Local Anesthetics & Spinal anesthesia

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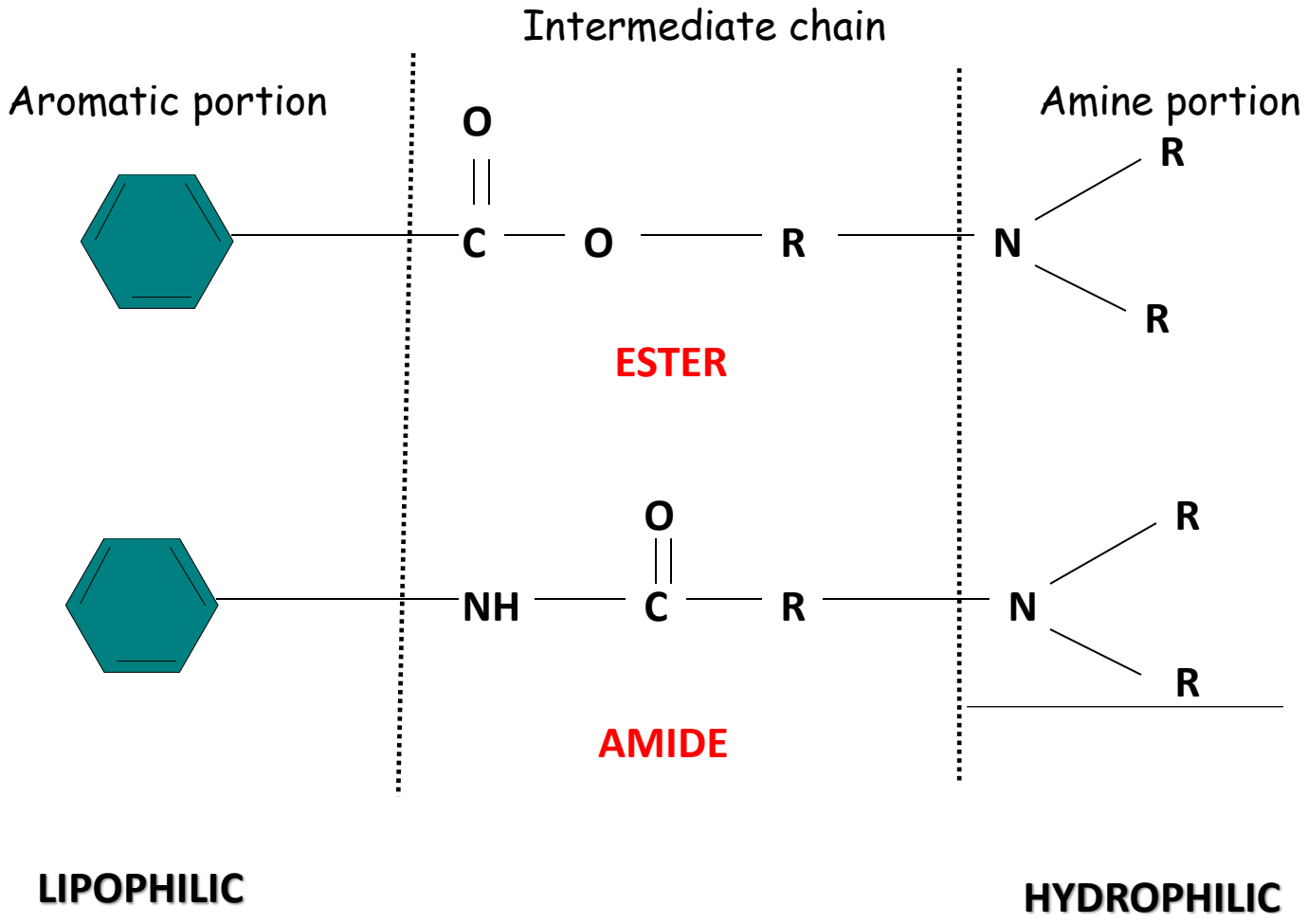
Outlines

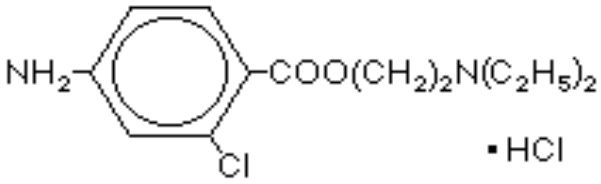
- Local Anesthetic Molecule
- Physiology of Nerve Conduction
- Mechanism of Nerve Blockade
- Physicochemical Properties of Local Anesthetics
- Pharmacology of local anesthetics
- LAST
- Indications and contraindications
- Technique of spinal anesthesia
- Physiologic changes during spinal anesthesia
- Spinal block monitoring
- Complications

Local anesthetics are Weak Bases



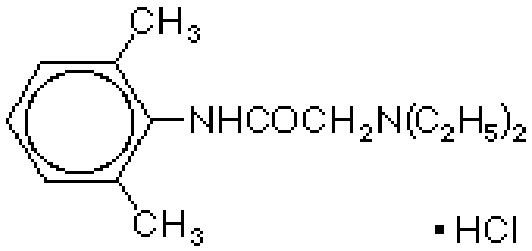
Local anesthetics

ESTER LINKAGE



$C_{13}H_{19}ClN_2O_2 \cdot HCl$

AMIDE LINKAGE

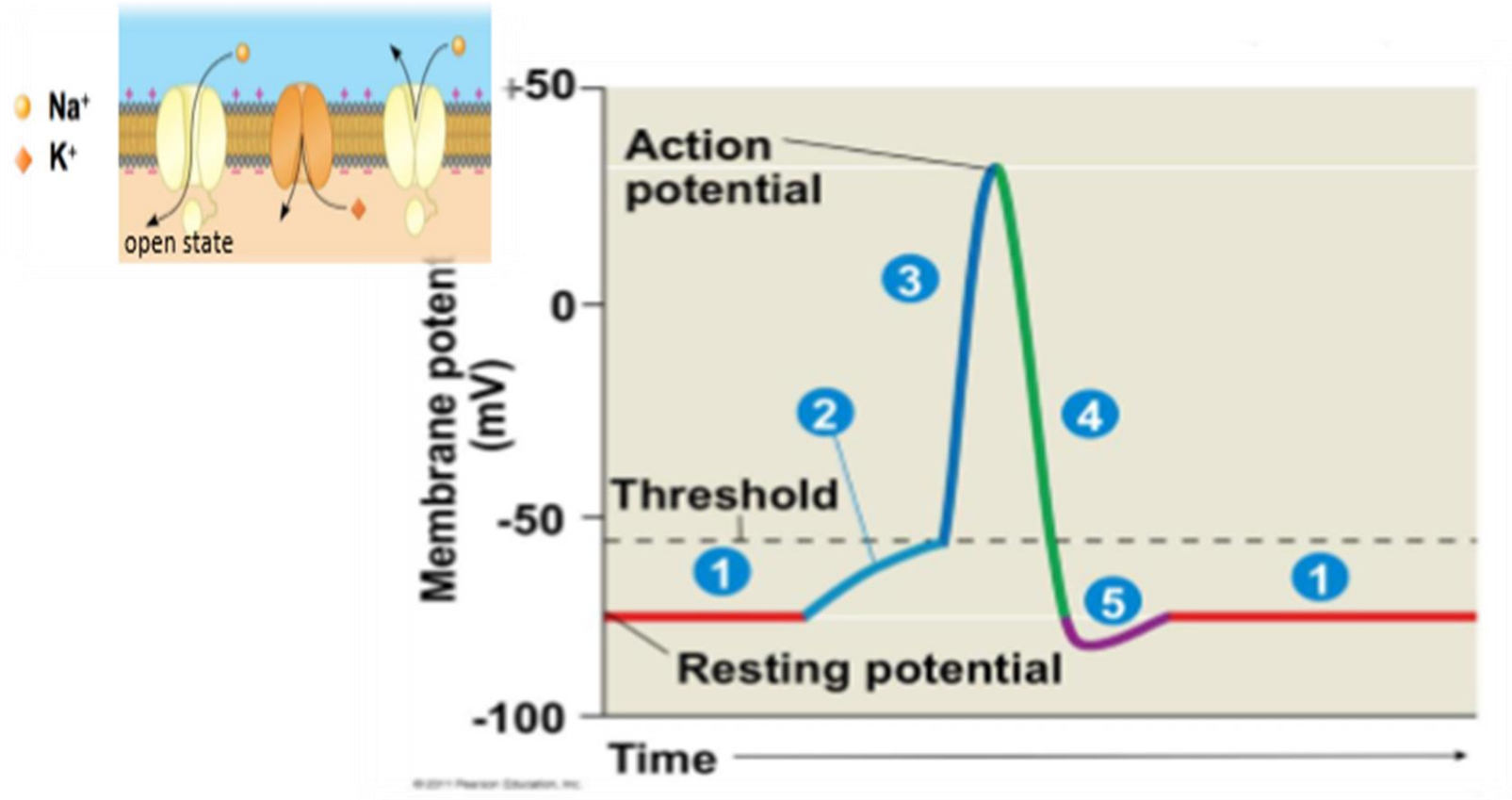


$C_{14}H_{22}N_2O \cdot HCl$

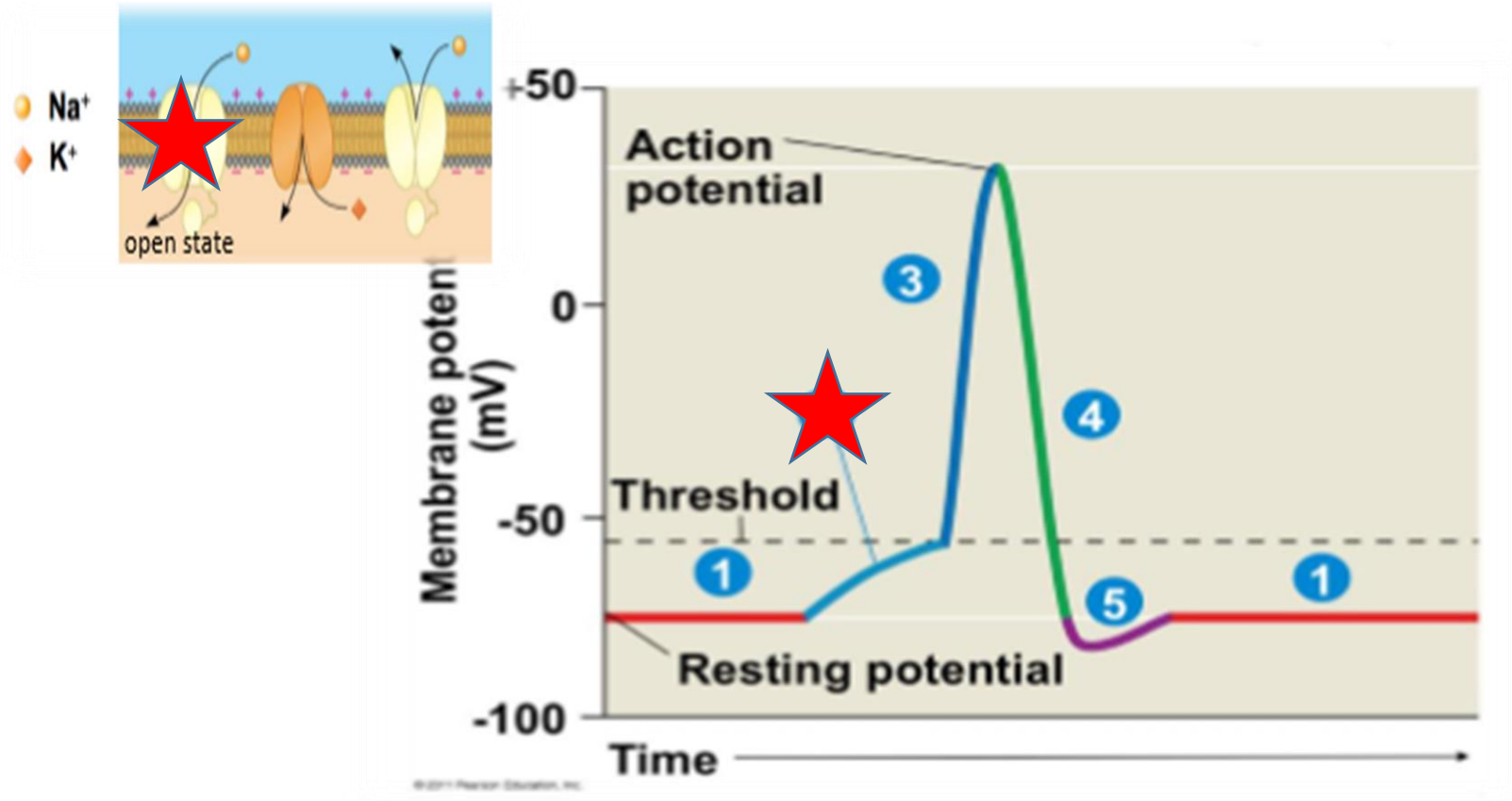
PROCAINE
procaine (Novocaine)
tetracaine (Pontocaine)
chloroprocaine
benzocaine
cocaine

LIDOCAINE
lidocaine (Xylocaine)
mepivacaine (Carbocaine)
bupivacaine (Marcaine)
etidocaine (Duranest)
ropivacaine (Naropin)

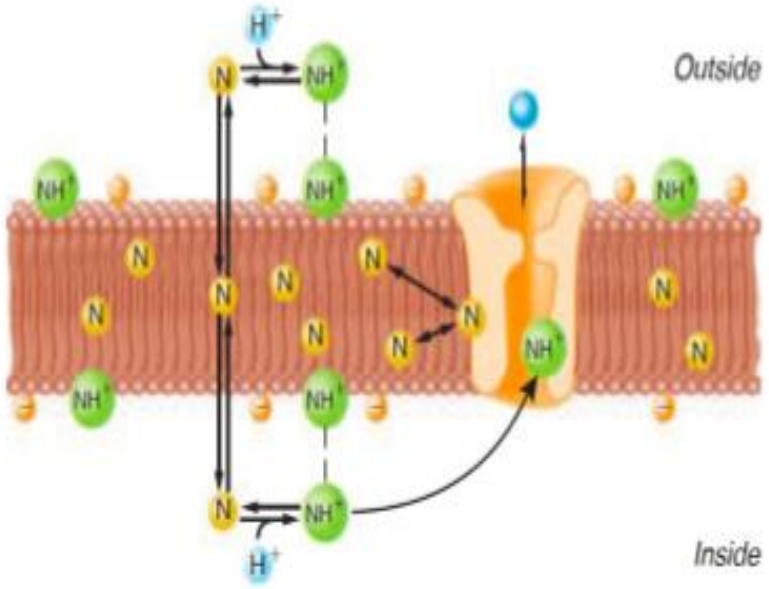
Physiology of nerve conduction



Mechanism of blockage



Mechanism of blockage



Block of sodium channel

- Bind to specific site in inner portion of channel
- Stabilize inactivated-closed state

Physicochemical Properties of Local Anesthetics

Onset

- pKa

Duration

- Protein binding
  - Unbound form is pharmacological active

Potency

- Lipid solubility, affect all of potency

Local anesthetics	pK_a	% Ionized (at pH 7.4)	Lipid solubility	% Protein binding
AMIDES				
Bupivacaine Levobupivacaine	8.1	83	3,420	95
Etidocaine	7.7	66	7,317	94
Lidocaine	7.9	76	366	64
Mepivacaine	7.6	61	130	77
Prilocaine	7.9	76	129	55
Ropivacaine	8.1	83	775	94
ESTERS				
Chloroprocaine	8.7	95	810	N/A
Procaine	8.9	97	100	6
Tetracaine	8.5	93	5,822	94

Pharmacology of local anesthetics

- Ester LA are predominantly metabolized by pseudocholinesterase (plasma cholinesterase or butyrylcholinesterase).

- Ester hydrolysis is very rapid, and the water-soluble metabolites are excreted in the urine.

- Procaine and benzocaine are metabolized to p-aminobenzoic acid (PABA), which has been associated with allergic reactions.

Pharmacology of local anesthetics

- **Lidocaine (Xylocaine)**
  - **Most widely used LA**
  - **Effective by all routes**
  - **Faster onset and longer lasting than procaine**
  - **More potent than procaine but about equal toxicity**
  - **More sedative than others**

Pharmacology of local anesthetics

- **Bupivacaine (Marcaine)**
  - **No topical effect**
  - **Slower onset and one of longer duration agents**
  - **Unique property of sensory and motor dissociation (provide sensory analgesia with minimal motor block)**
  - **More cardiotoxicity than other LA**

Local anesthetics Systemic toxicity (LAST)

CNS

- Light headedness, vertigo, tinnitus, slurred speech, seizure, CNS depression

CVS

- Relaxation of arteriolar smooth muscle
- Direct myocardial depression
- Prolonged P-R, widen QRS
- **Arteriolar dilation** (cocaine is exception: vasoconstriction)

Agents	Relative potency for CNS toxicity	CVS:CNS
Bupivacaine	4.0	2.0 : 1
Levobupivacaine	2.9	2.0 : 1
Chloroprocaine	0.3	3.7 : 1
Etidocaine	2.0	4.4 : 1
Lidocaine	1.0	7.1 : 1
Mepivacaine	1.4	7.1 : 1
Prilocaine	1.2	3.1 : 1
Procaine	0.3	3.7 : 1
Ropivacaine	2.9	2.0 : 1
Tetracaine	2.0	-

Dose-Dependent Systemic Effects of Lidocaine

Plasma concentration (μg/mL)	Effect
1–5	Analgesia
5–10	Lightheadedness Tinnitus Numbness of tongue
10–15	Seizures Unconsciousness
15–25	Coma Respiratory arrest
>25	Cardiovascular depression

The Pharmacologic Treatment of LAST is Different from Other Cardiac Arrest Scenarios

- **Reduce** individual **epinephrine** boluses to ≤ 1 mcg/kg
- **Avoid** vasopressin, calcium channel blockers, beta blockers, or other local anesthetics

- Stop injecting local anesthetic
- Get help
  - Consider lipid emulsion therapy at the first sign of a serious LAST event
  - Call for the LAST Rescue Kit
  - Alert the nearest cardiopulmonary bypass team - resuscitation may be prolonged
- Airway management
  - Ventilate with 100% oxygen / avoid hyperventilation / advanced airway device if necessary
- Control seizures
  - Benzodiazepines preferred
  - **Avoid** large doses of **propofol**, especially in hemodynamically unstable patients
- Treat hypotension and bradycardia – **If pulseless, start CPR**

Lipid Emulsion 20% (Precise volume and flow rate are not crucial)	
Greater than 70 kg patient	Less than 70 kg patient
Bolus 100 mL Lipid Emulsion 20% rapidly over 2-3 minutes • Lipid emulsion infusion 200-250 mL over 15-20 minutes	**Bolus 1.5 mL/kg Lipid Emulsion 20%** rapidly over 2-3 minutes • Lipid emulsion infusion ~0.25 mL/kg/min (ideal body weight)
If patient remains unstable: • Re-bolus once or twice at the same dose and double infusion rate; be aware of dosing limit (12mL/kg) • Total volume of lipid emulsion can approach 1 L in a prolonged resuscitation (e.g., > 30 minutes)	

- Continue monitoring
  - At least 4-6 hours after a cardiovascular event
  - Or, at least 2 hours after a limited CNS event
- Do not exceed 12 mL/kg lipid emulsion (particularly important in the small adult or child)
  - Much smaller doses are typically needed for LAST treatment
- See reverse side of this checklist for further details

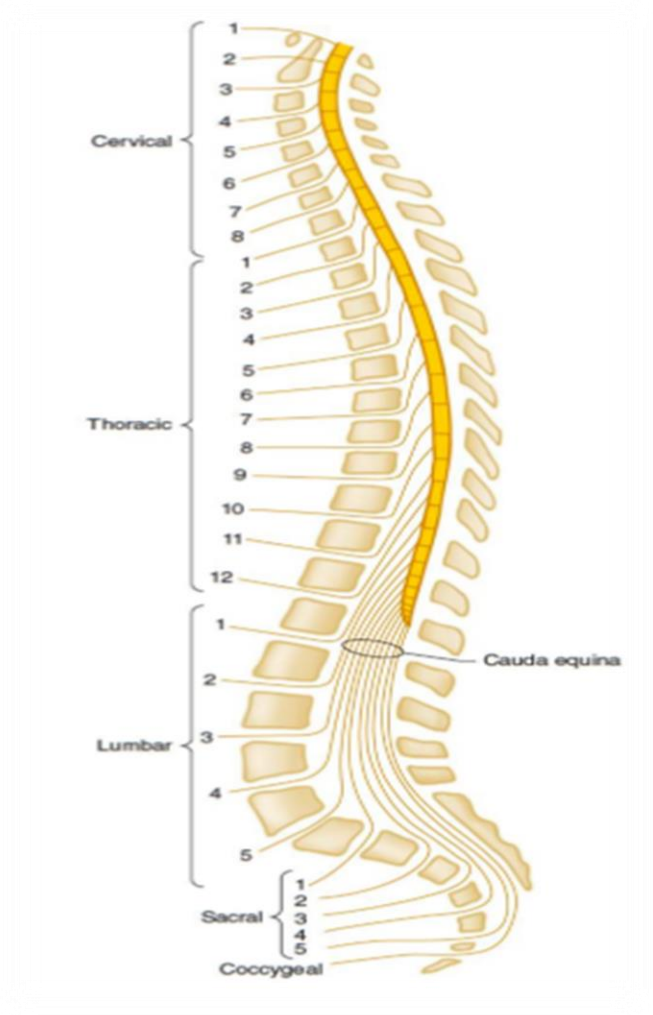
Indications and Contraindications

- **No absolute indications**
- **Contraindications**

  - **Patient refusal**
  - **Coagulopathy**
  - **Hemodynamic instability**
  - **Fixed cardiac output disease such as HOCM , severe AS/MS**
  - **Infection at site of injection**

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Relevant anatomy



- Cervical and lumbar lordosis
- Thoracic and sacral kyphosis

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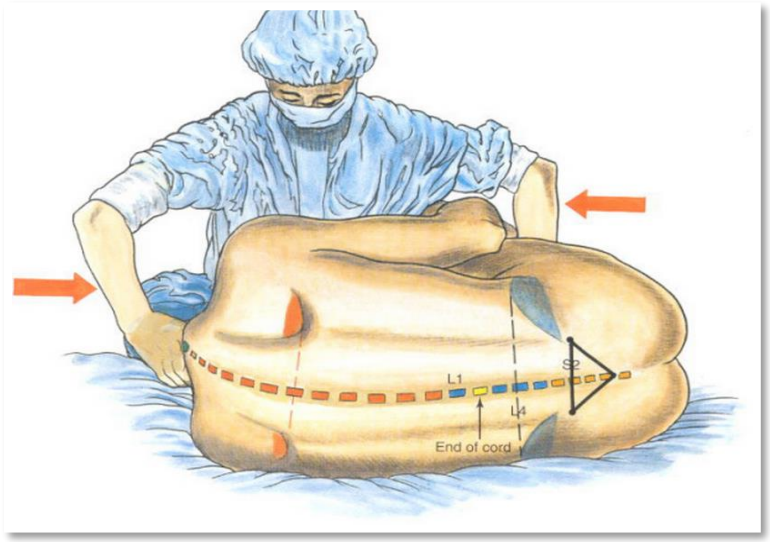
Relevant anatomy

➢ **Vertebrae**



- Many anesthesiologists use the line between the iliac crests (Tuffier line) to identify the L4–L5 interspace
- Obese patient, a midline skin crease and the gluteal cleft can help local the midline.

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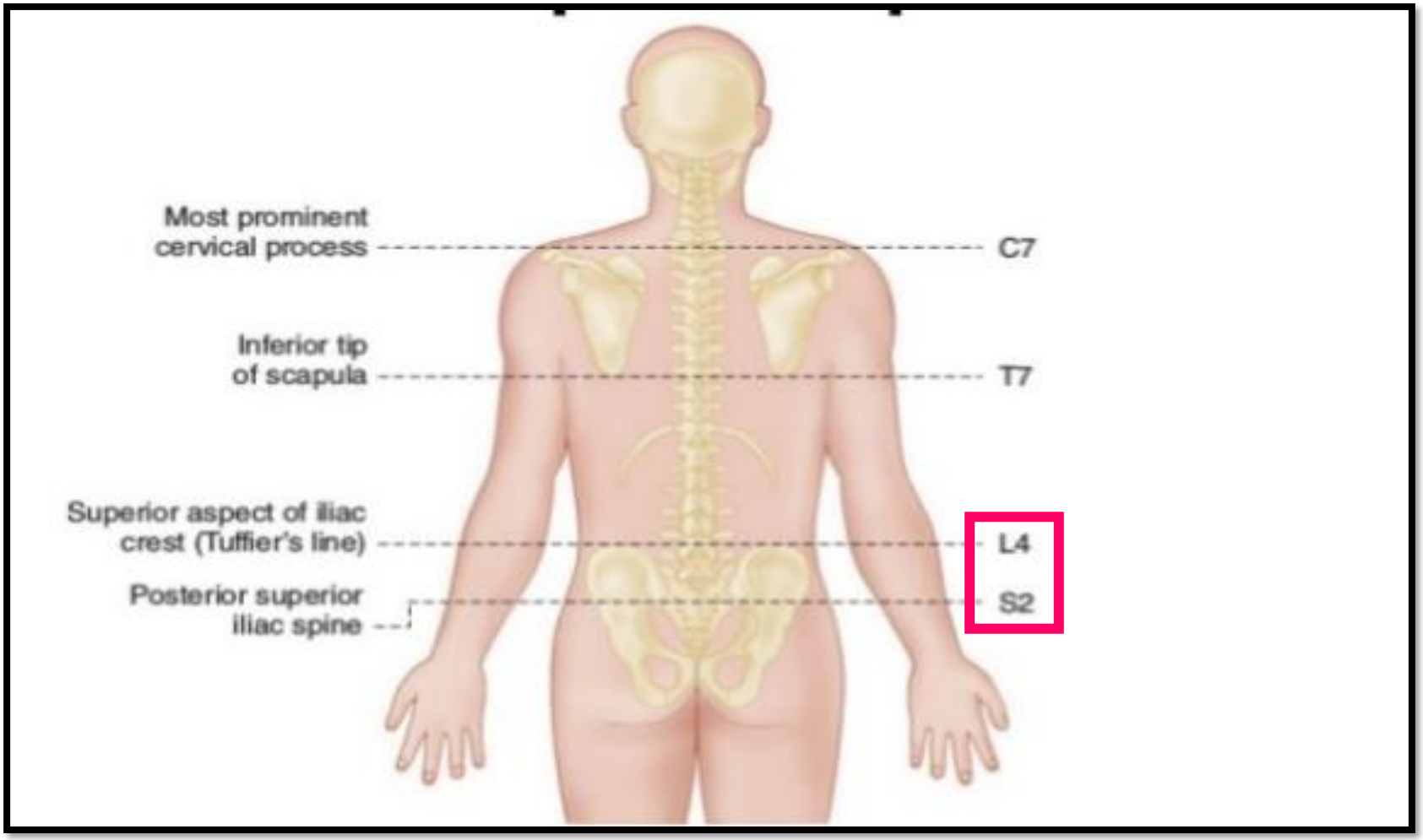
Relevant anatomy



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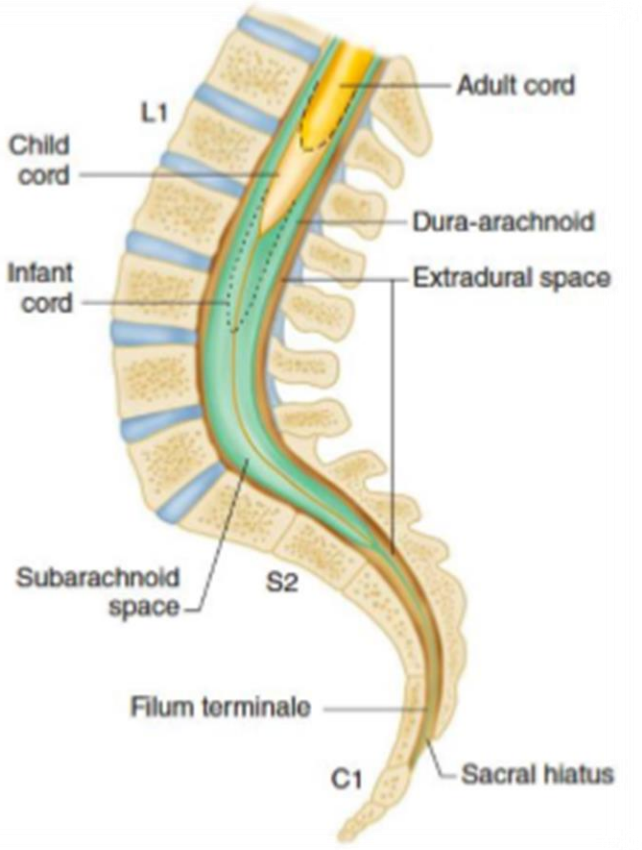
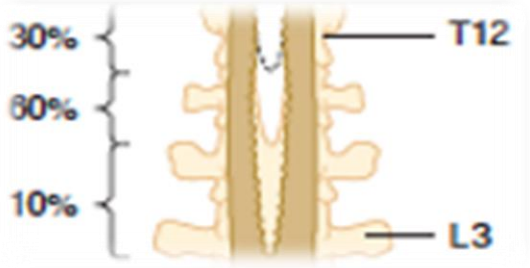
Relevant anatomy



- **The spinal cord terminates distally in the conus medullaris as the filum terminale and the cauda equine**
- **This distal termination varies from L3 in infants to the lower border of L1 in adults.**



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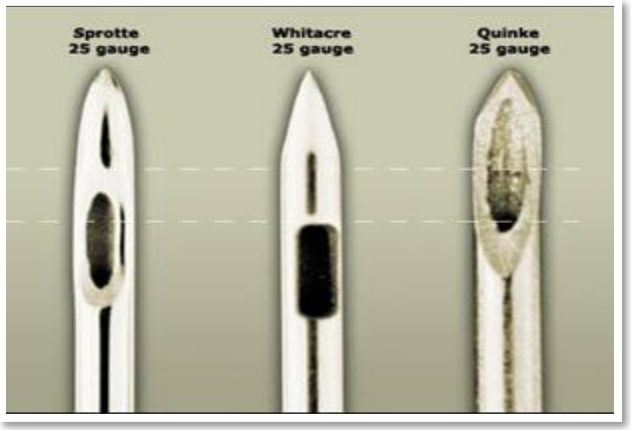
Technique of spinal anesthesia

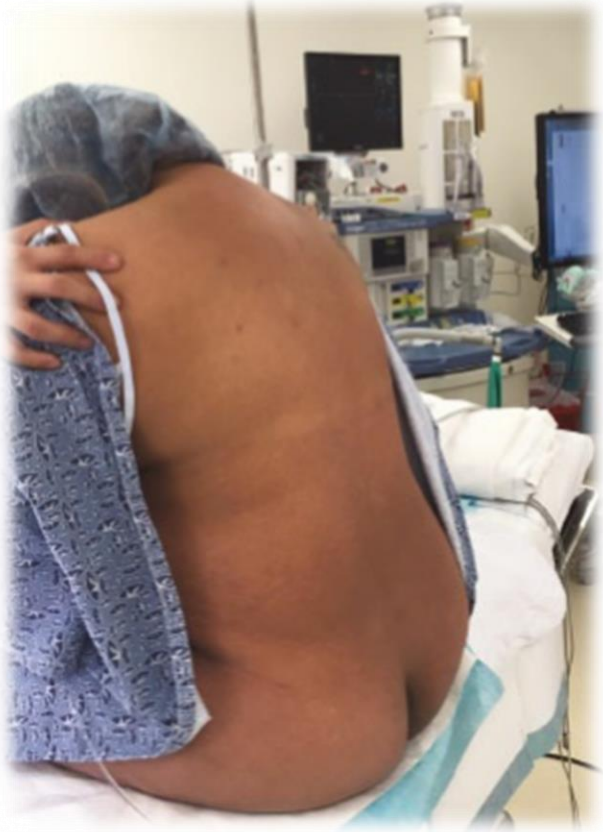
- Cutting/ traumatic
  - Quincke 
- Non-cutting/ atraumatic
  - Whitacre 
  - Sprotte

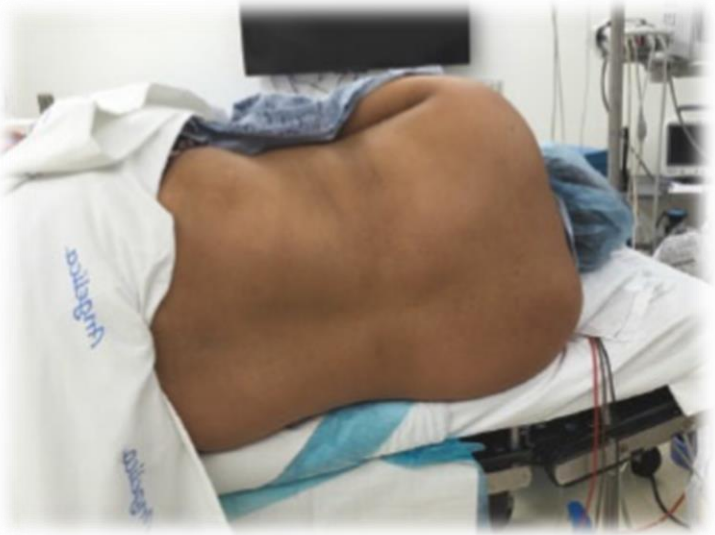


Amorim JA,Valença MM.Postdural puncture headache is a risk factor for new postdural puncture headache. *Cephalalgia.* 2008;28:5-8.

Technique of spinal anesthesia

- **Positioning**





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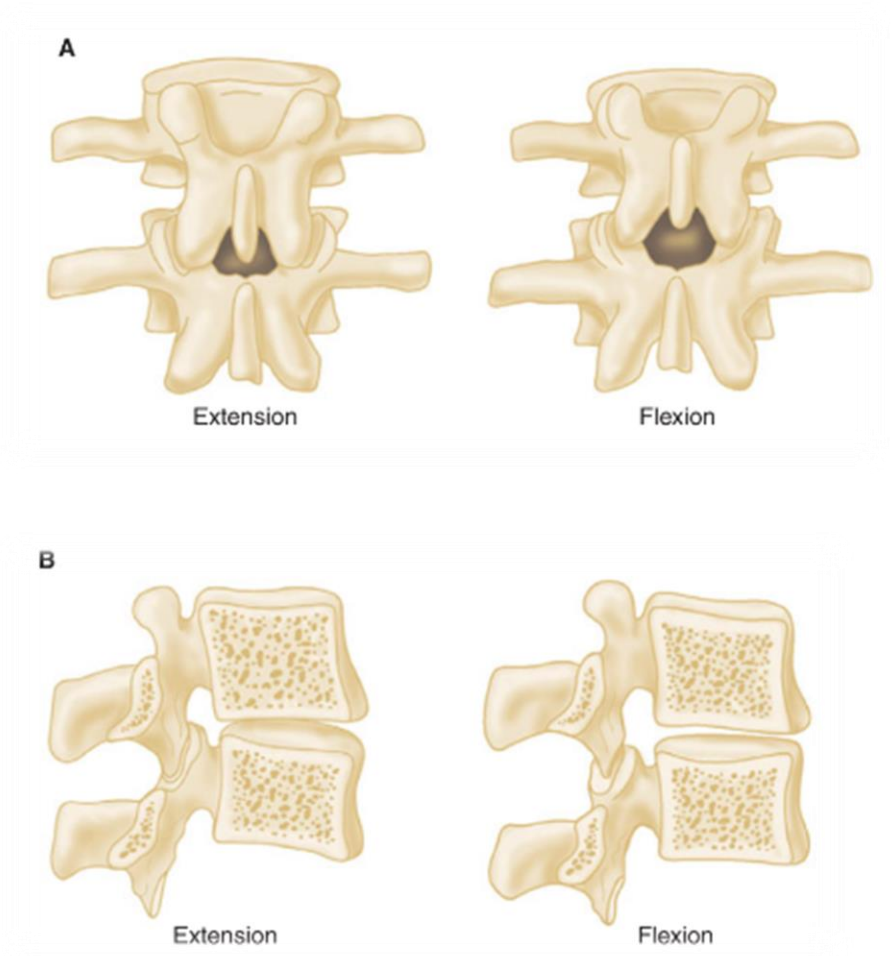
Technique of spinal anesthesia



Morgan & Mikhail's clinical anesthesiology. 5th ed. McGraw-Hill Education; 2013

Technique of spinal anesthesia

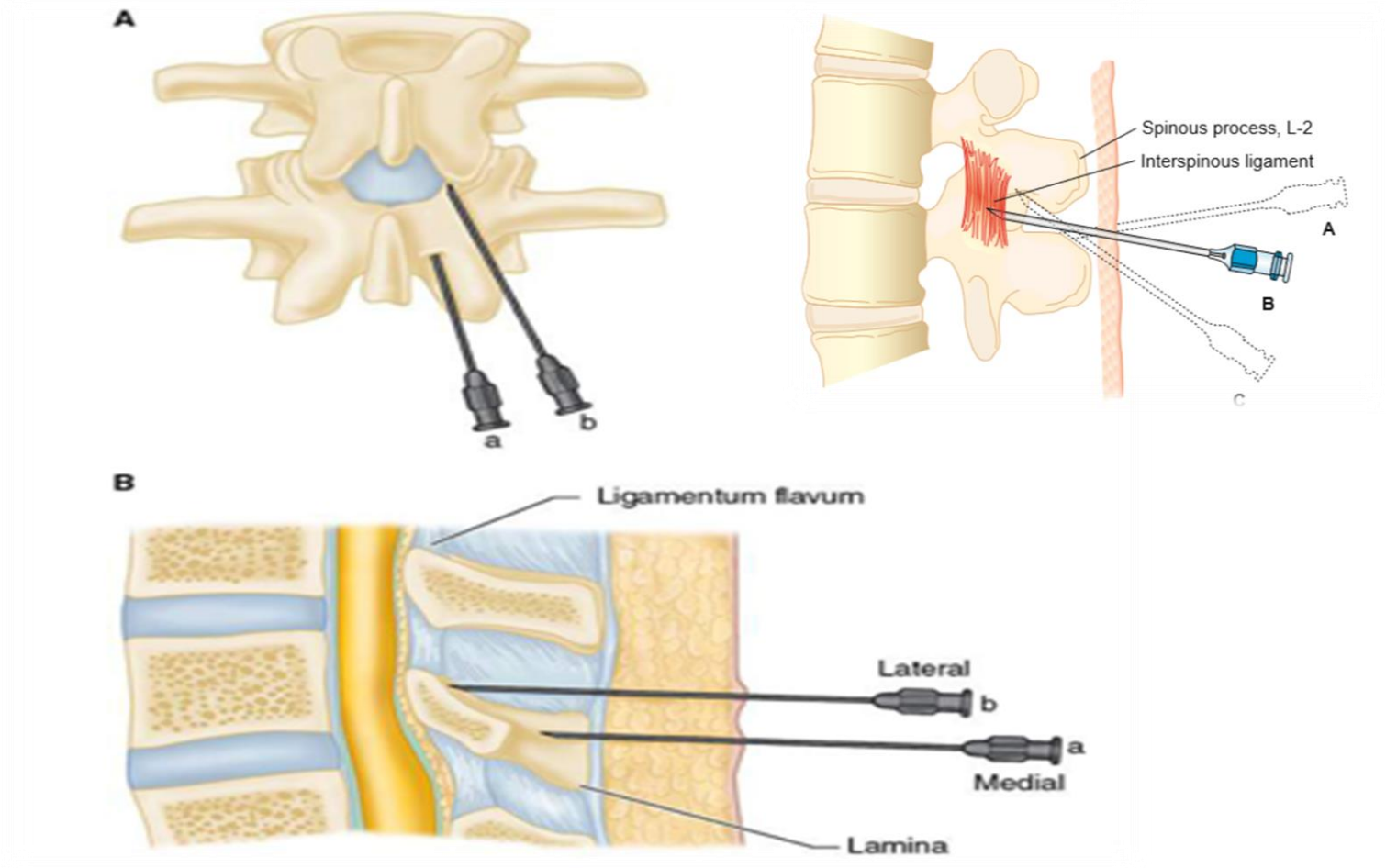
- **Skin Preparation**

  - **American Society of Anesthesiologists (ASA) recommends removing jewelry, hand washing, sterile gloves, caps, and masks**
  - **Both chlorhexidine with alcohol and povidone–iodine with alcohol provide effective skin decontamination**

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Technique of spinal anesthesia

- **Approach**



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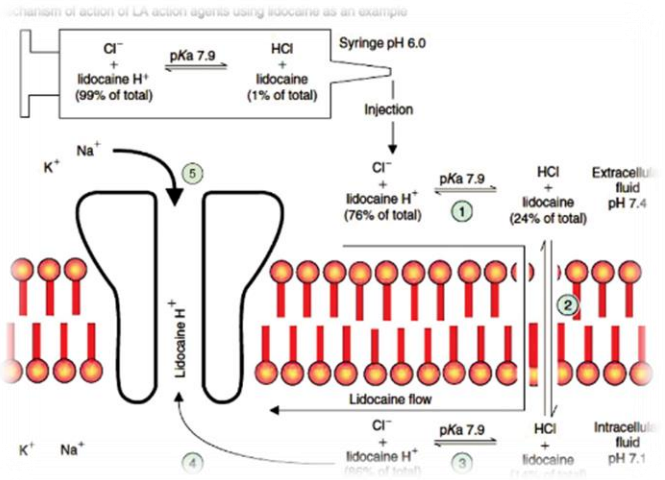
Pharmacology

➢Mechanism of action



- ❖ LA diffuses from CSF through pia mater to dorsal root ganglion
- ❖ Action : nerve root ganglion

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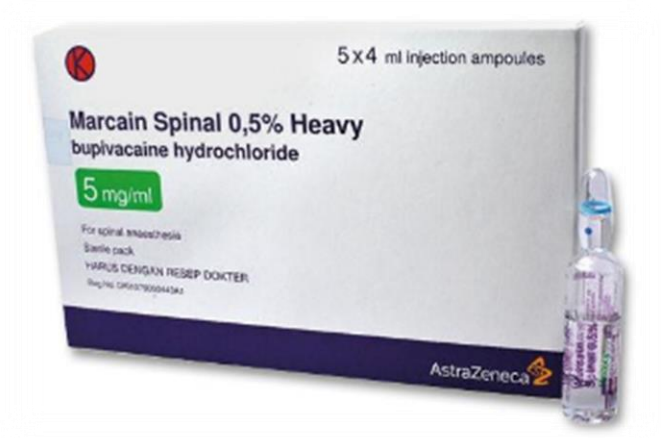
Pharmacology

➢Local anesthetic agent



- **Baricity** is defined as the ratio of the density (mass/ volume) of the local anesthetic solution divided by the density of CSF
- CSF density 1.0003 ± 0.0003 g/mL at 37°C.
- **Isobaric** : Solutions that have the same density as CSF have a baricity of 1
- **Hyperbaric** : Solutions that are denser than CSF
- **Hypobaric** : Solutions that are less dense than CSF

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Pharmacology

➢Local anesthetic agent

TABLE 34-3. BARICITY OF SOLUTIONS COMMONLY USED FOR SPINAL ANESTHESIA

	Baricity[a]
Hyperbaric	
Tetracaine: 0.5% in 5% dextrose	1.0133
Bupivacaine: 0.75% in 8.25% dextrose	1.0227
Lidocaine: 5% in 7.5% dextrose	1.0265
Procaine: 10% in water	1.0104
Isobaric[b]	
Tetracaine: 0.5% in normal saline	0.9997
Bupivacaine: 0.75% in saline	0.9988
Bupivacaine: 0.5% in saline	0.9983
Lidocaine: 2% in saline	0.9986
Hypobaric	
Tetracaine: 0.2% in water	0.9922
Bupivacaine: 0.3% in water	0.9946
Lidocaine: 0.5% in water	0.9985

✓ **Hyperbaric solutions are typically prepared by mixing the local anesthetic in 5% to 8% dextrose.**

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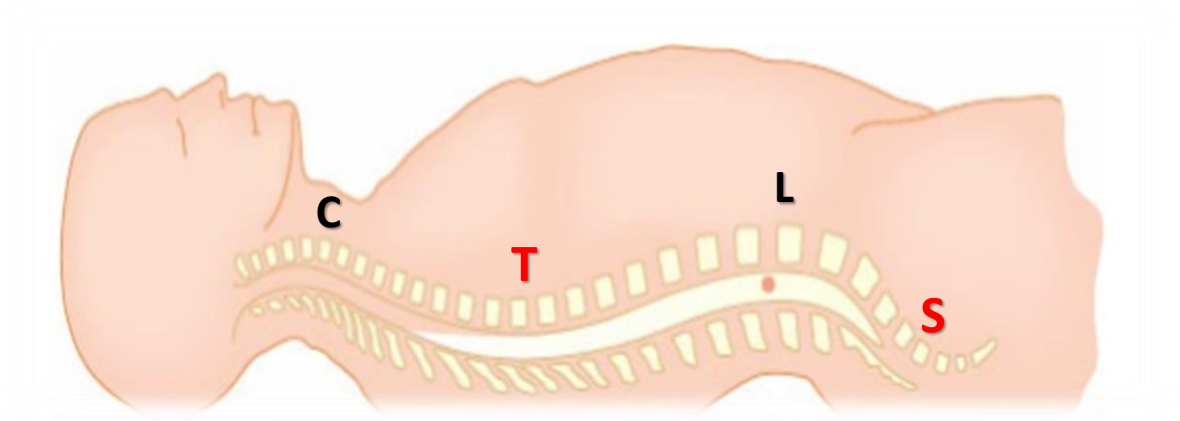
Pharmacology

- **Spreading of local anesthetic agent**



- **Baricity and dose are the two most important factors that determine the spread and duration of subarachnoid anesthesia.**

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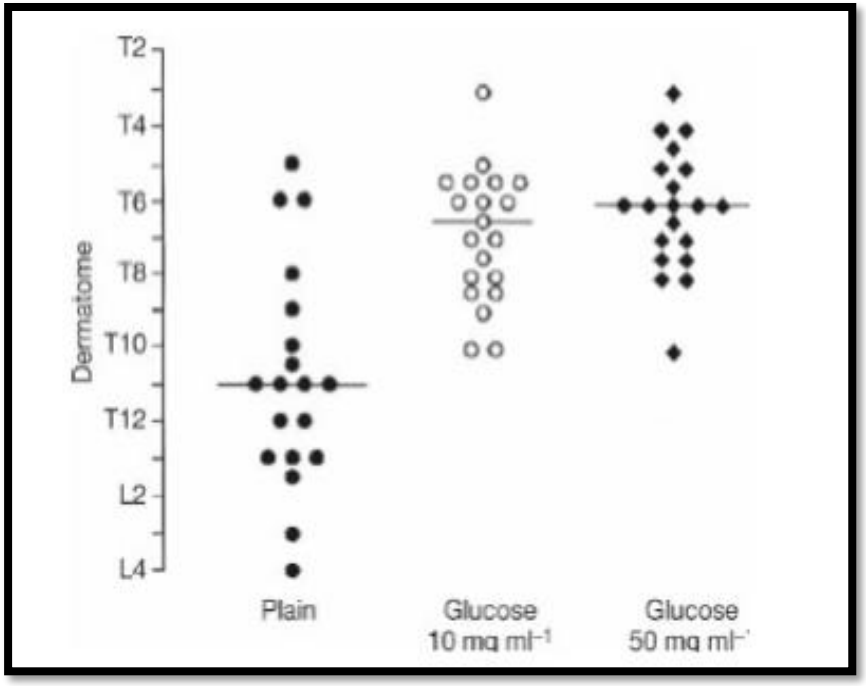
Pharmacology

- **Spreading of local anesthetic agent**



- Nonpregnant patients, **hyperbaric** local anesthetics **produce more** consistent **levels of sensory block** than isobaric drug

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Pharmacology

Procedure	Level	Local Anesthetic	Comments
Obstetrics			
Cesarean section	≥ T6	12–15 mg hyperbaric bupivacaine[a]	Lower doses may produce less hypotension but should be used as part of a combined subarachnoid epidural technique. Adding a lipophilic opioid may improve intraoperative analgesia.
Postpartum tubal ligation (mini laparotomy)	≥ T8	10–12 mg hyperbaric bupivacaine	
Cervical cerclage	≥ T10	5–7.5 mg hyperbaric bupivacaine	Faster discharge with smaller doses
Orthopedics			
Hip fracture or replacement	≥ T12	15–20 mg isobaric[b] bupivacaine	Sitting or lateral position with the operative side *up*
Knee replacement	≥ T12	12–15 mg hyperbaric bupivacaine	Higher level if tourniquet used
Knee arthroscopy	≥ T12	5–7.5 mg either isobaric or hyperbaric bupivacaine	Faster discharge with smaller dose. Lipophilic opioid may help with tourniquet pain. Some use 40 mg preservative-free 2-chloroprocaine for outpatient procedures.
Ankle surgery	≥ T12	At least 7.5 mg hyperbaric bupivacaine	Dose depends on expected duration of procedure. Combined subarachnoid epidural can add postoperative analgesia.
Urology			
Cystoscopy	≥ T10	2.5–5.0 mg isobaric or hyperbaric bupivacaine	Faster discharge with smaller dose
Trans urethral resection of bladder tumor or prostate	≥ T10	At least 7.5 mg isobaric or hyperbaric bupivacaine	Adjust dose according to expected duration of surgery.
Penile prosthesis	≥ T10	12–15 mg hyperbaric bupivacaine	
General Surgery			
Inguinal hernia repair/open appendectomy	≥ T8	12–15 mg hyperbaric bupivacaine	
Perianal/perirectal	Sacral	5–7.5 mg hyperbaric bupivacaine (lithotomy position)	Smaller doses of isobaric bupivacaine (2.5–5 mg) can be used if the patient will be prone.

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Pharmacology

- **Onset of spinal anesthesia**

- **Most patients can sense the onset of spinal block within a very few minutes after drug injection regardless of the local anesthetic used**
- **Peak block height : lidocaine 10-15 min , bupivacaine > 20 min**

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Pharmacology

- **Duration of spinal anesthesia**

  - **Local anesthetic agent**
  - **Drug dose**
  - **Block height**
  - **Adrenergic Agonists**
  - **Opioid**

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Pharmacology

- **Duration of spinal anesthesia**

- **Type of local anesthetic agent : Bupivacaine,tetracaine > lidocaine,mepivacaine> procaine**

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Pharmacology

- **Duration of spinal anesthesia**

➢ **Increasing local anesthetic dose clearly increases the duration of spinal block**

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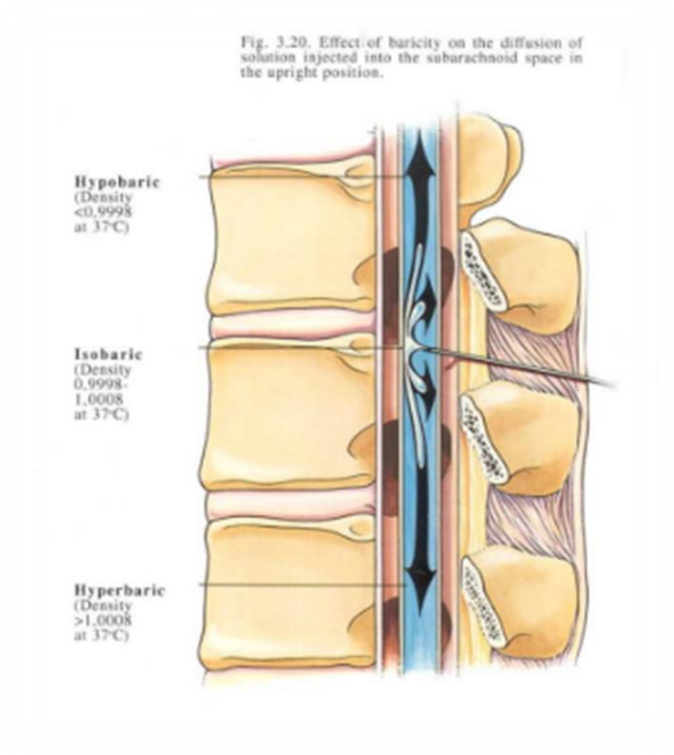
Pharmacology



- If drug dose is held constant, higher blocks tend to regress faster than lower blocks
- Consequently, isobaric local anesthetic solutions will generally produce longer blocks than hyperbaric solutions using the same dose.

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Pharmacology

- Adrenergic agonists, such as epinephrine is added to local anesthetics in an effort to prolong the duration of spinal anesthesia.
- More effective at prolonging block in the lumbar and sacral dermatomes than in thoracic dermatomes
- Mechanism : vasoconstrictor-mediated decreased drug clearance via the dural vasculature.

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Pharmacology

- Spinal administration of opioids provides analgesia primarily by attenuating C-fiber nociception and is independent of supraspinal mechanisms
- Coadministration of opioids with neuraxial local anesthetics results in synergistic analgesia
- Intrathecal morphine can provide prolonged (12 to 24 hours) postoperative analgesia but side effects, including itching and nausea and vomiting, are common and challenging to treat
- Intrathecal morphine produce delayed respiratory depression

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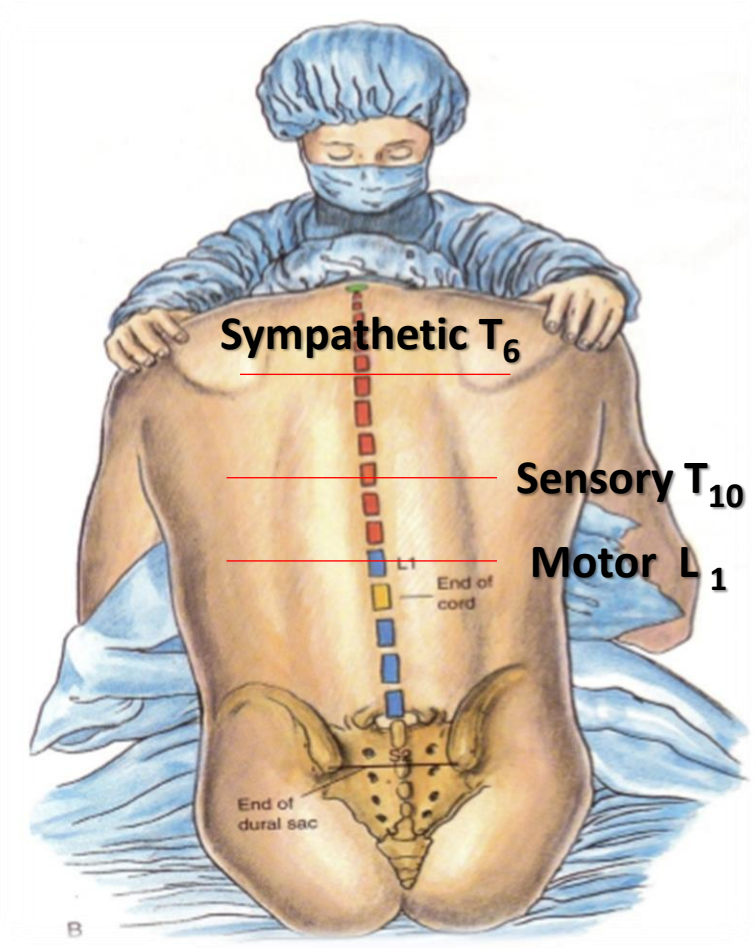
Physiologic changes during spinal anesthesia

- **Neurophysiology**



Differential nerve Block

- Sympathetic 2-6 dermatomes higher than the sensory block
- Motor 2 dermatomes lower than sensory block
- Sympathetic nerve fiber > pain > touch > motor

- Spatial separation is believed to result from a gradual decrease in local anesthetic concentration within the CSF as a function of distance from the site of injection.

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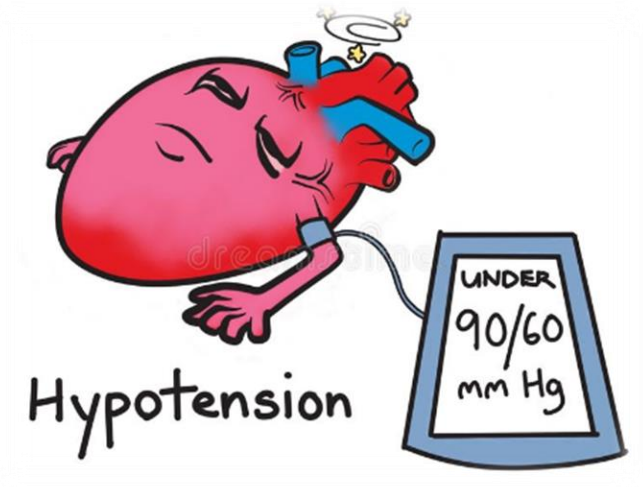
Physiologic changes during spinal anesthesia

- **Cardiovascular Physiology**



- **Blockade of sympathetic efferents is the principal mechanism by which spinal anesthesia produces cardiovascular derangements.**
- **Hypotension during spinal anesthesia is the result of both arterial and venodilation.**
- **This fall in preload is thought to be the principal cause of decreased cardiac output during high spinal anesthesia.**

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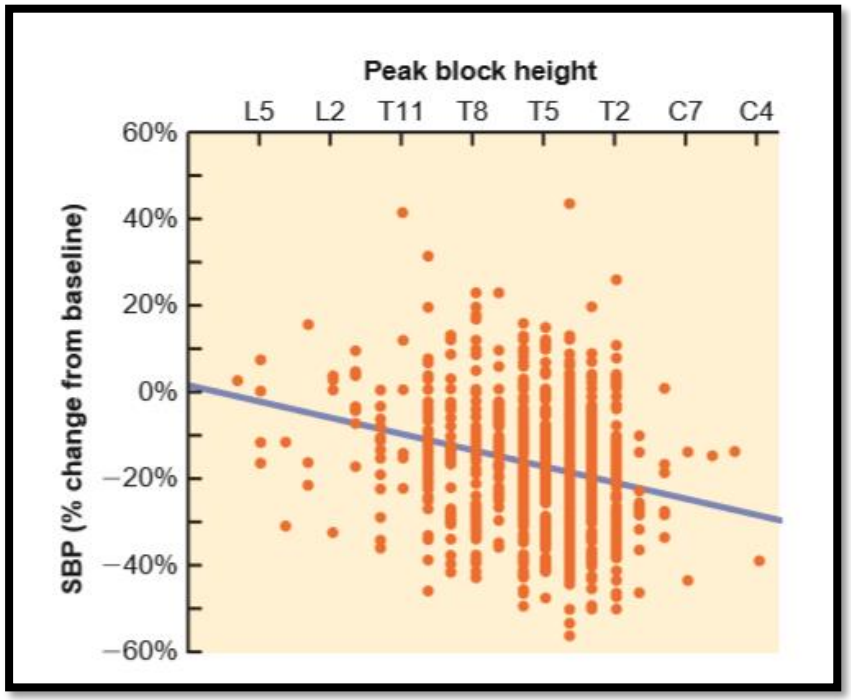
Physiologic changes during spinal anesthesia

- **Cardiovascular Physiology**



- **Statistically significant correlation between block height and decrease in systolic blood pressure**
- **Additional risk factors associated with hypotension include age >40 to 50 years, concurrent general anesthesia, obesity, hypovolemia**

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Physiologic changes during spinal anesthesia

- **Cardiovascular Physiology**

- Blockade of the sympathetic cardioaccelerator fibers originating from T1 to T4 spinal segments is often suggested as the cause
- Bezold–Jarisch reflex central volume depletion elicited a vagally mediated reflex slowing of heart rate
- Additional risk factors associated with bradycardia include age younger than 50 years, ASA 1 , concurrent use of beta-blockers

Spinal anesthesia can also produce second and third-degree heart blocks and that preexisting first-degree block may be a risk factor for progression to higher grade blocks during spinal anesthesia.

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Physiologic changes during spinal anesthesia

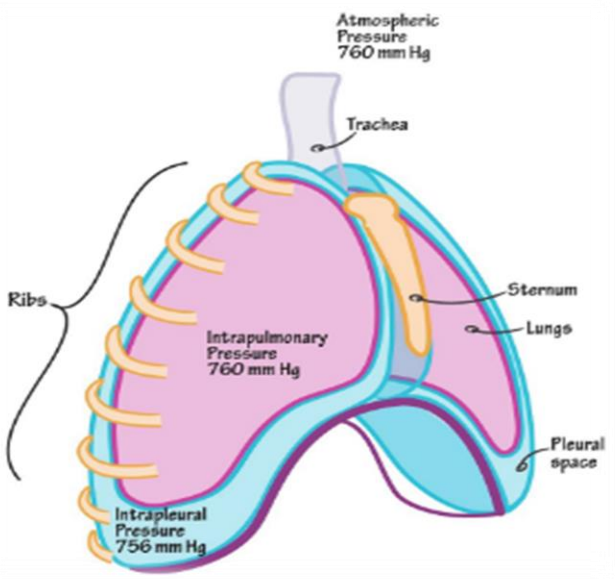
- **Treating Hemodynamic Changes**

  - **Prehydrating crystalloid administration has often been advocated to restore venous return and thus cardiac output during central neuraxial blockade but not reliably prevent hypotension**
  - **Colloid infusion is more effective, but hypotension is still common.**
  - **Ephedrine and phenylephrine are the most commonly used vasopressors for hypotension associated with neuraxial anesthesia**
  - **Vasopressor : SBP if it decreases more than 25% to 30% below baseline or normotensive patients, if SBP falls below 90 mm Hg**

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Physiologic changes during spinal anesthesia

- **Respiratory Physiology**

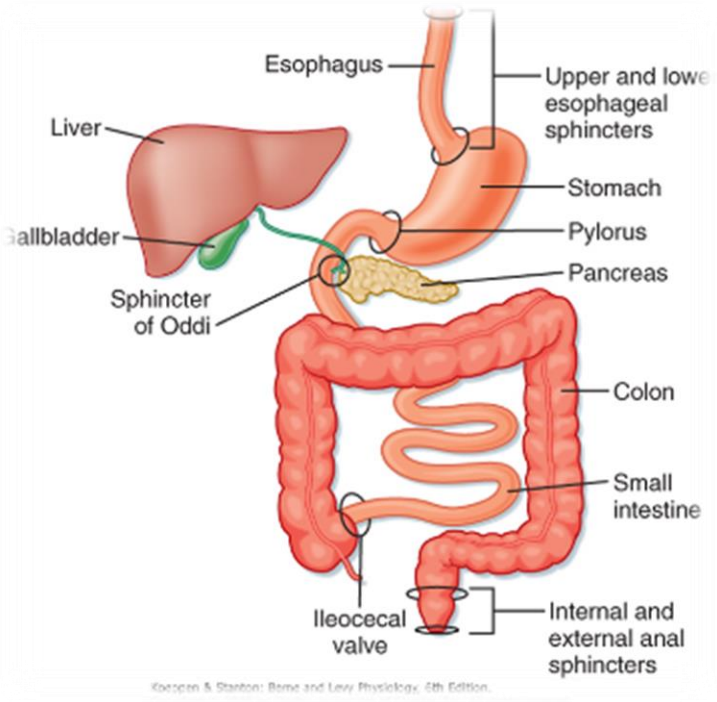


- **Spinal blocks to midthoracic levels have little effect on pulmonary function in patients without preexisting lung disease.**
- **Lung volumes, resting minute ventilation, dead space, arterial blood gas tensions, and shunt fraction show little or no change during spinal**
- **High blocks associated with abdominal and intercostal muscle paralysis can impair ventilatory functions requiring active exhalation**

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Physiologic changes during spinal anesthesia

- **Gastrointestinal Physiology**

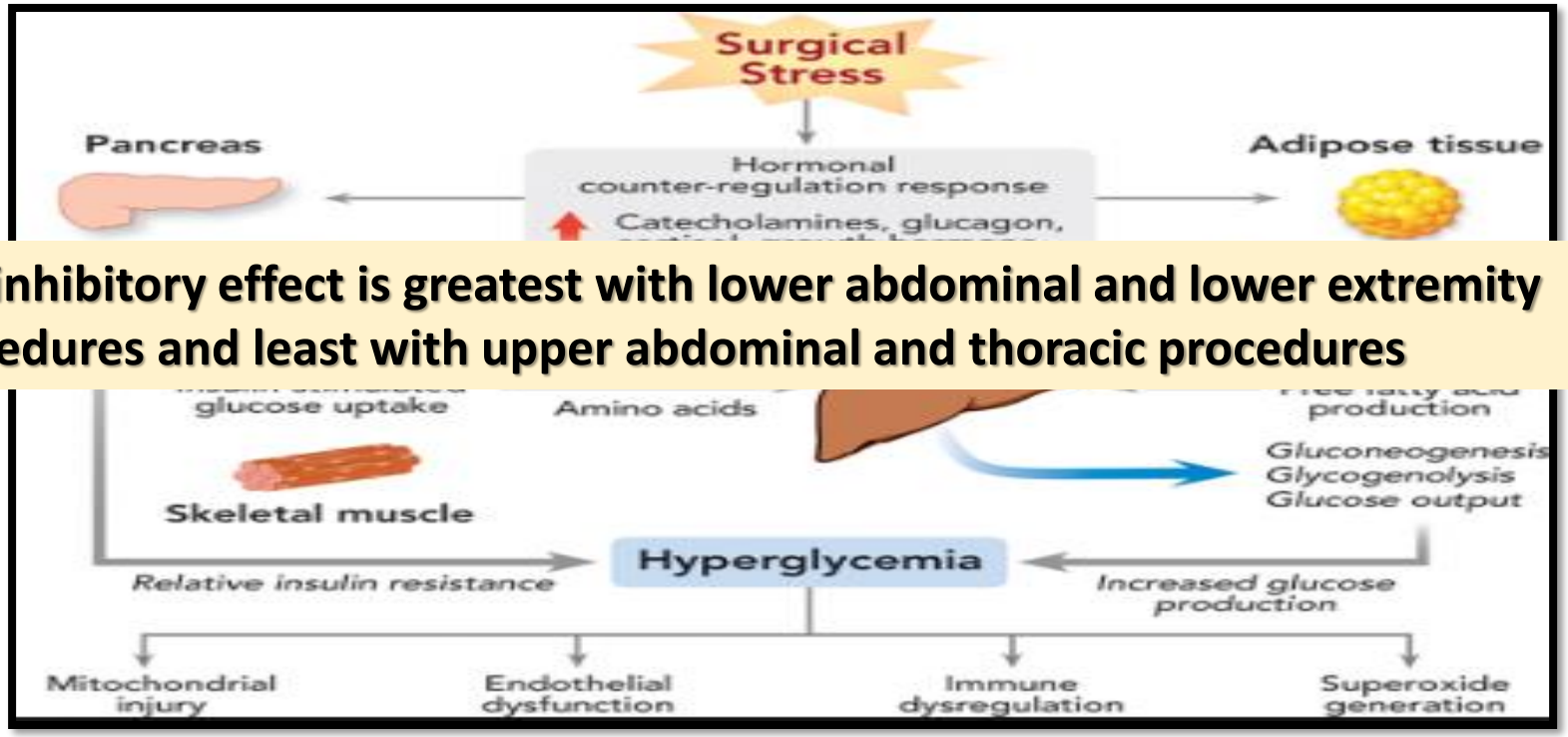


- **Abdominal organs derive their sympathetic innervation from T6 to L2**
- **Nausea is a common complication of spinal anesthesia**
- **Risk : blocks higher than T5, hypotension, opioid premedication, and a history of motion sickness**

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Physiologic changes during spinal anesthesia

- **Endocrine–Metabolic Physiology**



The inhibitory effect is greatest with lower abdominal and lower extremity procedures and least with upper abdominal and thoracic procedures

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Spinal block monitoring

- Monitor HR , BP typically 2 to 5 minutes for 15-20 minutes after spinal anesthesia
- Cold sensation and pinprick sensation
- Loss of sensation to cold is the most reliable method determine level of spinal anesthesia

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.
Miller's Anesthesia, 7th edition 2015.

Complication of spinal anesthesia

- **Backache**

- **The etiology of backache is not clear, although needle trauma, local anesthetic irritation, and ligamentous strain secondary to muscle relaxation have been offered as explanations.**

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Complication of spinal anesthesia

- **Postdural Puncture Headache**

- Typically appears or worsens when the patient moves from a supine to an upright position and is typically relieved when the patient lies down.
- Not develop immediately after dural puncture but 24-48 hours after the procedure with 90% of headaches presenting within 3 days.
- Quality may be burning, dull and/or throbbing at frontal or occipital with radiation to the neck and shoulders but temporal areas less common

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Complication of spinal anesthesia

- **Postdural Puncture Headache**

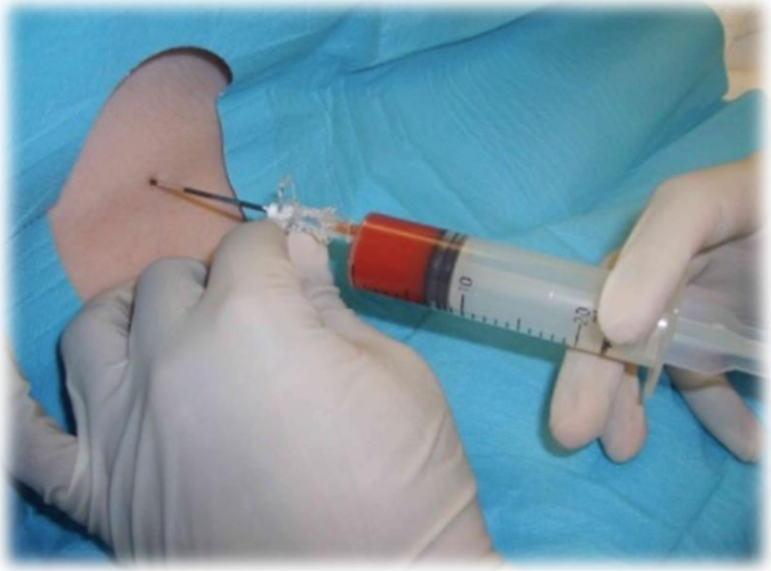
Risk factor

- Age
- Female gender
- Low body mass index (BMI)
- History of prior PDPH
- History of chronic headache
- Size of the spinal needle
- Bevel orientation and angle of insertion
- Stylet replacement
- Operator experience

Amorim JA,Valença MM.Postdural puncture headache is a risk factor for new postdural puncture headache. *Cephalalgia.* 2008;28:5-8.

Complication of spinal anesthesia

- **Postdural Puncture Headache**



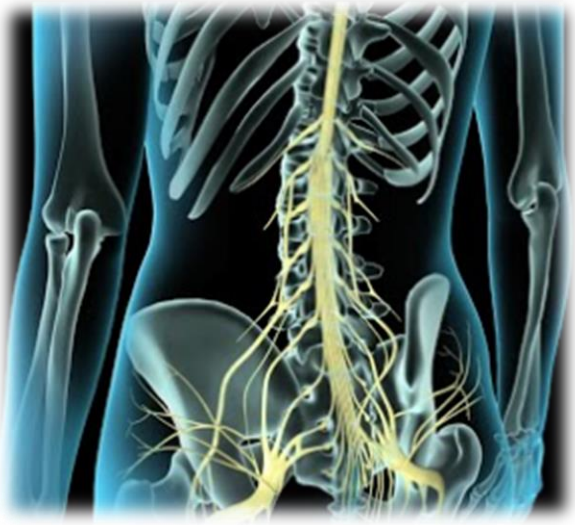
Treatment

- **Bed rest ???**
- **Oral or intravenous hydration has any effect on PDPH**
- **Acetaminophen, nonsteroidal anti-inflammatory drugs , and opioids can be effective**
- **Caffeine may relieve the symptoms of PDPH but the data supporting its use are weak.**
- **Epidural blood patch is the definitive therapy for PDPH**

Barash PG, eds. Clinical Anesthesia 8th ed. Philadelphia: Lippincott Williams & Wilkins;2017.

Complication of spinal anesthesia

- **Total spinal anesthesia**



- Spreads high enough to block the entire spinal cord and occasionally the brainstem
- Profound hypotension and bradycardia are common secondary to complete sympathetic blockade
- Respiratory arrest may occur as a result of respiratory muscle paralysis or dysfunction of brainstem respiratory control centers.
- Mx : supportive , vasopressor , atropine ,IV fluid

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Thank you for your attention