

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

M	id	az	nΙ	aı	n

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)

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

1-hydroxymidazolam

Metabolite

Metabolite

Sedation IV 0.01-0.1 mg/kg

dose

Pre-op Sedation (cont)

Respir- atory Effects	minimal but synergistic respiratory depression when combined with other sedatives
CV Effects	minimal
CNS Effects	anterograde amnesia, anticonvulsant properties, anxiolysis, antispasmodic effects <i>No analgesia</i>
	~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	Transport	patient to	OR via	stretcher	or amulation
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Transport patient to OR via stret	cher 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 <i>at least</i> 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



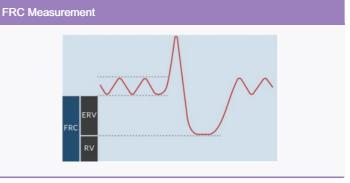
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Dunasinia	tion also Donites reputing (south)
Preoxygena	tion aka Denitrogenation (cont)
	 defined as the time until a patient reaches a saturation level of 88% - 90%, to allow for the placement of a definitive airway.
	$\ensuremath{\overline{\wp}}$ Below this level, oxygen saturation can decrease to critical levels <70% within moments.
Goals of preoxy-genation	
	□ Denitrogenate the residual capacity of the lungs, maximizing oxygen storage
	$\ensuremath{\overline{\wp}}$ Denitrogenate and maximally oxygenate the bloodstream.
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 particularly 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
	ObesePregnantPediatric



Functional Re	esidual 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 \$\tilde{\pi}\$ 1 minute if the patient was breathing room air
Desat formula	time until patient desats = FRC/VO2



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Hypermetabolic pts

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

Onioid	Dotonov



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	n Agents - Ge	neral Anesthesia (cont)
	Clearance	Liver
	Dosing	IV 1-2 mcg/kginduction 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 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 Drowsiness dizziness metallic taste Headache blurred vision paresthesia dysarthria euphoria Nausea Larger doses or if given rapidly Tinnitus Tremor Agitation Cardiovascular changes are usually minimal with the usual doses



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IV Induction	n Agents - G	eneral Anesthesia (cont)
	Uses	o 5% of patients have pain at propofol injection and of these, 1% of them have severe or excruciating pain √ 40 mg Lidocaine prevents this √ Also can mix Lidocaine and Propofol • Propofol and lidocaine= Magic o Add 1 ml of 1 % or 2% lidocaine to a 10 ml syringe of propofol √ Place the IV in an antecubital vein (vs the hand). √ Pretreat with IV opioids. √ 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) GABA-A receptor stimulation hyperplarizes neurons by increasing CI- conductance. More CI- 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	n Agents - Gene	eral Anesthesia (cont)
	Active Metabolite	None
	Induction dose	1.5-2.5 mg/kg IV
	Mainte- nance 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)		IV Induct	tion Agents - Gen	eral Anesthesia (cont)		
Side Effects		o Myoclonus (not a seizure) o Does not cause seizures if the patient	Resp Effects		drive, increased oral secretions (DROOL E, GIVE GLYCO)	
		does not have a history of seizures o Suppression of adrenocortical function	CV Effects	Increased SNS	tone, SVR, HR, and CO	
for up to 24 hrs. It should be avoided in sepsis and acute adrenal failure o N&V (greater than any other induction	sepsis and acute adrenal failure	CNS Effects	causes emerge	IOP, nystagmus and analgesia ence delirium and lowers seizure threshold, severe depression		
		o Acute intermittent porphyria	Food Alle	ergies & Propofol		
Ketamine	MOA	NMDA antagonist (creates dissociated state)	Oversee	n by the	o Propofol can cause anaphylactic reactions, the cause of these reactions is	
	MOA secondary	Many 2nd receptor targets including opioid, MAO, serotonin, NE, muscarinic, and NA channels	Allergy, A	American Academy of Allergy, Asthma and Immunology. They state:	unclear and appears not to be related to soy or egg allergy.	
	Onset IV	30-60 seconds	o Egg allergy		Patients with soy, peanut allergy or egg allergy can receive propofol without	
	Onset IM	2-4 minutes			any special precautions. – Probably safe	
	Onset PO	variable				
	Duration	10-20 minutes (can last 60-90 min to return to full orientation)			allergic to the albumin egg whites. Egg lecithin found in propofol is derived from the YOLK	
	Clearance	Liver	o Soy			
	Active Metabolite	Norketamine	producing an immune r removed during the refi		producing an immune response are removed during the refining process	
	Induction Doses	IV 1-2 mg/kg IM 4-8 mg/kg PO 10mg/kg				
	Opioid Sparing Dose	0.1-0.5 mg/kg or 1-3 mcg/kg/min				



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Food Allergi	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 ☐ Prop is safe to use in pts with a peanut allergy		
o Increased Risk of Bacterial Contam- ination	Propofol syringes must be discarded within 6 hrs 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 o Women (Kg)= Height (cm) - 105		
LBW	Lean body weight		
	√ LBW = 1.3 X IBW		
Drug	Dose	Recommendation	
Propofol	Induction Maintenance	LBW TBW	
Succinylc- holine	Intubation	TBW	
Rocuronium Vecuronium	Intubation Maintenance	LBW LBW	
Cisatr- acurium Atracurium	Intubation Maintenance	TBW TBWvsLBW	

LBW vs TBW (cont)		
Fentanyl ((nl))Suf- entanil	Loading Maintenance	TBW LBW
Remifentanil	Loading Maintenance	LBW LBW
Midazolam	Loading (not preop) Maintenance	TBW TBW
Epidural Local		75% of normal dose
Guadalle Stages of Anaethosia		

Epidurai Locai	75% of normal dose
Guedel's Stages of	f Anesthesia
Stage 1 - Analgesia or Disorientation	o Can be initiated in a preoperative holding area o Patient is given medication and may begin to feel its effects but has not yet become uncons- cious
o Induction stage	☼ Patients are sedated but conversational ☼ Breathing is slow and regular ☼ Patient progresses from analgesia free of amnesia to analgesia with concurrent amnesia ☼ 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

⇔ Blinking increases, and nystagmus may appear
 ⇔ Eyes eventually fix in the midline as the lids

close • GENTLE

☼ Patient becomes unresponsive, atonic, apneic, and the oculocephalic (or more precisely vestibularoculocephalic) and corneal reflexes are lost

Call patients name
 Eyelash reflex

√ Tape eyes- as soon as you lose consciousness

• If you struggle to ventilate they you could hurt their eyes

· Not on sedation cases

• Don't tape in endo watch the L eye

o Eye Protection after Loss of Consci-

ousness

 $\ensuremath{\overline{\bigcirc}}$ 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

o The combination of spastic movements, vomiting, and rapid, irregular respirations can compromise the patient's airway.]

o Fast-acting agents help reduce the time spent in stage 2 as much as possible and facilitate entry to stage 3.

o NEVER EXTUBATE AT THIS TIME

o If you are using gas induction no muscle relaxationyou can really see this

Its really short with IV induction

₽ FOR KIDS

Laryngospasm

· Don't touch them too soon

Stage 3 -Deep o Surgical Anesthesia targeted anesthetic level for procedures requiring general anesthesia

o Ceased eye movements and respiratory depression are the hallmarks of this stage.

o Airway manipulation is safe at this level

4 planes in stage



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Guedel's Stages of Anesthesia (cont)

- eyelid, conjunctival, and swallow reflexes usually disappear in this plane
- Just gazing
- \Box 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.
- \bigcirc Plane 4 is marked by irregular respiration, paradoxical rib cage movement, and full diaphragm paralysis resulting in apnea.

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

Mask Ventila	tion (cont)
Placement of LMA if unable to ventilate	LMA Difficult supraglottic airway placement Restricted mouth opening Obstruction Distorted airway Stiff lungs or C spine
Upper Airway Patency	 Pharynx Collapsible tube inside box Box is formed: Tongue Soft palate Pharyngeal tissue Cervical spine
During inspiration a negative gradient draws air into lungs	Tendency to make airway collapse In awake state 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 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)

Contro versy

- 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
- Tensure 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

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 manipulation

- o Good for specific types of surgery
- o No single agent is ideal for every situation

- What is the Neuromuscular Junction?
- o The neuromuscular junction is a synapse that develops between a motor neuron and a muscle fiber o Made up of several components: the presynaptic nerve terminal, the postsynaptic muscle membrane, and the intervening cleft (or gap)
- (NMJ) o End Plate
 - Acetylcholine is hydrolyzed rapidly by the enzyme acetylcholinesterase in the synaptic cleft
 - ☼ Not all acetylcholine that is released reaches the endplate, some is hydrolyzed en route.



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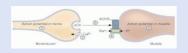


Why Neuromuscular Blockades (NMB)? (cont)

MuscleRelaxants

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

Neuromuscular junction



Muscle Relaxants



End Plate



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
- generating an action potentialdepolarization.
- 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
- Depolarizing neuro muscular blockers
- Bind to alpha subunits
- Cause Channel to remain open- mimics Ach
- Prolonged depolarization occurs

Chemical formula: C14H30N2O4



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Depolarizing l	Depolarizing NMB (cont)		
MOA	agonists at postsynaptic nicotinic acetylcholine receptors and cause prolonged membrane depolarization resulting in neuromuscular blockade		
Onset	IV 60-90 sec IM 2-3 min		
Duration	5 min		
Reversal	None		
Dose	IV 0.5-1.5 mg/kg Ped IV 4-5 mg/kg Laryngospasm: 15 mg/kg/IV or 4-6mg/kg IM		
Metabolism	Psuedocholinesterase		
Adverse Effects	୍ଦି Hyperkalemia ୍ଦି Malignant Hyperthermia ୍ଦି Apnea		

Non-De	polarizing	NMB
THOIT DO	polarizirig	TAIVID

- 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 Compet- itive Antagonist	☼ Two alpha subunits are binding sites for Ach ☼ Sites occupied by nondepolarizing neuro muscular blockers ☼ Cause channel to remain closed ఢ lon flow to produce depolarization can't occur	
Rocuronium	Rocuronium is the most widely used nondepolarizing relaxant in the United States. 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) o Competitive Antagonist – compete with ACH
	Onset	1-2 min
	Duration	20-35 min
	Dose	IV 0.6 - 1.2 mg/kg Infusion 5-12 mcg/kg/min Pretreatment 5mg no reconstitution
	Reversal	Sugammadex 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: .0812 mg/kg 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 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 Priming dose is given prior to succinylcholine rapid sequence induction to decrease the myalgias (5 mg)

Rapid Sequence Induction	—				
	Rank	i Sani	IANCA	Indii	CTION
	i vapic			mu	Guoir

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

Rapid Sequence Induction (cont) Rapid o Preoxygenation is critical o Suction and airway alternatives available Sequence Induction o Use adjuvant drugs to control BP, HR Method 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 o Same steps but with ventilation



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Sequence

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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



General Anesthesia - Inhalation Induction

□ Developmentally delayed adult

Pediatrics

Dotential airway obstruction e.g. epiglottitis

⟨□ Kids or special need,

Sevo dilates vein- if you cant get IV

Contraind-

♠ Aspiration risk

ications

ospasm)

Inhalation Induction Technique Prime circuit with anesthesia agent from vaporizer

at maximum setting

√ Oxygen at 8L/min

 $\ensuremath{ \begin{tabular}{l} \ensuremath{ \begin{tabular}{l} \ensuremath{ \ens$

occluded.

☐ Have patient exhale maximally, then apply face mask to patient and inhale maximally from primed

oirouit

followed by transient apnea, then pattern of rapid

shallow respirations.

They are crying then go dominate

7 Then you put the IV in and tube them

√ Need the pop up valve OPEN

General Anesthesia - Inhalation Induction (cont)

#2 occluded.

√ When patient is comfortable with situation, begin
volatile agent increasing vaporizer setting by 0.5%

every 3 or 4 breaths

Reassure patient with calm voice encouraging a

regular smooth breathing pattern.

√ Use of a deep breathing pattern here may lead to premature onset of apnea with prolonged phase.

ventilation

Don't use N2O if you are trying to get pregnant-

spont miscarriage

For adults or special needs

General Anesthesia - LMA Induction Sequence

Induction 🗸 Pre-Oxygenate

Lidocaine

Propofol

Loss of consciousness

₱ Eye protection

√ Usually don't ventilate

secretions go right in the airway

Fentanyl

LMA

 $\slash\hspace{-0.6em}$ Many anesthesia providers do not give fentanyl on

induction

Others give small dose



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