prevent rebreathing of CO_2. Minute ventilation in children is approximately 200 ml/kg body weight. Use a minimum of 3 liters flow.

(X) RESPIRATORY SYMPTOMS

A) Dyspnea is shortness of breath, difficulty with breathing, or pain while breathing. It may be caused by:
1) Exercise.
2) Airway obstruction.
3) Bronchiole constriction. During asthma, the muscles in the walls of the bronchioles constrict and cause wheezing. However, wheezing is not always asthma!
4) Heart failure that results in a decreased amount of blood and oxygen being delivered to the brain. The patient will attempt to obtain more oxygen by increasing his/her respiratory rate and depth.
5) A rapid and large blood loss. This will decrease the amount of blood and oxygen going to the brain and stimulate respiration.
6) Diabetic acidosis and metabolic acidosis. Acidosis causes the same response in the brain as increased carbon dioxide.

7) Brain hemorrhage.
8) Hysteria.
9) Chest wall injury causing pain.
10) Pneumothorax, hemothorax, or pyothorax. These will compress the lung, collapse alveoli and result in decreased oxygen and possibly increased arterial CO_2 levels.

B) Excessive or changed sputum and/or cough
1) Hemoptysis or bloody sputum, can be caused by tumors, pulmonary infarction, trauma, left heart failure, bronchitis, pneumonia, or tuberculosis. Enough blood can be lost for the patient to be in hypovolemic shock.
2) Green and/or thick sputum is seen with bacterial pneumonia, bronchitis, or sinus infections.

(XI) RESPIRATORY SIGNS

A) Fast and/or deep respirations may be signs of exercise, anesthesia, pain, fear, hysteria, brain damage, acidosis, decreased blood oxygen, or increased blood carbon dioxide.

B) Slow and/or shallow respirations may be signs of sleep, anesthesia, drug overdose, brain damage, or respiratory muscle weakness.

C) Paleness and cyanosis are unreliable signs but may indicate too little oxygen in the blood.

D) Forceful exhalations and/or wheezing are seen in asthma, presence of foreign bodies in the airway, heart failure, partial airway obstruction, and chronic bronchitis.

E) Rales or creps are continuous sounds heard when collapsed alveoli are opening.

F) Rhonchi are interrupted sounds heard when secretions are in bronchioles and bronchi.

G) Inadequate respirations can be recognized by absent or decreased:
1) Chest movements.
2) Abdominal movements.
3) Air movement from the nose and/or mouth.
4) Breath sounds in the lungs or trachea.

(XII) AIRWAY OBSTRUCTION

A) An obstructed airway can be recognized by:
1) Retractions in the chest and/or lower neck area.
2) Rocking motion of the abdomen.
3) Noises from the mouth or nose.
4) The patient desperately trying to change position, grabbing his/her throat, etc.
5) Cyanosis or paleness (often poor signs).
6) If it walks like a duck, talks like a duck and looks like a duck -- it is a duck!!
a) If the patient looks like he has an airway obstruction -- he does!!

B) An airway obstruction in an intubated patient can be recognized by:
1) Your inability to ventilate the intubated patient with an Ambu bag or by mouth to tube.
2) The ventilator alarming and/or requiring high pressures to ventilate the patient.
3) A complete obstruction in an intubated patient must be treated by immediately removing the endotracheal tube or tracheostomy tube and ventilating the patient with a bag and mask.
4) On rare occasions, an endotracheal tube or tracheostomy tube may be obstructed by an overinflated cuff.
a) Deflate the cuff before removing the endotracheal tube or tracheostomy tube.

C) Be **SURE** to watch the patient's chest and abdomen. If you have any doubts about the patient's breathing..... **VENTILATE THE PATIENT!!!**

(XIII) THERAPY FOR RESPIRATORY DISEASES

A) Oxygen.
1) An oxygen mask delivers about 50% oxygen with an eight liter/minute flow.
2) An Ambu-bag or an EMO machine (Epstein, Macintosh, Oxford), each with a reservoir hose, will deliver up to 60-80% oxygen depending upon the oxygen flow into the system and the patient's minute ventilation.
   a) The addition of just one liter/minute of oxygen to either of these systems will increase inspired oxygen concentration to approximately 30% from the 21% present in air.
3) An Ayres-T system will deliver up to 100% oxygen.
4) A Boyle's machine with a circle system and carbon dioxide absorber will deliver 100% oxygen with low oxygen flows.
5) If the patient's oral and nasal airways are clear, a nasal catheter or prongs will deliver approximately 40% oxygen at 3 liters/minute oxygen flow. Little benefit is derived from increasing the flow over 3 liters/minute.

B) Ventilation.
1) The Ambu-bag, mouth-to-mouth technique, EMO machine, Ayres-T system, and Boyle's machine will adequately ventilate a patient whose respirations are poor, absent, or obstructed if the person doing the ventilating knows what he/she is doing.

C) Position.
1) Those patients whose cough reflexes have been depressed by drugs (anesthesia, opiates, etc.) can manage their secretions much better in the lateral position.
2) All patients recovering from general anesthesia should be recovered on their sides to help prevent aspiration and airway obstruction.

D) Coughing.
1) Encouragement and forcing the patient to cough are the most effective ways to clear secretions from the lungs.
2) If a patient has pain from surgery or other causes that make it painful to cough, opiates should be used to decrease the pain of coughing.
3) Increased physical activity and postural drainage are very helpful.

E) Suction.
1) Suction of the trachea through the nose, endotracheal tube, or tracheostomy may be necessary in those patients who are unable to cough effectively. 2 or 3 ml of saline can be placed in the trachea to loosen secretions.
2) Suction of the trachea for longer than 10 seconds may result in the patient becoming hypoxic from collapse of alveoli.
3) Sterile suction catheters should be used each time the patient's trachea is suctioned to prevent the introduction of infection into the airway.

F) Tracheostomy or endotracheal intubation may be performed when the patient:
1) Is unable to cough his/her own secretions as in diphtheria or pneumonia.
2) Has an upper airway obstruction.
3) Is too weak to breathe on his own and needs a ventilator to breathe for him/her as in cardiac arrest, drug overdose, polio, or after some major surgeries.

G) Humidity.
1) If a patient has a tracheostomy or an endotracheal tube for a prolonged time, the inspired gas will not be humidified by the nose and this will result in dried and thick secretions.
2) The secretions may partially or completely obstruct an endotracheal tube or tracheostomy.
3) Bronchoscopy may be necessary to remove secretions from the patient's airway.

H) Chest tubes.
1) One end of the chest tube is placed in the pleural cavity to remove air, blood or pus after surgery, trauma or infection.

2) The other end is placed in sterile water in a bottle that is open to air and is 2-3 feet below the patient. This is to allow the patient to push or drain air, blood or pus out of the pleural cavity but to prevent air from being sucked in.
3) The bottle must remain below the patient's chest or the tube must be temporarily clamped if it is necessary to raise the bottle above the chest. This is to prevent the fluid in the bottle from flowing into the pleural cavity.

I) Drugs used in respiratory diseases:
1) Antibiotics.
2) Bronchodilators for treatment of wheezing.
a) Aminophylline, epinephrine, ephedrine, isoproterenol, atropine, and steroids have serious side effects so use them cautiously!
3) Opiates to relieve pain so that the patient can cough.
4) Oral cough suppressants.
a) All opiates will suppress respiration and cough. Codeine is usually used.
b) If sputum is present, cough should not be suppressed.
5) Oxygen!

PHARMACOLOGY CONSIDERATIONS

VASOPRESSORS

A) Vasopressors are short acting drugs that temporarily raise blood pressure.
   1) They are similar in action to the body's own epinephrine and norepinephrine.
   2) They are rarely needed during general anesthesia.
   3) They may occasionally cause cardiac arrhythmias.
   4) They have one or more actions:
      a) Increased heart rate
      b) Increased strength of the heart muscle
      c) Constriction of the muscles of the systemic arterioles and veins
   5) Ephedrine
      a) Has all three actions and is used for hypotension during spinal anesthesia.
      b) Is the drug of choice during C-section with spinal anesthesia because ephedrine does not decrease placental blood flow.
   6) Neosynephrine and Methoxamine
      a) Only vasoconstriction occurs. Watch the heart rate carefully because it may become very slow.
      b) Placental blood flow decreases.
   7) Adrenaline (Epinephrine)
      a) Has all three actions.
      b) Excellent bronchodilator.
      c) Very potent and relatively short acting. Best used when diluted.
      d) Decreases uterine blood flow.
   8) Dopamine
      a) Low doses may increase renal blood flow and may increase urine output.
      b) Higher doses increase cardiac output and may increase blood pressure.
      c) Much higher doses increase peripheral resistance and decrease renal blood flow.
      d) Decreases placental blood flow.

   9) Atropine
      a) Atropine is not a true vasopressor, but, by blocking the action of acetylcholine in the heart, it does allow increased activity of epinephrine and norepinephrine.
      b) Can be given i.v., i.m., sublingual, or diluted with saline into the endotracheal tube.
      c) Does not affect the neuromuscular junction.

KETAMINE

A) Ketamine has hypnotic (sleep producing), analgesic (pain relief) and amnesic (short term memory loss) effects. It also causes a trance like anesthesia (dissociative anesthesia).
   1) The patient's eyes may remain open and there may be movement of their limbs but the patient will not respond to pain.
   2) Other effects of Ketamine include bronchodilatation, cardiovascular stimulation, minimal respiratory depression and some preservation of airway reflexes.
   3) Disadvantages of Ketamine are increased skeletal muscle tone, increased salivation, increased intracranial and intraocular pressures, bad dreams, tachycardia and hypertension.

Ketamine Dosage

A) For surgical anesthesia, Ketamine may be given intravenously or sublingually (1 to 2mg/kg), intra-muscularly (5 to 10mg/kg) or as an intravenous infusion (1mg/min).
   1) Initial Ketamine doses should be decreased in the very ill and hypovolemic patients.
   2) To decrease salivation patients may need atropine (10 to 20micrograms/kg). Intramuscular atropine will not increase heart rate as much as intravenous atropine.
   3) Intravenous or sublingual Ketamine causes anesthesia in 2 to 3 minutes that lasts for 10 to 20 minutes.

4) Intramuscular Ketamine causes anesthesia in 3 to 5 minutes and provides 15 to 30 minutes of surgical anesthesia. Repeated doses of Ketamine (1/4th the intravenous dose or 1/2 of the intramuscular dose) can be given to prolong the anesthesia.
5) Ketamine may be used as premedication orally (5mg/Kg) or intramuscularly (2 to 4 mg/kg).
6) For pain control, 0.2 to 0.5mg/kg intramuscular doses of Ketamine can be used in selective post-operative and laboring patients.

B) Adults may require premedication with diazepam to reduce the incidence of bad dreams and "emergence delirium". They can be given diazepam 0.15mg/kg orally one hour pre-operatively or intravenous diazepam 0.1mg/kg. Unpleasant dreams may last for 24 hours.

Physiologic Effects of Ketamine

A) Ketamine usually maintains airway muscle tone and causes only a mild decrease in respiratory rate and tidal volume.
1) Patients usually will breathe adequately without assistance from the anesthetist.
a) Apnea can occur if ketamine is given rapidly.
b) Ketamine does not guarantee an unobstructed airway or protection from aspiration.

B) Ketamine stimulates the sympathetic nervous system causing a release of adrenaline and nor-adrenaline. This release increases the heart rate, peripheral resistance and blood pressure.
1) Ketamine is a good choice of induction agent for the hypovolemic patient.
2) Ketamine should be used with extreme caution in patients with severe hypertension, ischemic heart disease or pre-eclampsia.
3) Severely ill patients may have depleted stores of adrenaline and nor-adrenaline. Normal doses of Ketamine may result in severe decreases in blood pressure in these patients.

C) Ketamine increases intracranial and intraocular pressures and is not a good choice of anesthesia for neurosurgery or ophthalmic surgery.

DIAZEPAM

A) Diazepam is one of many drugs called benzodiazepines.
1) Diazepam causes sedation, amnesia, reduced anxiety and is an anticonvulsant.
2) The anticonvulsant dose is 0.25mg/kg intravenously. Diazepam is used for eclamptic seizures but magnesium sulphate is the preferred agent.
3) Diazepam is not an analgesic and must not be given to quieten a patient postoperatively who is in pain. It must be used with great care for patients who are agitated after surgery. The anesthetist must exclude other causes of agitation, especially hypoxia.

B) Diazepam causes dose dependent respiratory depression with reduction in tidal volume and respiratory rate.
1) Diazepam is very rarely required postoperatively unless it is used to treat seizures.

C) Diazepam causes dose related decreases in stroke volume, heart rate and peripheral resistance especially when used with inhalation agents or opioids.

D) Diazepam can be used for induction of anesthesia (0.1-0.15 mg/kg) but it causes pain on intravenous injection and may cause thrombophlebitis.
1) It is rapidly absorbed orally with peak concentrations in adults within 1 hour (premedication 0.1 to 0.2mg/kg).
2) Intra-muscular injections are very painful and absorption may be unpredictable so they should be avoided.

SKELETAL MUSCLE PARALYZING DRUGS

SUCCINYLCHOLINE

A) Succinylcholine, known as a depolarizing muscle relaxant, quickly produces total body skeletal muscle relaxation and stops the patient's breathing. It works by changing the electrical charges at the neuromuscular junction and is rapidly metabolized by plasma cholinesterase. This accounts for its short duration of action.
1) The muscle relaxation allows easier intubation of the trachea, mechanical ventilation and improved operating conditions.

2) Succinylcholine causes brief irregular muscle contractions (fasciculations) followed by relaxation.
3) Succinylcholine is not an anesthetic agent and has no effect on consciousness or pain relief.
4) The patient must not be extubated until the paralysis has been reversed. The patient must have adequate muscle strength to protect his/her airway and be able to cough and breathe normally.

B) Succinylcholine Dosages
1) Intravenous: 1-1.5mg/kg will produce paralysis in about 30 seconds for 3 to 5 minutes.
2) Intermittent intravenous: About 25% of the initial dose.
3) Sublingual: 1-1.5mg/kg/ will produce paralysis almost as fast as an intravenous dose
4) Continuous intravenous infusion of about 1-2mg/kg/hr.
5) Intramuscular: 2-3 mg/kg. Onset is slow and not predictable.

C) Complications of Succinylcholine
1) The total dose of Succinylcholine should not exceed 4 to 6mg/kg or recovery may be very slow (phase 2 block). Recovery may take 1-3 hours especially if the patient is hypothermic.
2) Bradycardia is common after the second dose, but may occur after the first dose, especially in children. This can be prevented by prior treatment with atropine.
3) About 1 in 3,000 patients have a 4-12 hours recovery time from a normal dose of succinylcholine.
4) Transient raised intragastric pressure with an intravenous dose.
5) Transient raised intracranial and intraocular pressure with an intravenous dose.
6) Muscle pains from the fasciculations.
7) Significant hyperkalaemia may occur in patients with unhealed third degree burns, spinal cord injury, muscle atrophy and severe intraabdominal sepsis. Succinylcholine is best avoided 48 hours after the injury and for the next 2 years.
8) Malignant hyperthermia can be triggered by Succinylcholine.
9) Succinylcholine cannot be reversed by anticholinesterase drugs.

CURARE AND SIMILAR DRUGS

A) Curare is one of several similar drugs that paralyze skeletal muscle by competing with acetylcholine at the neuromuscular junction. They are known as non-depolarizing muscle relaxants.
B) The muscle relaxation allows easier intubation of the trachea, mechanical ventilation and improved operating conditions.
C) Depending on the specific drug, its dose and the condition of the patient, paralysis will occur within 30 seconds to five minutes.
D) The paralysis can be reversed by anticholinesterase drugs.
E) Because some of the curare-type drugs are rapidly metabolized, the patient does not always need an anticholinesterase for reversal of the paralysis.
F) The patient must not be extubated until the paralysis has been reversed. The patient must have adequate muscle strength to protect his/her airway and be able to cough and breathe normally.
G) Curare is not an anesthetic agent and has no effect on consciousness or pain relief.
H) Dosages and specific uses of anticholinesterase and the multiple curare type drugs are beyond the scope of this publication.

HALOTHANE

A) Halothane is a quick acting, potent inhalation anesthetic gas that, because of its marked respiratory and cardiovascular effects, must be given by experienced anesthetists through a calibrated vaporizer.
1) It is easy to overdose the patient. However, the concentration can be changed easily and awakening is usually rapid.
2) Inhalation induction requires the gradual increase of inspired concentration up to 4%. A maintenance dose is 1 to 2% for spontaneously breathing patients and 0.5 to 1% during controlled ventilation.
3) Halothane is a poor analgesic and is usually used with other inhalation or intravenous drugs.

Physiologic effects of Halothane

A) Halothane causes dose dependent respiratory depression and blood flow changes in the lung resulting in hypoxia. Halothane must be used with oxygen.
   1) Respiratory rate is increased, tidal volume is reduced, minute ventilation is decreased and blood CO_2 is increased during spontaneous respiration.
B) Halothane produces dose dependent cardiovascular depression.
   1) Halothane decreases blood pressure by decreasing stroke volume and, in larger doses, heart rate.
   2) Peripheral vascular resistance may be slightly decreased by halothane.
   3) Blood pressure = stroke volume x heart rate x peripheral resistance
C) Halothane sensitizes the heart to epinephrine solutions injected during surgery.
D) Halothane will cause uterine relaxation.
   1) This may be useful to help manual removal of the placenta but can cause increased uterine hemorrhage when given in concentrations above 0.8%.
   2) The use of halothane in low concentrations during C-sections has no effect on the fetus and does not increase uterine bleeding.
E) Halothane increases cerebral blood flow with an increase in intracranial pressure but keeping the patient normocarbic or slightly hypocarbic can decrease the intracranial pressure.
F) Halothane hepatitis is extremely rare (1:30,000).
G) Halothane, like other volatile anesthetics, can trigger malignant hyperthermia.

HALOTHANE/ETHER AZEOTROPE

A) The halothane/ether azeotrope is a mixture of 2 parts halothane and 1 part ether.
   1) The azeotrope is given in standard calibrated halothane vaporizers including the OMV.
   2) It is not flammable in clinical concentrations.
   3) It should be mixed in a separate container and then poured into the halothane vaporizer.
   4) Like halothane, it should be used in low doses during C-sections.
   5) Mask induction is pleasant and non-irritating
   6) In comparison to halothane, the azeotrope:
      a) Improves analgesia
      b) Increases muscle relaxation and potentiates muscle relaxants
      c) Improves hemodynamic stability
      d) Partially reverses respiratory depression
      e) Improves early post-operative pain control
      f) Is cheaper
B) The azeotrope is, in the author's opinion, a superior anesthetic compared to halothane.

ETHER

A) Ether is an inexpensive agent with a strong irritant smell.
B) Ether has significant advantages.
   1) It is an anesthetic with excellent analgesia.
   2) It stimulates catecholamine release and maintains blood pressure with increases in cardiac output, heart rate and systemic vascular resistance.
   3) It causes bronchodilation and stimulates or maintains respiration. It is safe to use for spontaneous respiration without additional oxygen for most patients.
   4) It does not relax the uterus.
   5) It gives good to excellent abdominal muscle relaxation.
C) Ether has significant disadvantages
   1) Very high doses of ether will cause cardiac depression and respiratory arrest.
   2) Inhalation induction, by the inexperienced anesthetist, is difficult because of ether's unpleasant smell, slow induction, increased secretions, breath holding and coughing.
   3) Ether in high doses causes postoperative nausea and vomiting.
   4) When used as the only anesthesia, recovery is slow until the anesthetist becomes quite experienced.
   5) It is flammable within the patient's airways, lungs and stomach and within 30 cm of the anesthetic circuit.
D) If thiopentone and/or halothane are used for induction and if succinylcholine is used for intubation, ether can be used in low doses and many of its disadvantages are eliminated.

THIOPENTONE

A) Thiopentone, also known as Thiopental or Pentothal, is a member of a large group of drugs known as barbiturates.
B) Thiopentone is given intravenously to rapidly induce loss of consciousness and amnesia. A single dose will last about five minutes after which the patient will regain consciousness.
C) Thiopentone can be used alone for very short surgical procedures, to induce a patient for a long anesthetic and to control seizures.
D) Advantages of Thiopentone
    1) Rapid and pleasant induction and awakening
    2) Inexpensive
    3) Does not compromise the brain's blood circulation
    4) Significant allergic reactions are rare.
E) Disadvantages of Thiopentone
    1) Thiopentone causes dose related decreases in peripheral vascular resistance, stroke volume and blood pressure. Healthy patients can maintain their blood pressure by increasing their heart rates and peripheral resistance. Ill and hypovolemic patients may have severe hypotension and/or cardiac arrest because they cannot compensate.
    2) Thiopentone causes dose related decreases in respiratory rate and tidal volume. Induction of anesthesia with Thiopentone routinely causes respiratory arrest. The anesthetist must be an expert in airway management.
    3) Thiopentone does not suppress laryngeal reflexes. Laryngospasm during induction is common unless other drugs – opioids, inhalation gases, muscle relaxants – are used.
    4) Thiopentone must be mixed properly with distilled water to a concentration of 2.5% (25 mg/ml). Higher concentrations will cause severe pain or loss of tissue if injected subcutaneously. Intra-arterial injection will cause vasospasm and possible loss of a hand.
    5) Repeated doses of Thiopentone will cause prolonged anesthesia.
    6) Because Thiopentone is so easy to give and is usually safe, many people have died because of being given too high a dose.
    7) Thiopentone is a poor analgesic.
F) Dose of Thiopentone
    1) A healthy adult patient will need 3-5mg/kg for induction of anesthesia or control of seizures.
    2) Patients who old, young, hypovolemic, septic or who have cardiovascular disease may require much lower doses to prevent cardiovascular collapse and death. Vigilance is critical!!!!
G) Thiopentone for C-sections
    1) Thiopentone has been used successfully for induction of anesthesia in millions of C-sections. However, many mothers and fetuses have died because of Thiopentone overdose.
    2) Thiopentone crosses the placenta into the fetus. The highest Thiopentone fetal concentrations occur abou three minutes after the maternal dose.

OTHER CONSIDERATIONS

(I) Drug doses: Milliliters, grams, milligrams, micrograms and percentages

A) One milliliter (ml) of water weighs one gram (gm).
   1) One gram has one thousand milligrams (mg). Therefore:
      a) There are 1,000 mg of water in each ml of water.

B) One ml of a 1% solution has 10 mg of drug. The remainder of the ml is water. Therefore:
   1) 1 ml of a 5% solution has 50 mg of drug
   2) 3 ml of a 6% solution has 180 mg of drug
   3) 1 ml of a 0.1% solution has 1 mg of drug
   4) 1/4 ml of a 0.1% solution has 0.25 mg of drug
   5) 1 ml of a 0.9% solution has 9 mg of drug

C) Some drugs are measured in micrograms (mcg). There are one thousand micrograms in a milligram (mg). Therefore:
   1) 50 mcg of fentanyl is the same as 0.05 mg of fentanyl
   2) 200 mcg of fentanyl is the same as 0.2 mg of fentanyl
   3) 1000 mcg of fentanyl is the same as 1 mg of fentanyl

(II) Drug ratios

A) If a drug is stated as being part of a ratio it is written as: mg of drug: mg of water. For example:
   1) A 1:1,000 solution of epinephrine has 1 mg of epinephrine in 1 ml (1000 mg) of water or 0.25 mg of epinephrine in 0.25 ml of water.
   2) A 1:100,000 solution has 1 mg of drug for every 100,000 mg of water.
   3) 1 mg of epinephrine in 100 ml (100,000 mg) of water is a 1:100,000 solution of epinephrine in water.
   4) A 1:100,000 solution of epinephrine in saline has 0.50 mg of epinephrine in 50 ml of saline or 0.25 mg of epinephrine in 25 ml of saline.
   5) A 1:200,000 solution of epinephrine has 0.25 mg of epinephrine in 50 ml or 0.15 mg of epinephrine in 30 ml.

B) More than one drug may share the same solution:
   1) 1% lidocaine may have a 1:100,000 concentration of epinephrine in it. This solution has:
      a) 10 mg of lidocaine per ml and
      b) 1 mg of epinephrine per 100 ml -- or 0.5 mg of epinephrine per 50 ml -- or 0.25 mg epinephrine per 25 ml.

C) When the surgeon injects an epinephrine solution for hemostasis during a halothane anesthetic, no more than 0.5 ml per kg body weight of a 1:200,000 epinephrine solution should be used. If the solution has 1% or 2% lidocaine, it may help to counteract the high incidence of ventricular arrhythmias seen with the combination of halothane and epinephrine.

(III) N_2O & Pressure oxygen anesthesia machines

A) When using a Boyle's machine (or similar) the percentage of oxygen that is delivered is critical because the machine can easily deliver lethal concentrations of nitrous oxide (N_2O).
   1) To determine the percentage of O_2 delivered: divide the volume of O_2 delivered per minute by the total volume of gas delivered per minute and multiply by 100.
      a) If the machine delivers 2 liters (L) of O_2 per minute and 3L of N_2O per minute:
         $2/5 \times 100 = 40\%\ O_2$
      b) The % of O_2 in 1L O_2 per minute and 4L N_2O per minute is:
         $1/5 \times 100 = 20\%\ O_2$
      c) The % of N_20 in 1.5L O_2 per minute and 2.5L N_20 per minute is:
         $2.5/4 \times 100 = 62.5\%\ N_2O$

(IV) Copper Kettle & Pressure oxygen anesthesia machines

A) Some pressure oxygen anesthesia machines have Copper Kettle vaporizers in which a small amount of oxygen from a special flow meter (Vernitrol) is bubbled through either halothane or ether. This lethal concentration of anesthetic vapor is then diluted with gas from another flow meter of O_2 (or O_2 and N_2O) to reach the desired anesthetic concentration. These anesthesia machines are very dangerous and must be used with great caution and vigilance. The percentages given below are for a temperature of 22ºC in the liquid halothane or ether. Higher temperatures will result in higher percentages of either halothane or ether. Different O_2 flow rates result in different anesthetic percentages.

1) The safest procedure is for the anesthetist to first set the O_2 flows at either 5 liters, 2.5 liters or one liter and keep the same O_2 flow for the duration of the anesthetic.
   a) Then, the anesthetist can vary the percentage of halothane or ether delivered to the patient by changing the O_2 flow through the Copper Kettle.
   b) 4-5% halothane for brief periods are the maximum concentrations that should be used. Between 0.8% and 1.5% halothane are the usual maintenance concentrations.
   c) 15-20% ether for brief periods are the maximum concentrations that should be used. Between 1.5% and 3.5% ether are the usual maintenance concentrations.

B) When a 5-liter O_2 flow is used:

1) 100 ml Copper Kettle flow results in 1% halothane.
2) 300 ml Copper Kettle flow results in 3% halothane.

O_2 flow [O_2 and N_20]	Copper Kettle O_2 flow [Vernitrol flow	Halothane %	Ether %
5000 ml	25 ml	0.25%	0.8%
5000 ml	50 ml	0.5%	1.7%
5000 ml	100 ml	1.0%	3.5%
5000 ml	200 ml	2.0%	7.0%
5000 ml	300 ml	3.0%	10.0%
5000 ml	400 ml	4.0%	13.0%
5000 ml	500 ml	5.0%	17.0%
5000 ml	600 ml	6.0%	21.0%

C) When a 2.5-liter O_2 flow is used, the halothane concentration will be higher at the same Copper Kettle flows as in (#4) above. For example, when a 2.5-liter O_2 flow is used:

1) 100 ml Copper Kettle flow results in 2% halothane.
2) 300 ml Copper Kettle flow results in 6% halothane.

O_2 flow [O_2 and N_20]	Copper Kettle O_2 flow [Vernitrol flow	Halothane %	Ether %
2500 ml	25 ml	0.5%	1.7%
2500 ml	50 ml	1.0%	3.4%
2500 ml	100 ml	2.0%	7.0%
2500 ml	200 ml	4.0%	14.0%
2500 ml	300 ml	6.0%	20.0%
2500 ml	400 ml	8.0%	26.0%
2500 ml	500 ml	10.0%	>30.0%
2500 ml	600 ml	12.0%	>30.0%

D) When a 1.0-liter O_2 flow is used, the halothane concentration will be higher at the same Copper Kettle flows in (#4) or (#5) above. For example, when a 1.0-liter O_2 flow is used:

1) 100 ml Copper Kettle flow results in 5% halothane.
2) 300 ml Copper Kettle flow results in 15% halothane.

O_2 flow [O_2 and N_20]	Copper Kettle O_2 flow [Vernitrol flow	Halothane %	Ether %
1000 ml	10 ml	0.5%	1.7%
1000 ml	15 ml	0.8%	2.6%
1000 ml	25 ml	1.3%	4.3%
1000 ml	50 ml	2.5%	8.5%
1000 ml	100 ml	5.0%	17.0%
1000 ml	200 ml	10.0%	30.0%
1000 ml	300 ml	15.0%	>30.0%
1000 ml	400 ml	20.0%	>30.0%

(V) Vaporization of liquid anesthetic drugs

A) Vaporization is the conversion of a liquid to a gas. "Gas" and "vapor" are the same.
1) The molecules of a liquid are held together by cohesive forces.
2) To form a vapor, the cohesive forces in the liquid must be broken by heat.
3) If external heat is not available during vaporization, the heat required to separate the molecules comes from the liquid. The liquid becomes cool because of the heat loss.

B) In a closed but partially filled container, vaporization occurs until a balance is reached between a liquid and its vapor.

C) In an open container, a balance cannot be reached because the vapor is dispersed into the air. Vaporization will occur until the liquid is gone.

D) A flow of air or oxygen over the liquid in the container will increase the rate of vaporization by quickly removing molecules from the surface of the liquid. Higher flow results in more vaporization and a more rapid drop in the temperature of the liquid.

E) Anesthetic vaporizers are partially filled calibrated containers in which oxygen and/or air is pulled or forced over the liquid halothane or ether.
1) The temperatures of ether and halothane always decrease during anesthesia.
2) As the temperature decreases, fewer molecules become vapor and a lower amount of anesthetic gas is produced.

F) The temperature decrease during vaporization can be compensated for by:
1) Increasing the flow of air/oxygen over the liquid.
a) The EMO and the Fluotec vaporizers have temperature compensating valves which increase the air/oxygen flow as the temperature decreases.
2) Providing a water jacket that surrounds the liquid anesthetic. Heat is transferred from the water to the anesthetic.
a) The EMO and OMV vaporizers have water jackets. Many other vaporizers do not have water jackets.
3) Adding room temperature liquid anesthetic.

G) Room temperature slowly replaces the heat lost by the water and the liquid anesthetics.
1) Cold EMO, OMV and Fluotec vaporizers will usually warm to room temperature by the time the next procedure begins.

(VI) Liquids and gases

A) Liquids (like blood) and gases (like air) are both fluids. In contrast to solid objects, fluids can move easily from place to place.

B) Fluids can only move from one place to another if there is a pressure difference between the two places. For example:
1) Blood flows from the heart to the capillary beds because the heart creates a high pressure.

2) Air flows from the nose to the alveoli because the contracting diaphragm creates a negative pressure in the chest
3) Urine flows out through the urethra because muscles in the bladder wall create a high pressure in the bladder.

(VII) Gastrointestinal considerations

A) Preventing aspiration of gastric contents before, during and immediately after an anesthetic is the responsibility of the anesthetist.
1) Aspiration may cause the patient to die immediately from airway obstruction or later from pneumonia.
2) Aspiration can be prevented by waiting until the stomach is empty, by nasogastric suction, by cricoid pressure during induction and intubation and by extubating the patient when awake and on his/her side.
3) The cricoid is the only circular cartilage in the trachea. The esophagus is behind the trachea. Firm and steady pressure on the cricoid helps prevent aspiration by compressing the esophagus against the cervical vertebral column.

B) A normal healthy patient will empty his/her stomach of solid food in 8 hours. Water will be emptied in 2 hours.
1) These patients should be NPO for 8 hours before surgery but allowed to have water (only water - nothing else) until 2 hours before surgery.
a) The patients will come to surgery better hydrated and tolerate anesthesia better than if they had been NPO for the entire 8 hours.
b) If the patients have violated these guidelines, surgery must be delayed.

C) Patients who are pregnant, or in pain, or have obstructed bowels, or have had trauma and need immediate emergency surgery should not have their surgery delayed because of a full stomach.
1) No amount of waiting will guarantee an empty stomach in these patients.
a) Their stomachs ability to empty has been compromised by drugs, pain and/or their condition.
2) Depending on the circumstances, a nasogastric tube might be inserted preoperatively.
3) These patients must be well oxygenated before induction, have cricoid pressure during induction and intubation and be extubated when awake and on his/her side while being well oxygenated

ANESTHETIC QUESTIONS

1) What two basic principles must the anesthetist never forget?
2) Describe or give the formulas for: heart rate, stroke volume, peripheral resistance, cardiac output and blood pressure.
3) What three things do you know if the patient has good urine output without the use of diuretics?
4) What effects do halothane; ketamine, thiopentone, ether, pethidine and succinylcholine have on peripheral resistance, heart rate, stroke volume, blood pressure, minute ventilation and blood levels of carbon dioxide?
5) State the body's fluid spaces. What percentage of the body weight is in each of the spaces?
6) What are the advantages of the Boyle's machine? What are the advantages of the EMO/OMV machine?
7) Where is the narrowest part of: The pediatric airway? The adult airway?
8) What will most likely be the effects on stroke volume by: blood loss, halothane, surgical trauma, intravenous 0.9% saline, intravenous 5% dextrose, ether and blood?
9) What are the advantages and disadvantages of the Ayres T system? The circle system? Diagram both systems.
10) What is the technique of choosing a child's endotracheal tube?
11) What are the usual intravenous doses in a healthy patient of succinylcholine, ketamine and thiopentone?
12) Into what fluid spaces are intravenous 0.9% saline, 5% dextrose and blood distributed?
13) Diagram or describe the effects of the sympathetic nervous system on the heart, lungs, blood vessels and adrenal glands. What chemical is released by the sympathetic nervous system?
14) Diagram or describe the effects of the parasympathetic nervous system on the heart, lungs and blood vessels. What chemical is released by the parasympathetic system?
15) During spinal anesthesia, what are the effects on the blood pressure, pulse and respiration by a: T-6 block? T-1 block? C-1 block?
16) How are decreases in blood pressure and respiration managed during spinal anesthesia?
17) In what circumstances would you NOT use spinal anesthesia for a C-section?
18) What are the primary and the side effects of succinylcholine?
19) Define: respiratory rate; tidal volume; minute ventilation; vital capacity; functional residual capacity. What are the normal values in an adult? What is the purpose of the functional residual capacity?
20) Describe the initial and the anesthetic management of a patient with a severe head injury who arrives in the casualty department.
21) What is the primary control of minute ventilation?
22) If an anesthetic circuit does not eliminate the patient's CO_2, what are the effects on the patient?
23) How much O_2 does a normal patient use every minute while at rest? Does this change during surgery?
24) What are the common causes of cardiac arrest during general anesthesia? Spinal anesthesia?
25) Why do you not give blood to a normal eight years old boy with a Hb. of 6.5 having a hernia repair?
26) Describe the action of a non-depolarizing muscle relaxant at the neuromuscular junction.
27) Describe the action of neostigmine at the neuromuscular junction. Why is neostigmine used?
28) What drug is usually used with neostigmine? Why? What is the maximum adult dose of neostigmine?
29) What striated muscle is the most resistant to muscle relaxants? Which striated muscles are the least resistant?
30) What effect does term pregnancy have on: stroke volume, cardiac output, blood volume, Hb., FRC, minute ventilation, metabolic rate, size of epidural veins, volume of CSF, stomach emptying, rapidity of induction with anesthetic gases and rapidity of hypoxia during apnea?
31) What is the supine hypotensive syndrome? What is the treatment?
32) What are the reasons that cuffed endotracheal tubes are not used routinely in children?
33) Some hospitals allow patients for elective surgery to have water until two hours pre-operatively. Why?
34) Describe the intravenous fluid management for the pre-operative and intra-operative periods:
    a) A 50 kg woman who must wait two days for her hysterectomy. She is unable to eat or drink.
    b) A 20 kg girl who has a swollen and deformed upper leg after falling from a tree.
    c) A normal man of 60 kg who has sudden blood loss during a cholecystectomy.
    d) A 4 kg six months old boy with pyloric stenosis.
    e) An unconscious 10-year-old boy in septic shock from a perforated bowel. Weight 20 kg. Blood pressure 40/0. Pulse 160. No urine output.

35) Define eclampsia of pregnancy. What problems are there with spinal and general anesthesia for C- section in a patient with eclampsia?
36) What are the signs and symptoms of lidocaine overdose?
37) You have 50 mls 2% lidocaine & saline & 1:1,000 epinephrine. How do you obtain:
    a) 1% lidocaine?
    b) 0.5% lidocaine with 1:200,000 epinephrine?
    c) 1% lidocaine with 1:100,000 epinephrine?
38) You have 30 mls 2% lidocaine with 1:100,000 epinephrine & 0.9% saline. How do you obtain 1% lidocaine with 1:200,000 epinephrine?
39) What are the signs, symptoms and treatment of left ventricular failure? Right ventricular failure?
40) State two reasons that you might use lidocaine intravenously during general anesthesia.
41) Describe the management of a patient in the Intensive Care Unit with a crushed chest.
42) Define shock. How do you know when the patient has recovered from shock?
43) What is the % of oxygen in: the air?; the EMO/OMV machine without oxygen?; the EMO/OMV with 3 liters oxygen added to the intake port?; and, the Boyle's machine using oxygen?
44) Why is 2.5% thiopentone used rather than 5%?
45) How many mg in one ml of a 1% solution?
46) What main stem bronchus will usually be intubated if the endotracheal tube is down too far? Why? After an intubation, where is the first place you listen? Why?
47) What is unique about the cricoid cartilage? When is cricoid pressure used?
48) What drug should you always consider giving in a big dose? Why?
49) State six reasons that a patient might not breathe well at the end of an anesthetic.
50) Why do i.v. fluids decrease body temperature?
51) What is the sodium content of: blood?; 0.9% saline?; Ringers lactate? 5% dextrose in water? 5% dextrose in 0.45% saline? Muscle cells?
52) What is the disadvantage of the Trendelenberg position?
53) In contrast to the adult, what are the differences in a one-year-old's: respiratory rate, FRC, minute ventilation, metabolic rate, heart rate, blood volume and body surface area?
54) What are the signs of an upper airway obstruction?
55) What are the signs that a patient has recovered from a neuromuscular block?
56) What is the treatment for the metabolic acidosis created during shock?
57) What is the basic physiologic problem in eclampsia?
58) Why is the regulation of cardiac output different in a one-year-old than in an adult?
59) Why should the anesthetist keep the oxygen attached to the endotracheal tube during extubation?
60) Why do the EMO and OMV vaporizers become cold during an anesthetic?
61) Where do the phrenic nerves originate? Are they part of the autonomic nervous system?
62) Why do the EMO and OMV vaporizers have hoses of about two feet in length on their intake ports?
63) What is the third space?
64) What are the ABCDs?
65) List the risks of blood transfusion.
66) What percentage of the cardiac output goes to the kidneys?
67) What determines in which of the body's fluid spaces an intravenous fluid will remain?
68) Should you delay emergency surgery for a patient in severe pain who has a full stomach? Why or why not?
69) To increase blood volume as much as blood does, about _____ times as much Ringers lactate or 0.9% saline must be given.
70) Why is pulmonary artery pressure lower than aortic pressure?
71) State four different types of sensory nerves.
72) Why does intravenous atropine increase heart rate?
73) Which of the body's chemicals is increased during anaphylaxis? What are its effects?
74) Oxygen and food are used by the body's cell to produce --, --, --?
75) What is produced when cells have low levels of oxygen?
76) What is the percentage of nitrogen in the air?
77) Does arterial blood have: More or less oxygen than venous blood? More or less carbon dioxide than venous blood?
78) Why are blood carbon dioxide levels high in a patient who is spontaneously breathing halothane?

79) Start with 25 mls of 2% lidocaine & saline & one mg of epinephrine. How do you obtain;
    a) 25 mls of 1% lidocaine?
    b) 50 mls of 1% lidocaine with 1:100,000 epinephrine?
    c) 200 mls of 0.5% lidocaine with 1:200,000 epinephrine?
80) Why does the OMV (Oxford Miniature Vaporizer) have a water jacket?
81) What are the advantages and disadvantages of the; OMV, Ambu bag, Boyle's machine, Farman entrainer?

82) What dose of ketamine may be used during labor?
83) Why is epinephrine sometimes added to a spinal anesthetic? What is the dose?
84) What type of anesthetic would you give for a hemorrhoidectomy in a patient with severe asthma? Describe your technique.
85) State two reasons that the pulse oximeter may give you misleading information.
86) How does ether differ from halothane in: speed of induction, analgesia, muscle relaxation, blood pressure and minute ventilation?
87) How do you make the halothane/ether azeotrope?
88) Describe the normal activity at the neuromuscular junction.
89) How does succinylcholine effect the neuromuscular junction?
90) What are the maximum doses of intravenous lidocaine? Subcutaneous lidocaine with and without 1:200,000 epinephrine?
91) A 50-year-old man is bleeding from an open fracture of the femur. He ate two hours before the accident. He loses a large amount of blood during the surgery. Discuss your management of the anesthesia.
92) Which anesthetic drugs cause the release of the body's epinephrine?
93) What does "Preparation" mean to the anesthetist?
94) What are the causes of: Right ventricular failure? Left ventricular failure?
95) What does "Routines are traps" mean?
96) Why does a patient in heart failure have nocturia?
97) What is the central venous pressure?
98) What is the basic physiologic problem in diabetes?
99) What is the cause of hypoglycemia in the diabetic? Cause of hyperglycemia?
100) How do oral diabetic medications work?
101) Why might a diabetic patient develop metabolic acidosis?
102) What are the long-term complications of chronic hypertension? Of diabetes?
103) Describe a normal cough.
104) Describe the anesthetic management of:
    a) Eclampsia - 8 months pregnant. Seizures. BP 180/90.
    b) Ectopic pregnancy. Hb 10 gm%. BP 70/40. Pulse 150.
    c) Bowel obstruction who you are unable to intubate.
    d) Head injury. Unconscious with seizures.
    e) Elective surgery. BP 250/130.
    f) Multiple fractures including a fractured trachea (skin not open).
105) What are the contraindications to using the laryngeal mask airway?
106) What is the chemical in the oxygen concentrator? What does it do?
107) Why must the anesthetist accompany the patient to the recovery room or ward?
108) Does neostigmine reverse the effects of succinylcholine?
109) Is cardiac output the same in the right and left ventricles?
110) Describe a rapid ("crash") induction.
111) What are the airway pressure differences during spontaneous ventilation and positive pressure ventilation?
112) Why is ephedrine, rather than other vasopressors, used during spinal anesthesia for C-section?
113) Describe the steps in newborn resuscitation.
114) How can the use of Ketamine mislead you in a hypovolemic patient?

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