CHAPTER 1

Core Anesthesia Information

CONTENTS

1.1 Formulas and Conversions

Pound – Kilogram

$$1 \text{ kg} = 2.2 \text{ lb}$$
$$\text{lb} \div 2.2 = \text{kg}$$

Ideal Body Weight

Cm in height—100 = kg weight

OR

Males: 50 kg + 2.3 kg for each inch above 5 feet
Females: 45 kg + 2.3 kg for each inch above 5 feet

Body Surface Area or Mass Index

$$\text{BSA} = \text{m}^2$$
$$\textbf{BMI} = \frac{\text{weight in kg}}{\text{height in m}^2}$$

BMI categories of obesity:

- Normal BMI: 19–24
- Overweight BMI: 25–29
- Obese BMI: 30–39
- Extreme morbid obese BMI: 40–49
- Super morbidly obese BMI: > 50

Inch-Centimeter

$$1 \text{ in} = 2.54 \text{ cm}$$
$$\#\text{ inches} \times 2.54 = \text{cm}$$

Pressure Conversion

1 atmosphere = 760 mmHg (pressure/sq. in. @ sea level) = 14.7 psi

Temperature

$$F° = C \times 1.8 = + 32$$
$$C° = F - 32 = \div 1.8$$
$$- 273C = 0 \text{ K (zero Kelvin)} - K = C + 273$$

Intravenous Fluid Calculations

How to Figure mg or mcg per mL in IV bag, i.e., 400 mg dopamine mixed in 250 mL

$$\frac{400 \text{ mg}}{250 \text{ mL}} = 1.6 \text{ mg/mL}$$

To get mcg/mL: 1.6 × 1000 (for total mL in liter bag) = 1600 mcg/mL

IV Infusion Formula to find mL/hr

$$\frac{\text{Dose} \times 60 \text{ mins} \times \text{kg}}{\text{mcg/mL}}$$

That is, how many mL/hr to give 10 mcg/kg/min of Dopamine

pt kg weight: 70 kg
Dopamine mixed 400 mg in 250 mL
drip concentration: 1.6 mg/mL or 1600 mcg/mL

$$\frac{10 \times 60 \times 70}{1600} =$$

IV Infusion Formula to Find Dose Being Given at Certain mL Rate

$$\frac{\text{mcg/mL} \times \text{mL running}}{\text{kg wt} \times 60\ \text{mL}} = \text{mcg/kg/min}$$

Concentration: How Many Grams in IV Bag?

How many grams of dextrose are in a 1-liter bag of D5W?

D5 is 5% = 5 g/100 mL
5 g/100 mL × 10 for a liter bag = 50 g of dextrose in 1-liter bag
Or. . . 5 g/100 mL × 1000 mL/1 liter = 50 g

Ratio to mg/mL

Can express a ratio as a fraction: 1:1000 is also 1/1000 = 1 g in 1000 mL liquid

1,000 mg in 1,000 mL = 1 mg/1 mL

Quick method cheat:

1:1000 = 1 mg in *(take off 3 zeros)* in 1 mL = 1 mg in 1 mL
1:200,000 = 1 g/200,000 mL = 1 mg in 200 mL
1:300,000 = 1 mg in 300 mL

Ratio to mcg/mL

1:200,000 epinephrine is how many mcg/mL?

1 g/200,000 mL = 1,000,000 mcg/200,000 mL = 5 mcg/mL

Percent to mg/mL

move the decimal one place to right for mg/mL
5% = 50 mg/mL,
1% = 10 mg/mL; i.e., 25% albumin = 25 g/100 mL = 250 mg/mL

Mg to Percent Solution

figure mg/ml and move decimal one place to left
1 mg/mL = 0.1%

1.2 Neuromuscular Junction (NMJ)

Information regarding neuromuscular monitoring can be found in Chapter 3. 3. Neuromuscular drugs can be found in Chapter 5. 8. under Muscle Relaxants.

Includes: distal motor nerve terminal, synaptic cleft, and motor end-plate on muscle fiber.

As a nerve's action potential depolarizes, an influx of calcium ions moves transmitter storage vesicles to fuse with the terminal plasma membrane and release their

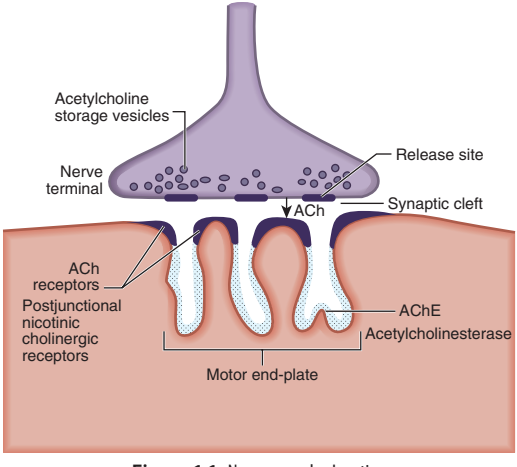


Figure 1-1 Neuromuscular Junction

contents—acetylcholine (ACh)—into the synaptic cleft. These ACh molecules move across the synaptic cleft to bind with nicotinic cholinergic receptors on the motor end-plate.

Each ACh receptor in the neuromuscular junction normally consists of five protein subunits. Only the two identical alpha subunits are capable of binding ACh molecules. When both alpha subunits are occupied by ACh (the channel will not open if ACh binds on only one site), the ion channel in the center opens and permits rapid entry of sodium ions, depolarizing the motor end-plate and bringing about muscle contraction. ***ACh is the key to unlocking the sodium channel to allow depolarization to progress.*

Acetylcholine is not tightly bound to the receptor and detaches easily, being rapidly hydrolyzed by acetylcholinesterase (AChE; also called true cholinesterase); metabolism occurs rapidly because AChE is embedded into the motor end-plate membrane adjacent to the ACh receptors. Acetylcholine's metabolites reenter the motor nerve endings to be synthesized into more ACh. Once ACh is no longer on the receptors, the receptor's ion channels close and the end-plate repolarizes.

The postjunctional nicotinic cholinergic receptors are the most prevalent and are of the most concern in normal neuromuscular functioning. However, there are also extrajunctional cholinergic receptors, but their synthesis is suppressed by neural activity. This becomes of importance in muscle wasting disease because the disuse of muscles allows these receptors to massively proliferate. This accounts for the substantial potassium release with succinylcholine in these disease states.

Neuromuscular Relaxation Principles

Both depolarizing and nondepolarizing neuromuscular blocking drugs act at several sites at the neuromuscular

junction but primarily at the postjunctional nicotinic cholinergic receptors. These drugs have no analgesic or amnestic properties.

Depolarizing Muscle Relaxant (DMR)

Depolarizing muscle relaxants act as ACh receptor agonists. The DMR (succinylcholine) closely resembles ACh molecules, which readily bind to ACh nicotinic receptors, inactivate voltage-gated sodium channels at the NMJ, increase potassium permeability in the surrounding membrane, and prevent a muscle action potential.

Unlike ACh, however, succinylcholine is not metabolized by AChE, and its concentration in the synaptic cleft does not fall as rapidly, resulting in a prolonged depolarization of the muscle end-plate. When the sodium channels sense the membrane depolarization, they first open, then close and become inactivated, which causes the muscles to become flaccid.

Metabolism of Succinylcholine

Plasma cholinesterase (also called pseudocholinesterase) has an enormous capacity to hydrolyze succinylcholine so much so that the recommended dosage is a "relative overdose" because only 10% of the injected dose ever reaches the neuromuscular junction. Plasma cholinesterase is synthesized in the liver and found in plasma; it hydrolyzes succinylcholine, ester local anesthetics, Novocaine (procaine), and cocaine. Half-life of pseudocholinesterase is 8–16 hours.

Reduced Pseudocholinesterase Levels

- Pregnancy
- Advanced age
- Liver disease
- Burns

- Certain drug therapies: echothiophate, metoclopramide, monamine oxidase inhibitors, oral contraceptives, cytotoxic drugs, and others)

Genetically Aberrant Enzyme

Patients with abnormal pseudocholinesterase (a genetic disease) are at increased risk of toxic side effects due to slower metabolism of succinylcholine or other drugs.

Dibucaine, a local anesthetic, normally inhibits normal pseudocholinesterase activity by 80% (so a patient with normal pseudocholinesterase has a dibucaine number of 80).

Dibucaine # = % inhibition of pseudocholinesterase = proportion of function. Only when there is a > 75% decrease in levels of normal pseudocholinesterase that is clinically evident is prolongation of succinylcholine seen.

Dibucaine inhibits the

*homozygous atypical enzyme by 20%—1:3200, prolonged block 2–8+ hours

*heterozygous by 40–60%—1:480, block slightly prolonged: 20–30 minutes

Nondepolarizing Muscle Relaxant (NDMR)

NDMRs function as competitive antagonists; NDMRs antagonize the action of ACh in a competitive manner at the postsynaptic nicotinic receptor. They do not produce conformational changes in the receptor, unlike depolarizing drugs. A NDMR binds to the receptor and thus prevents ACh from attaching to the receptor and affecting change.

With the antagonist blocking the receptor, there is a gradual reduction in end-plate potential until it fails to reach the threshold to fire off a propagating action potential to produce muscle contraction. Neuromuscular block, expressed as depression of the single twitch height, only becomes evident when 70–80% of receptors are occupied by nondepolarizing muscle relaxants.

NDMRs have a faster onset in the pediatric population; in an obese patient, you should dose 20% more than lean body weight.

Reversal of Neuromuscular Blockade

Anticholinesterase agents (also called cholinesterase inhibitors) include neostigmine, edrophonium, pyridostigmine, and sugammadex (edrophonium and physostigmine are also anticholinesterase agents but not ones given for muscle relaxant reversal).

The goal of reversal agents is to increase the amount of ACh at the NMJ so that it will compete with the nondepolarizing muscle relaxants for the receptor, thereby reestablishing neuromuscular transmission. Simply, its goal is to maximize nicotinic transmission.

Drugs such as neostigmine increase the local concentration of ACh in the synaptic cleft by one of two ways: either by slowing normal destruction of ACh (by the enzyme AChE) or by increasing its release from the nerve terminal.

Anticholinesterase agents act not only on nicotinic receptors for their intended action but they also act on muscarinic receptors, especially in the parasympathetic nervous system. The most prominent side effect of the anticholinesterase drugs is bradycardia, though salivation, increased bronchial secretions, and urinary and fecal incontinence also can occur. Anticholinesterase drugs need to be given with anticholinergics to offset anticholinesterase's muscarinic stimulation side effects.

Anticholinergics are given either immediately before or concurrently with an anticholinesterase agent to counteract unwanted muscarinic side effects: *cheat to remember unwanted muscarinic side effects: SLUDBBM—salivation, lacrimation, urination, defecation, bradycardia, bronchoconstriction, and miosis (pupil constriction).*

TABLE 1-1 Response to Muscle Relaxation in Disease States

Neuromuscular Disease	Depolarizer	Nondepolarizer
Lambert-Eaton (Myasthenic Syndrome) Decreased release of ACh	Hyperkalemia Very sensitive	Very sensitive
Myasthenia Gravis Decreased number of ACh receptors	Resistant or unpredictable	Extremely sensitive
Muscular Dystrophy (Duchenne's)	Rhabdomyolysis Hyperkalemia	Very sensitive
Myotonia	Malignant hyperthermia	Normal to very sensitive
Familial Periodic Paralysis	Hyperkalemia	Sensitive
DEMYELINATING		
Guillain-Barré syndrome	Hyperkalemia	Sensitive
Multiple Sclerosis	Hyperkalemia	Unpredictable
NEURODEGENERATIVE		
Parkinson's Disease	Hyperkalemia	No change
Huntington's Chorea	Prolonged effect	Sensitive
Amyotrophic Lateral Sclerosis	Hyperkalemia	Hypersensitivity
OTHER		
Burns	Hyperkalemia	Resistant
Cerebral Palsy	Hypersensitivity	Resistant
Epilepsy	No change	Resistant
Hemiplegia	Hyperkalemia	Resistance on affected side
Muscle Denervation	Hyperkalemia	Resistant

1.3 Postoperative Nausea and Vomiting (PONV) or Postoperative Vomiting (POV)

See Chapter 5 Antiemetics for more information.

Nausea and vomiting is triggered by stimulation of several sites within the brain, including:

- chemoreceptor trigger zone (CRTZ), in the area postrema in the floor of the fourth ventricle of the brain
- medulla oblongata of the brainstem
- nucleus tractus solitarius (NTS)

Other central and peripheral sites play a role as well.

These structures react to drugs, metabolites, hormones, or emetogenic toxins in the cerebral spinal fluid and blood. The neurotransmitters and receptors implicated in the control of nausea and vomiting include serotonin (5-HT3 receptor), acetylcholine (ACh), dopamine (D2), histamine (H-1 receptor), and substance P (NK-1 receptor).

Neuronal pathways connect the cerebral cortex associated with balance, salivation, respiration, and vasomotor to the emetic center.

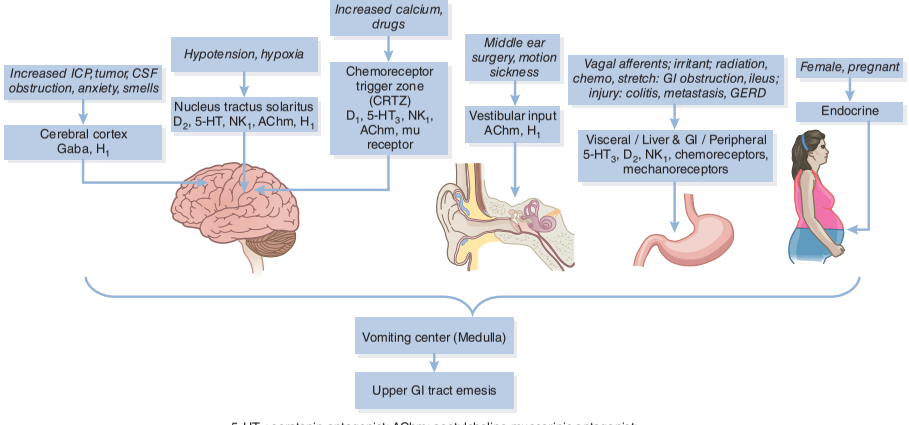
Antiemetic medications target these receptors to completely inhibit or greatly reduce vomiting.

Do we always give PONV prophylaxis? Use of prophylactic antiemetics should be based on valid assessment of the patient's risk for POV or PONV. Ideally, PONV can be prevented. Depending upon the level of risk, PONV prophylaxis should be initiated using interventions that reduce baseline risk (nonemetogenic anesthetic techniques), non-pharmacologic approaches, and monotherapy or combination antiemetic therapy.

Strategies to Reduce POV/PONV Risk

Avoidance of general anesthesia by the use of:

- Regional anesthesia
- Use of propofol for induction and maintenance of anesthesia (total intravenous anesthesia [TIVA])
- Avoidance of nitrous oxide



5-HT_3: seratonin antagonist; AChm: acetylcholine muscarinic antagonist;
D_2: dopamine antagonist; H_1: histamine 1 antagonist; NK_1: substance P antagonist

Figure 1-2 PONV

- Avoidance of volatile anesthetics
- Minimization of intraoperative and postoperative opioids
- Minimization of neostigmine
- Adequate hydration

Postoperative Nausea and Vomiting (PONV) Risk Factors

Patient-specific Risk Factors—the most important are bolded

- **Female gender**
- **Nonsmoking status**
- **History of PONV/motion sickness**
- Migraine
- Peds: 6–10 years old (34%)
- 11–14 years old (35–50%)
- Anxiety

Anesthetic Risk Factors—the most important are bolded

- **Use of intraoperative and postoperative opioids**
- **Use of volatile anesthetics**
- **Use of nitrous oxide**

Surgical Risk Factors

Duration of surgery: each 30-minute increase in surgery duration increases PONV risk.

Surgical Procedure

- Abdominal laparoscopic procedures
- Abdominal laparotomy
- Ear, nose, and throat
- Strabismus—eye
- Breast
- Plastics
- Maxillofacial
- Gynecological
- Neurologic/craniotomy
- Ophthalmologic
- Urologic

Decreases Risk
- Smoking

Risk Factors for POV/PONV Specific in Children
- Surgery > 30 minutes
- Age > 3 years
- Strabismus surgery
- History of POV or PONV in relatives

TABLE 1-2 Predictors for PONV

Number of independent predictors present	Risk for PONV in adults	Risk for PONV in pediatrics
0	10%	9%
1	20%	10%
2	40%	30%
3	60%	55%
4	80%	70%

- **Low risk for PONV (no risk factors)**—Some providers feel that patients with low risk for PONV do not require PONV prophylaxis. However, for patients in whom vomiting poses a particular medical risk, including those with wired jaws, increased intracranial pressure, gastric or esophageal surgery, more liberal prophylaxis is appropriate.
- **Moderate risk for PONV (1–2 risk factors)**—Patients at a moderate risk for PONV should receive 1 to 2 prophylactic agents.
- **High risk for PONV (2–3+ risk factors)** — Given that there are many antiemetic drugs that work on different receptor classes, many clinicians use multiple (two, three, even four different) pharmacologic agents for prophylaxis in patients who are at high risk for PONV.

Rescue therapy for PONV is not as effective as prophylaxis. Patients who have failed one class of drug as a prophylactic medication should be treated with a different class as the rescue antiemetic.

An emetic episode more than 6 hrs after surgery can be treated with any of the drugs used for prophylaxis except dexamethasone and transdermal scopolamine.

1.4 Anaphylaxis

Anaphylaxis is a systemic, immediate hypersensitivity reaction caused by immunoglobulin Ig E-dependent activation of effector cells of the immune system—predominantly mast cells and basophils. True antibody-mediated allergic reactions are extremely rare.

Anaphylactoid reactions mimic anaphylactic reactions but are not mediated by immunoglobin E (IgE)-dependent activation; the patient does not have to have been sensitized to a substance, and reexposure to a substance does not have to occur. The anaphylactoid reaction can be as equally life-threatening as anaphylaxis.

Regardless of the triggering agent, anaphylactic or anaphylactoid reactions occur as a consequence of inappropriate and overwhelming mast cell or basophile activation. These reactions usually occur immediately but may be delayed depending on the route of exposure.

Anaphylaxis/Anaphylactoid triggers in anesthesia

#1 Nondepolarizing muscle relaxants (60%)

High Risk: rocuronium (56%), succinylcholine (21%)

Intermediate Risk: vercuronium (11%), atracurium, and pancuronium

Low risk: mivacurium and cisatracurium

#2 Latex: patients who have had multiple prior surgical procedures can be sensitized to latex. Healthcare workers are also at increased risk. Recognized cross-sensitivity to bananas, avocado, and chestnuts.

#3 Antibiotics: penicillins, cephalosporin, vancomycin

#4 Colloids

#5 Hypnotics/induction: propofol, thiopental, midazolam

#6 Opioids: morphine, meperidine, codeine

#7 Radiocontrast dyes

Also: Blood products: red blood cells, platelets, cryoprecipitate, and fresh frozen plasma: anaphylactic reactions occur in 1:20,000–1:47,000 components transfused.

Direct skin contact: chloroprep, iodine, hibiclens, chlorhexidine

Physiologic Response

Cardiovascular Effects

Vasodilation and fluid extravasation due to increased vascular permeability with a hypovolemic shock pattern. Circulating blood volume may decrease by as much as 35% within 10 minutes.

Chemical mediators can cause an acute coronary spasm, which results in a myocardial infarction and acute coronary syndromes.

Hypotension and tachycardia may rapidly progress into severe arrhythmias and cardiovascular collapse if not treated quickly.

There is a direct correlation between the onset of symptoms and potential for death. The more rapidly anaphylaxis occurs, the more likely the reaction is to be severe.

Respiratory Effects

Smooth muscle spasm in the respiratory tract. Signs/symptoms include copious secretions from the mouth and nose, shortness of breath, elevated $ETCO_2$, and an increase in the peak airway pressure. This quickly progresses to bronchial spasm and hypoxia.

Angioedema is most apparent in the head and neck, including the face, lips, floor of the mouth, tongue, and larynx, but edema may involve any area of the body. Angioedema, if untreated, will advance to complete airway obstruction and death caused by laryngeal edema.

Laryngeal edema

Bronchospasm

Pulmonary edema

Rapidly declining oxygen saturation

Degrees of Anaphylaxis

Grade 1—Cutaneous signs, hives, itching, angioedema

Grade 2—Cutaneous signs along with hypotension, tachycardia, dizziness, wheezing, shortness of breath, GI symptoms

Grade 3—Above signs plus cardiovascular collapse, dysrhythmias, bronchospasm, $SA0_2 < 92$, loss of consciousness

Grade 4—Cardiac arrest, death

Preventing Perianesthetic Reactions

A careful history regarding adverse drug reactions and allergies should be conducted before any surgical procedures requiring anesthesia.

Even if the patient is not specifically known as allergic to a substance, identification of at-risk properties, such as morphine, antibiotics, and nondepolarizing muscle relaxants, should be administered as slowly as possible.

Recognition of Anaphylaxis in the Operating Room

A survey of anaphylaxis during anesthesia demonstrated that cardiovascular symptoms (73.6%), cutaneous symptoms (69.6%), and bronchospasm (44.2%) were the most common clinical features. Surgical drapes and patient position can hide cutaneous symptoms.

Perioperative anaphylaxis usually occurs within 1 minute after anesthetic induction and is primarily related to medications administered intravenously.

Treating the Patient

- Remove the offending trigger if it is identified
- Call for help
- Give oxygen
- Insert two large bore intravenous or intraosseous catheters:
  - Normal Saline Adults: 1000 mL IV, give wide open; may repeat as needed until the BP has stabilized. May require 5–7 L.
  - Normal Saline Children: at 30mL/kg in first hour.
  - *Do not use dextrose solutions in anaphylaxis (after dextrose metabolized, fluid remaining is just free water and hypotonic and will leak out into tissues).*
- **Epinephrine** 1:1000 (1 mg/mL): epinephrine is the drug of choice in treatment of anaphylaxis due to the alpha$_1$ effects that support blood pressure while beta$_2$ effects result in bronchial smooth muscle relaxation.
  - The decision to give by IM or IV depends on episode severity and if patient is in shock. In most instances, epinephrine can be given by IM route, but if the patient is in extremis and an IV is present, the IV route should be used.

- **Epinephrine IM route 1:1000:**
  - Adult: 0.3 to 0.5 mL given in the vastus lateralis muscle. The dose can be repeated every 5 to 15 minutes, depending on the response, × 3–4 doses.
  - Age 6–12 years of age dose: 0.3 mL IM given in the vastus lateralis muscle. The dose can be repeated every 5 to 15 minutes, depending on the response, × 3–4 doses.
  - Child < 6 years of age dose: 0.01 mg/kg IM given in the vastus lateralis muscle. The dose can be repeated every 5 to 15 minutes, depending on the response, × 3–4 doses.
- **Epinephrine IV route 1:10,000:** (avoid giving 1:1000 concentration intravenously)
  - **Adult:** give 0.5 to 2.5 mL, every 10 to 20 minutes.
  - **6–12 y:** 0.3 mL, every 10 to 20 minutes.
  - **< 6 y:** 0.15 mL
- **Benadryl** (Diphenhydramine)—H1 blockers
  - Adult dosage 50 mg IV push over 2 minutes × 1
  - Child—1.25 mg/kg, IV push over 2 minutes × 1 not to exceed 50 mg
- **Hydrocortisone** (Solu-Cortef): adult dose 100 mg IV push, q 15 minutes × 2
  - Child—6mg/kg IV push, not to exceed 100 mg per dose, q 15 minutes × 2
- **Zantac** (Ranitidine)—H2 blocker
  - Adult dose 50 mg/50 mL IV piggyback over 15 min (200 mL/hr.)
  - Child—1mg/kg/dose, not to exceed 50 mg IV piggyback over 15 minutes
- **Albuterol** nebulizer—beta$_2$ agonist/bronchodilator
  - (2.5 mg/3 mL) q 5 minutes × 3 for wheezing or bronchospasm
- Begin ACLS or PALS if needed

For Refractory Anaphylaxis in Patients on Beta Blockers
Glucagon may be useful for patients who become anaphylactic and who are also on beta blockers. Glucagon has positive chronotropic and inotropic effects that are independent of catecholamine receptors and bypass the receptors obstructed by beta blockers.

Adults: 1 mg IV, IM, SQ, IO, IN; give 1 to maximum 5 mg IV push q 5 mins. Infusion titrated 1 to 5 mg/hour, max benefit at 5–15 minutes. *IV: intravenous; IM: intramuscular; SQ: subcutaneous; IO: intraosseous, IN: intranasal*

Children: 6–12 y: 0.5–1 mg IV, start at 0.5 mg. Maximum administered over 5 minutes.

For Refractory Hypotension
Dopamine 1–20 mcg/kg/min IV infusion—adrenergic and dopaminergic agonist

Biphasic Anaphylaxis

After successful recovery from the original anaphylactic episode, a recurrence of anaphylactic symptoms can occur in approximately 20% of patients 1–72 hours after the original episode with no reexposure to allergen. If the patient had a near fatal anaphylactic response with original exposure, the risk of a fatal or near fatal biphasic reaction is up to 1/3 of patients.

The biphasic reaction can be less severe but can also be as severe as the original episode. Systemic corticosteroids do not seem to be able to prevent recurrence.

Serum Lab Test for Anaphylaxis

Lab tests that suggest an IgE-mediated anaphylactic reaction:

- Serum mast cell tryptase (a marker of mast cell activation) peaks at 30–60, half-life 2 hrs
- Compliment C3 and C4

1.5 Airway-Pulmonary

Laryngospasm

An acute total and profound glottic closure caused by contraction of both cricothyroid adductor muscles; stimulated by increased secretions or mechanical irritation of trachea or larynx.

Potential problems include hypoxia and negative-pressure pulmonary edema (NPPE).

- Partial spasm
  - Some air movement
  - Crowing/phonation
  - Paradoxical chest movement
- Complete
  - No air movement
  - Need to identify quickly

Treatment includes:

- Remove stimuli—pain, secretions, oral airway, etc.
- Jaw thrust with continuous positive pressure with mask and 100% FiO_2.
- If unsuccessful, give succinylcholine 0.5 mg/kg to relax muscles and mask ventilation.
- Humidified oxygen and nebulized racemic epinephrine for stridor.

Bronchospasm

A lower airway obstruction characterized by smooth muscle contraction and increased resistance to airflow through the bronchial tree. Occurs most often in patients with hyperactive airway (asthmatics, chronic bronchitis) due to sputum or secretions in the pulmonary tree. In an anesthetized patient, bronchospasm is detected by an increase in peak airway pressures (decreased lung compliance) and wheezing on both inspiration and

exhalation. It is best to try and avoid than to treat once it happens as severe bronchospasm can lead to hypercarbia, hypoxemia, impaired venous return (increased intrathoracic pressures), and decreased cardiac output. Chronic treatment includes bronchodilators and steroids. Acute treatment includes beta2 agonists such as albuterol, high concentrations of volatile inhaled agents, and 100% FiO_2. Epinephrine and aminophylline may also be used. Succinylcholine is NOT helpful in this scenario.

Asthma

Treat: bronchodilation with albuterol puffs,
IV aminophylline, racemic epinephrine nebulizer

Drugs to avoid in asthma patients

- beta2 blockers (i.e., propranolol or labetalol)
- histamine-releasing drugs
  - succinylcholine, atracurium, morphine, Demerol, mivacurium, trimethaphan (ganglionic blocker; antihypertensive)

Aspiration

Aspiration is also a cause for respiratory failure and death in patients. And while some complications may be unpredictable, aspiration has some degree of preventability and requires certain preparation and choice of anesthetic techniques from providers such as: patient positioning (head of bed up), not using an laryngeal mask airway (LMA), nasogastric tube placement before induction and immediately after induction, agents such as gastrokinetics (IV metoclopramide), histamine1-2 blockers, anticholinergics, oral antacids (preoperative PO Bicitra), proton pump inhibitors that block acid production in the stomach (Protonix, Prilosec,

Prevacid, etc.), and antiemetics—used alone or in various combinations—in patients who exhibit risk factors for aspiration.

Risk Factors for Aspiration

- all emergency surgery patients are considered "full stomach"
- unconscious patient
- nausea and vomiting
- obstetric patients
- full abdomen (ascites, small or large bowel obstruction, obesity)
- hiatal hernia
- gastroesophageal reflux disease (GERD)
- delayed gastric emptying (diabetic, chronic kidney disease)
- positive pressure ventilation
- difficult airway/intubation
- decreased lower esophageal tone

Lower Esophageal Sphincter (LES): The lower esophageal sphincter (LES) is a bundle of muscles at the low end of the esophagus where it meets the stomach. When the LES is closed, it prevents acid and stomach contents from traveling backwards from the stomach but when relaxed, gastric contents are allowed to move from the stomach into the esophagus.

*Anesthesia decreases LES tone and inhibits gag reflex.

Increased LES tone—decreases aspiration risk

- Metoclopramide, succinylcholine, histamine, ACh, pancuronium, neostigmine, edrophonium, metoprolol, prochlorperazine, alpha adrenergic stimulants

Decreased LES tone—increases aspiration risk:

- Conditions: pregnancy, obesity, intestinal obstruction, hiatal hernia, fatty meals, general anesthesia, reflux disease, Sellick's maneuver (cricoid pressure actually induces relaxation of the lower esophageal sphincter; its efficacy lacks scientific validation).
- Drugs: anticholinergics, nitroglycerin/nitroprusside, inhalation agents, opioids, beta agonists, benzodiazepines, high-dose propofol

1.6 Emboli

Venous Air Emboli (VAE)

Air emboli occur when a pressure gradient develops from surgical site to right atrium—for every inch difference in height is ~ 2 torr pressure difference (mmHg)

Surgical open space: non-collapsible bone or venous space held open either surgically or by venous sinuses/porous bone/bone compression or use of pressurized gas source.

- 30–50% sitting position posterior fossa procedures entrain air
- 8% side-lying posterior fossa procedures entrain air

Paradoxical emboli via Patent Foramen Ovale (PFO), which occurs in 25% population, or other shunts from the right to left heart are more susceptible to paradoxical air embolism, which leads to ischemia with devastating consequences.

Signs/Symptoms

- Air emboli can be as small as 0. 25 mL (range 0.1–0.5 mL).
- Right heart air emboli:
  - Symptomatic = 1 mL/kg
  - Lethal = 10 mL/kg

- $ETCO_2$ drops suddenly (due to fall in cardiac output and increased dead space) and nitrogen increases; large embolism gas lock of right heart produces decreased SaO_2, hypotension, tachycardia, arrhythmias, increased pulmonary artery pressures (PAP), and central venous pressure (CVP), cyanosis, hypoxia, wheezing; symptoms of pulmonary edema, and hypocapnia. CO will fall and circulatory collapse may result.
- Potential end organ damage from paradoxical air emboli to the microvascular circulation.

Detector of Air Embolism: From Most to Least Sensitive

- Transesophageal echocardiography (TEE) but too sensitive, picks up every single bubble
- Doppler: the #1 choice for detection of VAE. Placement? RSB 2–6th ICS
  - Precordial Doppler: measures velocity; can hear "mill wheel murmur" with VAE
  - Check Doppler 10 mL syringe of NS in so-called "whoosh" test: increases turbulence
    - flow so you can hear flow through Doppler. Inform surgeon before testing—surgeon is very tuned in to that sound.
- Capnography/Mass spectrometry/ETN2

Treat Air Emboli

- Inform everyone
- Stop N_2O if giving
- Give 100% O_2
- If CO_2 insufflated—immediately release peumoperitoneum
- Neck vein compression/NSS to field/Occlude open site/ stop pressurized gas
- Aspirate CVP
- Trendelenburg/left lateral decubitus position (if possible)

- Positive end expiratory pressure (PEEP) (questionable practice)
- Treat hypotension/arrhythmias
- Volume, inotropes, antiarrhythmics
- CPR—crack chest?

Air Embolism in Orthopedic Surgery
Etiology: during cement impaction, air is forced into medullary vessels or with high pressure lavage.

Carbon Dioxide Gas Embolism

There is a risk of carbon dioxide embolus during a laparoscopic procedure with carbon dioxide insufflation; gas may enter the circulation through any opening in an injured blood vessel. This type of embolism is the most dangerous complication associated with laparoscopy. It develops principally during induction of a pneumoperitoneum. With carbon dioxide embolus, "gas lock" occurs (as in air embolus) in the vena cava and right atrium, obstructing venous return. Normal blood flow cannot get into the pulmonary artery; cardiac output will fall and circulatory collapse will result.

Fat Embolism

Mobilized fat particles, which embolize and then lodge in the pulmonary vasculature. Fat globules arise and are mobilized from damaged bone marrow; enter circulation through medullary vessels; and then embolize to lung, where they obstruct.

- Toxic biochemical: free fatty acids (FFA) hurt pneumocytes— leads to adult respiratory distress syndrome (ARDS).

Etiology: Seen with long bone fractures, early as 12–48 hours after injury, most commonly 72 hours from injury.

Signs and Symptoms

- Triad is **dyspnea** (arterial hypoxemia), **confusion, dysrhythmias/tachycardia**; also see petechiae (axilla, conjunctiva, chest, upper extremities)—increased $PaCO_2$
- ARDS picture: interstitial edema, capillary leakiness, decreased surfactant, alveolar collapse, decreased compliance
- C-V Symptoms: EKG = Nonspecific changes, 'strain'/ischemia patterns
- Fat globules in urine, sputum
- Anemia/thrombocytopenia
- Ca+ (binds with the FFA)

Implicated Surgical Procedures

- Long bone fractures, total joint replacement, multiple fractures with pelvic injury. Also reported: intra-abdominal procedures, liposuction, post-CPR, bone marrow transplant, pancreatitis, burns, sickle cell disease/Hgb C disease.

Treatment: mainly supportive.

Prevention

- Early fracture splinting
- Adequate hydration
- Albumin can provide FFA binding sites
- Steroids (prophylactic)

Fat embolism has a 10–20% mortality.

Thromboembolism from Deep Vein Thrombosis (DVT)

Definition: a thrombus (clot) from a distant site migrating to the heart or pulmonary vasculature. Most DVT occur in first 24–48 hours postoperatively.

Origin:

- Clots form in deep calf/popliteal/ileofemoral veins—many undetectable.

DVT Risk Factors

- Trauma
- Immobility/recent surgery
- Previous DVT
- Peripheral vascular disease
- Obesity
- CHF
- Estrogen therapy
- Sepsis
- Cancer (lung, pancreas)
- Age
- Postpartum

Pulmonary Thromboemboli Classic Signs in Awake Patient

- Dyspnea, pleuritic chest pain, hemoptysis

Detection/Monitoring under General Anesthesia

- Cardiac
  - Tachydysrhythmias, "p pulmonale" (biphasic p), anterior lead T wave inversion, right axis deviation, right-bundle branch block, right heart failure, EKG changes indicating strain (ST changes), asystole, increases peak airway pressures
  - Hypotension
- Respiratory
  - Decreased $ETCO_2$, tachypnea, wheezing, crackles/rales
  - Hypoxemia/A-aDO_2 gap widens

Implicated Procedures

- Orthopedic (trauma)
- Vascular

- Procedures in which coagulopathy may occur
- Long-term lithotomy procedures (gynecological, urological)

Incidence of DVT:

- 20–80% total hip arthroplasty
- 50% total knee replacements and hip fractures
- 630,000 cases annually
- M & M = significant

Aggressive Treatment of Pulmonary Thromboemboli

- Thrombolytics
- TPA/urokinase/streptokinase: risk of bleeding
- ECMO/bypass
- Pulmonary embolectomy for saddle emboli

Prevention of DVT/Thromboemboli

- Pneumatic/compression stockings
- Appropriate positioning
- Low dose anticoagulation
  - Coumadin/Dextran/aspirin (ASA)
  - Lovenox, etc. (low molecular weight [LMW] heparin)
- Heparinization
  - Prevents further thrombi formation
  - May predispose to much greater estimated blood loss (EBL)
- Use of regional anesthesia
  - Improved regional blood flow
- Avoid succinylcholine if there are preexisting thrombi
  - Don't want fasciculation

1.7 Positioning Pearls

Teamwork and Communication

- Must use vigilance and planning
  - Do as much positioning as possible prior to induction while patient still awake.

- Use slow, physiologic, coordinated, smooth movement of the patient at all times
- Get all the help that you think that you will need.

Positioning "Dos"

- Adequate circulation
- Support of the head
- Good anatomical alignment of body parts
- Consider preexisting medical condition, individual physical limitations

Positioning "Don'ts"

- Excessive pressure on peripheral nerves or bony prominence
- Threats to eye integrity
- Pressure/abrasion/irritants
- Damage to appendages or extremities
- Joint/muscle strain or dislocation
- Don't let the patient FALL

Assess breath sounds for endotracheal tube (ETT) placement after position changes

- Head flexion can advance the ETT
- Head extension can pull the ETT back

May use reinforced ETT in the prone and lateral position to prevent tube kinking.

Record all position changes and/or q15 minute checks; include:

- Assessment and comparison of peripheral pulses
- Assess ALL bony prominences when possible
- Check for eye/ear pressure
- Chart positioning and ways you protected patient, especially in frail patients

Physiology and Positioning

Three major areas of concern:

- **Cardiovascular/Circulatory**
  - Impaired ANS/SNS
  - Loss of vasomotor tone
  - Depressed cardiac output
  - Redistribution of circulating volume
  - Compression of extremities or great vessels
  - Vasoconstriction
- **Respiratory**
  - Barriers to thoracic excursion
  - Loss of hypoxic pulmonary vasoconstriction
  - Alteration in V/Q ratio
- **Peripheral Nerve Injury**
  - Risk is present even with perfect positioning; nerve injury is second most common lawsuit. Successful nerve damage lawsuits are often due to undocumented padding or positioning or improper positioning.
  - Injury due to:
    - Direct compression of the nerve
    - Indirect: compartment syndrome
  - Increased risk
    - Lack of consciousness (patient can't adjust position and can't complain)
    - Reduction in skeletal muscle tone
    - Focal pressure can exceed capillary perfusion pressure
    - Preexisting conditions (diabetes, sickle cell, peripheral vascular disease)
    - BMI < 20 or > 35
    - Smoker
    - Areas with little tissue over bony prominences
      - heels
      - elbows

- sacrum
- occiput
- acromion
- malleolus

- Compression Injury: Areas at Risk
  - Eyes/ears
  - Penis/scrotum
  - Breasts
  - Fingers
  - Table injury to pendulous tissue

Upper Extremity Nerve Injury

Ulnar nerve: #1 upper extremity nerve injury

The ulnar nerve takes a superficial path near the medial epicondyle of the humerus.

Brachial Plexus: #2 upper extremity nerve injury

Brachial Plexus: formed by the anterior rami of cervical nerves 5–8 and first thoracic nerve T1. The brachial plexus is divided into 5 roots, 3 trunks, 6 divisions, 3 cords, and 5 branches innervating the upper limbs, some neck, and shoulder muscles.

Lower Extremity Nerve Injury

Common Peroneal: #1 lower extremity nerve injury

- Cause: Nerve compressed by the head of the fibula and hard surface (stirrup, candy cane leg holders, etc.)
- Symptoms: Foot drop/unable to dorsi-flexion/inability to evert the foot; sensory loss to the dorsal surface of the foot and portions of the anterior, lower-lateral leg

Sciatic

During lithotomy positioning/external leg rotation

Straight Knees: Weakness below the knee

Cause: Thigh and leg external rotation causing traction

TABLE 1-3 Distal Effects of Brachial Plexus Injury

Radial nerve	Controls movement of triceps Extends wrist and fingers Sensory to thumb and dorsal fingers	Trouble straightening the forearm, weak supination Wrist drop Unable to hitchhike (abduct thumb) Loss of sensation in lateral arm, posterior forearm, radial half of dorsum of hand, dorsal aspect of radial 3.5 fingers
Median nerve	Sensory to palm and ventral fingers	Unable to abduct and oppose thumb Sensory loss in thumb, index finger, long finger, and radial aspect of ring finger Weak forearm pronation
Ulnar Nerve	Sensory to pinky and ring finger	Motor: pinky Sensation: over medial half of fourth digit and entire fifth digit Atrophy of thenar eminence → claw hand

Lack of knee flexion in the sitting position

Symptom: foot drop

Saphenous

Medial sensory deficits

Compressed by the leg holder and the tibia

Medial knee sensory deficits

Femoral

Excessive thigh angulation: lose hip flexion and knee extension

Prone Checklist

- Eyes/ears
- Occluded/padded eyes
- Check all extremities

TABLE 1-4 Supine

Cardiac	Increased preload and CO Aortocaval compression with obesity, pregnancy, ascites, abdominal tumors
Pulmonary	Decreased functional residual capacity (FRC), can lead to V/Q mismatch and subsequent hypoxemia. PEEP settings can help mitigate.
Nerve	Increased risk of ulnar nerve injury, even with perfect positioning; lay arm supine and slightly flexed to decrease risk of injury. Extreme flexion of the elbow when the arm is folded tightly across the chest can also lead to nerve injury through surrounding ligament compressing ulnar nerve. THE BRACHIAL PLEXUS C4-T2— risk of stretch and compression with • Neck extension • > 90-degree abduction • External rotation of the arm To avoid brachial plexus injuries, arms should be < 90 degrees in abducted position, external rotation and posterior displacement avoided, and head should be maintained in a neutral position or turned toward the abducted arm. Pressure areas of concern include occiput, sacrum, and heels.

- Penis/breasts in the clear—move breasts in medially
- Catheter clear
- Chest rolls in good position—below clavicle and below inguinal space
- Brachial plexus checks—shoulders/elbows < 90 degree angles, head in midline position
- Clavicle/mandible checks
- Check eyes and nose for pressure every 15 min

TABLE 1-5 Trendelenburg—HOB Decrease > 15 Degrees

Cardiac	Increased venous return; increased central venous pressure Increased pulmonary artery pressures/wedge, and mean arterial pressure Decreased stroke volume due to pressure on heart Decreased MVO_2 Increased atrial pressures; increased left atrial pressure will be transmitted to pulmonary capillaries with an increased risk of congestive heart failure. Decreased cardiac output, heart rate, pulmonary vascular resistance Hypotension—Baroreceptors are activated and compensate with vasodilation
Pulmonary	Diaphragm now pushing on lungs and decreasing FRC 20% and pulmonary compliance decreased (the most of any position) Increased peak airway pressures; difficult ventilation Atelectasis Pulmonary engorgement
Nerve	Arms at patient sides or if on arm boards, shoulders < 90 degrees and placed on gel pads/egg crate and wrapped securely to prevent falling off arm board when bed tilted
Notes	Can increase intracranial pressure with increased cerebral blood flow Facial engorgement and airway edema risk is high; check to make sure patient will be able to ventilate once extubated! Assessed with a cuff leak test. For any doubt, keep patient intubated.

TABLE 1-6 Reverse Trendelenburg—HOB increase > 15 degrees	
Cardiac	Hypotension Venous pooling
Pulmonary	Good respiratory effect • Increased FRC • Increased compliance • Oxygenation improved with adequate cardiac output (CO) If ventilation setting on pressure control ventilation, watch for increased tidal volumes with head up position.
Nerve	None specific
Notes	Initial increase in every pressure, but baroreceptors now read elevated pressures and cause massive vasodilation. **If BP measured by cuff: calculate 0.75 mmHg for every 1 cm vertical height difference between where cuff pressure being taken and the blood pressure at the circle of Willis (measured at tragus of ear). There may be a significant difference between the arterial blood pressure at the level of the brain and the BP reading by cuff placed on a leg in a patient in the reverse Trendelenburg position. **If blood pressure is measured by an arterial line, level the transducer at the tragus of the ear to obtain a meaningful index of mean arterial blood pressure at the circle of Willis and cerebral perfusion pressures (CPP).

TABLE 1-7 Lithotomy	
Cardiac	Increased venous return as legs are lifted into lithotomy position and ~ 600 mL blood volume is redistributed centrally. Can see hypotension from fall in cardiac output when legs moved from lithotomy to supine position as blood shifts back into lower extremities.

TABLE 1-7 Lithotomy, continued

Pulmonary	Abdominal shift toward lungs • FRC decreased 20% • Vital capacity decreased • Atelectasis • Hypoxia
Nerve	Peripheral neuropathy from nerve injuries to the: • common peroneal—most often due to pressure on head of fibula • saphenous • sciatic—flexion and external rotation of hip • femoral • obturator • popliteal fossa pressure: due to prolonged bent knees Compartment syndrome—Surgical emergency Check pulses, edema, color, pain At end of case in lithotomy position • Feet are brought together and lowered to decrease lumbar torsion and strain Increased risk of deep vein thrombosis (prophylaxis with compression stockings or sequential compression devices) and hand/finger injury (pad hand/fingers and watch carefully as leg/bottom of bed raised or lowered to avoid crush injuries).
Notes	Always consider the normal range of movement for the patient, and limit the positioning to this.

Postoperative Visual Loss (POVL)—Prone Position

Complete or partial blindness is a potentially devastating complication for a patient in the prone position.

See more information under Anesthetic Complications—POVL

TABLE 1-8 Prone

Cardiac	Lower extremity/abdominal venous pooling from inferior vena cava Compression Decrease in cardiac output and venous return Epidural engorgement

Pulmonary	Chest rolls: with proper position with have free excursion and ventilation can actually improve (relative to supine) with prone positioning due to increase in FRC.
	• If chest not free:
	  • Decreased compliance from higher intrathoracic pressure
	  • High peak pressures
	  • Ventilation: Perfusion problems

Nerve	Possible damage to brachial plexus with arm/shoulder position—keep shoulders and elbows $<$ 90 degrees Risk of eye pressure and postoperative vision loss (POVL) Risk of pressure on nose Head and neck in neutral position
Notes	Variation with arm position in prone position • Both arms positioned at patient's sides • Both arms above head in "superman" position—elbows and shoulders $<$ 90 degrees • One arm above head and one arm at patient side Airway device must be properly secured to prevent inadvertent dislodgment during positioning.

TABLE 1-9 Lateral Decubitus

Cardiac	Minimal alterations unless hypovolemic. Consider placement of blood pressure cuff so that body is not compressing on arm or cuff, which will give inaccurate readings.
Pulmonary	Awake and spontaneously breathing patient in lateral position: Dependent lung is better ventilated and better perfused that the nondependent lung. Anesthetized and spontaneously breathing patient in lateral position: Ventilation shifts from the dependent lung to the nondependent lung but perfusion better to dependent lung. Anesthetized and mechanically ventilated patient in lateral position: Shunt worsens with more ventilation to nondependent lung and better perfusion to dependent lung. Dependent lung in anesthetized patient: Lower lung gets decreased ventilation but increased perfusion. *Dependent lung: lower lung; non-dependent lung: upper lung*
Nerve	Brachial plexus injury increased with improper padding. Suprascapular nerve: causes diffuse pain. Common peroneal injury if padding not placed along outside of lower leg (around knee just around head of fibula). The saphenous nerve can be protected by placing a pillow between the legs. Radial nerve injury is also increase risk in lateral position if shoulder is $>$ 90 degrees and forearm not supported in neutral position.

continues

TABLE 1-9 Lateral Decubitus, continued	
Notes	Lower lung excursion—axillary rolls to lift chest and decrease pressure on brachial plexus so that suprascapular neurovascular bundle is not stretching. Axillary roll can cut off circulation if not properly placed. Airway device must be properly secured to prevent inadvertent dislodgment during positioning. Need beanbags, special hip pads, pillows, overhead arm boards. Check dependent ear to make sure it's not folded over. Check to make sure no pressure on or around eyes.
Notes	FLEXED LATERAL DECUBITUS—Spreads thorax and costal margin to iliac crest distance; break in bed should be at flat area of the iliac crest.

TABLE 1-10 Sitting/Beach Chair	
Cardiac	Massive venous pooling Decreased venous return Decreased cardiac output and blood pressure Increased heart rate and systemic vascular resistance Initial increase in every pressure—baroreceptors now read elevated pressures and cause massive vasodilation.
Pulmonary	Increased FRC, lung volumes, and compliance Decreased work of breathing If ventilation setting on pressure control ventilation, watch for increased tidal volumes with head up position.
Nerve	Pressure points increased risk: occipitus, scapula, elbow/ulnar nerve; sacrum, ischial tuberosities, calcaneus (heel) Less risk to sciatic nerve with HOB up and knees bent If head falls forward in sitting position, must keep chin 1–2 fingers breadths from chest. Without this distance, there is too much strain on C5; can cause mid-cervical tetraplegia.

TABLE 1-10 Sitting/Beach Chair, continued

Notes	Airway device must be properly secured to prevent inadvertent dislodgment during positioning. Advantages: • Neuro: better exposure; blood and CSF drainage • Decreased morbidity in posterior fossae exploration Disadvantages: • Hypotension and cerebral ischemia • Risk of air emboli (40%) • Pneumocephalus • Ocular compression • Edema/ Macroglossia If BP measured by cuff: calculate 0.75 mmHg for every 1 cm vertical height difference between where cuff pressure being taken and the blood pressure at the circle of Willis (measured at tragus of ear). There can be a huge difference between the arterial blood pressure at the level of the brain and the BP reading by cuff placed on a leg in a patient in the sitting/ beach chair position. If blood pressure is measured by an arterial line, level the transducer at the tragus of the ear to obtain a meaningful index of mean arterial blood pressure at the circle of Willis and CPP.

TABLE 1-11 Jack-Knife

Cardiac	Venous pooling Mesenteric/epidural engorgement
Pulmonary	Visceral shift, FRC decreased by $< 20\%$ Decreased compliance Need free chest excursion
Nerve	Possible damage to brachial plexus with arm/ shoulder position.

1.8 Postoperative Visual Loss (POVL)

All complications of surgery and general anesthesia are unfortunate, and, thankfully, this complication is extremely rare, occurring in approximately 1 in 1,250,000 anesthetics.

POVL Risk factors

- Length of surgery
- Type of surgery
  - Spinal
  - Cardiac surgery with cardiopulmonary bypass
- Long periods of controlled hypotension
- Low hemoglobin levels
- Prone position
- Patient is a smoker, has high blood pressure, and/or is a diabetic

Two Causes of POVL

Ischemic optic neuropathy is most common cause of POVL and is most often associated with long-duration spinal surgery with instrumentation. These patients can have considerable blood loss leading to hypotension and low perfusion pressures.

- Prolonged hypotension, excessive blood loss, and lengthy prone positioning time
- Patient history: diabetes, cardiovascular disease, peripheral vascular disease, smoking
- Increase in blood viscosity, hemorrhage, anemia, hypotension

***Data shows that only 44% pts with ischemic optic neuropathy recovered vision.*

Retinal ischemia is the second most prevalent cause; increased intraocular pressure causes damage to the optic nerve (perfusion pressure of the eye = mean arterial blood pressure and intraocular pressure).

Medical–malpractice claims correlated with:

- direct pressure on the eye globe
- emboli
- low retinal perfusion pressures

***Data shows that no patient with central artery occlusion recovered vision.*

Methods to Help Prevent POVL

- Maintain mean arterial pressures at > 60–70 mm Hg especially for patient in prone or sitting positions.
- Maintain hemoglobin > 9.4.
- Keep neck in midline to prevent venous congestion in the head.
- Normothermia, euglycemia, and urinary output > 0.5 mL/kg/hr.
- Prevent any pressure placed on eye globe: document "eyes and nose check" along with vital signs frequently on anesthesia record in any patient in prone or sitting position.

1.9 Malignant Hyperthermia (MH)

Clinical syndrome in which there is a hypermetabolic self-perpetuating state involving skeletal muscle following certain anesthetics. Hyperthermia results from an acute uncontrolled increase in skeletal muscle metabolism from a pathological increase in skeletal muscle calcium.
Cause: succinylcholine and all inhaled agents

Signs*:* increased $ETCO_2$, tachycardia, arrhythmias, acidosis, shock, trunk or limb rigidity, masseter spasm, cyanosis; *increased temperature may be a late sign.*

Treatment: drug treatment for MH works on sarcoplasmic reticulum of skeletal muscle by reducing release of calcium or by inhibiting excitation–contracture coupling at the transverse tubule.

GET HELP—GET RYANODEX
NOTIFY SURGEON
Discontinue volatile agents and succinylcholine
Hyperventilate with 10 L flows of 100% oxygen
Halt the procedure as soon as possible
GET DANTROLENE for maintenance doses

New drug treatment for MH, Ryanodex® is a single vial of Dantrolene 250 mg, but it is reconstituted with 5 mL of sterile water and in ONE vial and can be administered in less than one minute—it will provide a therapeutic loading dose during a MH crisis for most patients much more quickly.

Dantrolene (or Revonto), start at 2.5 mg/kg q 5 mins, max 30 mg/kg.
Dantrolene 20 mg with 3 g Mannitol mixed in 60 cc sterile water—1 mg/3 mL.
In 70 kg patient—initial 2.5 mg/kg = 175 mg or 525 mL (9 vials).
Dantrolene® and Revonto® will be used for maintenance doses.

Complications of MH

Hyperkalemia: treat with

- hyperventilation
- bicarbonate (1–2 mg/kg IV)
- glucose/insulin
  - pediatric: insulin 0.1 u/kg and D50W 1 mL/kg
  - adult: insulin 10 u regular and D50W 50 mL

- calcium chloride 10 mg/kg or calcium gluconate 10–50 mg/kg
- check glucose levels hourly

Elevated temperatures

- **Cool** the patient with core temperatures >39 degrees with cold saline IV. Lavage open body cavities, stomach, bladder, and rectum. Apply ice to surface. Stop cooling if temp < 38 degrees.
- Note: use any standard drug therapy **except calcium channel blockers**, which may cause hyperkalemia or cardiac arrest in presence of dantrolene.

Prepare AGM for MH Patient

Ensure all vaporizers are disabled by removing or taping in "off" position. Some consultants recommend changing CO_2 absorbent. Flow 10 L/min oxygen through circuit for at least 20 minutes. If fresh gas hose is replaced, 10 minutes is adequate. During this time a disposable, unused breathing bag should be attached to Y-piece of circle system and ventilator set to inflate bag periodically. Use a new or disposable breathing circuit.

MH Emergency Hotline:
1-800-644-9737 (MH HYPER)

1.10 Awareness with Recall (AWR)

The type of surgery (in particular if the surgery is an emergency such as for obstetrics, cardiovascular, or major trauma), along with the patient's underlying medical condition, physical status, and age can increase the risk of AWR.

Many patients do not know there is a difference between levels and types of anesthesia and "awareness under anesthesia" was a source of postoperative complaints

in patients who had undergone "sedation" or "MAC: monitored anesthesia care." Clearly, the patients had not been prepared for the fact that "they may be aware of light and voices and hear other noises in the room—though they should not feel any pain . . ." and were distressed postoperatively thinking they had been "awake" during their surgery. It would be advantageous to help the patient understand, *preoperatively*, that during their surgery they may be aware of events in the operating room and to also assure them they should not be uncomfortable and that an anesthesia care provider will be with them throughout the case to help them be safe and comfortable. Thorough preoperative preparation and education will help to avoid disparities in their expectations regarding their anesthetic experience.

Majority of AWR Claims Involved

- Healthy women $<$ 60 years old
- Receiving total intravenous anesthesia (no volatile agent)
- Received muscle relaxants

Increased Risk of AWR under General Anesthesia

- Failure to turn on agent vaporizer
- Vaporizer malfunction
- Failure to anesthetize sufficiently during induction
- Inadvertent paralysis of conscious patient
- Under-dosing of anesthesia relative to specific patient requirement
- Difficult, repeated intubation attempts

***Substandard care judged in 42% of cases involving intraoperative awareness.*

Ways to Prevent AWR

- Use brain function monitor (i.e., BIS), maintain between 40 and 60. Use should be made on a patient-to-patient basis but encouraged in selected patients with an increased risk of AWR: history of AWR, patient who chronically uses or uses high doses of opioids, alcohol, anti-epileptic, or anxiolytic mediations. Also, a patient receiving muscle relaxation (and other intravenous anesthetics) without also receiving inhaled anesthetics.
- Monitor for unexpected tachycardia and or hypertension.
- Monitor volatile anesthetic levels in vaporizers.
- Use smallest dose of muscle relaxation necessary to provide satisfactory surgical conditions.

Posttraumatic stress disorder (PTSD) may result from AWR

Research demonstrates that AWR occurs in 1–2 patients per 1,000 receiving general anesthesia (highest percentage in trauma and cardiac surgical patients) and that approximately 50% of patients that experience AWR suffer psychological problems.

The actual incidence of AWR remains uncertain, though, and likely is underreported if there is no standardized approach of the patient in the postoperative period. The Brice questionnaire can help to detect intraoperative awareness:

- Explicitly inquiring about recall in adults should be part of your postoperative assessment (Brice, 1970).
  - *"What was the last thing you remember before going to sleep?"*
  - *"What is the first thing you remember after waking up?"*
  - *"Do you remember anything between going to sleep and waking up?"*

- *"Did you dream during your procedure?"*
- *"What was the worst thing about your operation?"*

- Evaluating the occurrence in children can be difficult due to developmental or language differences.

Upon discovery of AWR, there are important steps to take:

- Contact the anesthesia provider involved in the patient's case so they can speak to the patient directly.
- Document a detailed account of the events in the patient's medical record.
- Refer the patient to a mental health professional.
- Conduct a root-cause analysis to identify the causative factors that led to AWR.
- Follow up with the patient to ensure that appropriate care was received.

1.11 Intraoperative Burns

There is an increased possibility of intraoperative fire or patient burn in a "high-risk" procedure where an ignition source can come in close proximity to an oxidizer-enriched environment increasing the risk of fire (i.e., any mouth or throat surgery with lasers or cautery; tracheostomy; eye surgery; surgery around the head, neck, or face; or burr hole surgery); however, any surgery where oxygen is given in an open system (face mask or nasal cannula) is defined as a high-risk procedure.

Monitoring all surgical patients throughout the procedure with pulse oximetry, $ETCO_2$, and inspired, exhaled, and/or delivered oxygen concentration should be done.

Intraoperative burns are also attributable to

- IV bags or bottles
- Blanket or light warmers
- Defibrillator paddles
- EKG leads
- MRI at pulse oximetry site

Most burn claims result from operating room fires caused by surgeon's cautery or laser. All these claims involve the use of supplemental oxygen, and 95% occurred during head, eye, face, or neck surgery.

In the simplest of terms:

- surgeon provides ignition/heat source (electrocautery, laser)
  - there are also defibrillator pads, argon beam coagulators, fiberoptic light cables, heated probes, etc.
- nurses provide fuel source (prep, drapes)
  - there is also the patient's hair, dressings, gastrointestinal tract gases, blankets, gloves, tracheal tube, etc.
- anesthesia's liability arises from oxidizer source
  - from control of oxygen (>30%)—any FiO_2 > 30% is considered enriched oxygen
  - giving nitrous oxide
  - how oxygen/oxidizer is delivered (mask, nasal cannula, endotracheal tube)
  - not documenting ways we prevent burns and protect patient (chart decrease in FiO_2, etc.)

***Oxygen and nitrous oxide are both oxidizers; they both act as accelerant for fires and greatly increase the risk of combustion.*

Preventing Burns

Before a high-risk procedure, a team discussion of strategy for the prevention and management of a fire with continued communication throughout the case is crucial.

- Minimizing or avoiding an oxidizer-enriched atmosphere near surgical site
- Managing ignition sources
- Managing fuels

For airway surgery, use a cuffed tracheal tube, instead of uncuffed, which helps to keep supplemental oxygen below the level of the cuff.

For laser surgery, use a laser resistant tracheal tube, and fill the tracheal cuff with methylene blue-tinted saline vs. air when possible (helps to ascertain cuff puncture).

Moisten sponge or gauze if in proximity to ignition source.

Do not give oxygen throughout case or discontinue supplemental oxygen during period of increased risk (cautery or laser use). If supplemental oxygen is required, decrease FiO_2 as low as possible when either laser or cautery is used. Placing a suction catheter or Yankauer suction piece helps to pull gases away from the airway.

In surgery around the head, face, and neck, determine the need for sedation depth and oxygen dependence. If a face mask or nasal cannula must be used, again, communication with the surgeon is of paramount importance. Stop any oxidizer being given and wait a few minutes before approving the activation of the ignition source.

Arrange surgical drapes in an open configuration to avoid trapping high concentrations of oxygen; avoid giving nitrous oxide.

Do not use warming blanket tube without connecting to upper or lower body warming blanket.

Immediately available:
Several containers of sterile saline
Carbon dioxide extinguisher

For Fire in the Airway or Breathing Circuit

- Remove endotracheal tube.
- Stop flow of all airway gases.

- Remove all flammable/burning material from patient and surrounding area.
- Pour saline or water into patient's airway.
- Reestablish ventilation by circuit mask but avoid supplemental oxygen or nitrous oxide, if possible.
- Examine the tracheal tube to ensure that it is intact. If it is not intact, consider bronchoscopy to look for any fragments, remove debris, and assess injury.
- Reintubate the patient? Devise a plan for ongoing care.

For Fire Elsewhere on the Patient

- Stop flow of all airway gases.
- Remove and extinguish all flammable/burning material from patient and surrounding area.
- Assess for smoke inhalation injury.
- Devise a plan for ongoing care.