General Anesthesiology Information

Basic Setup

D. rugs:
1. An induction agent (Propofol, Etomidate, etc.)
2. Muscle relaxants:
   Depolarizing: Succinylcholine (fastest acting)
   Nondepolarizing: Vecuronium, Atracurium, etc.
3. Fentanyl
4. Midazolam
5. Lidocaine
6. Ephedrine
7. Atropine

A. irway:
1. Endotracheal tube with syringe to test/inflate cuff
2. Stylette fitted to endotracheal tube
3. Oral airway
4. Tongue depressor
5. Direct laryngoscope with Macintosh and/or Miller blade

M. achine:
Ventilator or Ambu-bag to deliver positive-pressure ventilation from oxygen source

M. onitors:
1. Blood pressure
2. Pulse oximetry
3. EKG
4. Endtidal carbon dioxide
5. Temperature (optional)

I. ntravenous fluid

S. uction

Note that these items must be readily available regardless of the anesthetic technique provided in case of emergency or the need to change technique and provide general anesthesia.

Five Goals of General Anesthesia

I. Amnesia
II. Anxiolysis/Sedation/Hypnosis: Essentially decreasing the patient's mental status. This goal is referred to by these various names indicating that this is a spectrum of effect from anxiolysis at minimum doses to hypnosis and sleep/coma at higher doses.
III. Analgesia
IV. Muscle Relaxation
V. Reflex Control: Spinal reflexes and cardiovascular reflexes such as increased blood pressure (hypertension) and heart rate (tachycardia).

Medication	Amnesia	Anxiolysis	Analgesia	Muscle Relaxation	Reflex Control
Volatile Agents	X	X	X	X	X
Nitrous Oxide	X	X	X		
Benzodiazepines	X	X		X	
Muscle Relaxants				X	X
Analgesics		X	X	X	X
Induction Agents	X	X	Ketamine	Propofol	Propofol
Local Anesthetics			X	X	X

Note: Ketamine is unique among induction agents for its analgesic properties and Propofol is unique for its ability to best provide muscle relaxation and suppress cough and gag refexes.

Note: Each goal can be achieved by the synergistic use of limited amounts of a particular medication from multiple classes. Thus, the side-effects or toxic-effects of employing large amounts of one medication are avoided. This technique is *balanced anesthesia*.

Note: "MAC" is used commonly to describe an individual's tolerance to anesthetic agents in general. Particularly when describing the amount of anesthesia required to have her/him tolerate a procedure. For example, someone who uses sedatives for sleeping could be described as having a "high MAC". This is different from the pharmacologic definition of MAC which refers to the dose of a particular volatile agent required to prevent motion in fifty percent of people under standard conditions.

Note: A single dose of any induction agent used to induce general anesthesia will result in both cessation of breathing and sleep. The termination of these clinical effects occurs by redistribution of the agent from the brain (receiving a large amount of cardiac output and therefore a large initial dose of the agent) to other blood compartments/organs that are less richly perfused. The initial dose however will not otherwise have been

metabolized when the clinical effects have resolved. This describes the difference between the cessation of clinical effect and pharmacologic metabolism. Spontaneous respiration begins in approximately five mintues and the patient will become arousable after about ten minutes.

Four Physiologic Stages of General Anesthesia

In order of increasing depth:

I. Sedation

Initiated in the Preoperative Anesthesiology Holding Room.
Used as an anesthetic technique itself during "local with sedation" otherwise known as "monitored anesthesia care/MAC"
The patient may not be amnestic. It is possible to have recall and memory of events during this stage.
An example of the use of sedation as an anesthetic technique can be summarized as follows:
A longer-acting sedative is employed to establish a foundation of anxiolysis and sedation (eg. Midazolam). A shorter-acting, more titrateable sedative is then used to make the patient comfortable during highly stimulating but short lasting portions of the procedure (eg. Propofol). Analgesia is provided as needed.

II. Excitation

Etiology remains to be discovered/clarified, however a popular theory describes that it is easier to suppress inhibitory neuronal pathways with an anesthetic that excitatory or neutral neuronal pathways. This results in a state of disinhibition. Clinically seen as:

Hyperreflexia: Autonomic Nervous System: Hypertension, tachycardia
Muscles: Spasticity/ increased tendon reflexes
Airway: Bronchospasm of concern in patients with asthma, COPD, or recent respiratory infection
Laryngospasm Partial spasm of vocal cords with audible sound "stridor" during inspiration denoting airway obstruction. Treated with continuous positive airway pressure of 15 to 25 cm water. Complete spasm of vocal cords with inability to breathe or ventilate. Treated with succinylcholine (one tenth of normal intubating dose up to full intubating dose) and supportive care (mask ventilation or intubation). Mostly occurs during emergence from anesthesia in children and young men due to secretions/blood in airway.

Secretion/excretion: Salivation, lacrimation (tearing), sweating, urination, defecation

Patient has divergent gaze (eyes looking in opposite directions).

III. Surgical

Five goals of anesthesiology are achieved and the patient can tolerate the procedure.

IV. Overdose

The pharmacology of anesthesia has at its basis the suppression of tissues with action potentials. The two of foremost interest are the central nervous system and the cardiovascular system. The nervous system is obtunded more readily (Stage 3), but if this depth is achieved cardiovascular collapse ensues. This occurs by two primary means: suppression of the cardiac pump and rate function and vasodilation in the peripheral bloodstream (decreased systemic vascular resistance). Clinically, this stage occurs most frequently immediately after IV induction and intubation when there is minimal stimulation of the patient to oppose the residual suppressant effects of the induction agents. Alternatively, overdose can occur whenever a patient has a low intravascular volume (prolonged NPO status, poorly-controlled hypertension, bleeding) or pre-existing cardiovascular disease.

Please note that with intravenous inductions patients jump from Stage I to Stage III while during mask (also known as "breathe-down") inductions patients progress through Stage II. Stage III is also the stage in which a patient should be intubated. Neither intubation nor extubation should occur during Stage II due to concern over illiciting or exacerbating the physiologic perturbations defining this stage. During emergence all patients progress through Stage II.

Four Clinical Stages of a General Anesthetic

I. Induction

After placement of monitors this can be achieved either through administering an anesthetic intravenously or by inhalation.

For Regular Intravenous Induction:

(Please note that the goal of IV induction is to deepen the patient from physiologic Stage I to Stage III).

Preoxygenate (also known as denitrogenate) patient.

This provides an oxygen reservoir within the lungs (Functional Residual Capacity) which supplies the patient with oxygen during the time it takes to intubate. This can be done by five minutes of normal breathing of 100% oxygen or 3 vital capacity breaths (maximum inhalation to maximum exhalation).

Administer the induction agent and wait at least 15 seconds for it to

circulate to the brain (can be prolonged with cardiopulmonary diseases/age).

Assess for the appropriate (Stage III) depth of anesthesia. The patient should not respond to voice and should have absent eyelid reflexes (a general index of the suppression of airway reflexes [cough, gag]).

Establish a patent airway (sniffing position) and confirm that you can mask ventilate the patient. This is good practice as you have verified that you can maintain the airway in the event that you cannot ultimately intubate the patient. If you cannot mask ventilate, consider allowing the patient to wake up and perform an "awake" (with sedation) fiberoptic intubation with an intubating scope. A patient will resume spontaneous ventilation after a single bolus of an induction agent within 10 minutes as the agent redistributes among the various physiologic blood compartments. If mask ventilation is difficult, consider using a short acting muscle relaxant (succinylcholine which wears off within 10 minutes). There is a correlation between difficult mask ventilation and difficult intubation.

Administer a muscle relaxant and mask ventilate the patient until it takes effect (30 to 60 seconds for succinylcholine [depolarizing] and 3 to 5 minutes for all other muscle relaxants [nondepolarizing]).
Intubate. Verify that the endotracheal tube is properly positioned by bilateral, equal breath sounds, fogging of exhaled air within the tube, equal chest rise on each side, and the presence of end-tidal carbon dioxide (gold-standard).

For "Rapid-Sequence": (a specialized IV induction for use when the patient is at risk of vomiting and aspiration) the induction includes the following exceptions:

Beginning with the administration of the induction agent, about 10 pounds of **sustained** hand pressure is applied to the cricoid cartilage of the neck thereby using this complete ring of cartilage to occlude the esophagus (Sellick maneuver).

Classic: no mask ventilation is attempted to minimize the likelihood of the aspiration of gastric contents. The actions are: induction agent, Sellick maneuver, succinylcholine, intubate as rapidly as possible.
After intubation is confirmed (by hearing breath sounds and seeing expired carbon dioxide per monitor) the Sellick maneuver is released.

Modified: the ability to mask ventilate is confirmed prior to administration of succinylcholine.

For Mask (Breathe-Down) Induction:

Place the mask of an anesthesia machine circuit primed with an anesthetic gas and oxygen over the patient's mouth and nose. Encourage the patient to take deep breaths.

Monitor the patient as they progress through deeper planes of anesthesia (from physiological Stage I to Stage II then to the "surgical" plane Stage III). Make the appropriate interventions to maintain a patent airway. Depending on the requirements, you could maintain this "mask" airway or intubate the patient.

II. Maintenance

Continuing to administer anesthetic agents to maintain the surgical stage of anesthesia.

III. Emergence

Discontinue administration of anesthetic agents with the goal of returning the patient to their baseline state of physiologic function or a state in which their neurological, cardiovascular, and respiratory functions (among others) are minimally perturbed. This involves but is not limited to reversal of muscle relaxant medications, provision of analgesia, and the restoration of spontaneous, independent ventilation. The patient is monitored as (s)he progress from physiologic Stage III up to Stage I. Interventions are made with regard to the cardiovascular and respiratory threats of Stage II and the airway is prepared for spontaneous ventilation. To allow for spontaneous ventilation the patient must have a "patient, powered, and protected airway". Patientcy is established by freeing the airway from obstruction (usually by the tongue and other soft tissue) with the "head-tilt/jaw-thrust" maneuver or airway stents and suctioning. Airway power is established by the resumption of the patient's muscle ability to cough (to clear their airway) and take deep breaths (to maintain oxygenation). Protection is established by resumption of airway reflexes and airway awareness.

IV. Recovery

This is an extension of emergence during which the goal of returning the patient as close as possible to their baseline state of physiologic function is achieved. Pain management and monitoring/managing complications are of high priority.

Anesthetic Agents

I. Inhalational:

Nitrous oxide: Traditionally advantageous because of its low solubility in blood and lipids. Therefore it has a fast onset/offset and can decrease the required amount of a longer acting volatile agent administered in conjunction, promoting a faster wakeup. Because of its preference for the gaseous phase, nitrous oxide will expand the volume of closed internal cavities. It is therefore contraindicated for such situations as patients with a pneumothorax, middle ear surgery (increased pain and dislodgement of prosthetic devices), retinal reattachments (repeat detachment), and intra-abdominal surgery (bowel expansion with obscured surgical field… this is controversial). It does support combustion.

Volatile agents: Desflurane, Isoflurane, Sevoflurane, Halothane
All decrease cardiac output by decreasing chronotropy, ionotropy, or vasodilation to varying degrees. Isoflurane causes the most vasodilation. Halothane sensitizes myocardium to catecholamines which increases the risk of arrhythmias. Sevoflurane has been implicated in renal failure (in mice) when used at low ventilator gas flows. Sevoflurane is the only agent that cannot cause volatile agent hepatitis (autoimmune response to hepatic metabolites) as it undergoes only minimal, renal metabolism. Desflurane has a low blood/lipid solubility approaching that of nitrous oxide, but can cause tachycardia. All support combustion at supraclinical concentrations. All volatile agents can cause malignant hyperthermia.

All inhalational agents increase the risk of postoperative nausea and vomiting.

II. Induction:

Propofol: Lipophilic. Has a large volume of distribution and short context-sensitive half-life. Wears off within 10-15 minutes unless supraclinical doses saturate its volume of distribution. Has the least incidence of postop nausea and vomiting of induction agents and can be used in treatment of nausea. Alcohol used to emulsify propofol in solution burns upon injection (lidocaine in IV first). Causes the most hypotension of all induction agents through vasodilation and myocardial depression (controversial). Does not have preservative and supports bacterial growth after six hours from opening.

Barbiturates: Thiopental and methohexital. Thiopental has a longer half life (10 hours vs. 4 hours). Both can cause hypotension from vasodilation. Can cause acute porphyric state in patients with porphyric disease (metabolism upregulates porphyia production hepatically).

Etomidate: Causes the least amount of hypotension of the induction agents via minimal vasodilation. Lipophilic, packaged with alcohol (burning) like Propofol.

Ketamine: Mediates "dissociative anesthesia" (profound separation of consciousness from body and its vital processes resulting in a cataleptic-like state). Causes the least respiratory depression (unless induction dose given) of induction agents. Only induction agent providing analgesia. Only induction agent that supports cardiovascular system by increasing sympathetic tone. Increases intracranial pressure. Only induction agent that works by antagonizing NMDA receptors (excitatory). All others have effect by upregulating GABA-receptor (inhibitory) function.

Benzodiazepines: Midazolam, lorazepam. Best agents at producing amnesia (lasting 30-45 minutes for midazolam). Have mild muscle relaxant ability. Only induction agent with reversal agent.

III. Muscle Relaxants:

Depolarizing: Succinylcholine. Fasting-acting relaxant (within 30-60 seconds). Contraindicated in denervating muscular disease/injury including CVA, 3rd-degree burns, myopathies). Can cause malignant hyperthermia.

Nondepolarizing:

Short acting (<30 minutes): Atracurium, rocuronium. Atracurium can cause hypotension due to histamine release and is metabolized by hydrolysis in plasma.
Intermediate acting (45 minutes): Vecuronium. Most cardiovascularly stable.
Long acting (1 hour): Pancuronium. Can cause tachycardia. Renal metabolism.

IV. Opioids:

Morphine: Can cause hypotension from histamine release.
Fentanyls: Alfentanyl, fentanyl, sufentanyl, remifentanyl. Can cause bradycardia. Remifentanyl has shortest half-life (4 minutes).
Hydromorphone (Dilaudid): Seven times as potent as morphine.
Meperidine (Demerol): Treatment for anesthetic-induced shivering. Normeperidine metabolite can cause seizures.

Local Anesthetic Toxic Doses

Agent	Maximum Dose Per Kilogram (Duration in minutes) Without Epinephrine	With Epinephrine
Ester		
Procaine (Novocaine)	7 (45)	10 (90)
Chloroprocaine (Nesacaine)	11 (60)	14 (90)
Amide		
Lidocaine(Xylocaine)	3-5 (120)	7 (240)
Mepivacaine(Polocaine)	5 (180)	5 (360)
Prilocaine(Citanest)	5 (90)	7 (360)
Bupivacaine (Marcaine/Sensorcaine)	2.5 (240)	3 (480)
Levobupivacaine (Chirocaine)	2.5 (240)	3 (480)
Ropivacaine(Naropin)	2.5 (240)	3 (480)

Note: These doses refer to infiltration techniques only.
Note: ½ of the maximum dose can be reinfiltrated after ½ of the block duration time (clinical half-life) has elapsed.
Note: The absolute maximum dose (regardless of obesity, etc.) is usually based on 70 kg.
Note: Local anesthetics with two "i's" in the name is an amide.
Note: Epinephrine is added at a concentration of 1:200,000 or 5 mcg/cc.
Note: Sodium bicarbonate can be added to local anesthetics as follows:

1 cc of 1 mEq/cc sodium bicarbonate solution to 9 cc of lidocaine
0.1 cc of sodium bicarbonate solution to 10 cc of bupivacaine (due to precipitation of bupivacaine in basic solution).

Note: The doses referenced are for adults but have been used in children. Pediatric doses of many local anesthetics need to be established.

Oxygen Delivery Devices for Spontaneously Breathing Patients

Device	Flow Rate (Liters/min)	Inspired Concentration%
"Blow-by"	15	Variable, > 21%
(Face in front of open-ended tube.) Used in Recovery Room in pediatrics and situations where other devices are not tolerated due to lack of comfort.		
Nasal cannula	2 to 6	28 to 44
Simple facemask	2 to 10	40 to 60
Partial rebreather facemask	6 to 15	Variable, 40 to 80
Similar to standard system in Recovery Room.		
Nonrebreather facemask	12 to15	80 to100
(with reservoir bag and one-way vent for exhaled air)		
"T" piece	15	Variable, > 70%
(Endotracheal tube attached to side of open-ended tube.) Used in Recovery Room on intubated patient awaiting extubation.		

Kurt Baker-Watson, MD
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