

paracervical block

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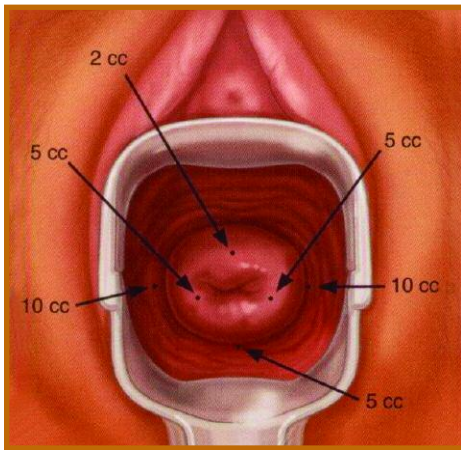
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Local anesthesia is an effective option for various minor gynecologic procedures. The relative ease of performing a paracervical block makes it particularly useful in the outpatient or office setting. The purpose of this Clinical Spotlight is to review the anatomy and pharmacology of the paracervical block to highlight techniques that may enhance the safety of the procedure.

anatomy

Pain associated with uterine contraction and cervical stretching is transmitted via visceral afferent nerve fibers which accompany sympathetic fibers sequentially passing through aggregates of nerve fibers and entering the spinal cord at T11-T12.

The ease with which these fibers, adjacent to the cervix, can be bathed in local anesthetic solution makes them readily available to local anesthetic blockade. The location of the broad ligaments just deep to the lateral fornices of the vagina is the landmark used to approximate the position of these aggregates of nerve fibers.



One PCB Injection Technique

In addition to the aggregate of nerve fibers, the uterine artery and vein and their branches are also located in the vaginal fornices. It is the presence of these vascular structures, in close proximity to the nerve bundles that create the potential for significant adverse outcomes when meticulous technique is ignored and high concentrations of the local anesthetic enter the circulation.

The technique for paracervical block has several variations recommending as few as two, to as many as six injections at a depth of 3-7 mm alongside the cervix in the vaginal fornices. Some physicians believe that more injections of smaller volumes at several sites (3, 5, 7, and 9 o'clock), rather than two large doses at 4 o'clock and 8 o'clock minimizes the risk of injecting a potentially toxic dose if the needle tip should inadvertently enter a blood vessel at any one site.

TIPS

While avoiding excessive negative pressure, frequent and careful aspiration of the syringe will assist recognition of an unintended intravascular placement.

pharmacology

Local anesthetics work by reversibly blocking sodium channels in the nerve membrane and inhibiting the development of action potentials and nerve impulse transmission.

The degree of blockade is affected by the diameter of the nerve. Larger fibers, carrying touch, pressure, and motor impulses require higher concentrations of local anesthetic to achieve a given degree of block compared to smaller diameter fibers that carry pain impulses. Because of these varying sensitivities to the concentration of drug at the nerve at any given time, the sensory modalities are lost as the effect of the block comes on, in the following order:

1. Pain (lost first)
2. Temperature
3. Touch
4. Deep pressure
5. Motor function (lost last)

This differential progression explains the observation of retained motor function in the presence of adequate pain relief when using lesser concentrations of a given agent.

In general, the duration of a block relates to its uptake, distribution, binding, and subsequent metabolism. Drugs that are strongly lipophilic* dissociate more slowly from their binding sites and will generally have a longer duration of action (e.g. Bupivacaine > Lidocaine). This often desired property conveys some liability/risk however for reasons that are discussed below.

***Drugs are referred to as lipophilic if easily dissolved in fat or oil, and hydrophilic if easily dissolved in water. Because nerves have a sheath made of fatty acid molecules, they readily bind lipophilic drugs**

Local anesthetic agents fall into two chemical classes: Esters and Amides.

Each class, ester and amide, has a distinct metabolism responsible for the degradation of the active drug. Esters undergo rapid breakdown in the blood and in some rare cases, a by product, p-aminobenzoic acid (PABA), can be allergenic. This rapid plasma degradation lends a degree of safety to the esters as blood levels fall quickly and central nervous system (CNS) and cardio-toxicity are thus tempered.

Amides rely on breakdown in the liver. Because amides remain in the blood longer, and are not metabolized in the blood, they have the potential to rise to toxic levels that may clinically manifest as CNS or cardiac events.

Drug	Class	Onset	Duration	Max Dose (without epinephrine)
2-Chloroprocaine 2% (Nesacaine® Anesthetic)	Ester	6-10 minutes	30-60 minutes 60-90 minutes with epi 1:100,000	11mg/kg (not to exceed 800 mg)
Lidocaine 1% (Xylocaine® Pharmaceutical)	Amide	4-10 minutes	60-120 minutes	4.5 mg/kg
Mepivacaine 1% (Carbocaine® & Polocaine® Anesthetics)	Amide	6-10 minutes	90-180 minutes	7 mg/kg (400 mg for any one procedure)
Ropivacaine 0.2-1% (Naropin® Anesthetic)	Amide	8-12 minutes	240-480 minutes	150-200 mg
Bupivacaine 0.25% (Marcaine® & Sensorcaine® Anesthetics)	Amide	8-12 minutes	240-480 minutes	2 mg/kg

TIPS

The common drugs at left are listed from lesser to greater lipophilic properties, indicating increased cardio-toxicity if injected intravascularly.

Data for Lidocaine, Mepivacaine, Bupivacaine from Norris (1) and Trott (2)

Data for Ropivacaine from Mather and Chang (3)

Data for 2-Chloroprocaine from AHFS Drug Information 2005 (4)

1. Norris RL Jr. Local anesthetics. Emerg Med Clin North Am 1992;10(4):707-18

2. Trott A. Infiltration and nerve block anesthesia. In: Trott A, ed. Wounds and lacerations: emergency care and closure. St Louis: Mosby Year Book, 1991:29-54

3. Mather LE, Chang DH. Cardiotoxicity with modern local anaesthetics: is there a safer choice? Drugs 2001;61:333-42.

4. McEvoy Gerald, Pharm.D. AHFS Drug Information 2005: American Society of Health-System Pharmacists®: 2005

systemic toxicity

Injected local anesthetics are absorbed into the circulation, and toxic effects on the central nervous system or cardiovascular system depend on the blood level of the drugs. The rate of change, as well as the total blood amount of the local agent, influences toxicity. More rapid accumulation is more toxic.

Specialized tissue in the heart initiates and conducts the electrical activity responsible for contraction and relaxation of the heart muscle. Nerve transmission in these tissues is regulated by sodium channels, small openings that allow the sodium to move into and out of the cells. As described, these can be blocked by local anesthetics. Without sodium crossing these channels, nerve transmission ceases and an anesthetic effect is obtained. In the case of inadvertent high blood levels of local anesthetic, a drug like lidocaine binds and releases from tissues rather quickly. Bupivacaine, however, releases much more slowly, allowing a more pronounced block to develop and persist leading to slowed cardiac nerve transmission and often irregular heartbeats that resist treatment and can be fatal.

The magnitude of the effect will depend on:

1. **Toxicity** of the drug (bupivacaine>lidocaine>mepivacaine> 2-chloroprocaine)
2. **Dose** administered (total milligrams of the drug) 1% concentration is 1 gram of drug /100 cc=1000 mg /100cc=10 mg/cc
3. **Rate of absorption** based on the speed of injection and site of administration (vascularity of the adjacent tissue beds or inadvertent direct intravascular injection)
4. **Physical status** of the patient (age and underlying medical conditions)

Toxicity with increasing plasma concentrations manifests as:
Early irritability due to blockade of central inhibitory neurons (resulting in stimulation)

1. **Numbness of the tongue**
2. **Lightheadedness**
3. **Visual and auditory disturbances** (ringing in ears)
4. **Muscular twitching**
5. **Seizures**

Followed by:

- Progression to central nervous system depression with blockade of both inhibitory and excitatory pathways
6. **Unconsciousness**
 7. **Coma**
 8. **Respiratory arrest**
 9. **Cardiovascular depression**

TIPS

Pay close attention to the patient whose demeanor seems different within the first 15 minutes of receiving a block. Garrulousness or complaints of a "metallic taste" may be an early sign of systematic toxicity.

management of adverse reactions

The majority of local anesthetic allergic reactions are associated with the ester metabolite PABA or the preservatives found in multidose vials, methylparaben or sodium meta-bisulfite. True allergy to amide type anesthetics is extremely rare. "Allergies" as often reported by patients are often confused with other "reactions" including:

1. **true systemic toxicity as described above and not necessarily reproducible on subsequent administrations.**
2. **vaso-vagal syncope**
3. **unpleasant side effects (flushing, tachycardia, dysphoria) following rapid absorption of vasoconstrictors (typically epinephrine) which are or may be added to local anesthetics to prolong their duration.**

TIPS

All anesthetizing locations should be equipped with (at a minimum):

- Stethoscope and mean of blood pressure measurement
- O2 source and Ambu® Bag
- Injectable Epinephrine 1:1000

Etiology	Main Clinical Features	Treatment
Local Anesthetic Toxicity Intravascular Injection Relative Overdose	Convulsions +/- cardiac arrhythmias Onset of irritability within 5-15 minutes progressing to seizures	Cardiopulmonary resuscitation, benzodiazepine to control seizure, Oxygen
Vaso-vagal Reaction	Rapid onset of bradycardia, hypotension, pallor, faintness	Leg elevation, atropine, Oxygen
Reaction to vasoconstrictor	Tachycardia, hypertension, headache, apprehension	Supportive measures, consider short acting beta-blocker if persistent tachycardia and hypertension, Oxygen
Anaphylaxis	Hypotension, bronchospasm, urticaria, edema	Epinephrine, Oxygen,

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