Episode 41: Local Anesthetics

On this episode: Dr. Jed Wolpaw

In this episode, episode 41, I review local anesthetics including the mechanism of action, commonly used agents, pharmacodynamics and kinetics, toxicity and treatment, and common blocks.

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Local Anesthetics Overview

0 – 3:22

- Weak bases used to block nerve conduction → block sensory +/- motor
- Work through Na^+ channel blockade from inside nerves
    - Easiest when channel is activated → nerves in use more often are more sensitive to local anesthetics because in activated configuration more often
- Two basic classes: amino-amides and amino-esters

Amino-amides	Amino-esters
- Have amide link between intermediate chain and aromatic end - Metabolized in liver - Very stable in solution	- Have ester link between intermediate chain and aromatic end - Metabolized in plasma via pseudocholinesterase - Unstable in solution - More likely to cause allergic hypersensitivity reactions
- Eg. Lidocaine, mepivacaine, prilocaine, bupivacaine, etidocaine, ropivacaine, levobupivacaine - Memory tip! all these words have 2 "l"s and amide has an "l"	- Eg. cocaine, procaine, tetracaine, chloroprocaine, benzocaine

Motor versus Sensory block

3:23 – 4:23

- Sensory comes first, especially with bupivacaine and ropivacaine
- Old reasoning is that local anesthetics affect small diameter c-fibers first → NOT TRUE
- Get sensory without motor by using less local anesthetics (↓ concentration, ↓ volume)

Pharmacokinetics

4:24 – 7:58

- **Onset of action** is determined by pKa → local anesthetics are weak bases with NH_3 group
    - More acidic pH → more H^+ ions → get NH_4^+
    - More acidic pH → less H^+ ions → remain as NH_3
- Higher pKa → more charged at physiological pH → slower onset of action as harder for charged particles to get into cells
    - pKa < pH of environment → remain uncharged
    - pKa > pH of environment → get H^+ ion more often
    - Eg. lidocaine has low pKa → fast onset of action
    - Eg. chloroprocaine has higher pKa than lidocaine, but used in obstetrical anesthesia when want faster block → has low toxicity so can use high concentrations (3% chloroprocaine vs. 1.5-2% lidocaine)
- **Potency**: at given pH, more lipid soluble → more potent
- **Duration of action:** ↑vasoconstriction and ↑ protein binding → longer duration

Types of Anesthetics

7:59 – 10:23

Type of Local Anesthetic	Onset	Max Dose		Duration of Action	
		Without epi (mg/kg)	With epi (mg/kg)	Without epi (hours)	With epi (hours)
Lidocaine	Rapid because low pKa	4.5	7	2	4
Mepivacaine	Rapid	5	7	3	6
Bupivacaine	Slow	2.5	3	4	8
Ropivacaine	Medium	2-3	No effect	3	6
Chloroprocaine	Rapid	10	15	30mins	60-90mins

Additives to Local Anesthetics

10:24 – 11:45

- **Bicarbonate** could increase speed of onset, but too much cause precipitate
    - Usual amount used is 1meq into 9 mL of local anesthetic
- Epinephrine increase duration of action because counteract vasodilatory properties of local anesthetic
    - Only local anesthetic without vasodilatory effects is cocaine as it is a vasoconstrictor
    - Centrally, have α2 receptors in spinal cord → epinephrine increases action of anesthesia

Absorption of Local Anesthetics

11:46 – 12:32

- Order of highest to lowest absorption: intercostal space > caudal epidural > lumbar and thoracic epidural > brachial plexus > subcutaneous

Toxicity and Side Effects

12:33 – 17:12

- CNS is affected first
    - Lightheadedness, dizziness → visual and auditory disturbances (eg. tinnitus)
- Cardiovascular affected later
    - ↓ rate of depolarization in fast conduction fibers of Purkinje fiber and ventricular muscle → bradycardia
    - Depress spontaneous pacemaker activity in sinus node → sinus bradycardia → arrest
- Treatment of cardiovascular collapse:
    - Start ACLS
    - 1.5ml/kg bolus of intralipid 20% followed by 0.25mL/kg/min infusion for 10 minutes
- Ropivacaine is S-isomer of bupivacaine → thought to be less cardiotoxic than bupivacaine
- Other effects:
    - Hypotension from high epidural or spinal block
    - Methemoglobinemia → prilocaine is worst offender at about doses > 600mg
    - Transient neurological symptoms (TNS) → more common with spinal lidocaine, incidence 13%, relative risk compared to prilocaine, bupivacaine, ropivacaine is 4

- Painful buttocks and thighs ± radiation to lower extremities
- Begins few hours after spinal anesthesia
- Lasts as long as 10 days, usually shorter
- No bowel or bladder dysfunction
- All electrophysiological studies are normal
- No studies showed permanent effects

Common Blocks

17:13 – 24:15

- Peripheral blocks:
  - 1.5-2% mepivacaine for short blocks (eg. upper extremity for AV fistula)
  - 0.5% ropivacaine or bupivacaine for longer blocks (eg. brachial plexus, femoral block)
    - Lasts up to 12 hours for ropivacaine and up to 16 hours for bupivacaine
    - Amounts:
      - Femoral → 20cc
      - Popliteal → 25-30cc
      - Saphenous → 20cc
      - Interscalene → 20-30cc
      - Supraclavicular → 30-40cc
      - Axillary → 30-40cc
      - Transversus abdominis plane → dilute to 0.25% bupivacaine and use 30cc each side
      - Paravertebral → 5cc per level
  - Catheters: (eg. popliteal catheter)
    - 0.2% ropivacaine at 8-12mL/hour + 2-3cc demand dose
- Spinals:
  - C-section spinals: 8 to 12mg of bupivacaine ± morphine and fentanyl to increase duration of action ± epinephrine to increase depth and duration of action
  - Non-C-section spinals: 0.75% bupivacaine 5mg to 22.5mg depending on length of surgery and levels
    - Hypobaric may travel up and cause high spinal
    - Avoid lidocaine and mepivacaine because increased risk of TNS
- Epidurals:
  - Labour epidurals: 2% lidocaine or 2-3% chloroprocaine
    - 10 to 15 cc bolus; then, titrate to effect
  - Lumbar epidurals: 1.5cc per level for 20 year old
  - Thoracic epidurals: 1cc per level for 20 year old
  - Rule of thumb: need 1% less per year
    - Eg. 50 year old patient will need 50% less
  - Rule of thumb: obesity and pregnancy will reduce dose by 1/3