Common Anesthesia Drugs

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Going over every single drug that can be used in an anesthetic would take an incredible amount of time. (Almost 3 years actually)

I’m going to include a good deal of information in the power point. We will not be talking about all of the drugs in depth. Please note this is NOT a complete list of drugs we will use, but an attempt to highlight common meds.

The information is provided so you may reference it later if you have questions.

Our plan today, is for me to briefly go over some of the major drugs, and try to answer any questions you may have.

Drug information provided here should not be considered complete.
Actions

Surrogate measures of pain suggest that, with the exception of nitrous oxide, inhaled anesthetics do not provide any significant analgesia. They do, however, produce immobility and amnesia. Other than nitrous oxide (which increases skeletal muscle tone), inhaled anesthetics either do not affect, or in some cases lower skeletal muscle tone.

Mechanism of Action

Inhaled anesthetics produce immobility via actions on the spinal cord [Campagna JA et al. N Engl J Med 348: 2110, 2003]. There is consensus that inhaled anesthetics produce anesthesia by enhancing inhibitory channels and attenuating excitatory channels, but whether or not this occurs through direct binding or membrane alterations is not known. [Miller]

Minimum Alveolar Concentration

The Minimum Alveolar Concentration (MAC) of an inhaled anesthetic is the alveolar (or end-expiratory) concentration at which 50% of patients will not show a motor response to a standardized surgical incision. The standard deviation of MAC is ~10%, thus 95% of patients will respond to 1.2 MAC, and 99% will respond to 1.3 MAC. Standard MAC values assume the absence of all other potentially sedative or hypnotic drugs. According to rat data, MAC values are additive in terms of preventing movement to incision (0.5 MAC of nitrous oxide plus 0.5 MAC of isoflurane = 1.0 MAC of any other agent) [Rampil IJ Anesthesiology 80: 606, 1994]
Anesthesia Gas

**ISOFLURANE**
Anesthesia Gas

› Desflurane, Isoflurane, Sevoflurane, Nitrous

Each of these gases have specific nuances way beyond the scope of a single talk. For example...
Anesthesia Gases

### Table 7–6. Clinical pharmacology of inhalational anesthetics.

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<tr>
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<th>Nitrous Oxide</th>
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<th>Isoflurane</th>
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<td>Metabolism(^{3})</td>
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<td>15-20%</td>
<td>0.2%</td>
<td>&lt; 0.1%</td>
<td>5%</td>
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1 N/C: no change.
2 Controlled ventilation.
3 Depolarizing blockade is probably also prolonged by these agents, but this is usually not clinically significant.
Anesthesia Gas

› The Quick and Simple...

› **Desflurane**: Airway irritant, (Stinks as well) can increase HR, very low solubility...meaning it is good for obese patients, absorbs quick, and goes away quick. VERY expensive. Needs a special heated vaporizer.

› **Sevoflurane**: Not irritable to the airway, good for pediatric induction, can cause emergence delirium in kids, middle of the line expensive, can hang around longer in obese patients. Has been indicated to cause nephrotoxicity in rats. Not proven in humans. Very commonly used.

› **Isoflurane**: Not used often, very lipid soluble (longer emergence), very cheap, still very effective if used correctly, very suitable for long cases where quick extubation is not expected. (Ex: Sick ICU patient going to stay on a ventilator)

› **Nitrous**: NOT a solo anesthesia gas. Used as an adjunct. Provides analgesia. Can be used in many different fashions. Allows decrease of primary anesthesia agents. Can be used to help wakeup. Has many benefits and downfalls. Most mentioned of these will be N/V, at higher doses. Not for use in neuro or most bowel cases.
### Factors which Increase Anesthetic Requirements

- Chronic ETOH
- Infant (highest MAC at 6 mo.)
- Red hair
- Hypernatremia
- Hyperthermia

### Factors which Decrease Anesthetic Requirements

- Acute ETOH
- Elderly Patients
- Hyponatremia
- Hypothermia
- Anemia (Hgb < 5 g/dL)
- Hypercarbia
- Hypoxia
- Pregnancy
Anesthesia Gas

› **Why gas?**
› Speed of onset.
› Titratable: Working on the very steep part of the dose response curve.
› Very potent: They do have a narrow therapeutic window.
› Little or no metabolism.
› Available Measure: Watching what the patient breaths in and out tells us what is in the patients brain.
Anesthesia Gas

› Brief Rundown...

› Differences...What to know...

› We don’t really know how they work...

› Provider preference...

› Pediatrics...(Sevo Vs. Des)
Propofol (Diprivan)

**Use:** IV induction of anesthesia, IV sedation and TIVA. (Street ready earlier, Less PONV)

**Preparation:** 10 mg/cc (20cc vial standard). 1% lipid solution

**Dose:**
- Induction = 2 mg/kg IV
- Sedation = 25 – 100 mcg/kg/min
- TIVA = 100 – 300 mcg/kg/min

*It is not a controlled substance. PAIN with IV pushes. 1-2% Lidocaine pretreatment works best. Doses rarely need to be changed with renal or liver disease. ↓dose in elderly patients. Propofol is very titratable.*

**Kinetics:** Patient is unconscious in 30 seconds. Elimination half-life = 0.5 to 1.5 hours. Protein Binding = 98%  Context Sensitive Half-Time is <40 minutes, does accumulate with long therapy.
Induction agents

- **Propofol continued...**
- Bronchodilator. Induction **drug of choice** in asthmatics
- Suppresses the SNS response to laryngoscopy; Best drug to blunt larynx
- Propofol has Anti Nausea properties, can be used as a rescue in low dose.
Induction agents

» Rare Problems with Propofol:
  » Critically ill adults with head injuries receiving long term (> 58 hours), high dose infusions (5 mg/kg/hr) tend to develop:
    » Lipidemia.
    » Fatty infiltrates of the liver.
    » Enlarged liver.
    » Metabolic acidosis.
    » Rhabdomyolysis.
    » Myoglobinuria.
  » Propofol Infusion Syndrome – sudden onset of bradycardia that progresses to asystole and is resistant to treatment. (Minerva Anestesiol. 2009 May;75(5):339-44)
**Induction agents**

- **Etomidate**
  - **Mechanism of Action**: GABA modulator (does not mimic – has to have GABA present). Carboxylated Imidazole = has a ring that is just like midazolam. The ring is open and water soluble in solution. The ring closes in the body and becomes very lipid soluble.
  - **Use**: Induction: Cardiovascular stability, induce seizure
  - **Preparation**: 0.2% solution
  - **Dose**: Adult dose = **0.3 mg/kg** There is no change for elderly or sick. This is the only dose. Pain on injection
  - **We use this drug in unstable patients, Trauma, elderly, cardiovascular instability. Has a possibly unfair bad reputation due to ICU studies.**
  - **Rare Problems with Etomidate:**
    - Adrenocortical Suppression – dose dependent inhibition of the enzyme – 11-B-Hydroxylase that convert cholesterol to cortisol. Suppression lasts 4 – 8 hours. Lowers post operative cortisol levels in healthy patients. No outcomes differences have been noted for major procedures.
  - **Other notes:**
    - PONV in 30% of patients (worst of all induction drugs)
Induction agents

- **Ketamine**
  
  - **Mechanism of Action:** Causes dissociative anesthesia. **Not a hypnotic.** Depresses the neuronal function of the cortex and the thalamus, however, it stimulates the limbic system. Activates opioid receptors and subcortical neurons in the spinal tract. These factors contribute to its analgesic effects. Provides intense analgesia and is profoundly amnestic. Patient will lie there with their eyes open and breath, but not remember a thing.

  - **Use:** Dissociative Anesthesia, Pain relief, adjunct to decrease other anesthesia drugs, chronic pain, decreases narcotic use

  - **Preparation:** Comes in 1%, 5%, and 10% solutions – 5% (50 mg/ml) is most common. Does not burn on injection

  - **Dose:**
    - **Adult induction dose** = **1.5 mg/kg IV**. (Onset is about 1 minute). Bolus dose will give dissociative anesthesia that lasts for 10 – 20 minutes. EPS symptoms will last up to an hour (i.e. critters).
    - **Intense analgesia** = **0.2 to 0.5 mg/kg**.
    - **Catch the kid** = **4 – 8 mg/kg IM** - given to calm a child. (Five minutes until onset of effects; but it lasts a little longer). **Ketamine dart**=
      - addition of versed and robbonol

  - *no pain on injection

  - **Kinetics:** Metabolizes 20-30% into nor-ketamine, another active metabolite. High Clearance (low protein bound). Induces CYP 450. Responsive in 30 min. redistribution. Elimination half time 2 hours – so elimination will affect their wake up from this drug. Tolerance can turn to tachyphlaxis

  - **Contraindications** No MAOI’s – increases epi.
Induction agents

› **Ketamine**

› Delirium: with small sub anesthetic dose it is rare. With induction dose, the delirium will come after the dissociation has worn off – post op / pacu issues.

› ↑SVR – PVR, ↑HR, ↑CO, ↑MVO2, ↑SNS outflow, ↑Epi,Norepi – inhibit reuptake
  – Little depression – but because of dissociation their air way is not protected.
  – ↑salivary secretions - Pre-treat with Robinol (anti-salagog).
  – Airway tone intact: Pharyngeal and Laryngeal reflexes are intact; but this is not a protected airway, again dissociation.

› **Problems with Ketamine:**

› Halucinations: administer with benzodiazepines. Vitamin V. Barbiturates/Propofol can also work. (just remember we may be using Ketamine because pt. couldn’t tolerate Barbs or Propofol
Induction agents (Not a true induction agent)

- **Dexmedetomidine (DMD) / Precedex:**

  - **Mechanism of Action:** Stimulation of α2 receptors causes pre-synaptic inhibition. (Turns down the SNS). It affects different classes of the α2 receptors:
    - α2A: Sedation, Hypnosis, Sympatholysis
    - α2B: Vasoconstriction, Anti-shivering, Analgesia, Ca linked—may be excitatory
    - α2C: Learning, Startle response
  - **Use:** ICU Sedation, Cardiovascular surgery, TIVA
  - **Preparation:** Supplied in 2 cc vials (100mcg/cc); mix with 50 cc NS = 4 mcg/cc.
  - **Dose:**
    - **Loading dose = **1 mcg/kg over 10 minutes**. If given too fast will cause hypertension initially. This is transient and is attenuated by giving is slowly.
    - **Infusion = **0.2 – 0.7 mcg/kg/hr**.
Induction agents

- **Precedex**
  - **Respiratory Effects of Dexmedetomidine:**
    - Minimal respiratory depressant effects (respiratory sparing) the least of all the induction drugs.
    - Sleep like depression.
    - Anti-salagog (Dries them up).
    - Watch for obstruction (sleep apnea) still not protected airway – especially in obese.
    - ABG normal, no big change in TV, RR
  
- **Other notes:**
  - 1. Widens thermoregulation / Anti shivering
  - 2. Decrease muscle rigidity seen with high opioids
  - 3. Drug addicts and alcoholic with drawl is well offset with this drug (alcoholic withdrawal under anesthesia is almost 50% fatal)
  - 4. Chronic pain patients become extremely tolerant. This can help increase drugs effects without all the side affects.
Methohexital (Brevital):
- 1% (10 mg/cc) or 2% (20 mg/cc) solution.
- Usually a 50cc vial of 1% solution.
- Adult dose = **1.5 mg/kg**.
- Distribution time = 5.6 minutes.
- Clearance = 11 ml/kg/minute.
- Elimination half-life = 4 hours.
- Causes myoclonus and hiccoughs even at low doses
- cause seizure activity at low doses = epileptogenic.
- Is used to treat seizures at high doses.
- Airway irritability
Induction agents

› **Methohexital Uses** – “rapid, non-painful procedures”.

› Electro Convulsive Therapy (ECT). Because it does not depress the seizure

› Cardioversion.

› Mapping seizure focus
Narcotics

- **Morphine**
- Opioid agonist
- Post-op pain dose: 5-20mg IV titrated
- Potential for delayed respiratory depression up to 24 hours after intrathecal administration
- Dose dependent histamine release
- Decreases PVR with high doses
- 2 active metabolites
- Prolonged half-life in elderly, liver failure, and renal failure
Narcotics

› **Hydromorphone/Dilaudid**
› Seven times more potent than morphine
› Less histamine release
› IV relief peaks at 5-20 minutes
› Duration 2-4 hours
› Favored by many anesthesia providers towards to end of surgery, carryover into PACU
› Very potent, lower doses needed, be cautious about delayed response. Dose slow and low.
Narcotics

› **Fentanyl / Sublimaze**
› Opioid agonist
› Dose: 1-5mcg/kg for 1\textsuperscript{st} hour of surgery
› Infusion: 3-6mcg/kg/hour (off 60 minutes before breathing)
› Supply: 50mcg/ml
› Decrease dose in elderly
› Dose on IBW
› Mainstay of intra-op anesthesia
Narcotics

› **Meperidine** / Demerol
› Affinity for mu, kappa, and delta opioid receptors
› Dose: 12.5mg IV for post-op shivering
› Structurally similar to atropine and local anesthetics
› Active metabolite → CNS stimulant (seizures)
› Contraindicated with MAOI’s
› Block reuptake of serotonin
› **Useful for post surgical/anesthesia shaking**
Narcotics

- **Sufentanil** / Sufenta
- Highly selective mu opioid receptor agonist
- Dose: 0.5-1mcg/kg
- Infusion: 0.5-1mcg/kg/hour (<2hrs = 30 min before breathing; >2hrs = 45 min before breathing)
- Supply: 50mcg/ml
- CV stable
- Good for infusion
- Good for post-op pain control after infusion (stays around longer than Remi)
Narcotics

- **Alfentanil / Alfenta**
- Opioid agonist
- Dose: 15-40mcg/kg
- Supply: 500mcg/ml
- Hard drug to time – more variable in elderly and obese
- Highly protein bound
- RAPID respiratory depression
- Good for short procedures: sedation for eye blocks, sedation for regional (probably not), outpatient procedures
Narcotics

- **Remifentanil / Ultiva**
- Mu opioid agonist
- Loading dose: 0.5-1mcg over 1 min
- Infusion: 0.125-0.375mcg/kg/min (off 6 min before breathing)
- Ultra-short → metabolized by plasma esterases (independent of organ function)
- **Quick** respiratory depression, **Bradycardia**
- Up to 70% reduction of MAC when using gtt
- Use for: neuro, carotid endarterectomy, eye blocks, TIVA
- **Must** cover for post op pain with another drug, **Quick on and Quick OFF**
- **Expensive!**
Narcotics

› Morphine family: Full agonists
  › Hydromorphone (Dilaudid)
  › Oxycodone
  › Hydrocodone
  › Codeine
  › Tramadol
Many other partial Agonist/Antagonist drugs
- Including Methadone, Stadol, Nubaine, Buprenex, etc.
- Unlikely to see these drugs often outside of OB
- They can be used to treat pruritis without reversing analgesia
OPIOID ANTAGONISTS

Naloxone / Narcan

- Mu, kappa, and delta opioid receptor antagonist
- Respiratory depression reversal: 40-80mcg IV
- Supply: 400mcg/ml (must dilute)
- Can see cardiac stimulation r/t abrupt reversal of analgesia
- Potential for flash pulmonary edema
- Reduces/reverses mu related SE’s: pruritis, urinary retention, muscle rigidity, biliary spasm
- Can wear off before narcotic, be vigilant!
Benzodiazepines: Midazolam (Versed), Diazepam (Valium), Lorazepam (Ativan), Flumazenil (Romazicon)

- Principle Pharmacology Effects:
  - Sedation.
  - Anxiolysis.
  - Hypnotic.
  - Anticonvulsant.
  - Spinal cord – mediated skeletal muscle relaxation.
Benzodiazepines

› **Potency:**
  
  › Lorazepam > Midazolam > Diazepam.
  
  › Lorazepam ~ 5 times more potent than Midazolam.
  
  › Midazolam ~ 5 times more potent than Diazepam.
Benzodiazepines

- **Cardiovascular Effects of Benzodiazepines:**
  - Minimal effects with sedative dose.
  - May get a small drop in blood pressure with sedation, but it is minimal.

- **Respiratory Effects of Benzodiazepines:**
  - Dose dependent respiratory depression.
  - Depresses airway reflexes and ability to swallow — usually seen at higher doses.
  - **Hypoxic Drive to Breath is depressed.** Normally breathing is driven of CO2, however with some patients they now use Oxygen/hypoxia to trigger breathing. 1. Chronic COPD, 2. Sleep Apnea, 3. Morbid Obesity. This drug will depress their drive to breath.
  - **ANY DOSE, ANY PATIENT, CAN CAUSE APNEA WITH MIDAZOLAM.**
  - Do not administer Midazolam and then walk away.
  - Patient must be monitored following administration of BZDs, especially respiratory function.
  - **SYNERGISTIC WITH OPIOIDS.**
  - Does not blunt the SNS Response to Intubation
  - PO Diazepam and lorazepam has almost no respiratory depression when used alone.
Flumazenil / Romazicon

› This is a reversal for the Benzodiazepines. We usually never use it as part of a surgical case because we will time our drugs better.

› **Dosing:**

› **0.2 mg iv** reverses the CNS effects of BZDs within two minutes.

› Supplied in 0.1 mg/cc vials.

› Onset of action = 2 minutes.

› **May repeat with 0.1 mg iv every 1 minute to a total of 1 mg in the first hour and 3 mg maximum dose.**

› Duration of action 30 – 60 minutes. *(Shorter ½ life than the drugs it reverses).*
N/V agents
N/V agents

- **Decadron** (Anti nausea, anti inflammatory, long acting)
- **Zofran**... Very safe (watch for QT), difficult to overdose, very effective.
- **Droperidol**... Tough to find anymore, Watch QT, can cause sedation/confusion
- **Reglan**... Forward motility, works on dopamine receptor, can cause anxiety, extrapyramidal side effects. Got a bad rap because of flawed studies.
- **Benadryl**... Antihistamine, Histamine receptors are in the chemoreceptor trigger zone
- **Scop patch**... Very effective for those with motion sickness, multi day treatment, remember education for removal.
- **Pepcid**...H2 blocker, decreases acid, but can reduce N/V, FDA approved for morning sickness
- **Phenergan**...Usually a rescue drug, sedation qualities, MUST dilute and make certain IV is working, extravasation can result in loss of limb. IV or IM use only.
- **Propofol**... Has anti-emetic properties, can be used as a last ditch rescue, can also be used in TIVA, avoiding gas to reduce N/V in high risk patients.
- **Emend (Aprepitant)**... Newer, expensive drug, for use in very high risk patients. A substance P/nk-1 antagonist (also a CTZ trigger). A previously unexplored pathway to prevent N/V
The earliest reports of the use of neuromuscular blockers date to the early 1500s, when natives of the South American Amazon used curare for hunting.

The first successful administration of curare to produce surgical relaxation in an anesthetized patient occurred in 1912, when Arthur Läwen, a German surgeon from Leipzig, used a partially purified preparation of the substance.

On 23 January 1942, Enid Johnson, following Canadian anesthesiologist Harold Griffith's instructions, used it in Montreal for a patient undergoing appendectomy. Since that time, neuromuscular blocking drugs have been used extensively and have undoubtedly been one of the major reasons for the tremendous progress in surgery.
**Paralytics**

- **Succinylcholine:**
  - Succinylcholine is 2 molecules of ACH bound together. It binds to the ACH-receptors at the MNJ and mimics ACH. It opens the channel and allows for depolarization of the motor end plate. It stays there and keeps the channel open – the receptor can not reset and the muscle is paralyzed.
  - **Mechanism of Action:**
    - Depolarizes presynaptic, postsynaptic and extrajunctional receptors.
    - Provides desensitization – prolonged contact between the agonist and the receptor.
    - Is antagonized by non-depolarizers.
    - Is augmented by acetylcholinesterase.
    - Onset of action is 30 – 60 seconds.
    - Duration of action is 7 – 10 minutes.
    - **MH Trigger, danger with use in burns, paralyzed patients..(up regulation) renal failure with increased K+... Suxx increases potassium levels and can result in arrhythmias, and arrest.
    - The Gold Standard for Rapid Sequence Induction and difficult airway manipulation.
      - Provides excellent intubating conditions within 60 seconds
Paralytics

- **Cisatracurium (Nimbex®):** Isomer of Atracurium and 4 times more potent.
- Does not release histamine.
- Little change with age or illness.
- Does not accumulate.
- $2 \times \text{ED}_{95}$: 0.2 mg/kg.
- After sux: 0.1 mg/kg.
- Infusion: 1 – 2 mcg/kg/min.
- Supplied: 2 mg/cc
- Keep in the refrigerator.
- Nimbex is drug of choice for Renal failure, as it is metabolized in the blood, organ independent.
- Nimbex is however, expensive
Paralytics

› **Vecuronium (Norcuron®):**
› Monoquaternary aminosteroid.
› $2 \times ED_{95} : 0.1 \text{ mg/kg.}$
› After sux: $0.05 \text{ mg/kg.}$
› Infusion: $1 – 2 \text{ mcg/kg/min.}$ Can be infused but not the best choice because of accumulation.
› Supplied as a powder – mix with water – $1 \text{ mg/cc.}$
› **Hepatic Metabolism (30 – 40%):**
  – Active metabolite.
  – 80% potency of the parent compound.
  – May accumulate in renal failure.
Paralytics

- **Rocuronium (Zemuron®):**
- 2 x ED$_{95}$: 0.6 mg/kg. Less potent gives us a faster onset. Sets up in 2min
- *RSI: 1.2 mg/kg* Sets up in 1min.
- After sux: 0.3 mg/kg
- Infusion: 5 – 12 mcg/kg/min. Much less accumulation then Vecuronium.
- Supplied: 10 mg/cc.
- Minimal to no metabolism:
  - No active metabolites.
  - Excreted unchanged by the kidneys.
  - Excreted unchanged in bile.
- Not affected by renal failure.
- Moderate prolongation in liver failure.
- **PACU Warning:**
  - Slightly prolonged in elderly.
  - Slightly prolonged in pregnancy.
Anticholinesterase: Acetylcholine esterase Inhibitors - bind Acetylcholine esterase at the NMJ and ↑ the concentration of ACH.

Nondepolarizing NMB’s: They are not direct inhibitors. They ↑ the NMB competitor - ACH. They are indirect antagonist.

Depolarizing NMB (Succinylcholine: These do not inhibit Succ, they augment it. Succ mimics ACH in order to depolarize the cell.

Edrophonium (Tensilon®). Used in Peds

Neostigmine (Prostigmin®). Most commonly used.

Pyridostigmine (Mestinon®). Rarely used (if we run out of neostigmine)

Phystostigmine (Antilirium®). We won’t use this (Pt’s on it at home).
Reversal of paralytics (Time and Dosing First)

- **Onset of Action:**
  - Edrophonium: 1 – 2 minutes.
    - Rapid onset.
    - May reflect a presynaptic effect.
    - Acetylcholine release.
  - Neostigmine: 7 – 11 minutes.
  - Pyridostigmine: 16 minutes.
- **Duration of Action:**
  - Edrophonium: 5 – 20 minutes.
    - Larger doses: up to 60 minutes.*
    - If a larger dose is needed, should be using neostigmine.
  - Neostigmine: 40 – 60 minutes.
  - Pyridostigmine: 90 minutes.

*Onset is significantly changed by how profound the neuromuscular blockade is.
Reversal of Paralytics (Time and Dosing First)

 › **Glycopyrrolate (Robinul®):**
   › Onset: 2 – 3 minutes.
   › Duration: 2 hours.
   › Supplied: 0.2 mg/cc.
   › Quaternary structure:
     – Does not cross the BBB. No sedation.
   › Tachycardia ++, Bronchodilation ++, Antisialagogue +++. (Less Tachycardia than Atropine)
   › *Used with Neostigmine....Neostigmine will cause bradycardia, severe...asystole severe. This drug combats that effect.
Sugammadex (New Drug)

- **Sugammadex/Bridion** is an agent for reversal of neuromuscular blockade by the agent rocuronium in general anesthesia. It is the first selective relaxant binding agent (SRBA).

- Essentially this new drug, surrounds rocuronium, rendering it inactive.

- Sugammadex, unlike neostigmine, does not inhibit acetylcholinesterase so cholinergic effects are not produced and co-administration of an antimuscarinic agent (robinol or atropine) is not needed. Sugammadex might therefore be expected to have fewer adverse effects than the traditional reversal agents.

- When muscle relaxant with rapid onset and short duration of action is required, there has been little choice apart from suxx but this drug has important contraindications; for example, it can trigger malignant hyperthermia in susceptible individuals, it has a prolonged duration of action in patients with pseudocholinesterase deficiency and it causes an increase in plasma potassium concentration which is dangerous in some circumstances.
Rocuronium has a comparably quick onset in high dose (0.6 mg kg\(^{-1}\) to 1 mg kg\(^{-1}\)) and can be rapidly reversed with sugammadex (16 mg kg\(^{-1}\)), so this drug combination offers an alternative to suxx.

Has been shown to work with Vecuronium and Pancuronium.

The routine reversal of a shallow/medium blockade is done by an administration of 2 mg/kg, while a deep blockade is reversed with a dosage of 4 mg/kg. To initiate an immediate reversal three minutes after administration of maximum 1.2 mg/kg rocuronium there is a dosage of 16 mg/kg sugammadex applied. Remark that all the administrations are done by a single rapid bolus injection and that it has to be injected directly into a vein or an IV line. There is no dose adjustment needed when the patient is older than 65 years, obese or based upon gender.

Just FDA approved in the US, but used overseas for sometime. Very expensive. Can be used as a rescue drug if Roc is given in a can’t intubate, can’t ventilate situation. Not yet available on formulary.
The “Usual Anesthetic”

- Let’s go through the traditional anesthesia induction and why.
- **Versed** preop... Amnesia, anxiolytic
- **Lidocaine**... Pain from induction agent and the blunting of sympathetic response
- **Fentanyl**...not just for pain...
- **Propofol, Emotidate, Ketamine**... why use each?
- **TIVA**- Remi, Sufenta, Prop, Precedex, Lidocaine, Ketamine, many options...
- **Paralytics**...Rocuronium, vecuronium, succinylcholine, pancuronium, etc...what to use each for?
- Titrate in longer acting pain agents... Dilaudid, Morphine, etc
- Anti-emetic agents... remember all drugs have side effects, sometime more is not better
- Reversal agents...
Many ways to skin a cat...
Many ways to skin a cat...

› There are many, many ways to perform an anesthetic...
› Lidocaine can be used for pain relief as an infusion...
› Magnesium can do the same..
› Is a block more effective than a general?
› Why do a total knee with a spinal verse an ETT?
› Going over each of these choices is beyond the scope of a single powerpoint review. Please note that many variables are always considered by the anesthetist to best suite the patient, and that there is not single perfect fit.
Thank You!

As is my usual...

“Questions, comments, concerns...Chocolate?”