## Preadmit Holding Area

| |
|---|
| Talk to patient |
| Check name band (identifier) |
| **Check consents - ALWAYS** - before sedation |
| Check if patient is marked |
| Check with holding are RN if patient is ready to go |
| Running IV? |
| Give pre-op sedation |

## Pre-op Sedation

**Only give once consent is confirmed to have been signed**

| | | |
|---|---|---|
| **Midazolam** | Administered by TBW because of an increased central volume of distribution. Just about all books seem to agree with this. Dosing in this way will prolong the elimination half-life and its duration of effect. In practice, it may cause over sedation in the obese pts who is sensitive to respiratory depressant drugs | |
| | TBW = total body weight (obese patients could overdose due to larger body weight and thus larger dose) | |
| | **MOA** | GABA-A Agonist |
| | | change frequency of channel opening - neuronal hyperpolarization |
| | | *most GABA-A agonists increase channel open time, benzos increase open frequency* |
| | **Onset** | 30-60 seconds |
| | **Duration** | 20-60 min |
| | **Clearance** | Liver |
| | **Active Metabolite** | 1-hydroxymidazolam |
| | **Sedation dose** | IV 0.01-0.1 mg/kg |

## Pre-op Sedation (cont)

| | |
|---|---|
| **Respiratory Effects** | minimal but synergistic respiratory depression when combined with other sedatives |
| **CV Effects** | minimal |
| **CNS Effects** | anterograde amnesia, anticonvulsant properties, anxiolysis, antispasmodic effects *No analgesia* |
| | ~anti spasmodic effects good for spinally mediated skeletal muscle relaxation (useful in CP patients) |

## Proceed to Operating Room

Transport patient to OR via stretcher or amulation

| | |
|---|---|
| Move patient to OR table and **ensure safety strap is secured** | usually placed across thighs 2 inches above the knees over the cover |
| | arms secured on padded arm boards or tucked |
| Apply Monitors | record vital signs *at least* every 5 minutes |
| | -EKG |
| | -BP |
| | -Pulse Ox |
| | -Capnography |
| | -Temperature |

## Preoxygenation aka Denitrogenation

o 1948: Fowler and Comroe demonstrated that inhalation of 100% oxygen (O2) resulted in a very rapid increase of arterial oxyhemoglobin saturation (Sao2) to between 98% and 99%, but that attainment of the last 1% to 2% was a much slower process
o 1950s: Rapid Sequence Induction (RSI) began being utilized in patients at risk for aspiration of gastric contents, preoxygenation became a component of the technique

*Preoxygenation* extends periods of **safe apnea**



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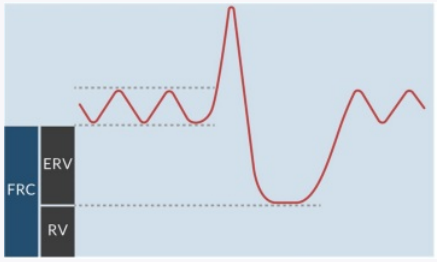
## Preoxygenation aka Denitrogenation (cont)

| | |
|---|---|
| | • defined as the time until a patient reaches a saturation level of 88% - 90%, to allow for the placement of a definitive airway. |
| | 👇 Below this level, oxygen saturation can decrease to critical levels <70% within moments. |
| **Goals of preoxy-genation** | 👇 Achieve 100% oxygenation saturation prior to procedure |
| | 👇 Denitrogenate the residual capacity of the lungs, maximizing oxygen storage |
| | 👇 Denitrogenate and maximally oxygenate the bloods-tream. |
| **Preoxy-genation techniques** | o Tidal volume breathing with 100% O2 for 3-5 minutes |
| | o 8 deep breaths of 100% O2 for 60 seconds |
| | o Sit up or reverse Trendelenburg to increase FRC |
| **Nasal oxygen @ 15L during intubation** | 👇 Preoxygenation and apneic oxygenation are partic-ularly beneficial if manual ventilation after induction of anesthesia is undesirable (eg during rapid sequence induction and intubation RSI), if difficulty with airway management is anticipated and for pts who are expected to desat rapidly |
| | • Obese<br>• Pregnant<br>• Pediatric<br>• Hypermetabolic pts |

## FRC Measurement




## Functional Residual Capacity

| | |
|---|---|
| **FRC** | Volume of air in lungs at end of expiration |
| | o FRC is the reservoir of oxygen that prevents hypoxemia during apnea |
| | o Diaphragmatic tone and position *also* effect FRC |
| | o FRC cannot be measured with spirometry because the residual volume cannot be exhaled and RV is a component of FRC |
| **Static equilibrium** | *At FRC* the inward elastic recoil of the lungs is balanced by the outward elastic recoil of the chest wall |
| **Normal FRC** | 35 ml/kg |
| **Indirect FRC measur-ement** | Nitrogen washout |
| | Helium wash in |
| | Body plethysmography |
| **How will FRC last during apnea?** | o We can estimate how long a pt can remain apneic before desaturation if we know the patients FRC and oxygen consumption (VO2) |
| | **o Healthy adult breathing 100% O2 takes 6.9 minutes to desaturate to 90% on pulse oximetry**<br>**👇 1 minute if the patient was breathing room air** |
| **Desat formula** | time until patient desats = FRC/VO2 |



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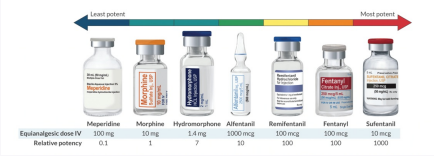
## Functional Residual Capacity (cont)

| | |
|---|---|
| **Conditions that decrease FRC** | *Obesity*<br>• Decreased chest wall compliance<br>• Increased airway collapsibility |
| | *Pregnancy*<br>• Diaphragm shifts cephalad due to gravid uterus<br>• First give O2!!!<br>• Decreased chest wall compliance |
| | *Neonates*<br>• Less alveoli<br>• Decreased lung compliance<br>• Cartilaginous ribcage prone to collapse during inspiration |
| **Postions that affect FRC** | *Decrease*<br>• Supine<br>• Trendelenburg<br>• Lithotomy |
| | *Increase*<br>• Prone<br>• Sitting<br>• Lateral- unchanged or increase |

## Opioid Potency




Opioid Potency Least potent (left) Most Potent (Right)
Meperidine 100mg / 0.1 RP
Morphine 10mg / 1
Hydromorphone 1.4m / 7
Alfentanil 1000mcg / 10
Remifentanil 100mg / 100
Fentanyl 100mcg / 100
Sufentanil 10mcg / 1000

## IV Induction Agents - General Anesthesia

| | | |
|---|---|---|
| **Opioids - Fentanyl** | **MOA** | mu receptor agonist |
| | **Onset** | 5 min |
| | **Duration** | 20-30 min |
| | **Active Metabolite** | CYP3A4 (P450) |

## IV Induction Agents - General Anesthesia (cont)

| | | |
|---|---|---|
| | **Clearance** | Liver |
| | **Dosing** | *IV* 1-2 mcg/kg<br>*induction* 10 mcg/kg (watch for chest wall or glottis rigidity) |
| | **Resp Effects** | respiratory depression |
| | **CV Effects** | bradycardia, vasodilation |
| | **CNS Effects** | analgesia, N/V |
| **Amine - Lidocaine** | **MOA** | **o Local anesthetics bind to alpha-subunit on inside of sodium channel**<br>o When critical number of sodium channels are blocked cell can't be depolarized and action potential cant be propagated |
| | **Adverse Effects** | *Mild CNS-related symptoms*<br>• Drowsiness<br>• dizziness<br>• metallic taste<br>• Headache<br>• blurred vision<br>• paresthesia dysarthria<br>• euphoria<br>• Nausea<br>*Larger doses or if given rapidly*<br>• Tinnitus<br>• Tremor<br>• Agitation<br>• Cardiovascular changes are usually minimal with the usual doses |



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## IV Induction Agents - General Anesthesia (cont)

| | | |
|---|---|---|
| | **Uses** | o 5% of patients have pain at propofol injection and of these, 1% of them have severe or excruciating pain<br>👇 40 mg Lidocaine prevents this<br>👇 Also can mix Lidocaine and Propofol<br>• Propofol and lidocaine= Magic<br>o Add 1 ml of 1 % or 2% lidocaine to a 10 ml syringe of propofol<br>👇 Place the IV in an antecubital vein (vs the hand).<br>👇 Pretreat with IV opioids.<br>👇 If the IV is in the hand, place a tourniquet proximally and pretreat with lidocaine |
| **Propofol** most common induction agent | **MOA** | GABA-A agonist (how long the channel stays open)<br>👇 GABA-A receptor stimulation hyperpolarizes neurons by increasing Cl- conductance. More Cl- inside the cell makes the cell more negative. This reduces resting membrane potential (RMP moves further away from TP) |
| | **Onset** | 30-60 seconds |
| | **Duration** | 5-10 min |
| | **Clearance** | Liver and extra hepatic metabolism |

## IV Induction Agents - General Anesthesia (cont)

| | | |
|---|---|---|
| | **Active Metabolite** | None |
| | **Induction dose** | 1.5-2.5 mg/kg IV |
| | **Maintenance dose** | 25-200 mcg/kg/min |
| | **Resp Effects** | decreased resp drive |
| | **CV Effects** | decreased BP, SVR, preload, contractility |
| | **CNS Effects** | decreased ICP and IOP, no analgesia, +/- seizure activity |
| **Etomidate** | **MOA** | GABA-A agonist |
| | **Onset** | 30-60 seconds |
| | **Duration** | 5-15 min |
| | **Clearance** | Liver & plasma esterases |
| | **Active Metabolite** | None |
| | **Induction dose** | 0.2-0.4 mg/kg IV |
| | **Resp Effects** | Mild Resp Depression |
| | **CV Effects** | Minimal |
| | **CNS Effects** | Decreased ICP, no analgesia |



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## IV Induction Agents - General Anesthesia (cont)

| | | |
|---|---|---|
| | **Side Effects** | o Myoclonus (not a seizure)<br>o Does not cause seizures if the patient does not have a history of seizures<br>o Suppression of adrenocortical function for up to 24 hrs. It should be avoided in sepsis and acute adrenal failure<br>o N&V (greater than any other induction agent)<br>o Acute intermittent porphyria |
| **Ketamine** | **MOA** | NMDA antagonist (creates dissociated state) |
| | **MOA secondary** | Many 2nd receptor targets including opioid, MAO, serotonin, NE, muscarinic, and NA channels |
| | **Onset IV** | 30-60 seconds |
| | **Onset IM** | 2-4 minutes |
| | **Onset PO** | variable |
| | **Duration** | 10-20 minutes (can last 60-90 min to return to full orientation) |
| | **Clearance** | Liver |
| | **Active Metabolite** | Norketamine |
| | **Induction Doses** | IV 1-2 mg/kg<br>IM 4-8 mg/kg<br>PO 10mg/kg |
| | **Opioid Sparing Dose** | 0.1-0.5 mg/kg or 1-3 mcg/kg/min |

## IV Induction Agents - General Anesthesia (cont)

| | |
|---|---|
| **Resp Effects** | maintains resp drive, increased oral secretions (DROOL EVERYWHERE, GIVE GLYCO) |
| **CV Effects** | Increased SNS tone, SVR, HR, and CO |
| **CNS Effects** | Increased ICP, IOP, nystagmus and analgesia<br>causes emergence delirium and lowers seizure threshold, can also treat severe depression |

## Food Allergies & Propofol

| | |
|---|---|
| Overseen by the American Academy of Allergy, Asthma and Immunology. They state: | o Propofol can cause anaphylactic reactions, the cause of these reactions is unclear and appears not to be related to soy or egg allergy. |
| o Egg allergy | 👇 Patients with soy, peanut allergy or egg allergy can receive propofol without any special precautions. – Probably safe<br>👇 Most people with egg allergies are allergic to the albumin egg whites. Egg lecithin found in propofol is derived from the YOLK |
| o Soy | 👇 Any soy proteins that are capable of producing an immune response are removed during the refining process<br>👇 Prop is safe to use in pts with soy allergy |



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## Food Allergies & Propofol (cont)

| | |
|---|---|
| o Peanut | 👎 Like soy peanuts are a type of legume. Some have speculated the potential of cross sensitivity between peanuts and soy (and thus propofol) although there is no evidence to support this<br>👎 Prop is safe to use in pts with a peanut allergy |
| o Increased Risk of Bacterial Contamination | 👎 Propofol syringes must be discarded within 6 hrs<br>👎 Infusions (and the tubing) must be discarded within 12 hrs |

## LBW vs TBW

| | |
|---|---|
| **TBW** | Total body weight **Maintenence** |
| | • Weight when individual steps on scale |
| **IBW** | Describes the BMI associated with the lowest risk of body weight related comorbidities. We can estimate the ideal body weight with the following formulas: |
| | o Men (kg)= height (cm) – 100<br>o Women (Kg)= Height (cm) - 105 |
| **LBW** | Lean body weight |
| | 👎 LBW = 1.3 X IBW |

| Drug | Dose | Recommendation |
|---|---|---|
| Propofol | Induction<br>Maintenance | LBW<br>TBW |
| Succinylcholine | Intubation | TBW |
| Rocuronium<br>Vecuronium | Intubation<br>Maintenance | LBW<br>LBW |
| Cisatracurium<br>Atracurium | Intubation<br>Maintenance | TBW<br>TBWvsLBW |

## LBW vs TBW (cont)

| | | |
|---|---|---|
| Fentanyl ((nl))Sufentanil | Loading<br>Maintenance | TBW<br>LBW |
| Remifentanil | Loading<br>Maintenance | LBW<br>LBW |
| Midazolam | Loading (not preop)<br>Maintenance | TBW<br>TBW |
| Epidural Local | | 75% of normal dose |

## Guedel's Stages of Anesthesia

| | |
|---|---|
| **Stage 1 - Analgesia or Disorientation** | o Can be initiated in a preoperative holding area<br>o Patient is given medication and may begin to feel its effects but has not yet become unconscious |
| *o Induction stage* | 👎 Patients are sedated but conversational<br>👎 Breathing is slow and regular<br>👎 Patient progresses from analgesia free of amnesia to analgesia with concurrent amnesia<br>👎 This stage comes to an end with the loss of consciousness. |



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## Guedel's Stages of Anesthesia (cont)

| | |
|---|---|
| *o Loss of Consciousness* | 👇 Count backwards from 100, the patient typically loses consciousness between 80 to 90, i.e. stops counting – the old way<br>👇 Blinking increases, and nystagmus may appear<br>👇 Eyes eventually fix in the midline as the lids close • GENTLE<br>👇 Patient becomes unresponsive, atonic, apneic, and the oculocephalic (or more precisely vestibular-oculocephalic) and corneal reflexes are lost<br>👇 Call patients name<br>👇 Eyelash reflex<br>👇 Tape eyes- as soon as you lose consciousness<br>• If you struggle to ventilate they you could hurt their eyes<br>• Not on sedation cases<br>• Don't tape in endo watch the L eye |
| *o Eye Protection after Loss of Consciousness* | 👇 Tape eyes horizontally after loss of consciousness<br>👇 Eyes should be protected before instrumenting the air way |

## Guedel's Stages of Anesthesia (cont)

| | |
|---|---|
| **Stage 2 - Excitement** | o There is a higher risk of laryngospasm (involuntary tonic closure of vocal cords) at this stage, which may be aggravated by any airway manipulation<br>o The combination of spastic movements, vomiting, and rapid, irregular respirations can compromise the patient's airway.]<br>o Fast-acting agents help reduce the time spent in stage 2 as much as possible and facilitate entry to stage 3.<br>o NEVER EXTUBATE AT THIS TIME |
| | o If you are using gas induction no muscle relaxation- you can really see this<br>👇 Its really short with IV induction<br>👇 FOR KIDS<br>• Laryngospasm<br>• Don't touch them too soon |
| **Stage 3 - Deep** | o *Surgical Anesthesia targeted anesthetic level for procedures requiring general anesthesia*<br>o Ceased eye movements and respiratory depression are the hallmarks of this stage.<br>o Airway manipulation is safe at this level |
| | **4 planes in stage** |



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## Guedel's Stages of Anesthesia (cont)

**Plane 1**, there is still regular spontaneous breathing, constricted pupils, and central gaze
• eyelid, conjunctival, and swallow reflexes usually disappear in this plane
• Just gazing

**Plane 2**, there are intermittent cessations of respiration along with the loss of corneal and laryngeal reflexes. Halted ocular movements and increased lacrimation may also occur.

**Plane 3** is marked by complete relaxation of the intercostal and abdominal muscles and loss of the pupillary light reflex. This plane is referred to as "true surgical anesthesia" because it is ideal for most surgeries.

**Plane 4** is marked by irregular respiration, paradoxical rib cage movement, and full diaphragm paralysis resulting in apnea.

## Mask Ventilation

| | |
|---|---|
| One hand | o C<br>o E<br>o If you are struggling put in oral airway |
| Two hands | o Get it less than 20<br>o Two people approach |
| Non- Invasive Airway Maneuvers | • Chin lift<br>• Not usually in induction<br>• Jaw Thrust |

## Mask Ventilation (cont)

| | |
|---|---|
| Placement of LMA if unable to ventilate | • LMA<br>• Difficult supraglottic airway placement<br>o Restricted mouth opening<br>o Obstruction<br>o Distorted airway<br>o Stiff lungs or C spine |
| Upper Airway Patency | • Pharynx<br>• Collapsible tube inside box<br>• Box is formed:<br>o Tongue<br>o Soft palate<br>o Pharyngeal tissue<br>o Cervical spine |
| During inspiration a negative gradient draws air into lungs | • Tendency to make airway collapse<br>• In awake state<br>o Counteracted by three sets of dilator muscle |
| If able to ventilate give muscle Relaxant | • Upper airway consists of the cartilaginous and bony structures of the nose and mouth, followed by the soft tissue of the oropharynx and laryngopharynx, and ending in the rigid trachea<br>• Soft tissue of the pharynx is prone to collapse in the unconscious, or anesthetized, patient and may be further compromised by obesity, a large tongue, airway edema, large neck circumference, external compression, and many other factors |



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## Mask Ventilation (cont)

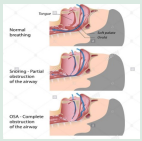
Controversy

• When placing an endotracheal tube after induction o Historically been instructed to refrain from administering muscle relaxation until adequate mask ventilation in the anesthetized patient was confirmed in order to both avoid
- Critical hypoxemic event
- Ensure an attempt at an escape wake up.

o There is little published evidence to support this practice, and the administration of muscle relaxation before ensuring adequate BVM ventilation remains controversial
o Neuromuscular Blockade and the Airway
- Regarding Mask Ventilate- There is evidence that paralysis of the upper airway musculature improves ability to ventilate
- A recent study published data indicating that NMB using rocuronium facilitated bag-mask ventilation in anesthetized patients

## Oral Airways

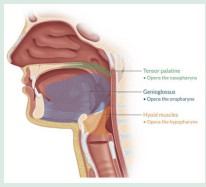
## Airway Obstruction



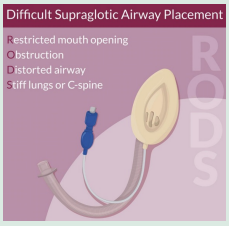
## Difficult Ventilation Mnemonic

Ventilate Patient with mask after loss of consciousness

## Upper Airway Patency



## Mneumonic for Difficult LMA Placement




## Why Neuromuscular Blockades (NMB)?

| | |
|---|---|
| • They allow for easy airway and operative field manipu-lation | o Good for specific types of surgery<br>o No single agent is ideal for every situation |
| • What is the Neurom-uscular Junction? (NMJ) | o The neuromuscular junction is a synapse that develops between a motor neuron and a muscle fiber<br>o Made up of several components: the presynaptic nerve terminal, the postsynaptic muscle membrane, and the intervening cleft (or gap)<br>o End Plate<br>- Acetylcholine is hydrolyzed rapidly by the enzyme acetylcholinesterase in the synaptic cleft<br>- Not all acetylcholine that is released reaches the endplate, some is hydrolyzed en route. |



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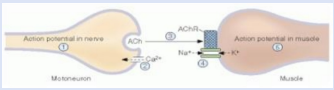
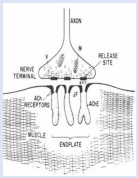
## Why Neuromuscular Blockades (NMB)? (cont)

| | |
|---|---|
| • Muscle Relaxants | o Disrupts the physiological sequence of neuromuscular transmission.<br>o Provides NO ANALGESIA or AMNESIA<br>o Used to optimize surgical condition and facilitate intubations.<br>o Mechanism of action occurs at the neuromuscular junction (NMJ)<br>o Post junction nicotinic receptors are composed of five subunits<br>o Lined up circumferentially around ion conducting core<br>o Two alpha subunits |

## Neuromuscular junction




## Muscle Relaxants




## End Plate

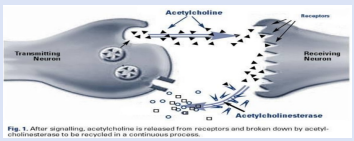
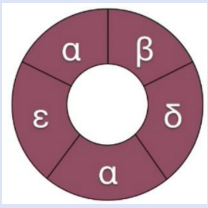



Fig. 1. After signalling, acetylcholine is released from receptors and broken down by acetylcholinesterase to be recycled in a continuous process.

## Post Junction Nicotinic Receptors




o Post junction nicotinic receptors are composed of five subunits
o Lined up circumferentially around ion conducting core
o Two alpha subunits

## Depolarizing NMB

| | |
|---|---|
| **Succinylcholine chloride (Anectine, Quelicin)** | o Depolarizing neuromuscular blockers act as **agonists** at postsynaptic nicotinic acetylcholine receptors and cause prolonged membrane depolarization resulting in neuromuscular blockade. |
| | • Resemble ACH bind to ACH receptors<br>• generating an action potential ....**depolarization**.<br>• Sodium channels are **open** as a result of depolarization, then **close** in a resting state and muscle relaxation occurs. |
| | • Ach binds to subunit-allows channel to open -*depolarization occurs*<br>• Depolarizing neuro muscular blockers<br>– Bind to alpha subunits<br>– Cause Channel to remain open- mimics Ach<br>– Prolonged depolarization occurs |
| | *Chemical formula*: C14H30N2O4 |



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## Depolarizing NMB (cont)

| | |
|---|---|
| **MOA** | agonists at postsynaptic nicotinic acetylcholine receptors and cause prolonged membrane depolarization resulting in neuromuscular blockade |
| **Onset** | IV 60-90 sec<br>IM 2-3 min |
| **Duration** | 5 min |
| **Reversal** | None |
| **Dose** | **IV 0.5-1.5 mg/kg**<br>Ped IV 4-5 mg/kg<br>Laryngospasm: 1-.5 mg/kg/IV or 4-6mg/kg IM |
| **Metabolism** | Psuedocholinesterase |
| **Adverse Effects** | 👎 Hyperkalemia<br>👎 Malignant Hyperthermia<br>👎 Apnea |

## Non-Depolarizing NMB

o NDMR compete with acetylcholine for the active binding sites at the postsynaptic nicotinic acetylcholine receptor

o Resemble ACH enough to **bind to the ACH receptor**, but **fail to activate** the receptor, thus blocking its action (paralyzing the muscle transmission)

o *"The key fits but won't open the door."*

**o Competitive Antagonist – compete with ACH**

**👎 SO THEY CAN BE REVERSED**

o The bond is very tight depending upon the drug, it will last from 20 to 90 minutes.

## Non-Depolarizing NMB (cont)

| | | |
|---|---|---|
| o Competitive Antagonist | 👎 Two alpha subunits are binding sites for Ach<br>👎 Sites occupied by nondepolarizing neuro muscular blockers<br>👎 Cause channel to remain closed<br>👎 Ion flow to produce depolarization can't occur | |
| **Rocuronium** | 👎 Rocuronium is the most widely used nondepolarizing relaxant in the United States.<br>👎 Can be used for rapid sequence induction (RSI) when succinylcholine is contraindicated. | |
| | **MOA** | o Resemble ACH enough to bind to the ACH receptor, but fail to activate the receptor, thus blocking its action (paralyzing the muscle transmission)<br>o Competitive Antagonist – compete with ACH |
| | **Onset** | 1-2 min |
| | **Duration** | 20-35 min |
| | **Dose** | **IV 0.6 - 1.2 mg/kg**<br>Infusion 5-12 mcg/kg/min<br>Pretreatment 5mg<br>*no reconstitution* |
| | **Reversal** | Sugammadex<br>Neostigmine (less effective) |



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## Non-Depolarizing NMB (cont)

| | | |
|---|---|---|
| | **Metabolism** | some de-acetylation |
| **Vecuronium** | **MOA** | |
| | **Onset** | 3-5 min |
| | **Duration** | 20-35 min |
| | **Metabolism** | Liver |
| | **Dose** | **IV: .08 - .12 mg/kg**<br>Infusion: 1-2 mcg/kg/min |
| | | • To shorten the onset time, the priming principle involves the administration of a small dose of rocuronium usually 3 minutes prior to induction<br>• The optimal priming dose which is the largest dose that it is given that will not produce weakness in an awake patient is very small<br>• Priming dose is given prior to succinylcholine rapid sequence induction to decrease the myalgias (5 mg) |

## Rapid Sequence Induction

**Indicators**

- o Patient at risk for regurgitation and aspiration who require GA History of
- o Recent vomiting or recent meal
- o Pregnancy
  - Over 18 weeks
  - Full stomach
  - Loose spincter
- o Increased intra-abdominal pressure
- o Abdominal distension
- o Poorly controlled GE reflux
- o Decreased level of consciousness
- o Gastroparesis
- o Bowel Obstruction
  - GOP1

## Rapid Sequence Induction (cont)

**Rapid Sequence Induction Method**

- o Preoxygenation is critical
- o Suction and airway alternatives available
- o Use adjuvant drugs to control BP, HR response: midazolam, narcotics, lidocaine, ketamine, etc
- o Explain and rehearse use of cricoid pressure with the patient.
- o Optimize position of upper airway.
- o Identify person to do cricoid pressure
- o Apply Cricoid while patient is awake
  - Conscious 20N (2 kg)
  - If you cant see they are pushing too hard
  - Tell them to keep holding pressure until you them to let go
- o Propofol 1.5-2.5 mg/kg
- o asleep 40N (4 kg) of pressure
- o **Succinylcholine 0.5 to 1.5 mg/kg** or **Rocuronium 1.2 mg/kg**
- o Loss of consciousness-fasciculations
- o Eye Protection
- o Intubate
- o Hold cricoid until endotracheal tube cuff is inflated and placement is confirmed

**Modified Rapid Sequence**

- o Same steps but with ventilation
- o Gentle IPPV (Paw 10-15 cm H2O) with 100% O2 until relaxant has peak effect.
- o If you cant see vent until glide scope

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## General Anesthesia - Inhalation Induction

| | |
|---|---|
| **Indications** | Difficult IV access<br>Developmentally delayed adult<br>Pediatrics<br>Potential airway obstruction e.g. epiglottitis<br>Kids or special need,<br>Sevo dilates vein- if you cant get IV |
| **Contraindications** | Aspiration risk<br>Active bleeding in airway (risk of cough, laryngospasm) |
| **Inhalation Induction Technique** | Prime circuit with anesthesia agent from vaporizer at maximum setting<br>Oxygen at 8L/min<br>Pop off valve open and patient end of circuit occluded.<br>Have patient exhale maximally, then apply face mask to patient and inhale maximally from primed circuit.<br>Expect prompt onset of sleep (60 seconds) followed by transient apnea, then pattern of rapid shallow respirations.<br>They are crying then go dominate<br>Then you put the IV in and tube them<br>Need the pop up valve OPEN |

## General Anesthesia - Inhalation Induction (cont)

| | |
|---|---|
| **Inhalation Induction Technique #2** | Prime circuit with N2O 70%,<br>FGF at 8L/min<br>Pop off valve open and patient end of circuit occluded.<br>When patient is comfortable with situation, begin volatile agent increasing vaporizer setting by 0.5% every 3 or 4 breaths<br>Reassure patient with calm voice encouraging a regular smooth breathing pattern.<br>Use of a deep breathing pattern here may lead to premature onset of apnea with prolonged phase.<br>Expect several minutes to fall asleep. Assist ventilation<br>Don't use N2O if you are trying to get pregnant- spont miscarriage<br>For adults or special needs |

## General Anesthesia - LMA Induction Sequence

| | |
|---|---|
| **Induction** | Pre-Oxygenate<br>Lidocaine<br>Propofol<br>Loss of consciousness<br>Eye protection<br>**Usually don't ventilate**<br>Open mouth insert LMA<br>When you take it out don't deflate cough- all the secretions go right in the airway |
| **Fentanyl LMA** | Many anesthesia providers do not give fentanyl on induction<br>Wait for return of spontaneous respiration<br>Others give small dose |

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