

**POCKET**  
**NOTEBOOK**

# **POCKET**

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# **ANESTHESIA**

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Wolters Kluwer  
Health

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# PREOPERATIVE PATIENT EVALUATION

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ASA Physical Status Classification		Example
I	No organic/physiologic/psychiatric problems	Healthy patient
II	Controlled medical conditions with mild systemic effects, no limitations in functional ability	Controlled HTN/smoker/obesity
III	Medical conditions with severe systemic effects, limitations in functional ability	Controlled CHF/stable angina/morbid obesity/COPD/chronic renal insuff.
IV	Poorly controlled medical conditions associated with significant impairment in functional ability that is potential threat to life	Unstable angina/symptomatic COPD or CHF
V	Critical condition, little chance of survival without surgical procedure	Ruptured AAA
VI	Brain dead, undergoing organ donation	
E	Emergency, trauma	Gunshot wound, GI perf.

## Preoperative Interview

<b>Current issue</b>	Indication for surgery
<b>Past medical history</b>	Presence & severity of medical comorbidities
<b>ROS</b>	Focus on general functional capacity
<b>CV</b>	Angina, SOB, exercise tolerance, activity level, limiting factors
<b>Pulmonary</b>	SOB, dyspnea on exertion, smoking, inhaler use, baseline O <sub>2</sub> use
<b>Neurologic</b>	TIA, stroke, pain, depression, anxiety
<b>GI</b>	GERD symptoms, NPO status
<b>Renal/GU</b>	Possibility of pregnancy
<b>Heme</b>	Easy bruising, easy bleeding, hx of anemia, clotting d/o
<b>Musculoskeletal</b>	Cervical range of motion
<b>Surgical history</b>	Previous surgeries, including complications/outcomes
<b>Anesthetic history</b>	Examine old records for hx of difficult airway management, PONV, any family hx suggestive of malignant hyperthermia
<b>Social history</b>	Tobacco/alcohol/illicit drug use
<b>Allergies</b>	Drug allergies (anaphylaxis, airway swelling, hives, pulmonary reactions) vs. side effects/intolerance, latex allergy
<b>Medications</b>	Especially cardiovascular meds, insulin, anticoagulation meds

## PREOPERATIVE PHYSICAL EXAMINATION

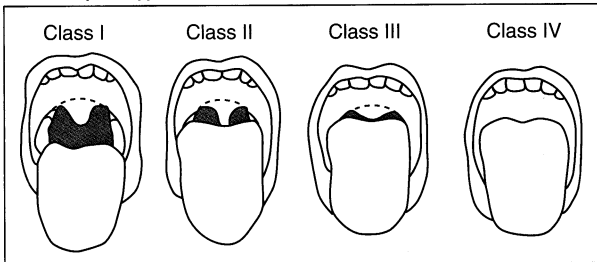
- Vital signs: Resting heart rate, BP, SpO<sub>2</sub> height, weight, body mass index
- CV & pulm: Heart & lung sounds, JVD, pulm/periph edema, carotid bruits
- Airway exam:
  1. Mallampati score (see below)
  2. Thyromental distance: Have pt extend neck & measure space between mental prominence and thyroid cartilage; <6 cm may indicate difficult intubation
  3. Cervical spine flexion/extension: Examine patient for ↓ range of motion that might limit head movement into sniffing position during intubation
  4. Miscellaneous: Oral opening, size of mandible (micrognathia) & tongue (macroglossia), dentition (loose, missing, prostheses)

### Mallampati Scoring System

Pt in an upright position, mouth open as wide as possible, not sticking tongue out

Grade View	Visible Structures	Intubation
1	Tonsillar pillars, soft palate, entire uvula	Unlikely difficult
2	Pillars & soft palate, only part of uvula	Unlikely difficult
3	Soft palate & base of uvula	Possibly difficult
4	Hard palate only	Difficult/impossible

**Figure 1-1 Mallampati classification of the oropharyngeal structures.** Peter Dunn, ed. (From *Clinical Anesthesia Procedures of the Massachusetts General Hospital 7th ed.* Philadelphia: Lippincott Williams & Wilkins, with permission.)



### Consensus Fasting Guidelines

Solid food, milk, infant formula	6 hr
Breast milk	4 hr
Clear liquids (water, soda, juices, black coffee)	2 hr
Emergency cases	rapid sequence intubation

### PREOPERATIVE LABORATORY TESTING

- Test selection based on PMH/current problem/nature of surgery/current meds
- Young, healthy asymptomatic pts → often require no testing

### Suggested Metrics for Preoperative Testing

ASA	Low Risk*	Mod. Risk*	High Risk*
I	ECG (M >45; F >55) BUN/Cr/Gluc >65	ECG (M >45; F >55) BUN/Cr/Gluc >65 CBC >65	ECG/CXR/ CBC/BUN/Cr/ Gluc >65 Blood bank T+S
II	ECG (M >45; F >55) BUN/Cr/Gluc >65 CXR	ECG (M >45; F >55) BUN/Cr/Gluc >65 CXR/CBC	ECG/CXR/CBC BUN/Cr/Gluc >65 Blood bank T+S
III	ECG/CBC/CXR Electrolytes/BUN/Cr/Gluc	ECG/CBC/CXR Electrolytes/BUN/Cr/Gluc	ECG/CBC/CXR Electrolytes/BUN/ Cr/Gluc Blood bank T+S
IV	Labs according to underlying condition		

\*See table below on page 1-4: "Predictors of Increased Cardiovascular Risk," for low-/mod-/high-risk categories.

Testing	Possible Indications
ECG	Cardiac history
Chest x-ray	Asthma/COPD, hx of malignancy
Pulmonary function testing	Severe asthma/COPD
Urine testing	Bladder/lower urinary tract symptoms
Pregnancy testing	Possibility of pregnancy
Hemoglobin	Anemia, abdominal/vascular/cardiac surgery
LFTs, albumin, bilirubin	Liver dz
Coagulation studies	Liver dz, anticoagulant use, plan for neuraxial anesthesia
Finger stick	DM, insulin therapy
Echocardiogram	Any murmur, acute coronary syndrome, CHF
Carotid Doppler US	Carotid bruits
Cervical spine flexion/ extension x-rays	Neck pain
Noninvasive cardiac testing	See Figure 1-2 Perioperative Risk-Assessment Algorithm

## INFECTIVE ENDOCARDITIS (IE) ANTIBIOTIC PROPHYLAXIS

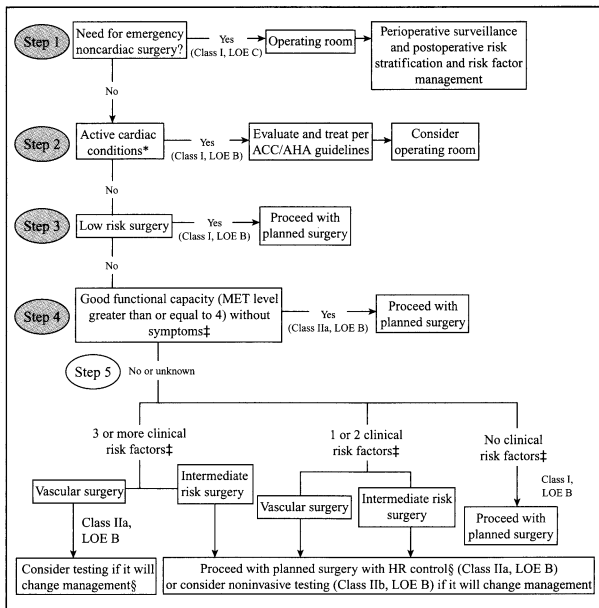
- Based on risk of developing IE & severity of outcome if IE were to occur
- Highest-risk patients (those requiring prophylaxis) include those with:
  - Prosthetic cardiac valve
  - Previous infective endocarditis
  - Congenital heart disease (CHD) (Unrepaired cyanotic CHD, completely repaired CHD with prosthetic device)
  - Repaired CHD with residual defect at site
  - Previous cardiac transplantation with subsequent cardiac valvulopathy.

Note: Current guidelines no longer include pts with common valvular lesions (bicuspid aortic valve, acquired aortic/mitral valve dz, mitral valve prolapse, hypertrophic cardiomyopathy)

Recommended Antibiotic Regimen (30–60 min prior to surgery)		
	First-Line Antibiotic	Alternative Antibiotic (PCN-allergic pts)
Oral	Amoxicillin 2 g Cephalexin 2 g	Clindamycin 600 mg Azithromycin 500 mg Clarithromycin 500 mg
IV/IM	<b>Ampicillin 2 g IV/IM</b> Cefazolin 2 g IV/IM Ceftriaxone 1 g IV/IM	Clindamycin 600 mg IV/IM

Source: Adapted from Wilson W, et al. Prevention of Infective Endocarditis. *Circulation* 2007;116(15):1736–1754.

**Figure 1-2 Perioperative risk-assessment algorithm. (From Fleisher LA, et al. ACC/AHA 2007 Perioperative Guidelines. *J Am Coll Cardiol* 2007; 50(17):1707–1732, with permission.)**



Predictors of Increased Cardiovascular Risk		
Risk	Procedural Component	Medical Component
MAJOR (cardiac risk >5%)	<ul style="list-style-type: none"> <li>Major abdominal/thoracic</li> <li>Emergent major operations</li> <li>Aortic/major vascular</li> <li>Intracranial</li> <li>Long procedures with large fluid shifts and EBL &gt;1000 mL</li> </ul>	<ul style="list-style-type: none"> <li>Ischemic heart dz (recent or acute MI, positive cardiac testing, symptomatic/unstable angina)</li> <li>Symptomatic valvular dz</li> <li>Decompensated CHF (pulmonary edema/peripheral edema, PND)</li> <li>Symptomatic/high-grade/new-onset arrhythmias</li> </ul>
INTERMEDIATE (cardiac risk <5%)	<ul style="list-style-type: none"> <li>Carotid endarterectomy,</li> <li>Head &amp; neck procedures</li> <li>Intraperitoneal/minor thoracic (lung bx/thoracoscopy)</li> <li>Orthopedic surgeries</li> </ul>	<ul style="list-style-type: none"> <li>Previous MI</li> <li>Diabetes</li> <li>Compensated CHF</li> <li>Renal insufficiency</li> </ul>
MINOR (cardiac risk <1%)	<ul style="list-style-type: none"> <li>Minimally invasive</li> <li>Endoscopic</li> <li>Breast-</li> <li>Eye procedures</li> <li>Hernia/thyroidectomy</li> <li>EBL &lt;200 mL</li> </ul>	<ul style="list-style-type: none"> <li>Abnormal ECG</li> <li>Age &gt;65</li> <li>Previous stroke</li> <li>Uncontrolled HTN</li> <li>Abnormal rhythm</li> </ul>

Source: Adapted from Eagle KA, et al. ACC/AHA Guidelines Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive Summary. *J Am Coll Cardiol* 2002;39:542–553.

### PERIOPERATIVE BETA-BLOCKER THERAPY (NONCARDIAC SURGERY)

- Perioperative  $\beta$ -blockers may  $\downarrow$  cardiac events & mortality  
(controversial, as recent data suggests may  $\uparrow$  morbidity)
- Pts already on  $\beta$ -blocker therapy should be continued
- Consider initiation of  $\beta$ -blocker therapy in:
  - Pts who have an indication for  $\beta$ -blocker
  - Major vascular surgery

# PHARMACOLOGY: INHALATIONAL ANESTHETICS

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Potent inhalational agents: **mechanism of action** still undetermined. Theorized membrane disruption or decreased membrane conductance, GABA<sub>A</sub> receptor action. Anesthetic binding might significantly modify membrane structure.

## Partition Coefficients

- Express relative solubility of anesthetic gas at equilibrium
- Lower partition coefficients imply low solubility, slower solution/tissue uptake, but *rapid* induction (e.g. desflurane)
- Higher partition coefficients imply high solubility, *prolonged* induction (e.g., halothane)
- **Tissue:blood partition coefficient** = time for equilibrium of tissue (brain, fat, muscle) with arterial blood

**Tissue/Blood Partition Coefficients at 37°C**

Agent	Blood/ Gas	Brain/ Blood	Muscle/ Blood	Fat/ Blood	Vapor Pressure (mm Hg, 20°C)	MAC (%)
Nitrous oxide	0.47	1.1	1.2	2.3	—	105
Halothane	2.4	2.9	3.5	60	243	0.75
Isoflurane	1.4	2.6	4.0	45	240	1.2
Desflurane	0.42	1.3	2.0	27	681	6.0
Sevoflurane	0.65	1.7	3.1	48	160	2.0

MAC: minimum alveolar concentration.

Source: Adapted from Morgan G, Mikhail M, Murray M. *Clinical Anesthesiology* 4th ed. McGraw-Hill Medical: New York, NY: 2005.

## FACTORS AFFECTING ANESTHETIC UPTAKE

- Agent levels in the brain depend on agent levels in the alveolus.
- Goal is to achieve rise in  $F_a$  (alveolar anesthetic concentration)/ $F_i$  (inspired anesthetic concentration).
- $\uparrow F_a/F_i \rightarrow \uparrow$  speed of induction.

### Factors that $\uparrow F_a/F_i$

- Low solubility in the blood; anesthetic potency correlates with lipid solubility.
- Potency: halothane > isoflurane > sevoflurane > desflurane > N<sub>2</sub>O. **Uptake and solubility** are inversely proportional to induction time—i.e., the lower the partition coefficient, the less soluble the agent and therefore the faster the rise in partial pressure.
- High alveolar ventilation and alveolar blood flow.
- High  $F_i$ .
- High fresh gas flow rates.
- The difference in partial pressure between alveolar gas and venous blood.
- Anesthetic uptake is directly proportional to **cardiac output** as a function of alveolar blood flow. Low-flow states (e.g., sepsis, sympathetic blockers, CHF) reduce uptake and  $\uparrow$  induction time because of the rapid rise of  $F_a$ . High CO  $\rightarrow \uparrow$  rate of anesthetic removal from alveolus  $\rightarrow \downarrow$  rate of induction.

### Concentration Effect

- Increasing the inspired concentration of a gas results in a disproportionate increase in the alveolar concentration.
- Most clinically significant with N<sub>2</sub>O, as can be used at much higher inspired concentrations than volatile anesthetics.

### Second Gas Effect

- The ability of a large-volume uptake of one gas (first gas) to accelerate the uptake of a coadministered gas (second gas).
- Example: N<sub>2</sub>O accelerates uptake of both volatile anesthetic and oxygen, although this is probably not clinically significant. The faster the alveolar/brain concentration reaches inspired concentration of agent, the faster the induction.

### Other Factors Affecting Anesthetic Uptake

- Tissue uptake (tissue/blood partition coefficient for fat vs muscle vs brain)
- Intracardiac shunts (R-to-L shunt  $\rightarrow \downarrow$  rate of induction)
- Pediatric pts  $\rightarrow$  faster induction due to  $\uparrow$  alveolar ventilation,  $\downarrow$  FRC,  $\uparrow$  % of blood flow to brain

**ELIMINATION/RECOVERY**

- Reduction of anesthetic in brain tissue is by biotransformation (P-450), transcutaneous loss, or exhalation, with exhalation being the most significant.
- Recovery expedited by high fresh gas flows, elimination of rebreathing, low absorption by the circuit, decreased solubility, high cerebral blood flow and increased ventilation.

**DIFFUSION HYPOXIA**

- As anesthetic diffuses out of the blood and enters the alveolus, it displaces and reduces the concentration of inspired oxygen and CO<sub>2</sub>.
- With rapid-offset agents such as N<sub>2</sub>O, where the concentration is high and blood solubility low, the diffusion of alveolar O<sub>2</sub> can lead to hypoxia.
- Administer high-flow 100% oxygen for 5 to 10 min after discontinuation of N<sub>2</sub>O to prevent significant hypoxia.

**MINIMUM ALVEOLAR CONCENTRATION (MAC)**

- The MAC of an inhaled anesthetic is the alveolar concentration at which 50% of patients will not move in response to a standardized stimulus (e.g., surgical incision).
- Developed to compare potency of agents.
- MAC values are roughly additive (i.e., 0.5 MAC of N<sub>2</sub>O plus 0.5 MAC of sevoflurane ≈ 1.0 MAC).
- At MAC 1.3, 95% of patients will not move in response to surgical stimulus.
- MAC-BAR (1.5–2.0 MAC): Concentration required to block autonomic reflexes to nociceptive stimuli.
- MAC-Aware: Concentration at which 50% of patients will not be forming long-term memory.
- MAC-Awake (0.3–0.5 MAC): concentration required to block voluntary reflexes and control perceptive awareness (i.e., opening eyes on command).
- MAC greatest at 1 year of age and reduced by 6% per decade of life.

**MAC VARIABILITY**

<b>Factors That Decrease MAC (Increasing Potency)</b>	
Anemia	Acute alcohol use
Elderly pts	Hypothermia
Benzodiazepines	Increased altitude
Intravenous anesthetics	Hyponatremia
Opiates	Severe hypotension
Pregnancy	Acidosis, hypoxia

<b>Factors That Increase MAC (Decreasing Potency)</b>
Chronic alcohol abuse
Very young age (closer to 1 year of age)
Increased temperature (>42°C)
Decreased altitude
Drugs: MAOIs, TCAs, cocaine, acute amphetamine use

**BENEFITS OF INHALATIONAL ANESTHETICS**

Nonflammable; reusable (via circle system); hypnotic; overall low degree of respiratory depression vs intravenous agents, potent bronchodilators

**SIDE EFFECTS OF VOLATILE ANESTHETICS**

- Hypotension 2° to vasodilation & myocardial depression
- Potentiates neuromuscular blockade
- During spontaneous ventilation → ↓ RR & ↓ TV, hypercarbia, ↓ ventilatory response to hypoxemia
- Inhibitor of hypoxic pulmonary vasoconstriction (HPV)
- May trigger malignant myperthermia (MH) (see Appendix D)

**INHALATIONAL ANESTHETICS, SPECIFIC COMMENTS**

**Nitrous Oxide (N<sub>2</sub>O)**

- **Key features:** MAC 105%; rapid onset/offset of action. Low solubility. Incomplete anesthetic, as 1 MAC requires a hypoxic gas mixture. Has analgesic properties.
- **Disadvantages:** Can rapidly diffuse into and expand air-containing cavities; → avoid in air embolism, pneumothorax, intestinal obstruction, intracranial air, pulmonary hypertension, some open eye procedures.

- Prolonged exposure → in bone marrow depression; inhibits B<sub>12</sub>-dependent enzymes, methionine synthetase (myelin formation), thymidylate synthetase (DNA synthesis).
- Possible teratogen.
- Supports combustion (although itself not flammable).
- May increase PONV risk.
- Decreases myocardial contractility, increases PVR in patients with preexisting pulmonary hypertension.
- **Interactions:** Potentiates neuromuscular blockade (less than volatiles). Use with epinephrine may increase incidence of arrhythmias as N<sub>2</sub>O increases endogenous catecholamine levels.

### Isoflurane (Forane)

- **Key features:** MAC 1.2%; inexpensive; slow onset/offset of action, pungent, causes tachycardia. Versatile use.
- **Disadvantages:** Potential coronary steal effect due to coronary artery dilation.

### Desflurane (Suprane)

- **Key features:** MAC 6.0%; most rapid onset/offset of action; very pungent.
- **Disadvantages:** Produces carbon monoxide in desiccated absorbent barium hydroxide lime (Baralyme) and also in sodium and potassium hydroxide.
- High vapor pressure requires an electrically heated vaporizer (eliminates substantial variation of delivered concentrations owing to fluctuations in ambient temperature).
- Pungency may be irritant in patients prone to bronchospasm.
- Rapid increase or high MAC (>1.25) may cause significant sympathetic stimulation.

### Sevoflurane (Ultane)

- **Key features:** MAC 2.0%; expensive, less pungent than desflurane: Best for inhalational induction. Fast onset/offset of action. Causes less tachycardia than desflurane or isoflurane (does not sensitize myocardium to catecholamines).
- **Disadvantages:** Compound A, a nephrotoxic by-product, produced when sevoflurane is degraded in barium lime and to a lesser extent soda lime, especially with low-flow anesthesia of less than 2 L/min over several hours.
- Toxicity demonstrated only in animal models; 5% metabolized with increased serum fluoride levels—questionable renal toxin. In theory, avoid in renal failure.

### Halothane

- **Key features:** MAC 0.75%; low pungency (ideal for gas induction)
- **Toxicity:** Postoperative hepatic dysfunction (halothane hepatitis) 1:35,000
- **Contraindications:** Liver dysfunction on prior halothane administration. Pre-existing ↑ ICP. Severe cardiac disease (due to negative inotropic effect).
- **Interactions:** Myocardial depression potentiated by β-blockers/Ca-blockers; tricyclic antidepressants, MAOI's, aminophylline + halothane → arrhythmias

Systemic Effects of Volatile Anesthetics*						
Agent	Cardio	Resp	Cerebral	NM	Renal	Hepatic
N <sub>2</sub> O	↔ BP ↔ SVR ↔ HR	↑RR ↓TV	↑ICP ↑CMRO <sub>2</sub>	↑Block	↓GFR ↓RBF	↓ ↔ HBF
Desflurane	↓↓BP ↓↓SVR ↑HR	↑RR ↓TV	↑ICP ↓↓CMRO <sub>2</sub>	↑↑Block	↓GFR ↓RBF	↓HBF
Sevoflurane	↓BP ↓SVR ↔ HR	↑RR ↓TV	↑ICP ↓↓CMRO <sub>2</sub>	↑↑Block	↓GFR ↓RBF	↓HBF
Isoflurane	↓↓BP ↓↓SVR ↑HR	↑RR ↓↓TV	↑ICP ↓↓CMRO <sub>2</sub>	↑↑↑Block	↓GFR ↓RBF	↓HBF
Halothane	↓↓BP ↔ SVR ↓HR	↑↑RR ↓↓TV	↑↑ICP ↓CMRO <sub>2</sub>	↑↑Block	↓GFR ↓RBF	↓↓HBF

SVR, systemic vascular resistance; NM, neuromuscular; RR, respiratory rate; TV, tidal volume; ICP, intracranial pressure; CMRO<sub>2</sub>, cerebral metabolic requirement of oxygen (oxygen consumption); GFR, glomerular filtration rate; RBF, renal blood flow; HBF, hepatic blood flow.

\*Key features shown in **bold** and *italics*.

Source: Adapted from Morgan G, Mikhail M, Murray M. *Clinical Anesthesiology* 4th ed. McGraw Hill Medical: New York, NY: 2005.



## Heliox (Helium–Oxygen Combination)

- Common mixtures: 70%/30% and 80%/20% helium–oxygen
- 80%/20%: Density 1/3 of air/oxygen mixture → reduced density helps ↓ airway resistance by promoting laminar flow
- Reduces airway resistance → used to treat upper airway obstruction, asthma, COPD
- Can help ↓ pressures needed to ventilate pts with small-diameter ETTs and decreases work of breathing

## Nitric Oxide (NO)

- Inhaled NO/O<sub>2</sub> blends are used to promote capillary and pulmonary dilation to treat pulmonary hypertension.
- Oxidative reaction of NO with oxyhemoglobin → nitrate.
- Selective relaxation of pulmonary vasculature and improved arterial oxygenation.
- Little systemic hemodynamic effects.
- Adult clinical outcomes do not show clear benefit.

# PHARMACOLOGY: INTRAVENOUS ANESTHETICS AND ANALGESICS

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## INTRAVENOUS ANESTHETICS

Pharmacokinetic Data for Intravenous Anesthetics									
Drug	Induction, Dose, mg/kg IV (70 kg Dose)	Infusion Dose (mcg/kg/min)	Sedation Dose (mcg/kg/min)	Duration of Action (min)	Vd (Steady State) L/kg	t <sub>1/2</sub> Distribution (min)	t <sub>1/2</sub> Elimination (hr)	Protein Binding (%)	Clearance (ml/kg/min)
Propofol	1-2.5 (70-125)	100-200	25-75	3-8	2-10	2-4	4-23	97	20-30
Thiopental	3-5 (210-350)	200-300 (1st 20 min) 30-70 (after 20 min)	30-80	5-10	2.5	2.4	11	83	3.4
Methohexital	1-1.5 (70-105)	50-150	10-50	4-7	2.2	5-6	4	73	4
Etomidate	0.2-0.3 (14-21)	10 (*)	2.5-7.5	3-8	2.5-4.5	2-4	2.9-5.3	77	18-25
Ketamine	1-2 (70-140)	10-100	5-20	5-10	3.1	11-16	2-4	12	12-17
Dexmedetomidine**	n/a	0.2-0.7 mcg/kg/h**	0.2-0.7 mcg/kg/h**	n/a	2-3	6	2-3	94	10-30
Midazolam	0.1-0.3 (7-21)	0.25-1	0.25-1	15-20	1.1-1.7	7-15	1.7-2.6	94	6.4-11

Adapted from Stoelting, Miller, Basics of Anesthesia, 5th ed, 2006 p99.

\*Infusion of etomidate is only recommended for short procedures due to risk of adrenal suppression.

\*\*Note dexmedetomidine is dosed mcg/kg/h.

Pharmacodynamic Effects of Commonly Used Intravenous Anesthetics

	Propofol	Thiopental	Midazolam	Ketamine	Etomidate	Dexmedetomidine
Ventilation	D	D	U	U	U/D	U
Respiratory rate	D	D	U	U	U/D	U
Response to CO <sub>2</sub>	D	D	U	U	D	U
CBF	D	D	U	I/U	D	U
CMRO <sub>2</sub>	D	D	U	I/U	D	U/D
ICP	D	D	U	U/I	D	U
Anticonvulsant	Unclear	Y	Y	Unclear	N	Unclear
Anxiolysis	N	N	Y	N	N	Y?
Analgesia	N	N	N	Y	N	N?
Emergence delirium	N?	N	N	Y	N	N
Nausea/Vomiting	D	U	U/D	U	I	U
Adrenocortical suppression	N	N	Y?	N	Y	N
Pain on injection	Y	N	N	N	N	N

Source: Stoelting and Miller. *Basics of Anesthesia*, 5th Ed. 2006.

D = Decreased; U = Unchanged; I = Increased; Y = Yes; N = No

Hemodynamic Effects of IV Induction Agents				
Agent	BP	HR	CO	Contractility
Propofol	↓↓	↓↓	↓↓	↓↓
Thiopental	↓↓	0/↑	↓↓	↓
Etomidate	0/↓	0	0	0
Ketamine	↑	↑	0	0/↓ if depleted catecholamines

## OPIATES

### Opiate Pharmacokinetics:

Clearance is primarily hepatic, although differences in lipid solubility affect pharmacokinetic variability.

### Benefits:

Analgesia and sedation (dose-dependent); reduces MAC; amnestic with large doses (unreliable); cough suppression.

Pharmacokinetics of Intravenous Opiates							
Drug	Onset	Elim $t_{1/2}$	Part. Coeff.	Context-sensitive $t_{1/2}$	Vd (L/kg)	Protein-Bound (%)	Potency (compared with IV morphine)
Fentanyl	3–7 min	475 min	820	1–2 hr	4.1	84	100
Remifentanyl	60–120 sec	3–10 min	17.8	3–6 min	0.3–0.4	80	250
Sufentanil	3–5 min	2.5–4.5 hr	1750	17 min	2.86	92	500–700
Alfentanil	1.5–2 min	90–111 min	130	12–18 min	0.86	92	10–25
Morphine	20–30 min	2–4 hr	1.4	—	2.8–4.2	26–36	1
Hydromorphone	15 min	2.64	1.3	—	3.7	8–19	5–7
Meperidine	15 min	3–5 hr	21	—	2.8–4.2	70	0.1

### Fentanyl (Sublimaze IV; Fentora Buccal; Actiq Lozenge; Duragesic/Ionsys Transdermal)

#### Dose: (70-kg pt)

Intubation: 1.5–3 mcg/kg (100–200 mcg)

Intrathecal: 25 mcg

Epidural: Epidural PCA (PCEA): 10–30 mcg q10min; epidural infusion: 5–10 mcg/mL local anesthetic

Post op: 0.5–1.5 kg (35–105 mcg)

Sedation/analgesia: 0.5 mcg/kg (load);

0.01–0.04 mcg/kg/min (maint)

GA (sole agent) Induction: 50–150

mcg/kg; Maint: 0.1–5 mcg/kg/h

Very high dose (150 mcg/kg) is used

rarely, e.g., open heart surgery

GA adjunct: Loading dose: 2–50 mcg/kg;

Maint: 0.03–0.1 kg/min

**Clearance:** Liver and intestinal mucosa by CYP-450 3A4; *metabolite:* Norfentanyl (active)

**Comments:** High lipid solubility causes rapid redistribution to inactive sites (fat, skeletal muscle); therefore, quick onset and quick redistribution below therapeutic index. Comparative potency (to morphine):  $\times 75$ – $\times 125$

### Remifentanyl (Ultiva)

#### Dose: (70-kg pt)

Induction: 1–3 mcg/kg over 1 min

(70–270 mcg)

GA Adjunct: Loading dose: 0.5–2 mcg/kg (35–140 mcg); bolus 0.5–1 mcg/kg;

Maint: 0.05–2 mcg/kg/min

Sedation: 0.5–1 mcg/kg load (35–70 mcg)

infused over 30–60 sec; 0.025–0.2

mcg/kg/min maint

May bolus 1 mcg/kg when changing infusion rates

**Clearance:** Rapid metabolism of ester linkage by nonspecific blood and tissue esterases (NOT plasma cholinesterase); carboxylic acid metabolite (inactive)

**Comments:** Contains glycine, therefore contraindicated for epidural/intrathecal administration. Do not administer concomitantly with blood as esterases may metabolize.

**Sufentanil** (Sufenta, IV, intranasal)**Dose: (70 kg pt)**

GA (minor proc) Ind: 1–2 mcg/kg (70–140), bolus: 10–25 mcg

GA (moderate proc): Ind: 2–8 mcg/kg (70–560), bolus: 10–50 mcg; Maint: 0.3–1.5 mcg/kg/hr

GA (major proc): 8–30 mcg/kg (560–2100); bolus 10–50 mcg; Maint: 0.5–2.5 mcg/kg/hr

Sedation: Load: 0.1–0.5 mcg/kg (7–35 mcg); Maint: 0.005–0.01 mcg/kg/min  
Epidural: 10–15 mcg/10 mL of 0.125% bupivacaine**Clearance:** Liver and small intestine**Comments:** Produces hypnosis at doses  $\geq$  8 mcg/kg**Alfentanil** (Alfenta)**Dose: (70 kg pt)**Intubation: 20–50 mcg/kg (1400–3500)  
Induction: 130–245 mcg/kg (9.1–17.1 mg)  
Infusion: Load 50–75 mcg/kg; Maint: 0.5–3 mcg/kg/minMAC: 3–5 mcg/kg IV q5–20min or 0.25–1 mcg/kg/min IV for maintenance; start: 3–8 mcg/kg IV  $\times$  1 for sedated, responsive, spontaneously breathing pts; usual total dose = 3–40 mcg/kgMinor proc: Load 20 mcg/kg; bolus 3–5 mcg/kg q5–20 min to max 8–40 mcg/kg; or 0.5–1 mcg/kg/min  
Major proc: Load 20–50 mcg/kg; Maint 5–15 mcg/kg q5–20min to max 75 mcg/kg**Clearance:** Liver**Comments:** May increase ICP. Not recommended for children < 12. Produced hypnosis as single agent in sufficient concentration. Erythromycin and cimetidine inhibit clearance.**Morphine** (Astramorph, Duramorph, MS Contin, others)**Dose: (70-kg pt)**Sedation/analgesia: 2–10 mg IV (**Peds:** 0.02–0.1 mg/kg IV)

Analgesic dosing: 2–20 mg q2–4hr IV, IM, SC

PCA dosing: Bolus 1–4 mg q6–20min;

Basal: 0–1 mg/hr (**Peds:** Demand 0.01–0.03 mg/kg q6–20min lockout, Basal 0–0.03 mg/kg/hr)Infusion: 0.8–10 mg/hr (**Peds:** Sickle cell/cancer pain 0.025–2 mg/kg/hr; postop: 0.01–0.04 mg/kg/hr)Intrathecal: 0.1–0.5 mg (**Peds:** 0.01 mg/kg)Epidural: 2–6 mg q8–24h (bolus); 0.2–1 mg/hr (infusion); (**Peds:** 0.03–0.05 mg/kg, max: 0.1 mg/kg or 5 mg/24hr)**Clearance:** Primarily renal; Metabolites: Morphine-3-glucuronide (55% to 75%, inactive) and morphine-6-glucuronide, active.**Comments:** May cause sphincter of Oddi (biliary) spasm. Adjust dosing in renal failure. Greatest histamine release of commonly administered opiates.**Hydromorphone** (Dilaudid)**Dose: (70-kg pt)**Analgesic dosing: 0.4–2 mg IV (**Peds,** 0.005–0.02 mg/kg)PCA dosing: Demand 0.2–0.6 mg q6–20 m lockout; Basal: 0–0.2 mg/hr (**Peds,** demand 0.005–0.02 mg/kg q6–20min lockout, basal 0–0.005 mg/kg/hr)**Clearance:** Liver metabolism, urine/bile excretion; Metabolites: Liver glucuronidation 3-glucuronide (major) and 6-hydroxy (minor)**Comments:** Useful alternative to morphine; less histamine release, safer in renal impairment, shorter time to peak effect.**Meperidine** (Demerol)**Dose: (70-kg pt)**Sedation/analgesic dosing: 50–150 mg IV/IM q3–4h (**Peds,** 0.5–2 mg/kg IV, IM),  
Infusion: 0.3–1.5 mg/kg/hrEpidural: Bolus, 20–50 mg; 10–50 mg/hr  
Postop shivering: 12.5–25 mg IV**Clearance:** Liver metabolism, urinary excretion; Metabolite: normeperidine**Comments:** Direct myocardial depressant; less incidence of spincter of Oddi spasm. Administration with MAOI may result in delirium or hyperthermia. Structurally similar to atropine, thus accounting for antispasmodic effect. Normeperidine is CNS stimulant at high concentrations, and is a potent convulsant. Antishivering action may be result of kappa receptor agonism.**Methadone** (Dolophine)**Indications:** Chronic pain, opioid withdrawal**Dose: Adults, opiate-naïve:** Start 2.5–10 mg PO or 2.5–5 mg IV/IM/SC q8–12h; titrate up q3–5d**Mechanism:** Opiate agonist and NMDA receptor antagonist**Clearance:** Liver

**Comments:** QT prolongation possible in doses greater than 90 mg/d. Less sedative and euphoric effects than morphine, but similar respiratory depression, miosis, constipation, and biliary tract spasm. Long half-life requires careful titration if increasing dose. Should wait 3–5 d before making dose changes. Use caution when converting long standing opiate users to methadone, as paradoxically lower dose conversion ratios are required (see table below). Use for detoxification may require participation of licensed opiate agonist therapy program.

Conversion of Oral Morphine Equivalent to Oral Methadone, Opiate-Tolerant Patient	
Daily Oral Morphine Equivalent	Conversion Ratio Morphine:Methadone
<30 mg	2:1
31–99 mg	4:1
100–299 mg	8:1
300–499 mg	12:1
500–999 mg	15:1
> 1000 mg	20:1

Use caution. Considerable individual variation and long half-life of methadone can result in dangerous dose escalation if sufficient time (~5 d) not allowed between dose changes. These tables should NOT be used in reverse (converting from methadone to morphine).

Source: From Fisch, M Cleeland. C. Managing cancer pain. In Skeel ed. *Handbook of Cancer Chemotherapy*, 6th ed. Philadelphia: Lippincott Williams & Wilkins, 2003:66.

### Tramadol (Ultram)

**Dose:** Start at 25 mg qAM, increasing 25 mg qd to 25 mg qid, then 50 mg/d × 3 d to 50 mg qid. Max 400 mg/d or 300 mg/d if age >75. May skip dose titration if immediate onset desired.

**Mechanism:** Opiate agonist and plus inhibition of norepinephrine and serotonin reuptake. Not completely antagonized by naloxone. Codeine analog.

**Clearance:** Liver; adjust dose in elderly, renal, and pts with hepatic disease.

Common Oral Opiates		
Brand Name	Generic	Adult Starting Dose
<b>Hydrocodone (HC) containing + acetaminophen (APAP)*</b>		
Norco	HC 10 mg + APAP 325 mg	1–2 tabs q4–6h prn
Lortab 2.5/500	HC 2.5 mg + APAP 500 mg	
Vicodin, Lortab 5/500, Zydone, Anexia 5/500, Hydrocet	HC 5 mg + APAP 500 mg	
Lortab 7.5/500, Vicodin ES, Anexia 7.5/650	HC 7.5 mg + APAP 500 or 650 mg	
Lortab 10/500, Vicodin HP	Dose: 1–2 tab q4h prn	
Lortab elixir	HC 2.5 mg + APAP 120 mg/5 mL	15 mL q4h prn
<b>Hydrocodone (HC) + Aspirin or Ibuprofen</b>		
Lortab ASA	HC 5 mg + ASA 500 mg	1 q4–6h
Vicoprofen	Hydrocodone 7.5 mg + ibuprofen 200 mg	Dose: 1–2 tab q4h prn
<b>Oxycodone-containing</b>		
Roxicodone	Oxycodone	5–10 mg q2–6h
OxyContin	Oxycodone (sustain release)	10 mg q8–12h
Percocet	Oxycodone/APAP 2.5, 5, 7.5, 10/325; 7.5/500; 10/650	1–2 PO q4–6h based on APAP dose*
Percodan	Oxycodone/aspirin 4.8355/325	1 PO q6h prn
OxyFast	Oxycodone liquid, 20 mg/mL	5–10 mg q2–6h
<b>Morphine-containing</b>		
Morphine	Morphine	10–30 q3–4h
Oramorph SR	Morphine sustained release	15–30 q8–12h
MSContin	Morphine sustained release	15–30 q8–12h
Roxanol	Morphine solution (20 mg/mL)	10–30 mg q4h prn

Common Oral Opiates (Continued)		
Brand Name	Generic	Adult Starting Dose
<b>Codeine-containing</b>		
Tylenol #2, #3, #4	APAP 300 mg + codeine 15, 30, 60 mg	30–60 mg codeine q4h*
	Codeine sulfate	15–60 mg q4h prn
Fioricet/Fiorinal with codeine	APAP 325, codeine 30, butalbital 50, caffeine 40	1–2 caps q4h prn, max 6/d
Soma with codeine	Carisoprodol 200 mg, ASA 325 mg, codeine 16 mg	1–2 caps qid
Synalgos-DC	Dihydrocodeine 16 mg, aspirin 356.4 mg, caffeine 30 mg	2 q4h
<b>Other</b>		
Dilaudid	Hydromorphone	6 mg q3–4
Demerol	Meperidine	100 mg q3h
LevoDromoran	Levorphanol	4 mg q6–8h
Dolophine	Methadone	10 mg q8–12h
Ultram	Tramadol	25 mg qAM

\*CAUTION: Do not exceed maximum daily dose for APAP: 4 g/d healthy adults; 2 g/d if hepatic disease

### COMMON BENZODIAZEPINES IN ANESTHESIA, SPECIFIC COMMENTS

#### Midazolam (Versed)

**Metabolites:** 1-OH-midazolam (inactive)

**Comments:** Highest lipid solubility of benzodiazepines. Increased lipid solubility after entering physiologic pH. Short-context sensitive half-time makes suitable for infusion. Approximately double affinity for gamma subunit of GABA<sub>A</sub> receptor explains greater potency of midazolam.

#### Diazepam (Valium)

**Metabolites:** Desmethyldiazepam (active), oxazepam (active)

**Comments:** Poor water solubility—propylene glycol solvent causes pain on injection

#### Lorazepam (Ativan)

**Metabolites:** Lorazepam glucuronide (inactive)

**Comments:** Infusion not recommended

### Short- and Long-Acting Benzodiazepines

Drug	Protein Binding (80%)	Half-life (hr)	Route	Dose (mg)	Onset (min)	Peak (hr)
Alprazolam (Xanax)	80	12–15	PO	0.25–1 q8h	2 hr	0.7–1.2
Lorazepam (Ativan)	93	10–20	PO	0.5–2 q8–12h	15–45	2–5
			IM	2–4 q8–12h	15–30	1–1.5
			IV	2–4 prn	1–5	Immediate
Oxazepam (Serax)	95–98	5–15	PO	10–30 q6–8h	45–90	2–4
Temazepam (Restoril)	96	10–20	PO	15–30 q6h	45–60	2–3
Triazolam (Halcion)	94–98	1.7–3	PO	0.25–0.5 q8h	18	1–3
Midazolam (Versed)	96–97	2	IM	5 × 1	15	0.5–1
			IV (induction)	0.1–0.3 mg/kg	0.5–2	Immediate
			IV (sedation)	1–4 × 1, repeat	0.5–2	Immediate
Chlordiazepoxide (Librium)	97	7–28	PO	50–100 q6h	15–45	0.5–2

(continued)

Short- and Long-Acting Benzodiazepines (Continued)						
Drug	Protein Binding (80%)	Half-life (hr)	Route	Dose (mg)	Onset (min)	Peak (hr)
			IM	25–100 q2–4h	15–30	Erratic
			IV	25–100 prn	1–5	immediate
Diazepam (Valium)	99	20–90	PO	2–10 q6h	15–45	0.5–1.5
			IM	2–10 q3–4h	20	0.5–1.5
			IV	2–10 prn	1–3	Immediate
Flurazepam (Dalmane)	88	Rapid, but active metabolites last 24–100hr	PO	15–30 qd	15–45	0.5–1

Source: Adapted from Attia RR, Grogono AW, Domer FR, eds. *Psychotropic Agents in Practical Anesthetic Pharmacology*, 2nd ed. Norwalk, CT: Appleton-Century-Crofts, 1987:149–169.

## ANTAGONISTS FOR ANALGESICS AND INTRAVENOUS ANESTHETICS

### Naloxone (Narcan)

**Indications:** (1) opiate overdose; (2) reversal of opiate respiratory depression; (3) treatment of opiate-induced pruritus.

**Dose: Adult:** (1) 0.4–2 mg IV q2–3min prn; (2) 0.04 to 0.4 mg doses IV, titrated q2–3min. *Infusion: Load* 5 mcg/kg, *infusion* 2.5–160 mcg/kg/hr; **Peds:** (1) **Birth to 5 yr:** <20 kg: 0.1 mg/kg IV q2–3min prn; >5 yr or >20 kg: 2 mg/dose q2–3min prn; *infusion* same as adult. (2) 1–10 mcg/kg IV titrated q2–3min (up to 0.4 mg).

**Mechanism:** Competitive inhibition of opioid receptors

**Clearance:** Hepatic metabolism (95%); primarily renal elimination.

**Comments:** May cause hypertension, dysrhythmias, rare pulmonary edema, delirium, reversal of analgesia, or withdrawal syndrome (in opioid-dependent patients). Renarcotization may occur because antagonist has short duration. Caution in hepatic failure and chronic cardiac disease.

### Methylnaltrexone (Relistor)

**Dose:** <38 kg: 0.15 mg/kg SC; 38–62 kg: 8 mg (0.4 mL) SC; 62–114 kg: 12 mg (0.6 mL) SC; *typical regimen:* every other day but no more frequently than once/24 hr.

**Mechanism:** Peripherally acting mu opioid receptor antagonist. Does not cross blood-brain barrier.

**Clearance:** Unknown metabolism. Excreted in urine/feces.

**Comments:** Indicated for treatment of opiate induced constipation and failed laxative therapy. Contraindicated in patients with known or suspected mechanical GI obstruction. In patients with severe renal impairment (creatinine clearance <30 mL/min), reduce dose by 50%. May cause diarrhea, abdominal pain, nausea, dizziness.

### Flumazenil (Mazicon)

**Indications:** (1) Reversal of benzodiazepine sedation; (2) reversal of benzodiazepine overdose

**Dose: Adult:** (1) 0.2–1.0 mg IV at 0.2 mg/min; wait at least 1 min between doses and repeat at least every 20 min to avoid resedation, with max dosing 3 mg/hr; (2) 3–5 mg IV at 0.5 mg/min, if no response, benzodiazepine overdose unlikely etiology.

**Peds:** 0.01 mg/kg IV at rate no faster than 0.2 mg/min to max 1 mg.

**Mechanism:** Competitive antagonism of GABA receptor, antagonizing benzodiazepine effect.

**Clearance:** 100% hepatic metabolism; 90%–95% renal elimination of metabolite.

**Onset:** 6–10 min for peak effect

**Comments:** Duration of action shorter than midazolam and other agonists which may lead to resedation after flumazenil cleared. May induce CNS excitation including seizures, acute withdrawal, nausea, dizziness, agitation, hypertension, arrhythmias. Only partial reversal of midazolam-induced ventilatory depression. Does not reverse nonbenzodiazepine induced CNS depression. Do not use in unknown drug overdose, suspected tricyclic antidepressant overdose, or seizure-prone patients.



## ADJUNCT MEDICATIONS IN TREATMENT OF PAIN

Anticonvulsants		
Drug	Starting Dose (adult)	Dose Range (comments)
<b>CALCIUM CHANNEL ACTION</b>		
<b>Gabapentin</b> (Neurontin)	300 mg qhs-tid	900-3600 mg/d divide tid
<b>Pregabalin</b> (Lyrica)	50 mg tid	Max/d 600 mg divide bid-tid
<b>SODIUM CHANNEL ACTION</b>		
<b>Carbamazepine</b> (Tegretol)	100-150 mg bid	200-1800 mg/d divide bid-tid
<b>Lamotrigine</b> (Lamictal)	25-mg qhs	25-600 mg/d divide bid (increase dose very slowly)
<b>Oxcarbazepine</b> (Trileptal)	75-150 mg qd or bid	600-2400 mg/d divide bid (increase weekly)
<b>MIXED SODIUM AND CALCIUM ACTION</b>		
<b>Topiramate</b> (Topamax)	15 mg bid or 50 mg qhs	15-800 mg/d divide bid (increase dose weekly by 50-mg increments)
<b>Zonisamide</b> (Zonegran)	100 mg/qod	100-600 mg/d (increase very slowly by 100 mg every other week)
<b>GABA ACTION</b>		
<b>Valproic acid</b> (Depakene)	15 mg/kg/bid-tid or 250 mg PO qd-bid	60 mg/kg/tid
<b>Clonazepam</b> (Klonopin)	0.5-1 mg qhs	0.5-4 mg/day divide daily-bid
<b>UNKNOWN ACTION (POSSIBLE GABA/CA<sup>2+</sup> ACTION)</b>		
<b>Levetiracetam</b> (Keppra)	250 mg bid	1000-4000 mg/d divide bid/tid

**Indications:** Anticonvulsant drugs have been found to be effective in treatment of neuropathic pain including painful diabetic neuropathy (PDN), postherpetic neuralgia (PHN), trigeminal neuralgia, etc.

**Mechanism:** Multiple receptor sites, generally antagonizing either calcium or sodium channels, GABA receptors, or other unknown sites of action.

**Clearance:** Mostly renal. Dosage adjustments may be required in renal impairment (especially with gabapentin and topiramate).

### MUSCLE RELAXANTS

Drug	Starting Dose (PO, adult)	Dose Range (PO)	Mechanism
<b>Baclofen</b> (Lioresal)	5 mg tid-qid	15-80 mg/day div tid-qid	GABA-B agonist
<b>Diazepam</b> (Valium)	2-5 mg bid-qid	2-10 mg bid-qid	GABA-A agonist
<b>Tizanidine</b> (Zanaflex)	4-8 mg qhs	12-36 mg/day div q8h	Alpha <sub>2</sub> adrenergic agonist
<b>Dantrolene</b> (Dantrium)		Max 400 mg div q6h	Inhibits Ca <sup>2+</sup> release
<b>Cyclobenzaprine</b> (Flexeril)	10 mg tid	20-60 mg/day	Similar to tricyclic antidepressants
<b>Carisoprodol</b> (Soma)	250 mg tid and qhs	250-350 mg PO tid and qhs	Metabolized to barbiturate meprobamate
<b>Metaxalone</b> (Skelazin)	800 mg tid/qid		Unknown
<b>Methocarbamol</b> (Robaxin)	1.5 g qid	4.5 g/day div q4-8h	Unknown
<b>Orphenadrine</b> (Norflex)	100 mg bid	100 mg bid	Unknown
<b>Chlorzoxazone</b> (Paraflex)	250-500 mg tid/qid	250-750 mg tid/qid	Unknown

Mixed Opiate Agonists–Antagonists				
Drug	Dose Equianalgesic to Morphine 10 mg IV	Onset (min)	T <sub>1/2</sub> (hr)	Typical Dosing/ Comments
<b>Buprenorphine</b> (Buprenex, IM/IV, Suprenex SL)	0.3–0.4 mg q6–8h IM/IV (May repeat initial dose in 30–60 min ×1. >0.3 mg/dose should only be given IM)	30	8	<b>2–12 yr/o:</b> 2–6 mcg/kg IM/IV q4–6h, max 6 mcg/kg/dose <b>&gt;13 yr/o:</b> 0.3 mg IV/IM q6–8h, max 300 mcg/dose Epidural: 0.3 mg
<b>Buprenorphine/ naloxone</b> (Suboxone)	<b>Access restricted in U.S. to certified opioid dependence centers. See <a href="http://www.suboxone.com">www.suboxone.com</a> for more info</b>			
<b>Butorphanol</b> (Stadol)	2 mg IM/IV q3–4h	20	2.5–3.5	1 mg IV or 2 mg IM. Max 4 mg/dose IM Nasal spray: 1 mg in 1 nostril q3–4h <b>As anesthesia adjunct,</b> 2 mg IV before induction then 0.5–1 mg IV prn
<b>Nalbuphine</b> (Nubain)	10 mg IV/IM/SC q3–6h	20	3–6	Max 20 mg/dose, 160 mg/day
<b>Pentazocine</b> (Talwin)	60 mg IV/IM/SC q3–6h	15	2–3	Max doses: 30 mg IV, 60 mg IM/IV SC dosing can cause tissue damage. Epidural: 0.3 mg/kg in 10 mL saline

**Indications:** Mild to moderate pain, esp. headaches

**Mechanism:** Bind to mu receptors with limited response (partial agonist) or no effect (competitive antagonist) and often kappa/delta receptor agonism as well.

**Comments:** Agonist–antagonist can decrease efficacy of subsequently administered opiates. Advantage is that these have limited respiratory depression and decreased potential for physical dependence. Unlike pure opiate agonists, agonist–antagonists have a ceiling effect in their dose–response relationship. Thus these medications are not recommended when pain may increase. Antagonist effects of these drugs may precipitate withdrawal in opiate dependent patients. Side effects otherwise similar to opiate agonists. Pentazocine and butorphanol (but not nalbuphine) increase systolic blood pressure, pulmonary artery blood pressure and cardiac output. Milder effects on GI and biliary systems than with morphine. Butorphanol commonly used in obstetrics.

## TRANSDERMAL/TOPICAL MEDICATIONS

### Fentanyl Transdermal (Duragesic)

**Indications:** Sustained release opiate therapy, treatment of chronic pain

**Dose:** See table below

**Mechanism:** Opiate agonist

**Clearance:** See fentanyl above

**Comments:** Available from 12.5 to 100 mcg/hr patches. Time to peak efficacy 12 hr. Change every 72 hr. See IV fentanyl for side effects. Conversion from total daily dose of morphine to fentanyl complicated with multiple possible formulas. See table below. Contraindicated for postoperative pain relief in opiate naïve patients as high risk for respiratory depression.

**Oral Morphine to Fentanyl Patch Conversion**

Oral 24-hr Morphine (mg/day)	Fentanyl Patch (mcg/hr)
60-134	25
135-224	50
225-314	75
315-404	100
405-494	125
495-584	150
585-674	175
675-764	200
765-854	225
855-944	250
945-1034	275
1035-1124	300

**Lidocaine patch, 5%** (Lidoderm)

**Indications:** Neuropathic pain, local inflammatory conditions.

**Dose:** 1-3 patches q24 h with 12 hr on and 12 hr off typical usage although greater than 3 patches and 24 hr usage have been studied and found to be safe and well tolerated.

**Mechanism:** Sodium channel blockade.

**Comments:** Produces analgesia but not anesthesia. Minimal systemic absorption. Main side effects are local skin irritation (e.g., burning, dermatitis, pruritus, rash).

**EMLA** (Eutectic mixture of local anesthetics, lidocaine 2.5%, prilocaine 2.5% cream)

**Indication:** Topical anesthesia for minor procedures, esp pediatric IV placement

**Dose:** Apply minimum of 1 hr prior to procedure and cover with occlusive dressing.  
**adult:** 2.5-10 g to intact skin.

**EMLA Pediatric Dosing**

Age/Weight	Max Dose (g)
0-3 mo/o, <5 kg	1
3-12 mo/o, >5 kg	2
1-6 y/o, >10 kg	10
7-12 y/o, >20 kg	20

**Mechanism:** Sodium channel blockade.

**Comments:** Use smallest amount necessary. Minimal systemic absorption. May cause pallor/blanching, erythema, edema, itching, and rarely rash.

**Nonsteroidal Anti-inflammatory Drugs (NSAIDs) and Acetaminophen**

**Indication:** Analgesia, anti-inflammatory, antipyretic.

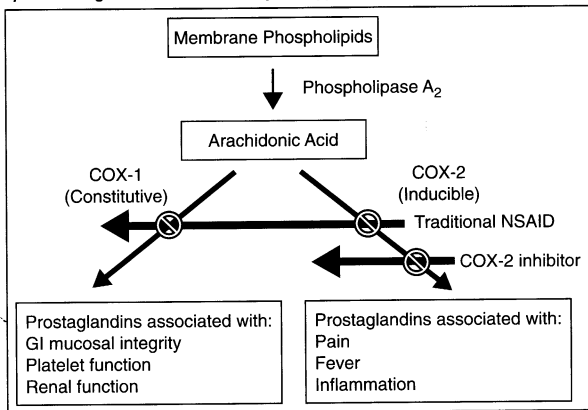
**Dosing:** See table below. Mostly PO, although Toradol may be administered IV and acetaminophen may be administered PR (IV in clinical trials in the U. S.). Generally have a ceiling effect (unlike opiates) beyond which further analgesia does not occur, but side effects worsen.

**Mechanism:** Inhibition (specific or nonspecific) of cyclooxygenase (aka COX) → decreased formation of inflammatory mediators (i.e., prostaglandins). COX is enzyme that catalyzes synthesis of prostaglandins from arachidonic acid. COX-2 pathway (inducible by injury) is typical target action. COX-1 pathway (constitutive) results in adverse effects.

**Clearance:** Primarily hepatic.

**Comments:** COX-1 inhibition is associated with majority of side effects: GI mucosal ulceration, decreased renal perfusion, and decreased platelet aggregation. Inhibition of prostaglandin synthesis suspected mechanism for NSAID-induced bronchospasm. Selective COX-2 inhibitors have been reconsidered in light of potentially increased risk of MI and CVA.

**Figure 2B-1** General mechanism of action of NSAIDs. (From *The MGH Handbook of Pain Management*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2002.)



INTRAVENOUS 2-15

Nonsteroidal Anti-inflammatory Medications				
Medication	Brand Name	Usual Daily Dose (Adults)	Serum Half-Life (hr)	Maximum Daily Dose (mg)
<b>Nonselective NSAIDs</b>				
<b>Carboxylic Acid Derivatives</b>				
Aspirin (ASA) (acetylsalicylic acid)	Multiple	MI prevention: 81–325 mg qd Pain: 325 mg–6 g/24 hr div q4–6h	4–15	3600
Salsalate	Disalcid	1.5–3.0 g/24 hr bid	Same	
Diflunisal	Dolobid	0.5–1.5 g/24 hr bid	7–15	2000
Choline magnesium trisalicylate	Trilisate	1.5–3 g/24 hr bid–tid		3000
<b>Propionic Acid Derivatives</b>				
Ibuprofen	Motrin, Rufen, OTC	OTC: 200–400 mg qid Rx: 400, 600, 800, maximum: 3200 mg	2	3200
Naproxen	Naprolan, Anaprox, Naprosyn EC	250, 375, 500 mg bid	13	1500
Fenoprofen	Nalfon	300–600 mg qid	3	3200
Ketoprofen	Orudis	75 mg tid	2	300
Flurbiprofen	Ansaid	100 mg bid–tid	3–9	300

<b>Nonsteroidal Anti-inflammatory Medications (Continued)</b>				
<b>Medication</b>	<b>Brand Name</b>	<b>Usual Daily Dose (Adults)</b>	<b>Serum Half-Life (hr)</b>	<b>Maximum Daily Dose (mg)</b>
Oxaprozin	Daypro	600–1800 mg/24 h	40–50	1800
Tolmetin	Tolectin	400, 600, 800 mg; 800–2400 mg	1	1800
<b>Acetic Acid Derivatives</b>				
Indomethacin	Indocin, Indocin SR Indocin SR	25–50 mg tid or qid SR: 75 mg bid; rarely >150 mg/24 hr	3–11	200 150
Tolmetin	See above		1	400
Sulindac	Clinoril	150, 200 mg bid to tid	16	2000
Diclofenac	Voltaren, Arthrotec	50 tid, 75 mg bid	1–2	200
Etodolac	Lodine	200–300 mg bid– tid–qid; maximum: 1200 mg	2–4	200
Ketorolac	Toradol	15–30 mg IM/IV q6h, 10 mg PO q4–6h	2	120
<b>Fenamates</b>				
Meclofenamate	Meclomen	50–100 mg tid–qid	2–3	400
Mefenamic acid	Ponstel	250 mg qid	2	1000
<b>Enolic Acid Derivatives</b>				
Piroxicam	Feldene	10, 20 mg qd	30–86	20
Phenylbutazone	Butazolidin	100 mg tid up to 600 mg/24 hr	40–80	
Meloxicam	Mobic	7.5–15 mg	20	15
<b>Naphthylkanones</b>				
Nabumetone	Relafen	500 mg bid, up to 1500 mg/24 hr	19–30	40
<b>COX-2 Selective NSAIDs</b>				
Celecoxib	Celebrex	100, 200 mg bid, 200 mg qd	11–12	800
<b>p-Aminophenols</b>				
Acetaminophen	Multiple	325–1000 mg PO/PR q4–6h	2–4	1000/dose, 4000 g/24 h

Source: Adapted from Warfield C, Fauset H. *Manual of Pain Management*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2002.

<b>Pediatric Dosing of NSAIDs</b>	
<b>Drug</b>	<b>Dose</b>
Acetaminophen	(PO) 10–15 mg/kg q4h (PR) 30–40 mg/kg* (IV) <i>In clinical trials in U.S.</i>
Aspirin**	10–15 mg/kg q4h
Ibuprofen	4–10 mg/kg q6–8h
Ketorolac (IV)	0.5 mg/kg q6–8h, not for >5 d

\*Avoid PR acetaminophen in pts with cancer, immunosuppressed neutropenic pts, etc.

\*\*Risk for Reye's syndrome if concurrent influenza, viral illness.

Source: Adapted from Berde C, Masek B. Pain in children. In Wall PD, Melzack R, eds. *Textbook of Pain*. Edinburgh: Churchill Livingstone, 1999.

## **NONSELECTIVE COX INHIBITORS**

**Acetaminophen** (Tylenol, Paracetamol)

**Dose:** q4–6h PO, PR. **Adults:** max 4 g/d or 2 g/d in pts with hepatic disease

**Mechanism:** Not true NSAID as lacks significant anti-inflammatory effect

**Clearance:** Liver

**Comments:** Does not produce GI irritation, affect platelet aggregation. Does contribute to nephrotoxicity. Single dose >15 g leads to hepatic necrosis due to

formation of N-acetyl-p-benzoquinone (scavenged by antioxidant glutathione). Toxicity occurs due to depletion of glutathione. Acetylcysteine may substitute for glutathione and prevent hepatotoxicity if administered within 8 hr of ingestion. Intravenous form in clinical trials in U.S. (available in Europe).

### **Propionic Acids (Ibuprofen, Naproxen, Fenoprofen, Ketoprofen, Diclofenac)**

**Comments:** Alter platelet function, but duration of COX inhibition varies with specific drugs. Naproxen may be dosed twice daily owing to longer half-life. May exacerbate renal disease. Extensive protein binding leads to adverse drug effects (e.g., reduce dosing of warfarin because of extensive displacement from protein binding sites and effects on platelet function). Ibuprofen has less protein binding than other propionic acids.

### **PYRROLOPYRROLE (e.g., KETOROLAC)**

**Ketorolac** (Toradol)

**Dose: Adult:** >50 kg: bolus 30–60 mg IV/IM, then 15–30 mg q6h, max 120 mg/d for pts <65 yr/o; max 60 mg/d for pt >65 yr/o or with renal disease; <50 kg: bolus 30 mg IV/IM, then 15 mg q6h, max 60 mg/d. PO: 10 mg q4–6h, max 40 mg/24 hr. **Peds:** 0.4–1 mg/kg/dose IV, then 0.2–0.5 mg/kg/dose q6h

**Clearance:** Less than 50% hepatic metabolism, renal metabolism; 91% renal elimination.

**Comments:** Parenteral administration makes useful short-term adjunct for severe pain when used with parenteral or epidural opioids. When administered IV or IM, analgesic effect is more potent than anti-inflammatory effect. Does not cause respiratory depression or biliary tract spasm, like opiates. Ketorolac 30 mg IM equianalgesic to 10 mg morphine or 100 mg meperidine. Decreased clearance in elderly, therefore reduce dosing. Effect on platelet function and prolonging bleeding time is observed with spinal anesthesia but not with general anesthesia. Renal compromise minimized with adequate hydration. **Max duration 5 d.**

### **CARBOXYLIC ACIDS**

**Acetylated: aspirin. Non-acetylated: sodium salicylate, salicylamide, diflunisal**

**Aspirin, acetylsalicylic acid**

**Indications:** Low intensity pain, headache, musculoskeletal pain; antiplatelet drug for MI/CVA prevention; antipyretic

**Mechanism:** Irreversibly acetylates cyclooxygenase

**Comments:** Usual stopped 5–10 days prior to surgery; antiplatelet effect exacerbated in Von Willebrand's dz & uremic pts; avoid in pts with severe hepatic dz, vit K deficiency, low prothrombin, hemophilia; doesn't inc. incidence of ESRD

**Selective COX-2 Inhibitors (celecoxib)**

**Mechanism:** Selectively inhibits cyclooxygenase-2 enzyme, inhibiting prostaglandin synthesis

**Comments:** May be safer in pts with history of gastric ulcers or gastritis; contraindicated in pts with sulfonamide allergy

# PHARMACOLOGY: LOCAL ANESTHETICS

JENNA HANSEN • SAMUEL C. SEIDEN

LOCAL 2-18

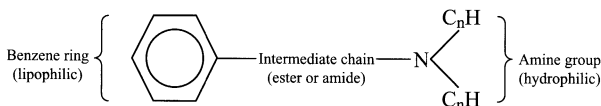
## Mechanism of Action

- Local anesthetics (LA) are weak bases, hydrophilic, tertiary amines
- Exert their action by binding to Na<sup>+</sup> channel, thereby blocking depolarization-induced influx of Na<sup>+</sup> and blocking propagation of nerve impulse

## Classification and Structure

LAs have a lipophilic benzene ring linked to an amine group by a hydrocarbon chain of amide or ester linkage. This linkage separates LAs into two groups:

- Esters** (procaine [Novocaine], chlorprocaine [Nesacaine], tetracaine [Pontocaine], cocaine)
  - They undergo hydrolysis by pseudocholinesterases found in plasma.
  - The pKa is usu >8.
  - A significant metabolite, PABA (para-amino benzoic acid), can cause an allergic reaction.
- Amides** (lidocaine [Xylocaine], bupivacaine [Marcaine], mepivacaine [Carbocaine], etidocaine [Duranest])
  - They undergo metabolism by hepatic microsomal enzymes.
  - The pKa is usu <8, may contain antibacterial preservative (methylparaben); allergic reactions less common.



## PHARMACODYNAMICS

- Ionization best correlates with onset of action. LAs exist in free equilibrium in both charged (ionized) and neutral (unionized) forms:
  - The charged form binds to the receptor and exerts the drug's action, but it is very hydrophilic and cannot penetrate the nerve membrane to exert its effect.
  - The uncharged lipid-soluble form allows the drug to penetrate nerve membrane.
- Lipid solubility best correlates with potency: The higher the solubility, the greater the potency.
- Protein binding best correlates with duration of action; only the unbound form is active.
- The relative proportion of charged and uncharged LA molecules is a function of the drug's pKa and the tissue pH.
- pKa = pH at which the concentrations of ionized and unionized forms are equal.

Commonly Used Local Anesthetics (See also Chapter 6)						
	pKa	Relative Potency	Onset (min)	Duration (hr)	Max Dose (mg/kg)	Concentrations with Clinical Uses
<b>Esters</b>						
Chlorprocaine (Nesacaine)	8.7	2	5-10	0.5-1	9, 15 (with epi)	Epidural 2%-3% Infiltration 1% Peripheral nerve block 2%
Cocaine	8.6	2	<2	0.5	1.5	Topical 4%-10%
Procaine (Novocaine)	8.9	1	2-5	0.75-1	7, 10 (with epi)	Spinal 10%
Tetracaine (Pontocaine)	8.5	8	5-10	3	1.5, 2.5 (with epi)	Spinal 0.5% Topical 2%

(continued)

Commonly Used Local Anesthetics (See also Chapter 6) (Continued)						
	pKa	Relative Potency	Onset (min)	Duration (hr)	Max Dose (mg/kg)	Concentrations with Clinical Uses
<b>Amides</b>						
Bupivacaine (Marcaine)	8.1	8	5–10	4–8	2.5, 3 (with epi)	Epidural/Spinal 0.5%–0.75% Infiltration 0.25% Peripheral nerve block 0.25%–0.5%
Lidocaine (Xylocaine)	7.9	2	<2	1–2	4.5, 7 (with epi)	Epidural/Spinal 1.5%–2% Infiltration 0.5%–1% IV regional 0.25%–0.5% Peripheral nerve block 1%–1.5% Topical 4%
Mepivacaine (Carbocaine)	7.6	2	3–5	1.5–3	5, 7 (with epi)	Epidural/Spinal 1.5%–2% Infiltration 0.5%–1% Peripheral nerve block 1%–1.5%
Prilocaine (Citanest)	7.9	2	<2	1–2	6, 9 (with epi)	Epidural 2%–3% Infiltration 0.5%–1% IV regional 0.25%–0.5% Peripheral nerve block 1.5%–2%
Ropivacaine (Naropin)	8.1	6	10–15	4–8	2.5, 3 (with epi)	Epidural 0.5%–1% Infiltration 0.2%–0.5% Peripheral nerve block 0.5%–1%

## SPECIFIC CONSIDERATIONS

### Chlorprocaine

- Contraindicated in plasma cholinesterase deficiency
- Useful in pts with significant liver disease and history of seizures
- Short-acting, undergoes rapid hydrolysis
- Possible neurotoxicity (arachnoiditis) from accidental intrathecal injection, possibly from bisulfite preservative
- Useful in obstetrics owing to ↓ risk of systemic toxicity (rapid hydrolysis) and fetal exposure

### Lidocaine

- Useful for topical, regional, intravenous, peripheral nerve block and spinal/epidural anesthesia.
- Transient neurologic symptoms (TNS); related to maldistribution of local anesthetic, spinal catheters, pt positioning (i.e., lithotomy).
- TNS:
  - Also occurs with use of other local anesthetics.
  - Pain symptoms occur within 12–24 hr → complete resolution in 3–7 days.
  - No motor or sensory deficits
  - Tx with NSAIDs and opioids as needed.

### Bupivacaine

- Reduce dose by 50% in neonates.
- High quality sensory anesthesia relative to motor blockade.
- More cardiotoxicity than ropivacaine.



**Ropivacaine**

- Less cardiotoxicity than bupivacaine (esp when large volumes are used).
- More vasoconstriction than bupivacaine → reduced systemic toxicity.
- More expensive than bupivacaine.

**Tetracaine**

- Long-acting, slow onset.
- Usual used for spinal → but high risk of TNS and systemic toxicity.

Speed of LA onset affected by:

1. pKa → The lower the pKa of the anesthetic, the greater the fraction of uncharged lipid-soluble drug at a given pH → easier membrane penetration → quicker the onset time.
2. Bicarbonate ( $\text{HCO}_3^-$ ) addition → increases the uncharged form → quicker onset time; but tissue acidosis (i.e., infection) → increase charged form → delayed onset.
3. Greater lipid solubility → increases membrane/nerve sheath penetration.
4. Presence of neural sheath delays onset.
5. Increased concentration and total dose of local → faster onset.
6. Site of injection and distance traveled to reach the nerve (subarachnoid → peripheral nerve).

Duration of LA action affected by:

1. Protein binding → higher protein binding → longer duration.
2. Site of local injection → vascular sites have shorter duration (more systemic absorption).
3. Degree of vasodilation (all locals except cocaine are vasodilators).
4. Lipid solubility → ↑ lipid solubility will ↑ duration.
5. Pseudocholinesterase deficiency → ↑ duration of ester anesthetics.
6. Liver disease → ↑ duration of amide anesthetics.

**ADDITIVES TO ENHANCE LAs**

- Epinephrine—vasoconstrictors added to local anesthetics limit systemic absorption → prolong duration and intensity of the block & ↓ likelihood of systemic toxicity. Vasoconstrictors have little effect on onset of action.

Epinephrine 1:1000 contains 1 mg of epinephrine per mL  
 1:10,000 solution = 0.1 mg (or 100 mcg)/mL  
 1:100,000 solution = 10 mcg/mL  
 1:200,000 solution = 5 mcg/mL (commonly used)  
 1:400,000 solution = 2.5 mcg/mL

- Bicarbonate—alkalinization of local anesthetic solution → ↑ percentage of uncharged form of the drug → allowing penetration & ↑ onset time.
- Variable response between LA and the location of injection as to whether vasoconstrictors ↑ duration of action.
  - Epidural blockade: Addition of epi to procaine, lidocaine, and bupivacaine → ↑ duration of block. Addition of epi to prilocaine, etidocaine, and bupivacaine → no significant increase.
  - Spinal blockade: Addition of epi to tetracaine → ↑ duration of block. Addition of epi to lidocaine or bupivacaine → no significant increase.
  - For peripheral blockade, the addition of epinephrine to most agents increases the duration of blockade.
- Phenylephrine (0.125%) in equipotent doses may be as effective as epi in tetracaine spinals.
- Opiates—enhance both surgical anesthesia and postoperative analgesia.
- Clonidine—improves anesthesia and prolongs duration of block.

**SYSTEMIC ABSORPTION AND TOXICITY**

Systemic toxicity results from excessive plasma concentrations (due to absorption of LAs from tissue injection sites or accidental intravascular injection). Factors relating to rate of absorption:

- Dose of LA. A 1% solution of any drug contains 1 g (1000 mg) of drug per 100 mL of solution, or 10 mg/mL. (Therefore, a solution of 1% lidocaine contains 10 mg/mL, and a 2% solution contains 20 mg/mL.)
- Addition of epi (which decreases systemic absorption). Also, the “test dose” of local with epi indicates likely intravascular injection if injection is associated with a significant and rapid (<2 min) increase in heart rate.
- Degree of vasodilation, lipid solubility of LA, presence of renal/hepatic disease, CHF.

- Site of injection (based on vascularity of the tissue), with the greatest degree of absorption as follows:

Intravascular > intercostal > caudal > epidural > brachial plexus > subcutaneous

Toxicity mainly affects CV system and CNS. CNS is more sensitive and usually affected first. Signs of local anesthetic toxicity with increasing plasma concentrations include:

Light-headedness → circumoral numbness → facial tingling → tinnitus → slurred speech  
→ seizures → unconsciousness → respiratory arrest → cardiovascular depression → circulatory arrest

- CNS toxicity: ↑ with hypercarbia & acidosis (↓ seizure threshold), LA potency, total dose, and rate of injection.
- CV toxicity:
  - HTN, ↑HR, myocardial depression, hypotension, dysrhythmias.
  - ECG Δs: ↑PR, ↑QRS.
- **LA toxicity treatment:**
  - Stop injecting LA; get help; maintain airway (intubate if necessary); give 100% oxygen and consider hyperventilation in presence of metabolic acidosis; treat seizures (benzodiazepines, propofol, thiopental in small doses).
  - If cardiac arrest with LA toxicity: CPR and treatment of arrhythmias with standard protocols. Consider cardiopulmonary bypass or treatment with lipid emulsion:
    - Lipid emulsion: Intralipid 20%, 1.5 mL/kg over 1 min (~100 mL in 70-kg pt); Start intralipid infusion at 0.25 mL/kg/min (17.5 mL/min for 70-kg pt); Repeat bolus (1.5 mL/kg) ×2 at 5-min intervals; after 15 min, increase infusion to 0.5 mL/kg/min (35 mL/min for 70-kg pt) if circulation not restored.
    - Continue CPR throughout intralipid infusion. Recovery from LA-induced cardiac arrest may take as long as 1 hr.
- **EMLA cream**—eutectic mixture of lidocaine and prilocaine → used for dermal anesthesia. Onset ~45–60 min, duration ~2 hr.
  - Risks/contraindications:
    - LA toxicity possible, including methemoglobinemia.
    - Avoid in G6PD deficiency.
    - Avoid with history of amide allergy.
- **Methemoglobinemia** (normal hemoglobin oxidized to methemoglobin)
 

Causes: LAs (benzocaine, prilocaine), antibiotics (dapson, trimethoprim), nitrates

Symptoms and signs: SOB, cyanosis, MS Δs, loss of consciousness; if >50% met-Hb → dysrhythmias, seizures, coma, and death.

Dx: Blood is “chocolate-brown” color.

ABG analysis will typically reveal normal PO<sub>2</sub> +/- metabolic acidosis.

Use co-oximetry and measure met-Hb level.

Tx: Supplemental oxygen.

1% methylene blue 1–2 mg/kg IV (restores iron in Hb to its normal reduced O<sub>2</sub>-carrying state)

Ascorbic acid.
- **Cocaine Pharmacology**

Ester LA, a vasoconstrictor (others are vasodilators)

CNS stimulant → ↑ release & ↓ reuptake of epi, norepi, dopamine, serotonin

Used as topical anesthetic (sinus surgery, awake nasal fiberoptic)

Side effects: HTN, tachycardia, arrhythmias, cardiomyopathy, coronary ischemia, stroke, cerebral & pulmonary edema, seizures, hyperthermia, myoglobinuria, renal failure, hallucinations, platelet dysfx

## ANESTHETIC CONSIDERATIONS IN COCAINE ABUSE

Cocaine use → depletion of catecholamines (use direct-acting sympathomimetics)

Consider: selective β<sub>1</sub> antagonists (esmolol) for HR control

direct vasodilator (nitroprusside, nitroglycerin) for HTN

a balanced anesthetic technique to blunt sympathetic stimulation

regional anesthesia if can correct coagulopathy & hypovolemia

Use caution with inhalational agents (can worsen myocardial depression, may have ↑ requirements to ↓ effects of high systemic catecholamines)

# PHARMACOLOGY: NEUROMUSCULAR BLOCKING DRUGS AND REVERSAL AGENTS

JENNA HANSEN • SAMUEL C. SEIDEN

NMBDs 2-22

## MECHANISM

Neuromuscular blocking drugs (NMBDs) work at the postsynaptic nicotinic cholinergic receptor of the neuromuscular junction (NMJ) → stop conduction of nerve impulses → leading to skeletal muscle paralysis.

- Nondepolarizing NMBDs (Pancuronium, Rocuronium, Vecuronium, Atracurium, Cisatracurium)
  - Interfere with the actions of acetylcholine (ACh) at the NMJ, which keeps ion channels closed so no depolarization can occur.
  - Because NMBDs compete with ACh for receptor binding, they are considered antagonists or competitive blockers.
  - NMBD action can be overcome by increasing ACh in the synaptic cleft (the mechanism behind reversal of neuromuscular blockade with acetylcholinesterase inhibitors).
- Depolarizing NMBDs (Succinylcholine)
  - Mimic ACh by binding to the  $\alpha$ -subunit of the nicotinic cholinergic receptor keeping the ion channel open.
  - Cause prolonged depolarization which initially manifests as diffuse muscle contractions known as fasciculations.
  - The depolarized, occupied membrane cannot react to further release of ACh, thereafter causing muscle paralysis.

**Aminosteroids** (Pancuronium, Rocuronium, Vecuronium)

- **Pancuronium**—long-acting (duration of action,  $\leq 90$  min.)
  - **Clinical Considerations**
    - Tachycardia,  $\uparrow$ BP,  $\uparrow$ CO.
    - Elimination is mostly renal (increased duration in renal disease, neonates, elderly).
- **Rocuronium**—intermediate-acting duration of action  $\leq 35$  min when dosing at 0.6 mg/kg. More rapid onset than other nondepolarizers if 1.2-mg/kg dose is used (however, this may prolong duration of action).
  - **Clinical Considerations**
    - Used in rapid sequence intubation instead of succinylcholine.
    - Does not release histamine or cause cardiovascular effects.
    - Biliary and renal excretion.
    - May promote muscarinic block.
- **Vecuronium**—intermediate acting (duration of action  $\leq 35$  min.)
  - **Clinical Considerations**
    - Does not release histamine or cause cardiovascular effects.
    - Biliary elimination; renal excretion of metabolites.
    - Increased duration in hepatic disease, esp with repeated dosing.

**Benzylisoquinolines** (Atracurium, Cisatracurium)

- **Atracurium**—intermediate acting (duration of action  $\leq 35$  min.)
  - **Clinical Considerations**
    - Metabolism is independent of liver and kidney function or plasma cholinesterase activity
    - Cleared by hydrolysis via nonspecific plasma esterases and Hofmann elimination (nonenzymatic spontaneous degradation at normal pH and temperature) to the metabolite laudanosine.
    - Laudanosine can cause CNS stimulation/seizures at high concentrations (worse in pts with renal failure).
    - Dose-dependent release of histamine → hypotension.
- **Cisatracurium**—intermediate acting (duration of action  $\leq 35$  min.)
  - **Clinical Considerations**
    - Metabolism independent of liver, kidney, or plasma cholinesterase function.
    - Useful in patients with renal failure.
    - Cleared mainly by Hofmann elimination to the metabolite laudanosine, but concentrations of laudanosine are less than with atracurium.
    - Cisatracurium does not release histamine.

### Succinylcholine (SCh)

- Only depolarizing drug available, used for its rapid onset and short duration (see table on next page).
- Dose of 0.5–1.5 mg/kg; onset → 30 sec, paralysis for 5–10 min.
- Rapidly hydrolyzed by plasma cholinesterase (pseudocholinesterase).
- Length of blockade is determined both by the amount of SCh that reaches the NMJ and the rate of diffusion of SCh away from the NMJ into the plasma (there is no pseudocholinesterase at the motor plate).
  - Patients with a genetic defect causing atypical plasma cholinesterase (pseudocholinesterase deficiency) may demonstrate marked prolonged paralysis after a standard intubating dose of SCh.
  - Diagnosis: Determine dibucaine number (~20% in affected homozygotes).
  - Dibucaine number indicates the quality but not the quantity of plasma cholinesterase → if pt has decreased levels of plasma cholinesterase due to liver disease, pregnancy, burns, etc, the dibucaine number would be normal (~80%).
- Prolonged effect also seen in:
  1. Severe liver disease
  2. Echothiophate use in glaucoma pts (binds irreversibly to cholinesterase)
  3. Organophosphate poisoning (irreversibly bind to cholinesterase)
  4. Anticholinesterase use (inhibition of pseudocholinesterase may occur)
  5. Lithium, magnesium
- Indications—used for rapid sequence induction when aspiration is a risk (i.e., full stomach, trauma, diabetes mellitus, hiatal hernia, obesity, pregnancy)
- Contraindicated in malignant hyperthermia susceptible pts (SCh = known MH trigger)
- Use caution in pt's with open eye injury, elevated ICP, extrajunctional receptor proliferation (burn pts → probably safe if given <24 hr or >6 mo of injury; spinal cord transection pts → probably safe if given <24 hr of injury).
- **Adverse Effects**
  - Cardiac—(sinus bradycardia, junctional rhythm, cardiac arrest) due to SCh's action on cardiac muscarinic receptors. More likely to occur when a second dose of SCh is given minutes after the first. Pretreatment with atropine may prevent such responses.
  - Hyperkalemia—may occur in patients with burns, trauma, prolonged inactivity, denervation and spinal cord injuries due to upregulation of extrajunctional receptors. Serum potassium concentrations transiently increase by 0.5–1.0 mEq/L.
  - Allergic reactions—NMBDs are responsible for >50% of the anaphylactic reactions occurring during anesthesia. SCh is most common cause, followed by rocuronium.
  - Myalgias—fasciculations caused by SCh may contribute to postoperative myalgias. Pretreatment with a subparalyzing dose of a nondepolarizing NMBD (i.e., 1 mg of vecuronium) may decrease the incidence.
  - Trismus—may be severe in pt's mouth; becomes difficult to open for intubation.
  - Increased intraocular pressure; this effect is transient and lasts only as long as the paralytic effects of SCh.
  - Increased intracranial pressure.
  - Increased intragastric pressure; because lower esophageal sphincter tone is also increased, there appears to be no increased risk of aspiration due to this effect.

<b>NMBDs</b>	<b>Intubating Dose (mg/kg) [RSI dose, mg/kg]</b>	<b>Maintenance Dose (mg/kg)</b>	<b>Onset (min)</b>	<b>Duration to Return <math>\geq 25\%</math> Twitch Height (min)</b>	<b>Duration to Return <math>\geq 0.9</math> TOF Ratio (minutes)</b>	<b>Continuous Infusion</b>	<b>Primary Mechanism of Elimination (%)</b>
<b>Depolarizing</b>							
Succinylcholine (Anectine)	1 [1-1.5]	—	0.5-1	5-10			Pseudocholinesterases
<b>Nondepolarizing</b>							
Pancuronium (Pavulon)	0.1	0.02	3-5	60-90	130-220		Renal (80) Biliary (10) Hepatic (10)
Rocuronium (Zemuron)	0.6-1.2 [0.6-1.2]	0.1	1-2	20-35	55-80	3-12	Biliary (50-70) Renal (10-25)
Vecuronium (Norcuron)	0.1 [0.3-0.4]	0.02	3-5	20-35	50-80	1	Biliary (40-75) Hepatic (20-30) Renal (15-25)
Atracurium (Tracrium)	0.5	0.1	3-5	20-35	55-80	4-12	Hofmann and plasma esterases
Cisatracurium (Nimbex)	0.1 [0.4]	0.02	3-5	20-35	60-90	0.4-4	Hofmann



## ANTAGONISM OF NONDEPOLARIZING NEUROMUSCULAR BLOCKADE

**Cholinesterase inhibitors** (Neostigmine 0.07 mg/kg, Edrophonium 0.5 mg/kg, Physostigmine)

- Inhibit acetylcholinesterase, thereby allowing ACh to build up at the NMJ.
- Because nondepolarizers work by competitive inhibition against ACh, increasing concentrations of ACh at the NMJ will favor ACh binding the alpha subunit of the nicotinic cholinergic receptor.
- Common cholinergic side effects of anticholinesterases:
  - Nausea.
  - Cardiac muscarinic effects (bradycardia). Bradycardia is minimized by concurrent dosing of an anticholinergic drug of similar onset time such as glycopyrrolate (dose of 0.01 mg/kg, given with neostigmine) or atropine (dose of 0.01 mg/kg, given with edrophonium).
  - Bronchospasm and increased secretions.
  - Miosis.
  - Nicotinic effects (muscle weakness, tachycardia).

*Physostigmine*

- Crosses the blood-brain barrier; may cause central cholinergic effects:
  - Delirium, seizures, impaired consciousness, respiratory depression.
  - Used to Tx central anticholinergic syndrome (see page 2-27).

### Figure 2D-1 Sensitivity of Muscles to Neuromuscular Blockade

Most Sensitive

Least Sensitive

Extraocular > Pharyngeal > Masseter > Adductor pollicis > Abdominal rectus > Orbicularis oculi > Diaphragm > Vocal Cord

### Figure 2D-2 Speed of Onset & Recovery of Neuromuscular Blockade

Fastest Onset

Slowest Onset

Larynx > Diaphragm > Orbicularis oculi > Adductor pollicis

Fastest Recovery

Slowest Recovery

Larynx > Orbicularis oculi = Diaphragm > Adductor pollicis

**Anticholinesterases**

Agent	Dosage (mg/kg)	Peak Antagonism (min)	Duration of Antagonism (min)	Dosage of Atropine (mcg/kg)	Dosage of Glycopyrrolate (mcg/kg)	Metabolism and Comments
Edrophonium (Tensilon)	0.5-1.0	1-3	45-60	7-10	10	30% hepatic*
Neostigmine (Prostigmin)	0.03-0.07 (up to 5 mg)	7-10	55-75	15-30	10-15	50% hepatic**
Pyridostigmine (Mestinon)	0.25	10-13	80-130	15-20	10	75% hepatic***

\*Duration prolonged in renal failure; may cause bradycardia, hypotension, CNS stimulation or depression; GI cramps or cholinergic crisis; because of difference in onset times, administer glycopyrrolate several minutes in advance.

\*\*Duration prolonged in renal failure; may cause bradycardia, hypotension, CNS stimulation or depression, GI cramps, or cholinergic crisis.

\*\*\*Duration prolonged in renal failure; does not significantly cross the blood-brain barrier; not recommended for use with atropine because of the differences in onset times; therapy for myasthenia gravis.

**Sugammadex** (Proposed Trade Name: Bridion)

- A new agent designed specifically to reverse the effects of rocuronium and vecuronium (not currently FDA approved.)
- Binds to rocuronium and vecuronium encapsulates it, rendering it incapable of binding at the NMJ.
- Advantages:
  - Can be given at any time after administration of rocuronium, thereby resulting in fast recovery of neuromuscular blockade.
  - Mechanism does not rely on acetylcholinesterase inhibition and therefore it does not have undesirable cardiac effects.
  - Studied dosing 2–8 mg/kg.

**ANTICHOLINERGIC AGENTS**

Block peripheral muscarinic effects of cholinergic agents.

**Atropine**

- Give 0.6–1.2 mg IV for each 0.5–2.5 mg neostigmine, 10–20 mg pyridostigmine, or 10–20 mg edrophonium dose.

*Central anticholinergic syndrome:*

- Occurs with administration of atropine or scopolamine (both cross blood–brain barrier).
- Manifested as delirium or somnolence.
- Elderly pts more susceptible.
- Responds to treatment with physostigmine.

**Glycopyrrolate**

- Give 0.2 mg IV for each 1 mg neostigmine or 5 mg pyridostigmine dose.

**Scopolamine**

- Anticholinergic dose: 0.3–0.6 mg IM/IV/SC tid–qid prn.
- Antisialagogue dose: 0.2–0.6 mg IM  $\times$  1; give 0.5–1 hr prior to anesthesia.
- Amnesia dose: 0.32–0.65 mg IM/IV/SC tid–qid prn.
- Sedation dose: 0.6 mg IM/IV/SC tid–qid prn.
- Antiemetic, transdermal administration, see page 2F-38.

Drug	Onset (min)	Duration (min)	Effect on Heart Rate	Effect on CNS	Effect on Secretions	Effect on Smooth Muscle Tone
Atropine	1–1.5	15–30	↑↑↑	Mild sedation	↓	↓↓
Glycopyrrolate	2–3	120–240	↑↑	None	↓↓	↓↓
Scopolamine (IV)	10	120	↑↑	Significant	↓↓↓	↓↓



# PHARMACOLOGY: VASOACTIVE, AUTONOMIC AND CARDIOVASCULAR DRUGS

SAMUEL C. SEIDEN

CARDIO 2-28

**Table 2E-1. Receptor Sites and Action**

Receptor Site	Action
$\alpha$ -1	Vasoconstricts vascular smooth muscle, GU contraction, GI relaxation, gluconeogenesis, glycogenolysis
$\alpha$ -2	Decreased insulin secretion, causes platelet aggregation; decreased NE release, vasoconstriction of vascular smooth muscle
$\beta$ -1	Increased cardiac contractility, heart rate, AV conduction; increased renin secretion; increased contractility and arrhythmias
$\beta$ -2	Relaxation of vascular smooth muscle; bronchial relaxation. GI & GU relaxation, gluconeogenesis, glycogenolysis
D-1	Dilation of vascular smooth muscle (renal, mesentery, coronary); renal tubules (naturesis, diuresis); juxtaglomerular cells (increased renin release)
D-2	Inhibits NE release, may constrict renal and mesenteric smooth muscle

**Table 2E-2. Receptor Activity of Adrenergic Agonists**

Drug	Direct	Indirect	$\alpha$ -1 (Arterial)	$\alpha$ -1 (Venous)	$\alpha$ -2	$\beta$ -1	$\beta$ -2
Phenylephrine	++++		++++	+++++	+0	0	0
Norepinephrine	++++		+++	+++	+++++	++++	0
Epinephrine <sub>1</sub>	++++		+	+	+	++++	++++
Epinephrine <sub>2</sub>	++++		+++	+++	++	++++	++++
Epinephrine <sub>3</sub>	++++		+++++	+++++	+++	++++	++++
Ephedrine	++	+++	++	++	?	+++	++
Dopamine <sub>1</sub>	++++		0	++++	Dopaminergic agonist		
Dopamine <sub>2</sub>	+++	+	+	++++	?	+++	+++++
Dopamine <sub>3</sub>	+++	+	+++++	++++	?	+++++	+++
Dobutamine	++++		+ 0 -	?	0	++++	++
Isoproterenol	++++		0	0	0	+++++	+++++

Source: Adapted from Cusick J. Anesthesia and critical care reference sheet, 2007. [www.accrs.com](http://www.accrs.com)  
 Epinephrine dose/response (mcg/kg/min): (1) 0.01-0.03 (2) 0.03-0.15 (3) 0.015-0.5; Dopamine dose/response (mcg/kg/min): (1) 0.5-3 (2) 3-10 (3) 10-20

**Table 2E-3. Hemodynamic Effects of Adrenergic Agonists**

Drug	Cardiac Output	Inotropy	HR	MAP	Preload (Venous Return)	SVR (Afterload)	PVR	RBF	Bronchodilation
Phenylephrine	0, ↑	0, ↑	↓ (reflex)	↑↑↑	↑	↑↑↑	↑↑	0, ↓	0
Norepinephrine	1, 0, ↓	↑	↓ (reflex)	↑↑	↑↑↑	↑↑↑	↑↑	↓↑	0
Epinephrine <sub>1</sub>	↑↑	↑↑	↑	↑↑	↑	0, ↓	↑	↑	↑
Epinephrine <sub>2</sub>	1, 0	↑↑	↑↑	↑	↑	↑, 0, ↓	↑, 0	↓, 0	↑↑
Epinephrine <sub>3</sub>	1, 0, ↓	↑↑↑	↑↑	↑	↑↑	↑↑↑	↑	↓↑	↑↑
Ephedrine	↑↑	↑↑	↑	↑↑	↑↑	↑	↑	↑, 0, ↓	↑↑
Dopamine <sub>1</sub>	↑	0	0	0, ↓	↑	0, ↓	0, ↓	↑↑	0
Dopamine <sub>2</sub>	↑↑	↑	↑	↑	↑	↓	↑, 0	↑	0
Dopamine <sub>3</sub>	1, 0, ↓	↑↑	↑↑	↑	↑	↑↑	↑↑	↓	0
Dobutamine	↑↑	↑↑↑	↑	1, 0, ↓	↓	↓↑↓	↓↑	0, ↑	0
Isoproterenol	↑↑	↑↑	↑↑↑	↑, ↓	↓	↓↑↓	↓↑	0, ↑	↑↑↑
Vasopressin	↓		0	↑↑		↑↑		↑	

HR, heart rate; MAP, mean arterial pressure; SVR, systemic vascular resistance; PVR, peripheral vascular resistance; RBF, renal blood flow. Epinephrine dose/response (mcg/kg/min): (1) 0.01–0.03 (2) 0.03–0.15 (3) 0.015–0.5; Dopamine dose/response (mcg/kg/min): (1) 0.5–3 (2) 3–10 (3) 10–20

Source: Adapted from Cusick J. Anesthesia and critical care reference sheet, 2007. [www.accrs.com](http://www.accrs.com)

Table 2E-4. Hemodynamic Effects of Other Vasoactive Drugs

Drug	Cardiac Output	Inotropy	HR	MAP	Preload	SVR Afterload	PVR	RBF
<b>Beta blockers</b>								
Esmolol	↓↓	↓	↓↓	↓	0	↓↓		
Labetolol	↓,0	↓	0,↓	↓↓		↑		
Metoprolol	↓	↓	↓	↓	0	↑,0,↓		↓
Propranolol	↓	↓	↓	↓				
<b>Vasodilators</b>								
Hydralazine	↑,0	↑ (reflex)	↑ (reflex)	↓↓	0	↓↓	↓↓	0,↑
Nitroglycerine	↓	↑ (reflex)	↑ (reflex)	↓	↓↓	↓	↓↓	0
Nitroprusside	0,↑	↑ (reflex)	↑ (reflex)	↓↓	↓	↓↓	↓↓	0,↑
<b>Calcium channel blockers</b>								
Diltiazem	0	↓,0	↓	↓↓	0	↓↓		
Nicardipine	↑	0	0	↓↓	0	↓↓		
Nifedipine		↑ (reflex)	↑ (reflex)	↓↓	0,↓	↓↓		
Verapamil	↓	↓	↓	↓↓	0,↑	↓↓		0
<b>Phosphodiesterase inhibitors</b>								
Inamrinone	↑↑	↑	0,↑	0,↓	↓	↓↓	↓↓	
Milrinone	↑↑	↑↑	0,↑	0,↓	↓	↓↓	↓↓	

HR, heart rate; MAP, mean arterial pressure; SVR, systemic vascular resistance; PVR, peripheral vascular resistance; RBF, renal blood flow. Source: Adapted from Cusick J. Anesthesia and critical care reference sheet, 2007. [www.accrs.com](http://www.accrs.com)

## ADRENERGIC AGONISTS

**General comments:** Act on alpha, beta, or dopaminergic receptors (see Table 2E-2). May cause hypertension, arrhythmias, myocardial ischemia, and tissue necrosis with extravasation (treat with phentolamine).

### Dobutamine (Dobutrex)

**Indications:** Heart failure

**Dose:** Infusion prep: 500 mg in 250 mL D5W or NS = 2000 mcg/mL (2 mg/mL)

**Adult:** 2 mcg/kg/min, titrate 2–20 mcg/kg/min, max 40 mcg/kg/min; **Peds:** 5–20 mcg/kg/min

**Mechanism:**  $\beta$ 1-Adrenergic agonist

**Clearance:** Hepatic metabolism. Renal elimination

**Duration:** <10 min

**Comments:** Less tachyarrhythmias than with dopamine. Can increase ventricular rate in atrial fibrillation. Do not mix with sodium bicarbonate. Lowers CVP and wedge pressure, but little change on pulmonary vascular resistance.

### Dopamine (Intropin)

**Indications:** (1) Hypotension, heart failure; (2) oliguria

**Dose:** Infusion prep: 400 mg in 250 mL D5W = 1600 mcg/mL;

(1) Infusion at 1–20 mcg/kg/min IV titrate to effect; (2) Infusion at 0.5–3 mcg/kg/min IV

**Mechanism:** Dopaminergic;  $\alpha$ - and  $\beta$ -adrenergic agonist

**Clearance:** MAO/COMT metabolism

**Duration:** <10 min

**Comments:** Contraindicated in pts with pheochromocytoma or ventricular fibrillation. Improved renal blood flow (controversial) primarily at lower doses. Beta activity predominates at doses <10 mcg/kg/min and mixed  $\alpha$ - and  $\beta$ -adrenergic effects at  $\geq$ 10 mcg/kg/min. Do not mix with sodium bicarbonate.

MAO = monoamine oxidase; COMT = catechol-O-methyltransferase.

### Ephedrine (Generic)

**Indications:** Short-term treatment of hypotension, e.g., after induction in patient with normal catecholamine stores

**Dose:** Bolus only **Adult:** 5–10 mg IV PRN, typically to max 50 mg or 0.1 mg/kg; 25–50 mg SC/IM q4–6h prn. **Peds:** 0.2–0.3 mg/kg/dose

**Mechanism:**  $\alpha$ - and  $\beta$ -adrenergic stimulation; norepinephrine release at sympathetic nerve endings

**Clearance:** Mostly renal elimination (unchanged)

**Duration:** 3–10 min

**Comments:** Tachyphylaxis with repeat dosing. Predominantly indirect action. May cause CNS stimulation, decrease in uterine activity, and mild bronchodilation. Minimal effect on uterine blood flow. Avoid in patients taking MAO inhibitors.

### Epinephrine (Adrenaline)

**Indications:** (1) cardiac arrest; (2) heart failure, hypotension; (3) bronchospasm, anaphylaxis; (4) bradycardia

**Dose:** Infusion prep: 4 mg in 250 mL D5W or NS = 16 mcg/mL

**Adult:** (1) standard dose 1 mg IV/IO q3–5min during resuscitation; intermediate dose: 2–5 mg IV q–5min; escalating dose 1 mg, 3 mg, 5 mg q3min; high dose: 0.1 mg/kg IV q3–5min; endotracheal dose 2–2.5  $\times$  IV dose; (2) 2–2.5 mg (1:10,000 sol) ETT q3–5min prn; (3) 0.020–0.3 mcg/kg/min = 20–300 ng/kg/min; (4) 0.1–0.5 mg IM/SC (1:1000 aqueous) q15min to q4h prn, 0.1–0.25 mg IV; (5) bolus 10–20 mcg IV; infuse 2–10 /min IV

**Peds:** (1) 1st dose 0.01 mg/kg IV/IO; subsequent doses 0.1–0.2 mg/kg IV/IO q3–5min; intratracheal: 0.1 mg/kg of 1:10,000 solution; (2) 0.1–1 mcg/kg/min, max 1.5 mcg/kg/min; (3) 0.01 mcg/kg SC (1:1000 aqueous) q15min to q4h prn; for anaphylaxis give 0.01 mcg/kg q15min  $\times$ 2 doses then q4h prn; (4) 0.01 mg/kg IV/IO or 0.1 mg/kg ET

**Neonates:** (1) 0.01–0.03 mg/kg IV/IO q3–5min; intratracheal: 0.1 mg/kg of 1:10,000 solution

**Mechanism:**  $\alpha$ - and  $\beta$ -adrenergic agonist

**Clearance:** MAO/COMT metabolism

**Duration:** 5–10 min

**Comments:** May cause increased lipolysis, glycogenolysis, and inhibition of insulin release. Dysrhythmias potentiated by halothane. Topical or local injection (1:80,000–1:500,000) causes vasoconstriction. Crosses the placenta. Predominantly beta activity at low doses, changes to combined beta/alpha activity at higher doses. Reserve 1 mg IV bolus for cardiac arrest to avoid significant hypertensive response. Endotracheal dosing 2–2.5  $\times$  IV per dose.

**Isoproterenol** (Isuprel)**Indications:** Indicated for heart failure, bradycardia, shock, pulmonary HTN, refractory asthma or COPD**Dose:** *Infusion prep* 1 mg in 250 mL = 4 mcg/mL**Adult:** AV nodal block: 5 mcg/min IV titrate up to 20 mcg/min (not weigh-based); *Shock:* 0.5–5  $\mu$ g/min IV. **Peds:** Start 0.02–0.1 mcg/kg/min; titrate to effect 0.05–2 mcg/kg/min**Mechanism:**  $\beta$ -Adrenergic agonist; positive chronotrope and inotrope. Decreases systemic and pulmonary vascular resistance. Increases coronary and renal blood flow.**Clearance:** Hepatic and pulmonary metabolism; 40%–50% renal excretion (unchanged).**Duration:** 1–5 min**Comments:** Refractory torsades de pointes,  $\beta$ -blocker overdose, 3rd degree AV block awaiting pacemaker. May cause CNS excitation, pulmonary edema.**Phenylephrine** (Neosynephrine)**Indication:** Hypotension, SVT, treatment of wide-angle glaucoma.**Dose:** **Bolus:** 50–100 mcg IV; 2–3 mg SC/IM q1–2h; *Infusion prep* 40 mg in 250 mL = 160 mcg/mL; *Infuse* 0.2–1 mcg/kg/min or 20–180 mcg/min **Peds:** bolus 0.5–10 mcg/kg IV *infuse* 0.1–0.5 mcg/kg/min**Mechanism:**  $\alpha$ 1-Adrenergic agonist**Duration:** <5 min**Clearance:** Hepatic metabolism; renal elimination**Comments:** May cause reflex bradycardia, microcirculatory constriction, uterine contraction, or uterine vasoconstriction. Contraindicated in pheochromocytoma, severe hypertension, bradycardia, ventricular tachyarrhythmias, uterine contraction, uterine vasoconstriction.**Norepinephrine** (Levarterenol, Levophed)**Indications:** Hypotension**Dose:** *Infusion prep* 4 mg in 250 mL NS or D5W = 16 mcg/mL; **Adult:** *infuse* 0.020–0.3 mcg/kg/min = 20–300 ng/kg/min or 4–12 mcg/min; **Peds:** 0.05–0.1 mcg/kg/min to max 2 mcg/kg/min**Mechanism:** Both  $\alpha$ - and  $\beta$ -adrenergic activity, with  $\alpha$ -adrenergic activity predominating**Peak Effect:** 3–10 min**Clearance:** MAO/COMT metabolism**Comments:** May cause increased uterine contractility, constricted microcirculation, or CNS stimulation. Preferably administered through central venous catheter**Vasopressin** (Antidiuretic Hormone, Pitressin)**Indications:** Diabetes insipidus, upper GI hemorrhage, pulseless ventricular tachycardia or ventricular fibrillation, shock refractory to fluid and vasopressor therapy.**Dose:** *Infusion prep* 100 IU in 100 mL NS = 1 U/mL**Adult:** (1) 5–10 U IM/SC q6–12h prn; (2) 0.1–0.4 U/min IV infusion; (3) 40 U IV/IO/ET bolus (single dose); (4) 0.01–0.04 U/min IV infusion (not weight-based). **Peds:** (1) 2.5–5 U IM/SC q6–12h prn; (2) 0.1–0.3 U/min, titrate 0.002–0.008 U/kg/min**Mechanism:** Synthetic analogue of arginine vasopressin. Antidiuretic, increases urine osmolality and decreases urine volume; smooth muscle constriction; vasoconstriction of splanchnic, coronary, muscular, and cutaneous vasculature**Clearance:** Hepatic and renal metabolism; renal elimination**Duration:** 10–20 min**Comments:** May cause oliguria, water intoxication, pulmonary edema; abdominal cramps (from increased peristalsis); anaphylaxis; contraction of gallbladder, urinary bladder, or uterus; vertigo or nausea. Patients with coronary artery disease are often treated with concurrent nitroglycerin. Do not abruptly discontinue IV infusion.**PHOSPHODIESTERASE INHIBITORS****Amrinone** (Inocor, Inamrinone)**Indications:** Indicated for tx of low cardiac output states, heart failure, and as adjunct in pulmonary hypertension**Dose:** **Adult/Peds:** Load 0.75 mg/kg IV bolus over 2–3 min, then infuse 5–15 mcg/kg/min. *Infusion prep* 100 mg in 250 mL in crystalloid *without dextrose* = 0.4 mg/mL; max dose: 10 mg/kg/24 hr. **Neonates:** Load 0.75 mg/kg IV bolus over 2–3 min, then infuse 3–5 mcg/kg/min**Mechanism:** Phosphodiesterase inhibitor (inhibits myocardial cAMP phosphodiesterase), increases cardiac output, increases contractility, decreases pulmonary vascular resistance and preload

**Clearance:** Variable hepatic metabolism; renal/fecal excretion. Reduce dose 50%–75% in ESRD

**Comments:** May cause hypotension, thrombocytopenia, and anaphylaxis (contains sulfites), worsening myocardial ischemia. Do not administer in same IV line as furosemide (Lasix)

### **Milrinone** (Primacor)

**Indications:** Indicated for congestive heart failure

**Dose:** *Infusion prep* 20 mg in 100 mL = 200 mcg/mL **Adult:** load: 50–75 mcg/kg IV over 10 min; *Infusion:* 0.375–0.75 mcg/kg/min titrate to effect. **Peds:** load: 50 mcg/kg IV over 10 min, followed by infusion of 0.5–1 mcg/kg/min and titrate to effect

**Mechanism:** Phosphodiesterase inhibition causing positive inotropy and vasodilation

**Clearance:** Renal elimination

**Comments:** Short-term therapy. May increase ventricular ectopy, may aggravate outflow tract obstruction in IHSS. May cause hypotension, headaches. Not recommended for acute MI.

## **ANTIHYPERTENSIVES**

### **Beta-blockers**

**General comments:** May cause bradycardia, AV conduction delays, hypotension, bronchospasm. Contraindicated in uncompensated CHF, cardiogenic shock, severe bradycardia or heart block greater than first degree.

### **Labetalol** (Normodyne, Trandate)

**Indications:** Hypertension, angina

**Dose:** **Adult:** IV: 5–20 mg increments or 1–2 mg/kg at 5–10-min intervals, to 40–80 mg/dose. Max total 300 mg; 200–400 mg PO q12h. *Infusion:* 2–150 mg/hr, or 0.05 mcg/kg/min, titrate to effect. **Peds:** 0.12–1 mg/kg/dose q10min PRN to max 10 mg/dose, *infusion* 0.4–1 mg/kg/hr, max 3 mg/kg/h

**Mechanism:** Selective  $\alpha$ 1-adrenergic blockade with nonselective  $\beta$ -adrenergic blockade. Ratio of  $\alpha/\beta$  blockade = 1:7

**Clearance:** Hepatic metabolism; renal elimination.

**Onset:** 1–2 min; **Duration:** 2–8 hr

**Comments:** May cause urinary retention, skin tingling. Crosses placenta. Avoid in CHF.

### **Metoprolol** (Lopressor)

**Indications:** Indicated for hypertension, angina pectoris, dysrhythmia, hypertrophic cardiomyopathy, myocardial infarction, pheochromocytoma

**Dose:** 2.5–5 mg IV boluses q2min, prn, up to 15 mg. 50–200 mg PO q8–24h

**Mechanism:**  $\beta$ 1-Adrenergic blockade ( $\beta$ 2-adrenergic antagonism at high doses)

**Clearance:** Hepatic metabolism, renal elimination

**Comments:** May cause clinically significant bronchoconstriction (with doses >100 mg/d), dizziness, fatigue, insomnia, increased risk of heart block. Crosses the placenta and blood–brain barrier.

### **Esmolol** (Brevibloc)

**Indications:** Supraventricular tachydysrhythmias, myocardial ischemia

**Dose:** *Infusion prep* 2500 mg in 250 mL = 10 mg/mL. **Adult:** Bolus: 5–10 mg IV bolus and increase q3min prn to total 100–300 mg. *Infusion:* load 500 mcg/kg over 1 min followed by 1–15 mg/min or 0.05–0.3 mg/kg/min, max 0.5 mg/kg/min. **Peds:** load 100–500 mcg/kg IV over 1 min; *infusion* start at 0.05 mg/kg/min, titrate to effect, max 0.5 mg/kg/min

**Onset:** 1 min; **Duration:** 12–20 min

**Mechanism:** Selective  $\beta$ 1-adrenergic blockade

**Clearance:** Degraded by RBC esterases; renal elimination

**Comments:**  $\beta$ 2 activity at high doses. Morphine may increase esmolol levels

### **Nadolol** (Corgard)

**Indications:** Indicated for angina pectoris, hypertension

**Dose:** 40–240 mg/d PO

**Peak Effect:** 4 hr

**Mechanism:** Prolonged (approximately 24 hr) nonselective  $\beta$ -adrenergic blockade

**Clearance:** No hepatic metabolism; renal elimination

**Comments:** See propranolol.

### **Propranolol** (Inderal)

**Indications:** (1) Hypertension; (2) tetralogy of Fallot cyanotic spells; (3) thyrotoxicosis; (4) atrial and ventricular dysrhythmias, as well as myocardial ischemia or infarction, hypertrophic cardiomyopathy, migraine headache, pheochromocytoma

**Dose: Adult:** Test dose of 0.25–0.5 mg IV, then titrate up by 0.5 mg/min to effect. PO: 10–40 mg q6–8h, prn; 1–3 mg slow IV; 1 mg/dose IV q5min to max 5 mg. **Peds:** 0.15–0.25 mg/kg/d slow IV, repeat prn; 0.01–0.1 mg/kg slow IV.

**Mechanism:** Nonspecific  $\beta$ -adrenergic blockade

**Clearance:** Hepatic metabolism; renal elimination

**Comments:** May cause hypoglycemia and drowsiness. Crosses the placenta and blood–brain barrier. Abrupt withdrawal can precipitate rebound angina.

## ALPHA AGONISTS

**Clonidine** (Catapres)

**Indications:** Hypertension

**Dose:** 5–25 mcg/kg/d PO div q6h. Start: 5–10 mcg/kg/d div q6h; Max: 0.9 mg/d; Info: increase gradually q5–7d; Transdermal: 1 patch/wk Start: 0.1 mg/24-hr patch, titrate q1–2wk; Max: 0.6 mg/24 hr (using two 0.3 mg/24-hr patches). Info: If switching from PO, continue PO  $\times$ 1–2 days; onset of hypotensive effect 2–3 days

**Mechanism:** Stimulates  $\alpha_2$ -adrenergic receptors (centrally acting antihypertensive)

**Clearance:** Hepatic; excretion renal 65%, biliary 20%

**Comments:** May cause rebound hypertension and arrhythmias with withdrawal, dry mouth, drowsiness, dizziness, constipation, sedation, weakness.

**Dexmedetomidine** (Precedex)

**Indications:** Sedation of intubated patients. Useful for procedural sedation, adjunct to GA

**Dose:** For sedation, may load 1 mcg/kg over 10 min and infuse 0.2–0.7 mcg/kg/hr, (although doses of 1–2 mcg/kg/hr have also been used, with increased risk of side effects)

**Mechanism:** Highly selective  $\alpha_2$ -adrenergic agonist

**Clearance:** Liver metabolism;  $t_{1/2}$ : 2 hr

**Comments:** Potential mild analgesia and amnesia. More similar to sleep state than other hypnotics. CNS: Decreases CBF. CV: Decreases HR, SVR, BP. Heart block, severe bradycardia, asystole reported. PULM: No significant respiratory depression. May cause severe bradycardia, sinus arrest, arrhythmias, atrial fibrillation, pleural effusion, pulmonary edema, respiratory acidosis, bronchospasm.

**Methyldopa** (Aldomet)

**Indications:** Hypertension.

**Dose:** 250–500 mg PO bid

**Mechanism:** Stimulates  $\alpha_2$ -adrenergic receptors (centrally acting antihypertensive).

**Clearance:** Metabolized by central adrenergic neurons and liver; primarily urine excretion

**Comments:** Adverse reactions include myocarditis, anemia, hemolytic, thrombocytopenia, hepatic necrosis, leukopenia, bradycardia, pancreatitis.

## CALCIUM CHANNEL BLOCKERS

**General comments:** May cause bradycardia and heart block. Coadministration with beta blockade increases risk of heart block. Contraindicated in sick sinus syndrome, 2nd or 3rd degree AV block (unless with functioning pacemaker).

**Diltiazem** (Cardizem)

**Indications:** Temporary control of rapid ventricular rate in atrial fibrillation/flutter, conversion of paroxysmal supraventricular tachycardia, hypertension, angina and variant angina from coronary artery spasm

**Dose:** Infusion prep 100 mg in 100 mL = 1000 mcg/mL (1 mg/mL); Load 2.5–25 mg (or 0.25 mg/kg) over 2 min, may rebolus 0.35 mg/kg in 15 min if no effect; infuse 2–15 mg/hr (no weight calculation) for up to 24 hr. PO: 30–120 mg PO q6–8h

**Mechanism:** Calcium channel antagonist that slows conduction through sinoatrial and AV nodes, dilates coronary and peripheral arterioles, and reduces myocardial contractility

**Clearance:** Primarily hepatic metabolism; renal elimination;  $t_{1/2}$  = 3.5–4 hr

**Comments:** Contraindicated in pts with WPW or short PR syndrome; wide QRS complex of unknown etiology; poison- or drug-induced tachycardia. May interact with beta blockers and digoxin to impair contractility. Causes transiently elevated LFTs. Avoid use in patients with accessory conduction tracts, or ventricular tachycardia. Active metabolite has 1/4–1/2 of the coronary dilation effect.

**Verapamil** (Isoptin, Calan)

**Indications:** Supraventricular tachycardia, atrial fibrillation or flutter, Wolff–Parkinson–White syndrome

**Dose:** *Adult:* 2.5–10 mg IV over  $\geq 2$  min. If no response in 30 min, repeat 5–10 mg (150 mcg/kg). **Peds:** **0–1 yr:** 0.1–0.2 mg/kg IV; **1–15 yr:** 0.1–0.3 mg/kg IV. Repeat once if no response in 30 min

**Mechanism:** Blocks slow calcium channels in heart. Prolongs PR interval. Negative inotrope and chronotrope; systemic and coronary vasodilator

**Clearance:** Hepatic metabolism; renal elimination.  $t_{1/2}$ : 5 hr

**Comments:** May increase ventricular response to atrial fibrillation or flutter in patients with accessory tracts. Active metabolite has 20% of the antihypertensive effect of the parent compound.

### Nicardipine (Cardene)

**Indications:** Short-term treatment of hypertension and chronic stable angina

**Dose:** *Infusion prep:* 25 mg in 250 mL = 0.1 mg/mL; 5–15 mg/hr (no weight calculation)

**Mechanism:** Dihydropyridine calcium channel blocker in systemic and coronary vascular smooth muscle and myocardium

**Clearance:** Hepatic metabolism, renal elimination  $t_{1/2}$ : 2 hr

**Comments:** Contraindicated in advanced aortic stenosis, severe hypotension or bradycardia, ventricular tachycardia or atrial fibrillation, cardiogenic shock. May cause peripheral edema, MI, tachycardia.

### Nifedipine (Procardia)

**Indications:** Indicated for coronary artery spasm, hypertension, myocardial ischemia

**Dose:** 10–40 mg PO tid; 10 mg SL

**Mechanism:** Blocks slow calcium channels, which produces systemic and coronary vasodilation and can increase myocardial perfusion.

**Clearance:** Hepatic metabolism  $t_{1/2}$ : 2 hr

**Comments:** May cause reflex tachycardia, gastrointestinal upset, and mild negative inotropic effects. Little effect on automaticity and atrial conduction. May be useful in asymmetric septal hypertrophy. Drug solution is light-sensitive. May rapidly produce severe hypotension in some patients, especially with sublingual administration.

## VASODILATORS

**General comments:** May cause hypotension, reflex tachycardia

### Fenoldopam (Corlopam)

**Indications:** Short-term management (<48 hr) of severe hypertension

**Infusion:** *Infusion prep:* 10 mg in 250 mL = 40 mcg/mL; 0.01–0.20 mcg/kg/min (Doses up to 1.6 mcg/kg/min studied in clinical trials.) Titrate to effect by 0.05–0.1 mcg/kg/min increments. Do not bolus

**Mechanism:** Dopamine (D1) receptor agonist causing rapid vasodilation of coronary, renal, mesenteric, and peripheral arteries

**Clearance:** Hepatic metabolism; 90% renal excretion

**Onset:** 15 min;  $t_{1/2}$ : 5 min

**Comments:** Contains sulfites. Use caution in pts with glaucoma (may increase IOP). May cause hypokalemia.

### Hydralazine (Apresoline)

**Indications:** Indicated for hypertension, pregnancy induced hypertension, and primary pulmonary hypertension

**Dose:** *Adult:* 2.5–20 mg IV q4h or prn. Max 40 mg/dose. PO available. *Pregnancy-induced hypertension* 5–10 mg IV q20–30min prn. **Peds:** 0.1–0.2 mg/kg/dose q4–6h, max 40 mg/dose

**Mechanism:** Direct reduction of vascular smooth muscle tone (arterial > venous)

**Clearance:** Extensive hepatic metabolism; renal elimination.

**Onset:** 5–20 min; **Duration:** 4–8 hr

**Comments:** May cause hypotension (diastolic more than systolic), systemic lupus erythematosus syndrome, thrombophlebitis, and hemolytic anemia. Increases coronary, splanchnic, cerebral, and renal blood flows.

### Isosorbide Dinitrate (Isordil)

**Indications:** Angina, hypertension, myocardial infarction, congestive heart failure

**Dose:** 5–20 mg PO q6h

**Mechanism:** See nitroglycerin

**Clearance:** Nearly 100% hepatic metabolism; renal elimination

**Comments:** See nitroglycerin. Tolerance may develop.

### Nitroglycerin (Tridil, Glycerol Trinitrate, Nitrostat, Nitrol, Nitro-Bid, Nitrolingual)

**Indications:** Angina, myocardial ischemia or infarction, hypertension, congestive heart failure, controlled hypotension, esophageal spasm



**Dose:** *Infusion prep:* 50 mg in 250 mL D5W or NS = 200 mcg/mL; IV infusion initially at 5 mcg/min. Titrate every 3–5 min by 10 mcg/min to max 200 mcg/min or 0.1–1 mcg/kg/min; SL: 0.15–0.6 mg/dose q5min to max 3 doses in 15 min. *Topical:* 2% ointment, 0.5–2.5 in. q6–8h, max 5 in. q4h

**Mechanism:** Direct vasodilator, venous > arterial. Produces smooth muscle relaxation by enzymatic release of NO, causing systemic, coronary, and pulmonary vasodilation; bronchodilation; biliary, gastrointestinal, and genitourinary tract relaxation.

**Clearance:** Nearly complete hepatic metabolism; renal elimination

**Onset:** 1 min; **Duration:** 3–5 min

**Comments:** Causes coronary vasodilation, headache, absorption into IV tubing. Tolerance with chronic use may be avoided with a 10- to 12-hr nitrate-free period. May cause methemoglobinemia at very high doses.

**Nitroprusside (Nipride, Nitropress)**

**Indications:** Hypertension, controlled hypotension, congestive heart failure

**Dose:** *Infusion prep* 50 mg in 250 mL D5W or NS = 200 mcg/mL; *Infusion* initially at 0.1–0.5 mcg/kg/min, then titrated to effect q3–5min to max 10 mcg/kg/min. Lower doses often adequate during general anesthesia

**Mechanism:** Direct NO donor causing smooth muscle relaxation equal to slightly greater arterial than venous relaxation

**Clearance:** RBC and tissue metabolism; renal elimination

**Onset:** 30–60 sec; **Duration:** 1–5 min

**Comments:** Very titratable with immediate onset and no CNS side effects. May cause excessive hypotension, reflex tachycardia. Contraindicated with decreased cerebral perfusion pressure, elevated ICP, AV shunt or aortic coarctation, hypovolemia, hypothyroid, B<sub>12</sub> deficiency. Nonenzymatically converted to cyanide, which is converted to thiocyanate leading to metabolic acidosis and methemoglobinemia. Accumulation of cyanide with liver dysfunction; thiocyanate with kidney dysfunction. Maintain thiocyanate levels <50 mg/L. Tx toxicity with sodium nitrite, sodium thiosulfate, hydroxocobalamin or methylene blue. Avoid with Leber's hereditary optic atrophy, hypothyroidism, or vitamin B<sub>12</sub> deficiency. Solution and powder are light sensitive and must be wrapped in opaque material.

## ANTIARRHYTHMICS

**Adenosine (Adenocard)**

**Indications:** PSVT, Wolff–Parkinson–White syndrome

**Dose: Adult:** 6 mg rapid IV push, may repeat 12 mg × 2 within 1–2 min; **Peds:** 0.1–0.2 mg/kg rapid IV push, increase by 50 mcg/kg q2min to max 250 mcg/kg

**Mechanism:** Slows AV nodal conduction by interrupting AV nodal reentry pathways

**Clearance:** Erythrocytes and vascular endothelial cells

**Comments:** Contraindicated in 2nd and 3rd degree AV block and sick sinus syndrome without pacing. Significant adverse reactions: hypotension, bronchoconstriction. 3–6 sec asystole after administration common.

**Amiodarone (Cordarone)**

**Indications:** Cardiac arrest; tachyarrhythmia

**Dose:** *Infusion prep:* 1200 mg in 250 mL D5W or NS = 4.8 mg/mL

**Adult:** 300 mg IVP, may repeat 150 mg IVP in 3–5 min to max 2.2 g/24 hr; *Load* 150 mg IV over 10 min, may repeat 150 mg q10min if VF/VT recurs. *Maintenance:* 1 mg/min × 6 hr (360 mg) then 0.5 mg/min × 18 hr (540 mg); may rebolus for recurrent VF/VT to max 15 mg/kg/d. **Peds:** 5 mg/kg IV/IO; *Load* 5 mg/kg IV over 20–60 min, *Maintenance* infuse 5–10 mcg/kg/min.

**Mechanism:** Prolongs action potential phase 3; alpha and beta adrenergic blockade, decreases AV conduction and sinus node function, prolongs PR, QRS and QT intervals

**Clearance:** Biliary

**Comments:** Indicated for refractory V-tach, V-fib, SVT, PSVT. Contraindicated in: 2nd and 3rd degree heart block, severe sinus node disease or sinus bradycardia, cardiogenic shock, thyroid disease. May increase serum levels of digoxin, diltiazem, oral anticoagulants, phenytoin.

**Lidocaine (Xylocaine)**

**Indications:** (1) Ventricular dysrhythmias. (2) Cough suppression. (3) Local anesthesia.

**Dose:** *Infusion prep:* 2 g in 250 mL D5W or NS = 8 mg/mL. **Adult:** (1) *load:* 1–1.5 mg/kg IV over 2–3 min; 2nd dose 5–30 min after 1st dose, 0.5–1.5 mg/kg to total 3 mg/kg; *Maintenance:* 15–50 mcg/kg/min IV (1–4 mg/min); (2) 1 mg/kg IV. (3) 4.5 mg/kg maximum dose for infiltration or conduction block. See also Chapter 2C, "Local Anesthetics". **Peds:** (1) *load:* 0.5–1 mg/kg IV, may repeat × 2 doses; *Maintenance:* 15–50 mcg/kg/min IV. (2) 1 mg/kg IV.

**Mechanism:** Decreases conductance of sodium channels, Antiarrhythmic effect, suppresses automaticity of conduction tissue

**Clearance:** Hepatic metabolism to active/toxic metabolites; renal elimination (10% unchanged). Decrease dose in pts with CHF, hepatic disease, shock.

**Comments:** May cause dizziness, seizures, disorientation, heart block (with myocardial conduction defect), hypotension, asystole, tinnitus, unusual taste, vomiting. Crosses the placenta. Therapeutic concentration = 1–5 mg/L. Caution in patients with Wolff–Parkinson–White syndrome, intraventricular heart blocks, pacemaker. Endotracheal dose  $2\text{--}2.5 \times$  IV dose diluted with 1–2 mL NS.

### Procainamide (Pronestyl)

**Indications:** Atrial and ventricular dysrhythmias

**Dose: Adult:** Load 20 mg/min IV, up to 17 mg/kg, until toxicity or desired effect occurs. Stop if  $\geq 50\%$  QRS widening or PR lengthening occurs; **Maintenance:** 1–4 mg/min.

**Peds:** Load: 3–6 mg/kg over 5 min, not to exceed 100 mg/dose; repeat q5–10min to maximum dose of 15 mg/kg; **Maintenance:** 20–80 mcg/kg/min; max 2 g/24 hr.

**Mechanism:** Blocks sodium channels; class I-A antidysrhythmic

**Clearance:** Hepatic conversion of 25% to active metabolite N-acetylprocainamide (NAPA), a class III antidysrhythmic; renal elimination (50% to 60% unchanged)

**Comments:** May cause increased ventricular response with atrial tachydysrhythmias unless receiving digitalis; asystole (with AV block); myocardial depression; CNS excitement; blood dyscrasia; lupus syndrome with +ANA; liver damage. Intravenous administration can cause hypotension from vasodilation, accentuated by general anesthesia. Decrease load by one-third in congestive heart failure or shock. Reduce doses in hepatic or renal impairment. Therapeutic concentration 4–10 mcg/mL (procainamide); 15–25 mcg/mL (NAPA); 10–30 mcg/mL (combined). Contains sulfite.

## HEART FAILURE

### Digoxin (Lanoxin)

**Indications:** CHF, tachydysrhythmias, atrial fibrillation/flutter

**Dose: Adult:** load 0.5–1.0 mg/d IV or PO in divided doses; **maintenance** 0.125–0.5 mg IV or PO qd; **Peds:** Load (Total daily doses usually divided into two or more doses);

**Neonates:** 15–30 mcg/kg/d; **Infants: 1 month–2 yr:** 30–50 /kg/d; **2–5 yr:** 25–35 mcg/kg/d; **Peds: 5–10 yr:** 15–30 mcg/kg/d; **> 10 yr:** 8–12 mcg/kg/d. **Maintenance:** 20–35% of LOAD qd (reduce in renal failure).

**Mechanism:** Increases myocardial contractility via inhibition of sodium/potassium ATPase leading to increased intracellular calcium; decrease chronotropy via decreases conduction in AV node and Purkinje fibers.

**Onset:** 30 min;  $t_{1/2}$ : 1.5–2 days

**Clearance:** Renal elimination (50%–70% unchanged)

**Comments:** May cause gastrointestinal intolerance, blurred vision, ECG changes, or dysrhythmias. Toxicity potentiated by hypokalemia, hypomagnesemia, hypercalcemia. Use cautiously in Wolff–Parkinson–White syndrome and with defibrillation. Heart block potentiated by beta blockade and calcium channel blockade. Therapeutic level: 0.8–2.0 ng/mL. Symptoms of toxicity include CNS depression, confusion, headache, anorexia, nausea vomiting, visual changes, arrhythmias, and seizures.

### Nesiritide (B-type natriuretic peptide, BNP)

**Indications:** Treatment of patients with acutely decompensated CHF with dyspnea at rest or minimal activity

**Dose:** Infusion prep 1.5 mg in 250 mL = 6 mcg/mL; load 2 mcg/kg over 1 min; Infuse 0.01 mcg/kg/min. May increase infusion by 0.005 mcg/kg/min, no more often than q3h up to max of 0.03 mcg/kg/min. Bolus 1 mcg/kg prior to changing rate

**Mechanism:** Binds to guanylate cyclase receptor; stimulates cGMP production, resulting in vascular smooth muscle relaxation (b-type natriuretic peptide); leads to dose-dependent reductions in pulmonary capillary wedge pressure and systemic arterial pressure heart failure patients

**Onset:** 60% effect achieved in 15 min, peak effect within 1 hr.  $t_{1/2}$ : 18 min

**Comments:** Contraindicated in patients with cardiogenic shock, or SBP <90. Physically and chemically incompatible with injectable formulations of bumetanide, enalaprilat, ethacrynate sodium, furosemide, hydralazine, heparin, and insulin. Administration with ACE inhibitors may cause increased hypotension. May cause hypotension, ventricular tachycardia, ventricular and atrial arrhythmias, angina, bradycardia, tachycardia, anemia, or azotemia. Use caution in pts with renal disease and pts with valvular heart disease as may not tolerate vasodilation.

# PHARMACOLOGY: COMMONLY ENCOUNTERED DRUGS IN ANESTHESIA

SAMUEL C. SEIDEN • STEPHEN FINK

COMMON 2-38

## ANTIEMETICS

(See also Chapter 27, "Ambulatory Anesthesia")

**5HT<sub>3</sub> Antagonists** (Ondansetron, Granisetron, Dolasetron)

**Mechanism:** Antagonism of 5HT<sub>3</sub> receptors centrally (chemoreceptor trigger zone) and peripherally (abdominal vagal nerve terminals)

Typical dose (given prior to emergence or as treatment for PONV):

Ondansetron (Zofran): **Adults:** 4 mg IV; **Peds:** 0.1 mg/kg, max 4 mg/dose

(Also available as orally dissolving tablet.)

Granisetron (Kytril): **Adults:** 1 mg IV; **Peds:** 10 mcg/kg × 1 to max 1 mg/dose

Dolasetron (Anzemet): **Adults:** 12.5 mg IV; **Peds:** 0.35 mg/kg (max 12.5 mg)

**Clearance:** Predominantly hepatic

**Comments:** *Adverse reactions:* Headaches, transient transaminitis, QT prolongation (especially dolasetron). For ondansetron and granisetron, IV and PO doses are identical. PO dolasetron is given as 100 mg. All appear equal in efficacy. Majority of studies in pediatric anesthesia involve ondansetron. Decreased dose in patients with severe hepatic impairment. Dosing of ondansetron >2 mg/24 hr may not provide additional efficacy. Consider other agents as well.

## Corticosteroids

Dexamethasone (Decadron)

**Dose:** 0.5–1 mg/kg (4–8 mg in adults) prior to induction

**Mechanism:** Corticosteroid. Antiemetic property not fully understood (likely central effect)

**Comments:** No studies have identified complications associated with the antiemetic dose of dexamethasone. Immunosuppression concern for postoperative infections.

## Butyrophenones (See Also Haloperidol)

Droperidol (Inapsine)

**Dose:** **Adults:** 1–2 mcg/kg IV (0.625–1.25 mg); **Peds:** 0.1 mg/kg max

**Mechanism:** Dopamine antagonist in chemoreceptor trigger zone. Centrally, also interferes with transmission of NE, 5HT, GABA. Peripherally exerts some  $\alpha$ -1 blockade.

**Clearance:** Hepatic

**Comments:** *Adverse reactions:* QT prolongation (black box warning), hypotension (mediated by  $\alpha$ -1 receptor blockade avoid in hypovolemic patients), rare extrapyramidal side effects. Avoid in pheochromocytoma, as droperidol can precipitate catecholamine release from adrenal medulla. Extrapyramidal Symptoms (EPS) can be treated with diphenhydramine. Antagonizes effects of L-dopa. All patients should undergo a 12-lead ECG prior to administration of droperidol to determine if a prolonged QT interval (QTc >440 msec for males or >450 msec for females) is present.

## PROKINETICS

**Metoclopramide** (Reglan)

**Dose:** **Adults:** 0.25 mg/kg IV/IM/PO (10–20 mg)

**Mechanism:** Central dopamine antagonist, peripheral cholinomimetic

**Metabolism:** Renal excretion

**Comments:** *Adverse Reactions:* Rare EPS, neuroleptic malignant syndrome; increases serum aldosterone leading to fluid retention (caution in CHF, cirrhotic patients). Increases LES tone and hastens gastric emptying by virtue of its peripheral cholinomimetic effects. Enhances peristalsis, decreases pyloric sphincter tone. Does not affect gastric pH. Contraindicated in patients with intestinal obstruction.

Decrease dose appropriately in patients with renal insufficiency. Avoid in patients with pheochromocytoma, as drug can induce hypertensive crisis.

## Phenothiazines

Promethazine (Phenergan)

**Dose:** **Adults:** 12.5–25 mg IV/IM/PO; **Peds:** 0.5 mg/LB

**Mechanism:** H<sub>1</sub> receptor antagonist. Antidopaminergic and  $\alpha$ -adrenergic blocking properties.

**Metabolism:** Hepatic

**Comments:** Adverse reactions: Resp depression (avoid in patients with COPD, OSA), CNS depression, rare neuroleptic malignant syndrome. Severe tissue damage with venous extravasation.

Contraindicated in children <2 yr/o owing to risk of fatal respiratory depression.

Prochlorperazine (Compazine) Dose		
	Adult	Pediatric
IM	5–10 mg q3–4h, max 10 mg/dose 40 mg/d	0.1–0.13 mg/kg q8–12h <b>Max doses: mg/kg/d</b> <b>&gt;2 yr/o, 9–14 kg: 7.5</b> <b>&gt;2 yr/o, 14–18 kg: 10</b> <b>18–39 kg: 15</b> <b>39 kg: 20</b>
IV	2.5–10 mg q3–5h	—
PO	5–10 mg PO q6–8h, max 40 mg/d	<b>&gt;2 yr/o, 9–14 kg:</b> 2.5 mg PO q12–24h, max 7.5 mg/d; <b>&gt;2 yr/o, 14–18 kg:</b> 2.5 mg PO q8–12h, max 10 mg/d; <b>18–39 kg: 2.5 mg PO q8h,</b> max 15 mg/d; <b>&gt;39 kg: 5 mg</b> PO q6–8h, max 20 mg/d
PR	25 mg q12h	—

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## ANTIHISTAMINES

(See Also Hydroxyzine)

**Diphenhydramine** (Benadryl)

(Also discussed below)

**Dose: Adult:** 25–50 mg PO/IM/IV q4–6h, max 300 mg/d. **Peds:** 1 mg/kg/dose PO/IV/IM

**Clearance:** Hepatic metabolism; renal excretion.

**Comments:** May cause hypotension, tachycardia, dizziness, urinary retention, seizures.

## VASOPRESSORS (FOR TREATMENT OF PONV)

**Ephedrine**

**Dose:** 50 mg IM; in pts <50 kg, 35 mg IM; may repeat ×1 in 4 hr

**Comments:** May cause HTN. Contraindicated in patients with hypertensive disease or cardiovascular disease.

## ANTICHOLINERGICS

**Scopolamine Patch**

(Other uses discussed later)

**Dose: Adult:** 1.5-mg transdermal patch. **Peds:** Not available. Patch cannot be cut.

**Mechanism:** Peripheral and central cholinergic (muscarinic) antagonist, antisialagogue, histamine and serotonin antagonist.

**Clearance:** Hepatic metabolism; renal elimination.

**Comments:** Used as antisialagogue, sedative, antiemetic, anti-motion sickness.

Excessive CNS depression can be reversed by physostigmine. May cause excitement, delirium, transient tachycardia, hyperthermia, urinary retention, blurred vision, photophobia. Care when handling patch because contact with eyes may cause long-lasting mydriasis and cycloplegia. Crosses the blood-brain barrier and placenta.

## COAGULATION RELATED

**Abciximab** (Reopro)

**Indications:** Prevents thrombus formation after percutaneous coronary intervention (PCI).

**Dose:** Bolus 0.25 mg/kg administered 10–60 min prior to PCI, then 10 mcg/min IV infusion.

**Mechanism:** Inhibits platelet glycoprotein IIb/IIIa; prevents platelet adhesion and aggregation via inhibition of binding of fibrinogen, von Willebrand factor, and other adhesive molecules.

**Clearance:** Remains in circulation for ≥15 d in a platelet-bound state, but platelet function recovers in about 48 hr.

**Comments:** Anaphylaxis may occur; hypotension with bolus dose. Bleeding complications and thrombocytopenia are common side effects. Contraindicated if active internal bleeding, GI/GU bleeding in last 6 weeks, CVA within 2 years, oral anti-coagulants within 7 d unless  $PT \leq 1.2 \times$  normal, platelets  $< 100,000$ , intracranial AVM, neoplasm, aneurysm, severe uncontrolled HTN, use of IV dextran prior to PCI or intent to use, vasculitis, bleeding diathesis.

### Aminocaproic Acid (Amicar)

**Dose:** 5 g/100–250 mL of NS IV to load prior to skin incision, followed by 1 g/hr infusion, or load 100–150 mg/kg IV over 30–60 min and infuse at 33.3 mg/kg/hr  $\times$  8 hr or until bleeding controlled.

**Mechanism:** Stabilizes clot formation by inhibiting plasminogen activators and plasmin. Fibrinolysis inhibitor.

**Clearance:** Primarily renal (decrease dose by 75% in ESRD)

**Comments:** Indicated for hemorrhage secondary to fibrinolysis.

Contraindicated in DIC. May cause hypotension, bradycardia, arrhythmias, thrombosis, LFT elevation, decreased platelet function

### Aprotinin (Trasylol)

*Discontinued from U.S. market 5/2008 secondary to increased risk of death compared with aminocaproic acid and tranexamic acid*

### Argatroban (Argatroban)

**Indications:** (1) Treatment or prophylaxis of thrombosis in heparin-induced thrombocytopenia; (2) PCI in patients with or at risk for heparin-induced thrombocytopenia.

**Dose:** (1) 2 mcg/kg/min IV continuous infusion, MAX 10 mcg/kg/min, adjust until steady-state aPTT is 1.5–3 $\times$  initial baseline value (not to exceed 100 sec). (2) 350 mcg/kg IV over 3–5 min and 25 mcg/kg/min IV continuous infusion, maintain ACT between 300 and 450 sec. Check ACT 5–10 min after bolus; may rebolus 150 mcg/kg and increase infusion to 30 mcg/kg/min for ACT  $< 300$ . For ACT  $> 450$  decrease infusion to 15 mcg/kg/min and recheck ACT in 5–10 min. **Peds:** Safety and efficacy in children  $< 18$  yr/o have not been established.

**Mechanism:** Direct, highly selective thrombin inhibitor. Inhibits fibrin formation; activation of factors V, VIII, and XIII; protein C; and platelet aggregation.

**Clearance:** Hepatic metabolism with 65% biliary excretion and 22% renal (16% excreted as unchanged drug).

**Comments:** Bleeding is the major adverse effect. May also cause hypotension, minor GI/GU bleeding, minor decrease in hematocrit. Not to be administered with other parenteral anticoagulants. Caution when switching to or from other anticoagulants: Allow heparin's effect on the aPTT to decrease; loading doses of warfarin should not be used. Caution in patients with severe HTN, recent lumbar puncture, or major surgery. Reduce dose in hepatic dysfunction.

### Clopidogrel (Plavix)

**Indications:** (1) Acute coronary syndrome. (2) PCI. (3) Recent MI, recent thromboembolic stroke, or established arterial disease.

**Dose:** (1) and (2) load: 300 mg PO; Maintenance: 75 mg PO qd. (3) 75 mg PO qd.

**Mechanism:** Antiplatelet agent. ADP receptor blocker: Prevents fibrinogen binding, thus reducing the possibility of platelet adhesion and aggregation.

**Clearance:** Hepatic metabolism; renal excretion.

**Comments:** Major side effect is bleeding. Concurrent use with heparin and aspirin is accepted, particularly in treatment of ACS. Caution in bleeding states. Gastrointestinal intolerance in  $> 20\%$  of patients. Reduce dose in hepatic insufficiency. Recommend discontinuing 7 d prior to neuraxial anesthesia.

### Dalteparin (Fragmin)

**Indications:** (1) DVT prophylaxis; (2) DVT tx; (3) ACS

**Dose:** (1) 2500–5000 U SC qd; (2) 100 U/kg SC bid or 200 U/kg SC qd; (3) 120 U/kg SC to max 10,000 U SC q12h  $\times$  5–8 d combined with aspirin therapy

**Mechanism:** Anticoagulant via factor Xa and IIa inhibition.

**Metabolism:** Hepatic with renal elimination

**Comments:** As effective as unfractionated heparin with more predictable dose–response relationship. Occurrence of hematoma with neuraxial anesthesia, especially with indwelling catheters, has been reported. Incidence of thrombocytopenia rare. Not studied in children.

### **Eptifibatid (Integrilin)**

**Indications:** Prevents thrombus formation after PCI and treatment of patients with ACS

**Dose:** Bolus 180 mcg/kg, then 2 mcg/kg/min infusion and repeat bolus of 180 mcg/kg 10 min after 1st bolus.

**Mechanism:** Prevents platelet adhesion and aggregation by reversible inhibition of glycoprotein IIb/IIIa, fibrinogen, and von Willebrand factor.

**Clearance:** Platelet function recovers within 4 to 8 hr after discontinuation of infusion.

**Comments:** Bleeding complications and thrombocytopenia are common side effects. Contraindicated in bleeding diathesis, severe uncontrolled HTN, severe bleeding within preceding 6 wk, renal dialysis dependency, CVA within last 30 d. Decrease infusion to 1 mcg/kg in pts with creatinine clearance <50 mL/min, but bolus doses are the same.

### **Enoxaparin (Lovenox)**

**Indications:** (1) DVT prophylaxis; (2) DVT Tx; (3) ACS

**Dosage: Adult:** (1) 30 mg SC bid or 40 mg SC qd for 7–10 d; (2) 1 mg/kg SC q12h or 1.5 mg/kg SC qd; (3) 1 mg/kg SC bid for  $\leq 2$  d in conjunction with aspirin therapy.

**Peds:** (1) <2 mo: 0.75 mg/kg/dose SC q12h; (2) 1.5 mg/kg/dose SC q12h; >2 mo (1) 0.5 mg/kg/dose SC q12h; (2) 1 mg/kg/dose SC q12h

**Mechanism:** Anticoagulant; inhibits both factor Xa and factor IIa.

**Clearance:** Hepatic; renal excretion.

**Comments:** As effective as unfractionated heparin with more predictable dose–response relationship. Appears superior to unfractionated heparin in aspirin-treated patients with unstable angina or non-Q-wave MI. Occurrence of hematoma with neuraxial anesthesia, especially with indwelling catheters, has been reported. Incidence of thrombocytopenia rare. Not studied in children. Does not significantly effect bleeding time, platelet function, PT, or aPTT. May cause fever, confusion, hypochromic anemia, pain/erythema at injection site.

### **Heparin, Unfractionated**

**Indications:** Anticoagulation for (1) thrombosis, thromboembolism; (2) CPB; (3) DIC; (4) thromboembolism prophylaxis; (5) line patency

**Dose: Adult:** (1) INFUSION: Load: 50–150 U/kg IV (or bolus 5000 U); Maintenance: 15–25 U/kg/hr IV (or ~1000 U/hr). Titrate to PTT or activated clotting time (ACT). INTERMITTENT IV: bolus 10,000 U IV, then 5000–10,000 U (50–70 U/kg) q4–6h; (2) Load: 300 U/kg IV; Maintenance: 100 U/kg/hr IV, titrate to ACT. (3) Load: 50–100 U/kg IV; Maintenance: 15–25 U/kg/hr IV; titrate to ACT. (4) 5000 U q8–12h SC; (5) 100 U/mL for line flushing, 1 U/mL for arterial lines; **Peds:** (1) INFUSION: Load: 75 U/kg over 10 min, then 15–25 U/kg/hr titrate to ACT. INTERMITTENT IV: Load: 50–100 U/kg, then 50–100 U/kg q4h (5) >10 kg, 100 U/mL for line flushing, 1 U/mL for arterial lines; **Infants/Neonates:** (1) Load: 50–75 U/kg IV over 10 min; Maintenance: 28 U/kg/hr (range 15–35 U/kg). Titrate to ACT. (5) <10 kg, line flushing 10 U/mL; art lines 0.5–2 U/mL.

**Mechanism:** Potentiates action of antithrombin III; blocks conversion of prothrombin and activation of other coagulation factors IX to XII and plasmin; prevents conversion of fibrinogen to fibrin; stimulates lipoprotein lipase release; decreases platelet aggregation.

**Clearance:** Primarily by reticuloendothelial uptake, hepatic biotransformation.

**Comments:** 1 mg = 100 U. May cause bleeding, thrombocytopenia, allergic reactions, diuresis (36–48 hr after a large dose), hypotension. Half-life increased in renal failure and decreased in thromboembolism and liver disease. Does not cross placenta. Reversed by protamine. Occurrence of hematoma with neuraxial anesthesia and lumbar puncture, especially with indwelling catheters.

### **Prostaglandin E1 (Alprostadil, Prostin VR)**

**Dose: Neonates:** Starting dose 0.05–0.1 mcg/kg/min. Titrate to effect (typical 0.1–0.4 mcg/kg/min or maximum 0.6 mcg/kg/min. Customary mix: 500 mcg/99 mL of NS or D5W = 5 mcg/mL.

**Mechanism:** Vasodilation, inhibits platelet aggregation, vascular smooth muscle relaxation, and uterine and intestinal smooth muscle relaxation.

**Clearance:** Pulmonary metabolism; renal elimination.

**Comments:** Indicated for pulmonary vasodilation, maintenance of patent ductus arteriosus. May cause hypotension, apnea (premature newborns), flushing, and bradycardia. Decrease to lowest effective dose if increase in PaO<sub>2</sub> noted.

## Protamine Sulfate

**Dose:** Time elapsed since last heparin dose (see table below); Based on ACT: 1.3 mg/100 U heparin calculated from ACT. Slow IV,  $\leq 5$  mg/min.

Time Elapsed (since last heparin dose)	Protamine (mg) to Neutralize 100 U Heparin
<30 min	1-1.5
30-60 min	0.5-0.75
60-120 min	0.375-0.5
>2 hr	0.25-0.375

**Mechanism:** Heparin antagonist. Polybasic compound forms complex with polyacidic heparin.

**Clearance:** Fate of the heparin-protamine complex is unknown.

**Comments:** May cause anaphylaxis, anaphylactoid reaction, myocardial depression and peripheral vasodilation with sudden hypotension (secondary to histamine release) or bradycardia. May cause severe pulmonary HTN, particularly in the setting of cardiopulmonary bypass. Protamine-heparin complex antigenically active (particularly in pts receiving procaine and with fish allergy). Transient reversal of heparin may be followed by rebound heparinization. Can cause anticoagulation if given in excess relative to amount of circulating heparin (controversial). Monitor response with activated partial thromboplastin time or activated clotting time.

**Tissue Plasminogen Activator** (Alteplase, Activase, t-PA)

**Indications:** (1) Lysis of coronary arterial thrombi in hemodynamically unstable patients with acute MI. (2) Management of acute massive PE in adults. (3) Acute embolic stroke.

**Dose:** (1) *Load:* 15 mg (30 mL of the infusion) IV over 1 min followed by 0.75 mg/kg (not to exceed 50 mg) given over 30 min. *Maintenance:* 0.5 mg/kg IV up to 35 mg/hr for 1 hr immediately following the loading dose. Total dose not to exceed 100 mg. (2) 100 mg IV continuous infusion over 2 hr. (3) Total dose of 0.9 mg/kg IV (maximum 90 mg); administer 10% as a bolus and the remainder over 60 min.

**Mechanism:** Tissue plasminogen activator

**Clearance:** Rapid hepatic clearance.

**Comments:** Doses > 150 mg have been associated with an increased incidence of intracranial hemorrhage. Contraindicated with active internal bleeding, history of hemorrhagic stroke, intracranial neoplasm, aneurysm, or recent (within 2 mo) intracranial or intraspinal surgery, or trauma. Should be used with caution in pts who have received chest compressions and in pts who are currently receiving heparin, warfarin, or antiplatelet drugs.

**Vitamin K** (Phytonadione, AquaMEPHYTON)

**Dose: Adult:** 2.5-10 mg IM/SC/ or 5-25 mg/d PO, or 1-10 mg IV at  $\leq 1$  mg/min (with caution). If international normalized ratio (INR) time is not improved 8 hr after initial dose, repeat prn.

**Mechanism:** Vitamin K is required for synthesis of clotting factors II, VII, IX, X.

**Clearance:** Hepatic metabolism

**Comments:** Indicated for deficiency of vitamin K-dependent clotting factors, reversal of warfarin anticoagulation. Excessive doses can make pt refractory to further oral anticoagulation. May fail with hepatocellular disease. Rapid IV bolus can cause profound hypotension, fever, diaphoresis, bronchospasm, anaphylaxis, and pain at injection site. Crosses the placenta. Reserve IV administration for emergency use.

**Warfarin** (Coumadin)

**Dose:** *Load:* 5 mg PO  $\times$  2-5 d; *Maintenance:* 2-10 mg PO, titrated to prothrombin time (INR should be 2 to 3, based on indication).

**Mechanism:** Interferes with utilization of vitamin K by the liver and inhibits synthesis of factors II, VII, IX, X, proteins C & S, prothrombin.

**Clearance:** Hepatic metabolism; renal elimination.

**Comments:** Indication for anticoagulation. Contraindicated in severe renal or hepatic disease, GI ulcers, neurosurgical procedures, malignant HTN, pregnancy (teratogenic). Multiple drug interactions causing potentiation/antagonism. Crosses the placenta. Thrombostatic only, no lysis of existing thrombus.

## DIURETICS

### Acetazolamide (Diamox)

**Indications:** (1) Secondary metabolic alkalosis; (2) Increased ICP

**Dose:** (1) 3–5 mg/kg/dose q6h × 4 doses; (2) 20 mg/kg/24 hr div q8h; Max 100 mg/kg/24 hr to max 2 g/24 hr

**Mechanism:** Carbonic anhydrase inhibitor; increases HCO<sub>3</sub> excretion

**Metabolism:** Renal clearance, unchanged

**Comments:** Indicated for respiratory acidosis with metabolic alkalosis, increased ICP, IOP. Contraindicated in hepatic failure, renal failure. Rare reaction in pts with sulfa allergy.

### Bumetanide (Bumex)

**Indications:** Edema, HTN, intracranial HTN.

**Dose:** 0.5–1.0 mg IV, repeated to a maximum of 10 mg/d. PO also available.

**Mechanism:** Loop diuretic; inhibits Na<sup>+</sup> and Cl<sup>-</sup> reabsorption in ascending limb of loop of Henle and increases K<sup>+</sup> excretion.

**Clearance:** Hepatic metabolism; 81% renal excretion (45% unchanged).

**Comments:** May cause electrolyte imbalance (especially hyponatremia, hypokalemia), dehydration, ototoxicity (in animal studies with higher dosing), and rarely thrombocytopenia. Patients who are allergic to sulfonamides may show hypersensitivity to bumetanide. Effective in renal insufficiency but contraindicated in anuria; and bumetanide should be discontinued if renal function worsens during therapy. Dose of 1 mg bumetanide approximately equal to 40 mg furosemide. Oral and IV/IM have similar bioavailability.

### Chlorothiazide (Diuril)

(hydrochlorothiazide for PO dosing)

**Dose: Adult:** 100–500 mg IV at 50–100 mg/min in 1–2 doses; max 2 g/24 hr. 500 mg–2 g/d PO. **Peds:** 4 mg/kg/d IV div q12h, 20 mg/kg/d PO div q12h. **Infant <6 mo:** 2–8 mg/kg/d IV div q12h, 20–40 mg/kg/d PO, div q12h

**Mechanism:** Thiazide diuretic; inhibits distal tubule sodium reabsorption causing increased excretion of water and sodium as well as hydrogen ions, potassium, magnesium, calcium, and phosphate.

**Metabolism:** Renal elimination

**Comments:** Useful in tx of edema associated with CHF, pregnancy, nephrotic syndrome. Also in acute or chronic renal failure and HTN. Antihypertensive activity enhanced. May also enhance loop diuretic activity in pts with renal failure. Diabetic patients may have increased insulin requirements. Use with caution in patients with severe renal or hepatic disease. May increase serum calcium, bilirubin, glucose, uric acid, alkalosis; may decrease potassium and magnesium. May cause pancreatitis, dizziness.

### Furosemide (Lasix)

**Dose: Adult:** 2–100 mg IV q6–12h max 1000 mg/d (initial dose; dosage individualized). Infusion: Load 0.1 mg/kg and 0.1 mg/kg/hr doubling every hr to max 0.4 mg/kg/hr.

**Peds:** 0.5–2 mg/kg/dose q6–12h, max 6 mg/kg/dose. **Neonates:** 0.5–1 mg/kg/dose IV/PO q8–12h, max PO: 6 mg/kg/dose; max IV: 2 mg/kg/dose

**Mechanism:** Increases excretion of Na<sup>+</sup>, Cl<sup>-</sup>, K<sup>+</sup>, PO<sub>4</sub><sup>3-</sup>, Ca<sup>2+</sup>, and H<sub>2</sub>O by inhibiting reabsorption in loop of Henle. Decreases CSF production

**Clearance:** Hepatic metabolism; 88% renal elimination.

**Comments:** Indicated for tx of CHF, edema, HTN, intracranial HTN, renal failure, hypercalcemia. May cause electrolyte imbalance, dehydration, transient hypotension, deafness, hyperglycemia, or hyperuricemia. Hypersensitivity possible in sulfa-allergic patients.

### Mannitol (Osmitol)

**Indications:** (1) Increased intracranial pressure. (2) Oliguria or anuria associated with acute renal injury.

**Dose: Adult:** (1) 0.25–1 g/kg IV as 20% solution over 30–60 min (in acute situation, can bolus 1.25–25 g over 5–10 min). Max 1–2 g/kg in 2–6 hr (2) 0.2 g/kg (or ~12.5 g) test dose over 3–5 min to obtain urine output 30–50 mL/hr for 1–3 hr; then load 0.5–1 g/kg (50–100 g) IV over 30 min and infuse 0.25–0.5 g/kg q4–6h. **Peds:** (1) 0.2 g/kg test dose over 3–5 min to obtain urine output 1 mL/kg for 1–3 hr; then load 0.5–1 g/kg IV over 30 min and infuse 0.25–0.5 g/kg q4–6h

**Mechanism:** Increases serum osmolality, reducing cerebral edema and lowering intracranial and intraocular pressure; also causes osmotic diuresis (inhibits tubular water reabsorption) and transient expansion of intravascular volume

**Clearance:** Renal elimination



**Comments:** Contraindicated in severe renal disease, active intracranial bleed, dehydration, severe pulmonary edema. Rapid administration may cause vasodilation and hypotension. May worsen or cause pulmonary edema, intracranial hemorrhage, systemic HTN, or rebound intracranial HTN. Check preparation for unwanted crystal sedimentation.

### Spironolactone (Aldactone)

**Indications:** (1) edema; (2) HTN; (3) diuretic-induced hypokalemia; (4) CHF  
**Dose: Adult:** (May divide dosing bid) (1) 25–200 mg PO qd; (2) 25–50 mg PO qd, start 12.5 mg/d; (3) 25–100 mg PO qd; (4) 25 mg PO qd (may increase to 50 mg/d after 8 wk if K<sup>+</sup> stable or decrease to 25 mg qod if hyperkalemia). **Peds:** (1) 1–3.3 mg/kg/d POo div bid–qid, max 3.3 mg/kg/d or 200 mg/d.

**Mechanism:** Aldosterone antagonist. Potassium-sparing diuretic.

**Clearance:** Liver. Canrenone active metabolite.

**Comments:** For treatment of edema associated with CHF, cirrhosis, or nephrotic syndrome. Takes 2–4 days to see full effect of drug and lasts 48–72 hr after discontinuing. May cause hyperkalemia.

### OTHER

#### Albuterol (Proventil, Ventolin)

**Dose:** Nebulizer: 2.5–5 mg in 3 mL saline q 6h (or more frequently PRN)  
 < 1 yr: 0.05–0.15 mg/kg/dose q4–6 hrs; 1–5 yrs: 1.25–2.5 mg q4–6h; Metered dose inhaler: 90–180 mcg (1–2 puffs) via inhaler q4–6h.

**Mechanism:** Beta-2 receptor agonist

**Clearance:** Liver metabolism to inactive sulfate

**Comments:** May cause tachyarrhythmias (consider ipratropium, aka Atrovent, if concerned), HTN, chest pain, headache, increased blood glucose. Can administer MDI to intubated pt by inserting canister into ~60 mL syringe and connecting in place of gas sampling line.

#### Atropine Sulfate

**Indications:** (1) Antisialagogue. (2) Bradycardia/PEA/asystole. (3) Adjunct with edrophonium in reversal of neuromuscular blockade

**Dose: Adult:** (1) 0.2–0.4 mg IV. (2) 0.4–1.0 mg IV q3–5min **Peds:** (1) 0.01 mg/kg/dose IV/IM (max 0.4 mg). (2) 0.02 mg/kg/dose IV q3–5min (max single dose, children: 0.5 mg; adolescents 1 mg), (3) 0.015–0.03 mg/kg  
 Minimum dose 0.1 mg IV (Peds and adult)

**Mechanism:** Competitive blockade of acetylcholine at muscarinic receptors

**Clearance:** 50%–70% hepatic metabolism; renal elimination.

**Comments:** May cause tachydysrhythmias, AV dissociation, premature ventricular contractions, dry mouth, or urinary retention. CNS effects occur at high doses. Contraindicated in patients sensitive to sulfites. Ineffective in 2nd degree type II AV block. Avoid in new-onset 3rd degree AV block with wide QRS complexes and hypothermic bradycardia.

#### Bicarbonate (Sodium Bicarbonate)

**Dose:** Metabolic acidosis: **Adult:**  $0.2 \times \text{wt (kg)} \times \text{base deficit (mEq/L)}$  **Peds:**  $0.3 \times \text{wt (kg)} \times \text{base deficit (mEq/L)}$

Cardiac arrest: **Adult:** 1 mEq/kg/dose; **Peds:** 0.5–1 mEq/kg/dose. Use 4.2% solution in neonates.

**Mechanism:** Neutralization of hydrogen ions

**Comments:** Contraindicated in respiratory alkalosis, unknown abdominal pain, CPR with inadequate ventilation, excessive chloride losses. Caution in CHF, renal impairment, cirrhosis, hypocalcemia, HTN, concurrent corticosteroid use. 8.4% solution is ~1.0 mEq/mL. Can cause intraventricular hemorrhage in neonates. Crosses placenta. May cause metabolic alkalosis, hypercarbia, and hyperosmolality, hypokalemia, hypocalcemia, tissue necrosis (extravasation). May decrease cardiac output, systemic vascular resistance, and myocardial contractility.

#### Bicitra (Sodium Dihydrate/Citric Acid)

**Dose: Adult:** 15–30 mL PO 15–30 min prior to induction; **Peds:** 5–15 mL PO 15–30 min prior to induction

**Mechanism:** Neutralization of gastric acid.

**Clearance:** Metabolized to sodium bicarbonate

**Comments:** Nonparticulate antacid useful for preoperative administration as inadvertent aspiration will not cause as significant chemical pneumonitis as conventional (aluminum, calcium, etc.) antacids will. Contraindicated in pts with severe renal disease, sodium restriction. Do not combine with aluminum-containing antacids. May

have laxative effect, cause hypocalcemia, metabolic acidosis. 10 mL bicitra: 1 g sodium citrate, 666 mg citric acid, 10 mEq Na, equivalent to 10 mEq of bicarbonate.

### **Calcium Chloride (CaCl<sub>2</sub>); Calcium Gluconate** (Kalcinate)

**Indications:** Hypocalcemia, hyperkalemia, hypermagnesemia, cardiac arrest (CaCl<sub>2</sub>)

**Dose:** Calcium chloride: 5–10 mg/kg IV prn (10% CaCl<sub>2</sub> = 1.36 mEq Ca<sup>2+</sup>/mL). *Cardiac arrest:* **Adult:** 250–500 mg/dose (or 2–4 mg/kg/dose, may repeat q10min prn; **Peds:** 20 mg/kg/dose q10min prn. Calcium gluconate: 15–30 mg/kg IV prn (10% calcium gluconate = 0.45 mEq Ca<sup>2+</sup>/mL). *Hypocalcemia secondary to citrated blood transfusion:* CaCl<sub>2</sub>: 33 mg/100 mL blood transfused. Ca gluconate: 100 mg/100 mL blood transfused.

**Actions:** Maintains cell membrane integrity, muscular excitation–contraction coupling, glandular stimulation–secretion coupling, and enzyme function. Increases blood pressure.

**Clearance:** Incorporated into muscle, bone, and other tissues. Renal excretion.

**Comments:** May cause bradycardia or arrhythmia (especially with digitalis), increased risk of ventricular fibrillation, HTN. Irritating to veins with necrosis risk if extravasates. Central line is preferred route, especially for calcium chloride. Ca<sup>2+</sup> less available with calcium gluconate than with calcium chloride due to binding of gluconate. Not routinely used in ACLS.

### **Chloral Hydrate** (Noctec)

**Indications:** (1) Sedation; (2) hypnosis

**Dose: Adult:** (1) 250 mg PO tid; (2) 500–1000 mg PO/PR qhs or 30 min prior to procedure (max 2 g/24 hr). **Peds:** (1) 5–15 mg/kg/dose PO/PR q8h (max 500 mg) to 50–75 mg/kg/dose 30–60 min prior to procedure, repeat 30 min prior to procedure prn. (2) 20–40 mg/kg/dose PO/PR to max 50 mg/kg/24 hr. **Neonate:** (1) 25 mg/kg/dose PO/PR prior to procedure (2) 50 mg/kg/dose PO/PR.

**Mechanism:** Unknown. CNS depressant due to active metabolite trichloroethanol.

**Onset:** 30–60 min; **Duration:** 4–8 hr

**Comments:** Frequently used for procedural sedation in pediatrics by nonanesthetists. Use only for nonpainful procedures, as does not provide analgesia. Contraindicated in patients with renal or hepatic disease. May cause GI irritation, paradoxical excitement, hypotension, depression of respiratory/myocardial function.

### **Dantrolene** (Dantrium)

**Dose: Adult/Peds:** 2.5 mg/kg IV bolus (mix 20 mg/60 mL sterile water = 525-mL bolus for 70-kg pt). Repeat dose q5min until symptoms improve up to max 10 mg/kg (although 30 mg/kg sometimes required). Postacute reaction 1 mg/kg q6h for 24–48 hr, then taper or change to oral tx.

**Mechanism/Indication:** Reduction of Ca<sup>2+</sup> release from sarcoplasmic reticulum. Tx for malignant hyperthermia (MH) and skeletal muscle rigidity.

**Metabolism:** Hepatic with renal elimination

**Comments:** Powder dissolves slowly into solution. Note large volume to be administered. Prophylactic treatment not recommended. See also "Malignant Hyperthermia Protocol," Appendix D.

### **Desmopressin Acetate** (DDAVP)

**Indications:** (1) Treatment of coagulopathy in von Willebrand disease, hemophilia A, renal failure. (2) Antidiuretic for tx of central diabetes insipidus.

**Dose:** Dilute to 0.5 mcg/mL NS. **Adult:** (1) 0.3 mcg/kg IV (diluted 50 mL NS), infused over 15–30 min preop. (2) 2–4 mg/d div q12h. **Peds:** <10 kg, dilute adult dose in 10 mL NS; >10 kg, see adult dose.

**Mechanism:** Increases plasma levels of factor VIII activity by causing release of von Willebrand factor (factor VII) from endothelial cells; increases renal water reabsorption by increasing water permeability of collecting duct

**Clearance:** Renal elimination.

**Comments:** Chlorpropamide, carbamazepine, and clofibrate potentiate the antidiuretic effect. Repeat doses q12–24h will have diminished effect compared with initial dose. Use with caution in HTN, CAD, pts at risk for thrombus formation. May cause headache, nasal congestion, abdominal cramps, HTN.

### **Dexamethasone** (Decadron)

**Indications:** (1) Cerebral edema from CNS tumors; (2) airway edema; (3) prophylaxis of postoperative nausea and vomiting; (4) septic shock; (5) croup

**Dose: Adult:** (1) Load: 10 mg IV; *Maintenance:* 4 mg IV q6h (tapered over 6 d); (2) 4–6 mg IV/IM; (3) 4 mg IV; (4) 1–6 mg/kg IV (to max 40 mg) q2–6h during shock physiology. **Peds:** (1) Load: 1–2 mg/kg/dose IV; *Maintenance:* 1–1.5 mg/kg/d (max 16 mg/d) div q4–6h; (2) 0.25 mg/kg/dose IV/IM; (3) 0.15 mg/kg IV; (4) 0.6 mg/kg/dose IV/IM

**Mechanism:** Decreases inflammation via suppression of PMN migration and reversing increase capillary permeability

**Clearance:** Primarily hepatic metabolism; renal elimination.

**Comments:** Immunosuppressant; may delay wound healing. Has 20 to 25 times the glucocorticoid potency of hydrocortisone. Minimal mineralocorticoid effect. May cause seizures, osteoporosis, hyperglycemia, diarrhea, nausea, GI bleeding, Cushingoid effects. Caution in pts with hypothyroidism, cirrhosis, HTN, CHF, ulcerative colitis, thromboembolic disease. Risk of adrenocortical insufficiency if abrupt withdrawal. Causes GI/GU burning sensation with rapid IV push in awake pts.

**Diphenhydramine** (Benadryl)

**Indications:** (1) Antihistamine; (2) pruritus; (3) antiemetic, sedative

**Dose: Adult:** (1, 2) 10–50 mg IV q4–8h, max 100 mg/dose, 400 mg/d; (3) 25–50 mg PO/IV/IV q4–6h, max 300 mg/d. **Peds:** (> 10 kg) (1) 5 mg/kg/d IV div q6–8h (max 300 mg/d 75 mg/dose); (2) 0.5–1 mg/kg/dose PO/IV/IM q4–6h; (3) 1 mg/kg/dose PO/IV/IM

**Mechanism:** Histamine-1 (H1) receptor antagonist; anticholinergic; CNS depressant.

**Clearance:** Hepatic metabolism; renal excretion.

**Comments:** Indicated for allergic reactions, drug-induced extrapyramidal reactions; sedation, as antiemetic. May cause hypotension, tachycardia, dizziness, urinary retention, seizures.

**Epinephrine, Racemic** (Vaponefrin)

**Dose:** Inhaled via nebulizer. **Adult:** 0.5 mL of 2.25% solution in 2.5–3.5 mL of NS q1–4h prn. **Peds:** > 2 yr/o: 0.25–0.5 mL of 2.25% solution in 2.5–3.5 mL of NS q4h prn; < 2 yr/o: 0.25 mL of 2.25% solution in 2.5–3.5 mL of NS q4h prn

**Mechanism:** Mucosal vasoconstriction

**Clearance:** MAO/COMT metabolism

**Comments:** Indicated for airway edema, bronchospasm. Rebound airway edema may occur after discontinuation.

**Ethacrynic Acid** (Edecrin)

**Dose: Adult:** PO: 50–200 mg/d in 1–2 div doses; IV: 25–100 mg IV over 5–10 min; max 400 mg/d (IV/PO) **Peds:** PO: 1 mg/kg/dose qd, may increase to max 3 mg/kg/d; IV: 1 mg/kg/dose (repeat doses with caution due to potential for ototoxicity). Max 3 mg/kg/d (IV/PO)

**Mechanism:** Diuretic. Inhibits sodium and chloride reabsorption from ascending loop of Henle and distal renal tubule.

**Clearance:** Hepatically metabolized to active cysteine conjugate (35%–40%); 30%–60% excreted unchanged in bile and urine.

**Comments:** Indicated for edema, CHF, acute/chronic renal failure, ascites. May potentiate the activity of antihypertensives, neuromuscular blocking agents, digoxin, and increase insulin requirements in diabetic patients. Risk for ototoxicity.

**Famotidine** (Pepcid)

**Dose: Adult:** 20 mg IV/PO q12h (dilute in 1–10 mL of D5W or NS). **Peds:** 0.6–0.8 mg/kg/d IV div q8–12h (max 40 mg/d)

**Mechanism:** Histamine-2 (H2) receptor antagonist

**Clearance:** 30%–35% hepatic metabolism; 65%–70% renal elimination.

**Comments:** Pulmonary aspiration prophylaxis, peptic ulcer disease. May cause confusion, dizziness, headache, diarrhea. Administer slow IV: rapid IV administration may increase risk of cardiac dysrhythmias and hypotension

**Glucagon**

**Indications:** (1) Duodenal or choledochal relaxation. (2) Refractory  $\beta$ -adrenergic blocker toxicity. (3) Hypoglycemia

**Dose:** (1) 0.25–0.5 mg IV q20min prn. (2) 5 mg IV bolus, with 1–10 mg/hr titrated to patient response. (3) **Adults:** 0.5–1 mg, repeat q20min prn. **Peds:** 0.025–0.1 mg/kg/dose to max 1 mg/dose. Repeat q20min prn. **Neonates:** 0.025–0.3 mg/kg/dose, max 1 mg/dose.

**Mechanism:** Stimulates adenylate cyclase  $\rightarrow$  increased cAMP  $\rightarrow$  promotes hepatic gluconeogenesis, glycogenolysis, catecholamine release. Positive inotrope and chronotrope. Relaxes smooth muscle of stomach, duodenum, small intestine, and colon.

**Clearance:** Hepatic and renal proteolysis.

**Comments:** May cause anaphylaxis, nausea, vomiting, hyperglycemia, or positive inotropic and chronotropic effects. High doses potentiate oral anticoagulants. Use with caution in presence of insulinoma or pheochromocytoma. Do not mix with normal saline; use sterile water.

**Glycopyrrolate** (Robinul)

**Indications:** (1) Decreases gastrointestinal motility, antisialagogue; (2) bradycardia; (3) adjunct to reversal of neuromuscular blockade

**Dose: Adult:** (1) 0.1–0.2 mg IV/IM/SC; 2.5–10 mcg/kg/dose IV/IM q3–4h; 1–2 mg PO; (2) 0.1–0.2 mg/dose IV; (3) 0.2 mg IV for each 1 mg neostigmine or 5 mg pyridostigmine or 0.01–0.02 mg/kg IV. **Peds:** (1) 4–10 mcg/kg/dose IV/IM q3–4h, max 0.2 mg/dose or 0.8 mg q24h; 40–100 mcg/kg/dose PO tid–qid. (2) 0.01–0.02 mg/kg IV. **Neonates/infants:** 4–10 mcg/kg/dose IV/IM q4–8 h; 40–100 mcg/kg/dose PO q8–12h

**Mechanism:** Blocks acetylcholine action at smooth muscle parasympathetic sites, secretory glands, and CNS.

**Clearance:** Renal elimination.

**Comments:** Longer duration, better antisialagogue with less chronotropic effect than atropine. Does not cross blood–brain barrier or placenta. Unreliable oral absorption. May cause bronchospasm, blurred vision, constipation. Caution with asthma, ulcerative colitis, glaucoma, ileus, or urinary retention.

**Haloperidol** (Haldol)

**Dose: Adult:** 2–5 mg IM/IV q4–8h prn; 0.5–5 mg PO bid–tid max 30 mg/d; **ICU sedation, mild agitation:** 0.5–2 mg IV/IM; **moderate agitation:** 2–5 mg IV/IM; **severe agitation:** 10–20 mg IV/IM. **Continuous infusion:** 1–40 mg/hr (100 mg/100 mL D5W). **PONV** 1 mg IV; **Peds:** 3–12 yr/o, 15–40 kg 0.025–0.07 mg/kg/d div q8h to max 0.15 mg/kg/d. **Antiemetic:** 0.01 mg/kg/dose IV q8–12h

**Mechanism:** Dopaminergic (D1 and D2) antagonist. Depresses reticular activating system.

**Clearance:** Hepatic metabolism; renal/biliary elimination.

**Comments:** Indicated for psychosis, agitation, postoperative nausea and vomiting, ICU sedation. May cause extrapyramidal reactions or mild  $\alpha$ -adrenergic antagonism. Can prolong QT interval and produce ventricular arrhythmias, particularly torsades de pointes. Lowers seizure threshold. May precipitate neuroleptic malignant syndrome. Contraindicated in Parkinson's disease; caution in cardiac patients.

**Hydrocortisone** (Solu-Cortef)

**Indications:** (1) acute adrenal insufficiency; (2) physiologic replacement; (3) shock; (4) stress dosing; (5) status asthmaticus. **Dose: Adult:** (1) bolus 100 mg IV, then 300 mg/d IV div q8h; (2) 500–2000 mg IV/IM q2–6h; (3) infuse 1.5–4 mg/kg/d from start of surgery for 24 hr or 40–100 mg/m<sup>2</sup>/d div q6–8h; (4) 1–2 mg/kg/dose IV q6h, then maintenance 0.5–1 mg/kg IV q6h. **Peds:** (1) *Older children:* 1–2 mg/kg/dose IV then 150–250 mg/d div q6–8h; *Infants/young children:* 1–2 mg/kg/dose IV, then 25–150 mg/d div q6–8h; (2) 0.5–0.75 mg/kg/d PO div q8h or 0.25–0.35 mg/kg/d IM qd; (3) 50 mg/kg IV/IM, may repeat q4h

**Mechanism:** Anti-inflammatory and anti-allergic effect; mineralocorticoid effect; stimulates gluconeogenesis; inhibits peripheral protein synthesis; has membrane-stabilizing effect.

**Clearance:** Hepatic metabolism; renal elimination.

**Comments:** May cause adrenocortical insufficiency (adisonian crisis) with abrupt withdrawal, delayed wound healing, CNS disturbances, osteoporosis, or electrolyte disturbances.

**Hydroxyzine** (Vistaril, Atarax)

**Dose: Adult** PO: 25–200 mg q6–8h; IM: 25–100 mg q4–6h. Not an IV drug. **Peds:** 2–4 mg/kg/d PO div q6–8h; 0.5–1 mg/kg/dose IM q4–6h prn

**Mechanism:** Histamine-1 (H1) receptor antagonist. CNS depression, antiemetic. Some evidence for analgesic effect.

**Clearance:** Hepatic (P-450) metabolism; renal elimination.

**Comments:** Indicated for anxiety, nausea and vomiting, allergies, sedation. May cause dry mouth, drowsiness, tremor, convulsions. Minimal cardiorespiratory depression. Intravenous injection may cause thrombosis, hence not recommended. May cause pain at IM injection site. Crosses placenta.

**Indigo Carmine**

**Dose:** 40 mg IV slowly (5 mL of 0.8% solution).

**Mechanism:** Rapid glomerular filtration produces blue urine.

**Clearance:** Renal elimination

**Comments:** Indicated for evaluation of urine output and localization of ureteral orifices during cystoscopy. May cause HTN from  $\alpha$ -adrenergic stimulation, lasts 15–30 min after IV dose and transient decrease in measured (not actual) pulse oximetry.

**Indocyanine Green** (Cardio-Green)

**Dose:** 5 mg IV (diluted in 1 mL of normal saline) rapidly injected into central circulation.

**Mechanism:** Plasma proteins bound, distributes within plasma volume.

**Clearance:** Hepatic elimination

**Comments:** Used to measure cardiac output by indicator dye dilution. May cause allergic reactions or transient increases in bilirubin levels. Absorption spectra changed by heparin. Caution in iodine-allergic (contains 5% sodium iodide).

**Insulin, Regular**

**Indications:** (1) Hyperglycemia; (2) diabetic ketoacidosis; (3) hyperkalemia

**Dose:** (1) (individualized) usually 5–10 IU IV/SC prn; (2) *Load:* 0.1 IU/kg IV; *Maintenance:* 0.05–0.2 IU /kg/hr IV, goal decrease blood glucose 80–100 mg/dL/hr; (3) 0.05–0.1 IU/kg/hr infused with glucose

**Mechanism:** Facilitates glucose transport intracellularly. Shifts  $K^+$  and  $Mg^{2+}$  intracellularly.

**Clearance:** Hepatic and renal metabolism; 30%–80% renal elimination. Unchanged insulin is reabsorbed.

**Comments:** May cause hypoglycemia, allergic reactions, or synthesis of insulin antibodies. May be absorbed by plastic in IV tubing. Prep: 50 IU reg insulin/250 mL D5W = 0.2 IU/mL; 5 mL/hr = 1 IU/hr

**Ketorolac** (Toradol)

**Dose: Adult:** >50 kg: bolus 30–60 mg IV/IM, then 15–30 mg q6h, max 120 mg/d for pts <65 yr/o; max 60 mg/d for pts >65 yr/o or with renal disease; pts <50 kg: bolus 30 mg IV/IM, then 15 mg q6h, max 60 mg/d. PO: 10 mg q4–6h, max 40 mg/24 hr. **Peds:** 0.4–1 mg/kg/dose IV, then 0.2–0.5 mg/kg/dose q6h

**Mechanism:** NSAID. Limits prostaglandin synthesis by cyclooxygenase inhibition.

**Clearance:** <50% hepatic metabolism, renal metabolism; 91% renal elimination.

**Comments:** Useful short-term adjunct for severe pain when used with parenteral or epidural opioids. Contraindicated for surgical cases where hemostasis is critical, pts with nasal polyps, hx of bronchospasm or angioedema with NSAIDs, active peptic ulcer disease or GI bleeding, advanced renal disease, hepatic failure, labor and delivery or nursing mothers, with neuraxial anesthetic. May cause peptic ulceration, GI bleeding, nausea, decreased platelet function, interstitial nephritis, decreased renal blood flow. **Max duration 5 d.**

**Levothyroxine** (Synthroid, T4)

**Dose: Adult:** PO: average 0.1–0.2 mg/d; IV: 50–75% of adult oral dose. *Myxedema coma/stupor* 200–500 mcg IV  $\times$  1 then 100–300  $\mu$ g subsequent d prn. **Peds:** PO: 0–6 mo: 25–50 mcg/d or 8–10 mcg/kg/d; 6–12 mo: 50–75 mcg/d or 6–8 mcg/kg/d; 1–5 yr: 75–100 mcg/d or 5–6 mcg/kg/d; 6–12 yr: 100–150 mcg/d or 4–5 mcg/kg/d; >12 yr: over 150 mcg/d or 2–3 mcg/kg/d; IV: 50–75% of oral dose

**Mechanism:** Exogenous thyroxine hormone

**Clearance:** Metabolized in the liver to triiodothyronine (active); eliminated in feces and urine.

**Comments:** Indicated for hypothyroidism. Contraindicated with recent myocardial infarction, thyrotoxicosis, or uncorrected adrenal insufficiency. Phenytoin may decrease levothyroxine levels. Increases effects of oral anticoagulants. Tricyclic antidepressants may increase toxic potential of both drugs. May cause HTN, arrhythmias, diarrhea, weight loss. 100 mcg levothyroxine = 65 mg thyroid USP.

**Lidocaine** (Xylocaine)

**Indications:** (1) Ventricular dysrhythmias. (2) Cough suppression. (3) Local anesthesia.

**Dose: Adult:** (1) *Load:* 1–1.5 mg/kg IV over 2–3 min; 2nd dose 5–30 min after 1st dose, 0.5–1.5 mg/kg to total 3 mg/kg; *Maintenance:* 15–50 mcg/kg/min IV (1–4 mg/min); (2) 1 mg/kg IV. 3.5 mg/kg max dose for infiltration or conduction block, (3) See Chapter 2C, on local anesthesia. **Peds:** (1) *Load:* 0.5–1 mg/kg IV, may repeat  $\times$  2 doses; *maintenance:* 15–50 mcg/kg/min IV. (2) 1 mg/kg IV.

**Mechanism:** Decreases conductance of sodium channels. Antiarrhythmic effect, suppresses automaticity of conduction tissue.

**Clearance:** Hepatic metabolism to active/toxic metabolites; renal elimination (10% unchanged). Decrease dose in pts with CHF, hepatic disease, shock.

**Comments:** May cause dizziness, seizures, disorientation, heart block (with myocardial conduction defect), hypotension, asystole, tinnitus, unusual taste, vomiting. Crosses the placenta. Therapeutic concentration = 1–5 mg/L. Caution in patients with Wolff–Parkinson–White syndrome, intraventricular heart block, pacemaker. Endotracheal dose 2–2.5  $\times$  IV dose diluted with 1–2 mL NS.

**Methylene Blue** (Methylthionine Chloride, Urolene Blue)**Indications:** (1) Surgical marker for genitourinary surgery; (2) methemoglobinemia.**Dose:** (1) 100 mg (10 mL of 1% solution) IV; (2) 1–2 mg/kg IV of 1% solution over 10 min; repeat q1h, prn.**Mechanism:** Low dose promotes conversion of methemoglobin to hemoglobin. High dose promotes conversion of hemoglobin to methemoglobin.**Clearance:** Tissue reduction; urinary and biliary elimination.**Comments:** Prolonged use may cause RBC destruction. May cause HTN, bladder irritation, nausea, diaphoresis. May inhibit nitrate-induced coronary artery relaxation. Transient (1–2 min) decrease in measured (not actual) pulse oximetry. Can cause hemolysis in patients with G6PD deficiency.**Methylprednisolone** (Solu-Medrol)**Dose:** **Adult:** 40–60 mg IV q6h. Higher doses in transplant patients. **Peds:** 0.16–0.8 mg/kg/d. Status asthmaticus: **Load:** 2 mg/kg; **Maintenance:** 0.5–1 mg/kg q6h. Spinal cord injury: **Load:** 30 mg/kg IV over 15 min; after 45 min begin. **Maintenance:** 5.4 mg/kg/hr × 23 or 47 hr.**Mechanism:** Suppresses migration of PMNs. Five times glucocorticoid potency of hydrocortisone. Almost no mineralocorticoid activity.**Clearance:** Hepatic metabolism; renal elimination (dose- and route-dependent).**Comments:** See hydrocortisone.**Naloxone** (Narcan)**Indications:** (1) opiate overdose; (2) reversal of opiate respiratory depression; **Dose:****Adult:** (1) 0.4–2 mg IV q2–3min prn; (2) 0.04- to 0.4-mg doses IV, titrated q2–3min. **Infusion:** Load 5 mcg/kg, infusion 2.5–160 mcg/kg/hr. **Peds:** (1) **birth–5 yr/0:** <20 kg: 0.1 mg/kg IV q2–3min prn; >5 yr/0 or >20 kg: 2 mg/dose q2–3min prn; **infusion** same as adult. (2) 1–10 mcg/kg IV titrated q2–3min (up to 0.4 mg).**Mechanism:** Competitive inhibition of opioid receptors.**Clearance:** Hepatic metabolism (95%); primarily renal elimination.**Comments:** May cause HTN, dysrhythmias, rare pulmonary edema, delirium, reversal of analgesia, or withdrawal syndrome (in opioid-dependent patients). Renarcotization may occur because antagonist has short duration. Caution in hepatic failure and chronic cardiac disease.**Octreotide** (Sandostatin)**Dose:** **Bolus** 25–50 mcg IV. **Infusion** 25–50 mcg/hr**Mechanism:** Somatostatin analogue that suppresses release of serotonin, gastrin, vasoactive intestinal peptide, insulin, glucagon, and secretin.**Clearance:** Hepatic and renal (32% eliminated unchanged); decreased in renal failure**Comments:** Indication upper GI bleeding, acute variceal hemorrhage. May cause nausea, decreased GI motility, transient hyperglycemia, cholelithiasis, abdominal pain, headache, pain at injection site. Duration of therapy should be no longer than 72 hr because of lack of documented efficacy beyond this time.**Omeprazole** (Prilosec)**Dose:** **Adult:** 20–40 mg PO qd. **Peds:** 0.6–0.7 mg/kg/dose qd; may increase to 0.6–0.7 mg/kg/dose bid to max 3.3 mg/kg/d**Mechanism:** Proton pump inhibitor. Inhibits H<sup>+</sup> secretion by irreversibly binding parietal H<sup>+</sup>/K<sup>+</sup> ATPase.**Clearance:** Extensive hepatic metabolism; 72% to 80% renal elimination; 18% to 23% fecal elimination.**Comments:** More rapid healing of gastric ulcers than with H<sub>2</sub> blockers. Effective in ulcers resistant to H<sub>2</sub> blocker therapy. Inhibits some cytochrome P-450 enzymes.**Phenoxybenzamine** (Dibenzyline)**Dose:** **Adult:** 10–40 mg/d PO (start at 10 mg/d and increase by 10 mg/d q4d prn).Usual dose 20–40 mg bid–tid. **Peds:** 0.2 mg/kg PO qd, max 10 mg; increase by 0.2 mg/kg to typical maintenance of 0.4–1.2 mg/kg/d q6–8h.**Mechanism:** Nonselective, noncompetitive, long lasting  $\alpha$ -adrenergic antagonist.**Clearance:** Hepatic metabolism; renal/biliary excretion.**Comments:** Indication for preoperative preparation for pheochromocytoma resection, tx of hypertensive crisis caused by sympathomimetic amines. May cause orthostatic hypotension (which may be refractory to norepinephrine), reflex tachycardia, syncope. Caution in pts with renal disease or coronary or cerebral artery diseases.

**Phentolamine** (Regitine)

**Indications:** (1) HTN from catecholamine excess as in pheochromocytoma. (2) Tx for extravasation of drugs with  $\alpha$ -adrenergic effects.

**Dose: Adult:** (1) 1–5 mg IV/IM q2–3h for HTN (5 mg IV/IM for diagnostic purposes). (2) 5–10 mg in 10 mL of NS infiltrated into affected area within 12 hr of extravasation; **Peds:** (1) 0.05–0.1 mg/kg/dose IV/IM 1–2 hr preprocedure q2–4h to max 5 mg; (0.05–0.1 mg/kg/dose IV/IM  $\times$  1 for diagnostic purposes); (2) 0.1–0.2 mg/kg diluted in 10 mL NS infiltrated into area of extravasation within 12 hr

**Mechanism:** Nonselective, competitive  $\alpha$ -adrenergic antagonist. Briefly antagonizes circulating epinephrine and norepinephrine to reduce HTN.

**Clearance:** Unknown metabolism; 10% renally eliminated (unchanged).

**Comments:** Indicated for diagnosis and tx of HTN associated with pheochromocytoma and tx. May cause hypotension, reflex tachycardia, cerebrovascular spasm, dysrhythmias, stimulation of gastrointestinal tract, or hypoglycemia. Caution in pts with renal disease or coronary or cerebral artery diseases.

**Phenytoin** (Dilantin)

**Indications:** (1) Status epilepticus; seizure prophylaxis. (2) Arrhythmias (digoxin-induced)

**Dose: Adult:** (1) *Load:* 10–20 mg/kg IV at  $<$ 50 mg/min (up to 1000 mg cautiously, with ECG monitoring); *Maintenance:* 300 mg/d or 5–6 mg/kg/d div q8h; for neurosurgical prophylaxis, 100–200 mg IV q4h (at  $<$ 50 mg/min); (2) 1.5 mg/kg or 50–100 mg IV at  $<$ 50 mg/min q5–15min until dysrhythmia is abolished, side effects occur, or a maximal dose of 10–15 mg/kg is given. **Peds/Neonates:** *Load:* 15–20 mg/kg mg IV; *Maintenance:* age-specific.

**Mechanism:** Anticonvulsant effect via membrane stabilization, inhibiting depolarization. Antidysrhythmic effect, blocking calcium uptake during repolarization prolonging refractory period.

**Clearance:** Hepatic metabolism; renal elimination (enhanced by alkaline urine).

**Comments:** Contraindicated in heart block, sinus bradycardia. May cause nystagmus, diplopia, ataxia, drowsiness, gingival hyperplasia, GI upset, hyperglycemia, or hepatic microsomal enzyme induction. Intravenous bolus may cause bradycardia, hypotension, respiratory arrest, cardiac arrest, or CNS depression. Venous irritant. Crosses the placenta. Significant interpatient variation in dose needed to achieve therapeutic concentration of 7.5–20.0 mcg/mL. Determination of unbound phenytoin levels may be helpful in patients with renal failure or hypoalbuminemia. Caution in renal and hepatic disease.

**Phosphorus** (Phospho-Soda, Neutra-Phos, Potassium Phosphate, Sodium Phosphate)

**Indications:** (1) Treatment and prevention of hypophosphatemia. (2) Bowel preparation

**Dose:** (1) *Mild to moderate hypophosphatemia:* **Pts  $>$ 4 yr/o and adults:** 250–500 mg (phosphorus) PO tid or 0.08–0.15 mmol/kg IV over 6 hr. *Moderate to severe:* **Pts  $>$ 4 yr/o and adults:** 0.15–0.25 mmol/kg IV over 6–12 h. (2) 45 mL diluted to 90 mL with water PO the evening prior to the examination and repeated the following morning. **Peds:**  $<$ 4 yr/o: (1) *Mild to moderate:* 250 mg (phosphorus) PO 3–4  $\times$ /d; *Moderate to severe:* 0.15–0.3 mmol/kg IV over 6 hr

**Mechanism:** Electrolyte replacement.

**Clearance:** Kidneys reabsorb 80% of dose.

**Comments:** Infuse doses of IV phosphate over 4–6 hr ( $<$ 3.1 mg/kg/hr); risks of rapid IV infusion include hypocalcemia, hypotension, muscular irritability, calcium deposition, deteriorating renal function, hyperkalemia. Orders for IV phosphate preparations should be written in mmol (1 mmol = 31 mg). Use with caution in patients with cardiac disease and renal insufficiency. Do not give with magnesium- and aluminum-containing antacids or sucralfate, which can bind with phosphate.

**Physostigmine** (Antilirium)

**Indications:** (1) Reversal of nondepolarizing neuromuscular blockade; (2) postoperative delirium, tricyclic antidepressant overdose, reversal of CNS effects of anticholinergic drugs.

**Dose:** (1) 0.01–0.03 mg/kg IV. (2) **Adult:** 0.5–2 mg IV/IM q15min prn until response or adverse effects. Repeat 1–4 mg q30–60min as symptoms recur. **Peds:** 0.01–0.03 mg/kg/dose IV q5–10min until response or adverse effects to max 2 mg

**Mechanism:** Prolongs central and peripheral cholinergic effects; inhibits cholinesterase

**Clearance:** Plasma esterases

**Comments:** Crosses blood–brain barrier, therefore useful for CNS anticholinergic toxicity. May cause bradycardia, tremor, convulsions, hallucinations, CNS depression,

mild ganglionic blockade, or cholinergic crisis. Crosses blood-brain barrier. Antagonized by atropine. Contains sulfite.

**Potassium Chloride (KCl)**

**Dose: Adult:** 20 mEq of KCl administered IV over 30–60 min. Usual infusion 10 mEq/hr. **Peds:** 0.5–1 mEq/kg/dose infused at 0.5 mEq/kg/hr (max 1 mEq/kg/hr).

**Mechanism:** Electrolyte replacement

**Clearance:** Renal

**Comments:** Indicated for hypokalemia, digoxin toxicity. IV bolus administration may cause cardiac arrest; Infusion rate should not exceed 1 mEq/min in adults. Central line preferred route for concentrated solutions. May cause pain/phlebitis at injection site.

**Scopolamine (Hyoscine)**

**Dose: Adult:** 0.3–0.6 mg IV/IM q6–8h, 1.5-mg transdermal patch. **Peds:** 6 mcg/kg/dose IM/IV/SC <q6–8h, max 0.3 mg/dose

**Mechanism:** Peripheral and central cholinergic (muscarinic) antagonist, antispasmodic, histamine and serotonin antagonist.

**Clearance:** Hepatic metabolism; renal elimination.

**Comments:** Used as antispasmodic, sedative, antiemetic, anti-motion sickness. Excessive CNS depression can be reversed by physostigmine. May cause excitement, delirium, transient tachycardia, hyperthermia, urinary retention, blurred vision, photophobia. Patch must be handled with care because contact with eyes may cause long-lasting mydriasis and cycloplegia. Crosses blood-brain barrier and placenta.



# PHARMACOLOGY: ANTIBIOTICS AND HERBAL MEDICINES

RICHARD D. URMAN • JESSE M. EHRENFELD

The following tables of spectra of activity for different antibiotics are generalizations. Sensitivity data at your own institution should be used to guide therapy.

Penicillins		
Generation	Properties	Spectrum
Natural	Some GPC, GPR, GNC, most anaerobes (except <i>Bacteroides</i> )	Group A streptococci Enterococci, <i>Listeria</i> <i>Pasteurella</i> <i>Actinomyces</i> , Syphilis
Anti-Staph	Active vs. PCNase-producing Staph Little activity vs. Gram $\ominus$	Staphylococci (except MRSA) Streptococci
Amino	Penetrates porin channel of Gram $\ominus$ Not stable against PCNases	<i>E. coli</i> , <i>Proteus</i> , <i>H. influenzae</i> <i>Salmonella</i> , <i>Shigella</i> Enterococci, <i>Listeria</i>
Extended	Penetrates porin channel of Gram $\ominus$ More resistant to PCNases	Most GNR incl. <i>Enterobacter</i> , <i>Pseudomonas</i> , <i>Serratia</i>
Carbapenem	Resistant to most $\beta$ -lactamases	Most gram $\oplus$ and $\ominus$ bacteria incl. anaerobes (except MRSA and VRE)
Monobactams	Active vs. Gram $\ominus$ but not Gram $\oplus$	Gram $\ominus$ bacterial infxn in Pt w/ PCN or Ceph allergy
$\beta$ -lact. Inhib.	Inhib. plasma-mediated $\beta$ -lactamases	Adds Staph, <i>B. fragilis</i> and some GNR ( <i>H. influenzae</i> , <i>M. catarrhalis</i> , some <i>Klebsiella</i> )

Cephalosporins		
Resistant to most $\beta$ -lactamases. No activity vs. MRSA or enterococci.		
Gen.	Spectrum	Indications
First	Most GPC (incl. Staph & Strep) Some GNR (incl. <i>E. coli</i> , <i>Proteus</i> , <i>Klebsiella</i> )	Used for surgical prophylaxis & skin infxns
Second	$\downarrow$ activity vs. GPC, $\uparrow$ vs. GNR. 2 subgroups: Respiratory: $\uparrow$ activity vs. <i>H. influenzae</i> & <i>M. catarrhalis</i> GI/GU: $\uparrow$ activity vs. <i>B. fragilis</i>	PNA/COPD flare  Abdominal infxns
Third	Broad activity vs. GNR and some anaerobes Ceftazidime active vs. <i>Pseudomonas</i>	PNA, sepsis, meningitis
Fourth	$\uparrow$ resistance to $\beta$ -lactamases (incl. of Staph and <i>Enterobacter</i> )	Similar to 3rd gen. MonoRx for nonlocalizing febrile neutropenia

Other Antibiotics	
Antibiotic	Spectrum
Vancomycin	Gram $\oplus$ bacteria incl. MRSA, PCNase-producing pneumococci and enterococci (except VRE)
Linezolid	
Daptomycin	GPC incl. MRSA & VRE
Quinopristin/ Dalfopristin	
Quinolones	Enteric GNR & atypicals. 3rd & 4th gen. $\uparrow$ activity vs. Gram $\oplus$ .
Aminoglycosides	GNR. Synergy w/ cell-wall active abx ( $\beta$ -lactam, vanco) vs. GPC. $\downarrow$ activity in low pH (e.g., abscess). No activity vs. anaerobes.
Macrolides	GPC, some respiratory Gram $\ominus$ , atypicals
TMP-SMZ	Some enteric GNR, PCP, <i>Nocardia</i> , <i>Toxoplasma</i> , most community-acquired MRSA
Clindamycin	Gram $\oplus$ (except enterococci) & anaerobes (incl. <i>B. fragilis</i> )
Metronidazole	Anaerobes (incl. <i>B. fragilis</i> )
Doxycycline	<i>Rickettsia</i> , <i>Ehrlichia</i> , <i>Chlamydia</i> , <i>Mycoplasma</i> , <i>Nocardia</i> , Lyme

<b>Penicillins</b>				
Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
<b>Natural penicillins</b>				
Penicillin G	0.4-4 MU IM/IV q4h	NC	NC	1-2 MU q4h
Penicillin V	250-500 mg PO q6h	NC	NC	NC
<b>Anti-staphylococcal</b>				
Dicloxacillin	250-500 mg PO q6h	NC	NC	NC
Nafcillin	1-2 g IM/IV q4h	NC	NC	NC
Oxacillin	1-2 g IM/IV q4h	NC	NC	NC
<b>Aminopenicillins</b>				
Amoxicillin	250-500 mg PO q8h	NC	250-500 mg q8-12h	250 mg q12h
Amox-clav	250-500 mg PO q8h	NC	250-500 mg q8-12h	250 mg q12h
Ampicillin	1-2 g IM/IV q4h	NC	1-2 g q8h	1-2 g q12h
Amp-sulbact	1.5-3 g IM/IV q6h	NC	1.5-3 g q12h	1.5-3 g q24h
<b>Extended-spectrum penicillins</b>				
Piperacillin	2-4 g IM/IV q4-6h	NC	2-4 g q8h	2-4 g q12h
Pip-tazo	3.375 g IV q6h	NC	2.25 g q6h	2.25 g q8h
Ticarcillin	2-4 g IM/IV q4h	NC	2-3 g q6h	2 g q12h
Ticar-clav	3.1 g IV q4h	NC	3.1 g q6h	2 g q12h
<b>Other <math>\beta</math>-lactams</b>				
Aztreonam	1-2 g IM/IV q8h	NC	1 g q8h	0.5 g q8h
Ertapenem	1 g IV/IM q24h	NC	0.5 g q24h (if CrCl <30)	
Imipenem	250-500 mg IV q6h	NC	250-500 mg q8-12h	250-500 mg q12h
Meropenem	1 g IV q8h	NC	0.5-1 g IV q12h	0.5 g IV q24h

<b>Macrolides</b>				
Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
Azithromycin	500 mg IV qd 500 mg PO on d 1, then 250 mg PO qd	NC	NC	NC
Clarithromycin	250-500 mg PO bid	? ↓	? ↓	? ↓
Erythromycin	0.5-1 g IV q6h 250-500 mg PO qid	NC	NC	250-500 mg IV or 250 mg PO q6h
Telithromycin*	800 mg PO qd	NC	600 mg qd (if CrCl <30)	

\*Ketolide (ketone derivative of macrolide nucleus)

<b>Tetracyclines</b>				
Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
Doxycycline	100 mg PO/IV q12-24h	NC	NC	NC
Tigecycline	100 mg IV $\times$ 1 then 50 mg q12h	NC	NC	NC

<b>Cephalosporins</b>				
<b>Antibiotic</b>	<b>Normal dose</b>	<b>Dose in renal failure (by GFR)</b>		
		<b>&gt;50</b>	<b>10-50</b>	<b>&lt;10</b>
<b>1st generation</b>				
Cefadroxil	0.5-1 g PO q12h	NC	0.5 g q12-24h	0.5 g q36h
Cefazolin	1 g IM/IV q8h	NC	1 g q12h	1 g q24h
Cephalexin	250-500 mg PO q6h	NC	NC	NC
<b>2nd generation</b>				
Cefaclor	250-500 mg PO q8h	NC	NC	NC
Cefotetan	1-2 g IM/IV q12h	NC	1-2 g q24h	1 g q24h
Cefoxitin	1-2 g IM/IV q4h	1-2 g q6h	1-2 g q8h	1 g q12h
Cefprozil	250-500 mg PO q12-24h	NC	NC	250 mg q12h
Cefuroxime	750-1500 mg IM/IV q6h	NC	750-1500 mg q8h	750 mg q24h
Loracarbef	200-400 mg PO q12h	NC	200 mg q12h	200 mg q3-5d
<b>3rd generation</b>				
Cefdinir	600 mg PO qd	NC	NC	300 mg qd
Cefditoren	200-400 mg PO bid	NC	200 mg bid	200 mg qd
Cefixime	400 mg PO q24h	NC	300 mg q24h	200 mg q24h
Cefoperazone	1-3 g IV q8h	NC	NC	NC
Cefotaxime	1-2 g IM/IV q6h	NC	NC	1-2 g q12h
Cefpodoxime	100-400 mg PO q12h	NC	NC	400 mg q24h
Ceftazidime	1-2 g IV q8h	NC	1-2 g q12h	1 g q24h
Ceftibuten	400 mg PO qd	NC	200 mg qd	100 mg qd
Ceftizoxime	1-2 g IV q6h	NC	1 g q12h	0.5 g q12h
Ceftriaxone	1-2 g IM/IV q12-24h	NC	NC	NC
<b>4th generation</b>				
Cefepime	1-2 g IM/IV q12h	NC	1-2 g q16-24h	1-2 g q24-48h

<b>Fluoroquinolones</b>				
<b>Antibiotic</b>	<b>Normal dose</b>	<b>Dose in renal failure (by GFR)</b>		
		<b>&gt;50</b>	<b>10-50</b>	<b>&lt;10</b>
<b>1st generation</b>				
Nalidixic acid	1 g PO qid	n/a	n/a	n/a
<b>2nd generation</b>				
Ciprofloxacin	500-750 mg PO q12h 200-400 mg IV q12h	NC	250-500 mg q12h	250-500 mg q24h
Lomefloxacin	400 mg PO qd	NC	200-400 mg qd	200 mg qd
Norfloxacin	400 mg PO q12h	NC	400 mg q12-24h	400 mg q24h
Ofloxacin	200-400 mg PO/IV q12h	NC	400 mg q24h	200 mg q24h
<b>3rd generation</b>				
Levofloxacin	250-500 mg PO/IV q24h	NC	250 mg q24h	250 mg q48h
<b>4th generation</b>				
Gatifloxacin	400 mg PO/IV qd	NC	200 mg qd	200 mg qd
Gemifloxacin	320 mg PO qd	NC	160 mg qd	
Moxifloxacin	400 mg PO qd	NC	NC	NC

<b>Aminoglycosides</b>				
<b>Antibiotic</b>	<b>Normal dose</b>	<b>Dose in renal failure (by GFR)</b>		
		<b>&gt;50</b>	<b>10-50</b>	<b>&lt;10</b>
Gentamicin	1-1.7 mg/kg q8h	60-90%	30-70%	20-30%
Tobramycin		q8-12h	q12-18h	q24-48h
		or ~1-1.7 mg/kg q(8 × serum Cr)h		
Amikacin	5 mg/kg q8h	60-90%	30-70%	20-30%
		q8-12h	q12-18h	q24-48h
		or ~5 mg/kg q(8 × serum Cr)h		

<b>Other Antibiotics</b>				
<b>Antibiotic</b>	<b>Normal dose</b>	<b>Dose in renal failure (by GFR)</b>		
		<b>&gt;50</b>	<b>10-50</b>	<b>&lt;10</b>
Chloramphenicol	0.5-1 g IV/PO q6h	NC	NC	NC
Clindamycin	600 mg IV q8h 150-300 mg PO qid	NC	NC	NC
Daptomycin	4 mg/kg IV q24h	NC	4 mg/kg q48h (if CrCl <30)	
Linezolid	400-600 mg IV/PO q12h	NC	NC	NC
Metronidazole	1000 mg load then 500 mg IV/PO q6h	NC	NC	NC
Nitrofurantoin	50-100 mg PO qid	NC	avoid	avoid
Quinopristin/ Dalfopristin	7.5 mg/kg IV q8-12h	NC	NC	NC
TMP-SMX*	2-5 mg TMP/kg PO/IV q6h	NC	2-5 mg TMP/kg q12h	avoid
Vancomycin	1 g IV q12h	NC	1 g q24-72h ✓ trough (goal 5-10, ? <15 for some infxns) adjust dose & interval	

(\*Single strength tablet = 1 ampule = 80 mg of TMP + 400 mg SMX).

## HERBAL MEDICINES AND SIDE EFFECTS

<b>Name of Herb</b>	<b>Common Uses</b>	<b>Possible Side Effects or Drug Interactions</b>
Echinacea	Boosts the immune system and helps fight colds and flu, aids wound healing.	May cause inflammation of the liver if used with certain other medications such as anabolic steroids, methotrexate or others.
Ephedra	Used in many over the counter diet aids as an appetite suppressant; also for asthma or bronchitis.	May interact with certain antidepressant medications or certain high blood pressure medications to cause dangerous elevations in blood pressure or heart rate. Could cause death in certain individuals.
Feverfew	Used to ward off migraine headaches and for arthritis, rheumatic disease and allergies.	May increase bleeding, especially in patients already taking anticlotting medications.

<b>Name of Herb</b>	<b>Common Uses</b>	<b>Possible Side Effects or Drug Interactions</b>
GBL, BD, and GHB	Bodybuilding, weight loss aid and sleep aid.	These are abbreviations for illegally distributed, unapproved drugs (not supplements) that may cause death, seizures or unconsciousness.
Garlic	For lowering cholesterol, triglyceride levels and blood pressure.	May increase bleeding, especially in patient already taking certain anticlotting medications. May decrease effectiveness of certain AIDS-fighting drugs, e.g., saquinavir.
Ginkgo (also called ginkgo biloba)	For increasing blood circulation and oxygenation and for improving memory and mental alertness.	May increase bleeding, especially in patients already taking certain anticlotting medications.
Ginseng	Increased physical stamina and mental concentration.	May increase bleeding, especially in patients already taking certain anticlotting medications. May see increased heart rate or high blood pressure. May cause bleeding in women after menopause.
Goldenseal	Used as a mild laxative and also reduces inflammation.	May worsen swelling and/or high blood pressure.
Kava-kava	For nervousness, anxiety or restlessness; also a muscle relaxant.	May increase the effects of certain antiseizure medications and/or prolong the effects of certain anesthetics. May cause serious liver injury. May worsen the symptoms of Parkinson's disease. Can enhance the effects of alcohol. May increase the risk of suicide for people with certain types of depressions.
Licorice	For treating stomach ulcers.	Certain licorice compounds may cause high blood pressure, swelling or electrolyte imbalances.
Saw palmetto	For enlarged prostate and urinary inflammation.	May see effects with other hormone therapies.
St. John's wort	For mild to moderate depression or anxiety and sleep disorders.	May decrease effectiveness of all currently marketed HIV protease inhibitors and nonnucleoside reverse transcriptase inhibitors (powerful AIDS-fighting drugs). May possibly prolong effects of anesthesia (not proven). May unknowingly decrease levels of digoxin, a powerful heart medication.

(continued)

<b>Name of Herb</b>	<b>Common Uses</b>	<b>Possible Side Effects or Drug Interactions</b>
Valerian	Mild sedative or sleep-aid; also a muscle relaxant.	May increase the effects of certain antiseizure medications or prolong the effects of certain anesthetic agents.
Vitamin E	Used to prevent stroke and blood clots in the lungs. Also used to slow the aging process and for protection against environment pollution.	May increase bleeding, especially in patients already taking certain anticlotting medications. May affect thyroid gland function in otherwise healthy individuals. In doses higher than 400 IU per day, may cause problems with increased blood pressure in people who already have high blood pressure.

From: American Society of Anesthesiologists. What you should know about Herbal and Dietary Supplement Use and Anesthesia; 2003.

## GAS SUPPLY

### Medical Gases (O<sub>2</sub> & N<sub>2</sub>O):

- Oxygenate patient, serve as media to deliver volatile anesthetics, drive ventilator

### O<sub>2</sub> & N<sub>2</sub>O have Two Separate Supply Sources:

- Pipeline supply (50 psi) = main supply source for anesthesia machine
- Cylinder supply = backup source (in event of pipeline failure)

→ O<sub>2</sub> cylinder (size E) pressure proportional to volume

*Example of calculating remaining volume: if gauge reads 1000 psi,  
liters O<sub>2</sub> left = 1000 psi/2200 psi × 660 L = 300 L*

*At 5 L/min flows, 300/5 = 60 min remaining*

→ N<sub>2</sub>O cylinder (size E) pressure remains at 750 psi until all liquid vaporized; pressure starts to fall when ≈25% of contents remain

### Properties of Compressed Gases in E Cylinders

Characteristic	Oxygen	Nitrous Oxide	Air
Color	Green	Blue	Yellow
Physical state	Gas	Liquid and gas	Gas
Volume (L)	660	1590	625
Pressure full (psi)	2200	750	1800

## FLOW-CONTROL CIRCUITS IN ANESTHESIA MACHINE

### Pressure Regulators

- Reduce pipeline & cylinder tank pressures to safe & consistent levels before delivery to pt

### O<sub>2</sub> Supply Failure Protection Devices

- Minimize likelihood of delivering hypoxic gas mixtures
- Prevent delivery of N<sub>2</sub>O without concomitant O<sub>2</sub> flow
- Alarm sounds when inlet gas pressure drops below threshold (usu. 20–30 psi)

### Flow Valves & Meters

- Control amount of gases delivered to breathing limb
- Flow-control valves divide anesthesia machine into two parts
  - High-pressure circuit (upstream to flow valves)
  - Low-pressure circuit (downstream to flow valves)
- Second-stage regulator = downregulates O<sub>2</sub> supply (pipeline or cylinder) to a precise pressure (usu. 14 psi) to ensure that a constant O<sub>2</sub> pressure is supplied
- Fail-safe valve = 1st & primary interface between O<sub>2</sub> & N<sub>2</sub>O supply; will either shut off or proportionally ↓ N<sub>2</sub>O flow in event of O<sub>2</sub> supply failure
- Check valves = one-way valves to prevent retrograde flow into vaporizers
- O<sub>2</sub> & N<sub>2</sub>O flowmeters are mechanically linked & O<sub>2</sub> flowmeters always located downstream (closest to pt), so in case of leak, risk of hypoxic delivery is minimized

### Vaporizers

- Convert liquid volatile anesthetic to gas
  - Agent-specific; filling vaporizer with wrong agent may lead to incorrect dosing: placing inhalational agent with higher vapor pressure (VP) into vaporizer used for agent with lower VP → overdose
  - VP determined by temperature and physical properties of liquid
- Deliver constant agent conc. independent of temp, flow, altitude
- Variable bypass: (total gas flow divided into two portions)
  - Carrier gas (flows over liquid anesthetic in vaporizing chamber & saturates with anesthetic at conc. based on control knob)
  - Balance gas (exits vaporizer unchanged)
  - Two flows mix & exit machine via common fresh gas outlet
- Tipping of vaporizer → ↑↑ output → overdose
- At very low/very high flows → output is < dial setting
- "Pumping effect": Gas is compressed by back pressure during positive-pressure ventilation/or use of O<sub>2</sub> flush valve → ↑↑ output
- Desflurane vaporizer (Tec 6)
  - Desflurane vapor pressure so high → almost boils at room temp

- Vaporizer heats liquid above boiling point, allows reliable delivery
- At higher altitude, need to dial up (otherwise you underdose)
- National Institute of Occupational Safety and Health (NIOSH) recommendations: halogenated agents when used alone = 2 parts per million (ppm) in the ambient air; with N<sub>2</sub>O = 0.5 ppm. N<sub>2</sub>O used alone = 25 ppm

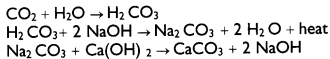
### Common Fresh Gas Outlet

- Multiple gas inlets into machine but only one common gas outlet out of machine
- Supplies gas & anesthetic vapor to breathing circuit
- O<sub>2</sub> flush valve precedes common gas outlet
  - Provides high-flow (50 L/min) O<sub>2</sub> bypassing flowmeters & vaporizers

### BREATHING CIRCUIT: CONNECTS ANESTHESIA MACHINE TO PATIENT

**Circle System:** Most commonly used, components arranged in a circle prevents rebreathing of exhaled CO<sub>2</sub>

- Adjustable pressure limiting valve (APL valve or pop off valve):
  - Can be adjusted to coordinate manual bag compression to assist or take over ventilation of pts lungs by manual bag compression → allows venting of excess gas into waste scavenging system
- Bag/vent switch: Will exclude/include gas reservoir bag & APL from system
- Reservoir bag: Maintains reserve volume of gas
  - Inspiratory flow rate can be up to 60 L/min
- Inspiratory one-way valve: Open during inspiration/closed during expiration
  - Prevents expiratory gas from mixing with fresh gas in inspiratory limb
- Expiratory one-way valve: Open during expiration/closed during inspiration
  - Gas is then either vented through APL valve or passes to CO<sub>2</sub> absorber
- CO<sub>2</sub> absorbent: removes CO<sub>2</sub> from breathing circuit (chemical neutralization)
  - Most common absorbent = soda lime (Ca,Na,K-hydroxide + H<sub>2</sub>O)



- Oxygen analyzer: Determines inspired & expired O<sub>2</sub> conc.
- Spirometer: Measures exhaled tidal volume & respiratory rate
- Circuit pressure gauge: Measures circuit airway pressure in cm H<sub>2</sub>O

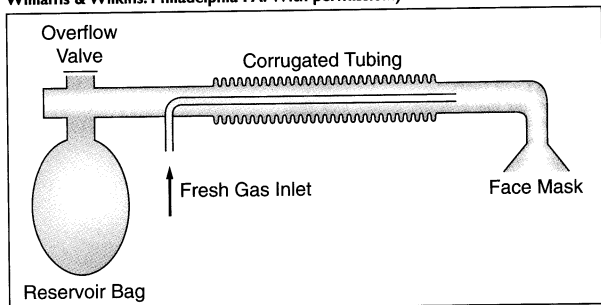
### Open Breathing Systems (not typically used in modern medicine)

- Insufflation = blowing of anesthetic gas across person's face
- Open drop anesthesia = volatile anesthetic dripped onto gauze covered mask applied to pt's face

### Mapleson A to E: 5 Systems Described in 1950s

- Differ in arrangement of fresh gas inflow tubing, reservoir tubing, location of face mask, reservoir bag, & expiratory valve
- Characterized by (1) absence of valves directing gases to and from pt & (2) absence of CO<sub>2</sub> neutralization
- Mapleson **A** circuit = most efficient for spontaneous ventilation (fresh gas flow = minute ventilation; which is sufficient to prevent CO<sub>2</sub> rebreathing)

**Figure 3-1 Bain circuit.** (From Barash DG. *Clinical Anesthesia*, 5th ed. Lippincott Williams & Wilkins: Philadelphia PA. With permission.)





- Mapleson D circuit = most efficient for controlled ventilation (fresh gas flow forces alveolar air away from pt & toward pressure release valve)

### Bain Circuit: Modification of Mapleson D

- Fresh gas supply runs coaxially inside corrugated expiratory tubing
- Advantages: ↓ circuit bulk, retains humidity better than Mapleson D
- Disadvantages: kinking/disconnect of coaxial tubing (i.e., fresh gas inlet)

### BAIN CIRCUIT

Closed-circuit anesthesia involves (1) total rebreathing of exhaled gases after absorption of carbon dioxide and (2) APL valve or ventilator relief valve closed

- Advantages: ↑ Humidification of gases, less pollution
- Disadvantages: Inability to rapidly change gas conc., may provide hypoxic/hypercarbic mix, excessive gas conc.

### ROUTE OF OXYGEN MOLECULE FROM HOSPITAL SUPPLY TO PATIENT

main hospital supply to OR → O<sub>2</sub> pipeline supply inlet (50 psi) → into machine → O<sub>2</sub> pressure regulator(s) → flow control valve → calibrated vaporizer (saturating with anesthetic) → check valve → common gas outlet → breathing circuit inspiratory limb → inspiratory one-way valve → endotracheal tube → patient

### VENTILATORS

- Most ventilators pneumatically driven by O<sub>2</sub> (double-circuit ventilators)
  - Driving gas circuit: Drives force for ventilator bellows & machine
  - Patient gas circuit: Gas supply to pt
- Inspiration: Space inside rigid container containing compressible bellows hit with pressurized O<sub>2</sub> from driving circuit → bellows empty
- Exhalation:
  - Ascending bellows: Rise during exhalation (preferred)
  - Bellows will not fill or rise if circuit disconnect or leak occurs
  - If hole develops in bellows, high gas pressure can be transmitted to pt airway (risk of barotraumas); will be indicated by ↑ FiO<sub>2</sub>
  - Descending bellows: Hang during exhalation
  - Will fill by gravity even in presence of circuit disconnect or leak

### Pressure & Volume Monitoring

- Peak inspiratory pressure (PIP) = highest pressure in circuit during inspiration
- Plateau pressure = pressure measured during inspiratory pause (no gas flow)

Causes of Increased Peak Inspiratory Airway Pressure (PIP)	
<b>↑ PIP &amp; ↑ Plateau Pressure</b> ↑ Tidal volume ↓ Pulmonary compliance • Mechanical causes: • Abdominal gas insufflation • Trendelenburg position • Abdominal packing • Endobronchial intubation • Physiologic causes: • Pulmonary edema • Pleural effusion • Ascites • Tension pneumothorax	<b>↑ PIP &amp; Unchanged Plateau Pressure</b> ↑ Inspiratory gas flow rate ↑ Airway resistance • Mechanical causes: • Kinked endotracheal tube • Airway compression • Foreign body aspiration • Physiologic causes: • Bronchospasm • Secretions

### Machine Leak Tests

- **Performing low-pressure system leak test:**
  1. Verify machine master switch & flow control valves are off
  2. Attach suction bulb to common (fresh) gas outlet
  3. Squeeze bulb repeatedly until fully collapsed
  4. Verifying bulb stays fully collapsed for at least 10 sec
  5. Open one vaporizer at a time & repeat steps 3 & 4
  6. Remove suction bulb & reconnect fresh gas hose
- **Performing breathing system leak test:**
  1. Set all gas flows to zero (or minimum)
  2. Close APL (pop-off valve) & occlude Y-piece
  3. Pressurize breathing system to about 30 cm H<sub>2</sub>O with O<sub>2</sub> flush

4. Ensure pressure remains fixed for at least 10 sec
5. Open APL (pop-off valve) & ensure pressure drops

### Problems Associated With Anesthesia Ventilators

- Ventilator—fresh gas flow (FGF) coupling:
  - High fresh FGF can: ↑ tidal volume above setting, ↑ min. ventilation, ↑ PIPs
- Hole in bellows:
  - Hyperventilation/barotrauma due to delivery of high driving gas pressures to pt
- Excessive positive pressure:
  - Intermittent/sustained high inspiratory pressures ↑ risk of barotrauma (*do not use O<sub>2</sub> flush valve when ventilator is on → risk of barotrauma*)
- Tidal volume discrepancies:
  - Difference between set & actual volumes due to breathing circuit compliance, gas compression, ventilator fresh gas flow coupling, leaks
- Sources of disconnects:
  - All connection sites, loose or cracked bellows, incompetent system components (including scavenger system, pop-off valve)

### Ventilator Alarms

- "Disconnect" alarms: Low PIP, low exhaled tidal volume, low EtCO<sub>2</sub>
- Additional alarms: High PIP, high PEEP, sustained high airway pressure, negative pressure, & low O<sub>2</sub>
- Consider disconnect/leak when capnograph shows decrease or loss of EtCO<sub>2</sub>

### Waste Gas Scavengers

Prevent pollution of OR with anesthetic gases; components include

- Waste gas collecting assembly
- Transfer tubing
- Waste gas scavenging interface, can be open or closed to the room:
  - Open*—does not require pressure-relief valve
  - Closed*—requires pressure relief valve
- Gas disposal tubing
- Disposal assembly, can be:
  - Passive*—disposal tubing directed outside via ventilation duct
  - Active*—disposal tubing connected to hospital vacuum system

Note: Must adjust vacuum control valve to ↓ risk of transmitting negative pressure to pt during low flows

## ELECTRICAL SAFETY IN THE OR

### Electrosurgery

- Electrosurgery & surgical diathermy:
  - High frequency alternating current to cut or cauterize small blood vessels
- Electrosurgical units (ESUs) generate high-frequency current
  - Tip of small electrode → through pt → out large electrode (grounding pad)
- Malfunction of grounding pad: Inadequate contact/conducting gel/disconnect current will exit pt through alternate path (ECG pads, OR table) → may cause burn
- Bipolar electrodes limit current propagation to few millimeters
- ESU may interfere with pacemaker & ECG recordings

### Risk of Electrocutation

- Body contact with two conductive materials at different voltage potentials may complete circuit & result in electric shock
- Leakage current present in all electrical equipment
  - Fibrillation threshold is 100 milliamps (above leakage current magnitude)
- Current applied to heart (bypassing high resistance offered by skin) may be fatal
  - Even as low as 100 microamps

Macroshock (current applied outside body)	Microshock (current applied inside body)
1 milliamp = perception	10 microamps = max leakage current
10 milliamps = one would let go	
100 milliamps = cause V-fib	100 microamps = cause V-fib

### Ungrounded Power & Protection From Electric Shock

- Isolation transformer: Isolates OR power supply from grounds
  - If live wire unintentionally contacted by grounded pt, isolation transformer prevents current flow through pt

- Line isolation monitors (LIMs) measure potential for current flow from isolated power supply to ground
- Alarm sounds if unacceptable current flow to ground becomes possible
- Alarm *does not* interrupt power unless ground leakage circuit breaker activated
- Even isolated power circuits do not protect against *microshock*
- Note: New building codes no longer required ORs to have ungrounded power

## **STANDARDS FOR BASIC ANESTHETIC MONITORING (ADOPTED BY THE ASA)**

- Best monitor is a vigilant anesthesiologist; anesthesia personnel must be in room throughout all general/regional/monitored anesthetics
- During all anesthetics, patients oxygenation, ventilation, circulation, & temp should be continuously monitored

### **Oxygenation**

- Inspired gas oxygen conc. must be measured
- Blood oxygenation must be measured such as with pulse oximetry

### **Ventilation**

- Adequacy of ventilation must be evaluated with presence of CO<sub>2</sub> in exhaled gas
- Position of ETT or LMA must be verified with presence of CO<sub>2</sub> in exhaled gas
- Use of ventilator disconnect alarms must be employed

### **Circulation**

- ECG should be continuously displayed from beginning of anesthesia through patient leaving room
- BP & heart rate should be monitored and recorded at least every 5 min

### **Temperature**

- Temp should be monitored when significant changes in body temp anticipated/ intended

# AIRWAY MANAGEMENT

TARUN BHALLA

AIRWAY 4-1

<b>Airway Anatomy</b>	
<b>Pharynx</b>	Divided into nasopharynx, oropharynx, & laryngopharynx
<b>Epiglottis</b>	Separates laryngopharynx into hypopharynx (to esophagus) & larynx (to trachea)
<b>Larynx</b>	(C4–C6); laryngeal skeleton consists of 9 cartilages: 3 paired (corniculates, arytenoids, cuneiforms) & 3 unpaired (epiglottis, thyroid, cricoid); protects entrance of respiratory tract & allows phonation
<b>Thyroid cartilage</b>	Largest & most prominent for lateral & anterior walls
<b>Cricothyroid membrane</b>	Connects thyroid & cricoid cartilage; ≈ 1 to 1.5 fingerbreadths below laryngeal prominence; any incisions/needle punctures should be made in inferior third & directed posteriorly (due to cricothyroid arteries & vocal folds)
<b>Cricoid cartilage</b>	(C5–C6); shaped like signet ring, inferior to thyroid cartilage only complete cartilaginous ring along laryngotracheal tree
<b>Arytenoids</b>	Originate on posterior aspect of larynx & posterior attachments of vocal cords; may be only visible structures in pts with an "anterior" airway
<b>Laryngeal muscles</b>	Lateral cricoarytenoid (adduction), posterior cricoarytenoid (abduction), transverse arytenoids → open/close the glottis, cricothyroid, thyroarytenoid, vocalis → control vocal ligament tension

<b>Airway Innervation—Sensory</b>	
Glossopharyngeal nerve (CN IX)	Posterior third of tongue, oropharynx from nasopharyngeal surface to junction of pharynx & epiglottis, including vallecula; tonsillar area; gag reflex
Superior laryngeal nerve, internal branch (CN X/vagus)	Mucosa from epiglottis to vocal cords (sensory innervation of larynx above vocal cords), including base of tongue, supraglottic mucosa, cricothyroid joint
Superior laryngeal nerve, external branch (CN X/vagus)	Anterior subglottic mucosa
Recurrent laryngeal nerve (CN X/vagus)	Subglottic mucosa, muscle spindles
Trigeminal nerve (CN V)	Nares & nasopharynx

<b>Airway Innervation—Motor</b>	
Superior laryngeal nerve, external branch (CN X/vagus)	Cricothyroid muscles → tensing of vocal cords, inferior pharyngeal constrictors
Recurrent laryngeal nerve (CN X/vagus)	All other intrinsic muscles of larynx: thyroarytenoid, lateral cricoarytenoid, interarytenoid, posterior cricoarytenoid
Glossopharyngeal (CN IX) & superior laryngeal, internal branch (CN X/vagus)	No motor innervational contribution

Note: all laryngeal innervation is by 2 branches of vagus: superior laryngeal & recurrent laryngeal nerves.

- Injury of SLN (external branch) → hoarseness
- Injury of RLN → unilateral paralysis → paralysis of ipsilateral vocal cord → hoarse voice; bilateral paralysis → stridor & respiratory distress

## Airway Assessment

- History
  - Adverse events related to prior airway management
  - Radiation/surgical history
  - Burns/swelling/tumor/masses

- Obstructive sleep apnea (snoring)
- Temporomandibular joint dysfunction
- Dysphagia
- Problems with phonation
- C-spine disease (disk dz, osteoarthritis, rheumatoid arthritis, Down's syndrome)
- Physical examination
  - Mallampati score (see *Chapter 1, on preoperative assessment*)
  - Symmetry of mouth opening (3 fingerbreadths)
  - Loose/missing/cracked/implanted teeth
  - Macroglossia (associated with difficult laryngoscopy)
  - High-arched palate (associated with difficulty visualizing larynx)
  - Mandible size
    - Thyromental distance <3 fingerbreadths suggests poor laryngeal visualization
- Neck examination
  - Prior surgeries/tracheostomy scars
  - Abnormal masses (hematoma, abscess, goiter, tumor) or tracheal deviation
  - Neck circumference & length
  - Range of motion (flexion/extension/rotation)

### Signs of a Potentially Difficult Airway

- |   |  |
|---|--|
| • Abnormal face shape                                   | • Narrow mouth   |
| • Sunken cheeks   | • Obesity  |
| • Edentulous  | • Receding mandible  |
| • "Buck teeth"  | • Facial/neck pathology                                    |
| • Mouth opening <3 fingerbreadths                       | • Thyroid cartilage-mouth floor distance <2 fingerbreadths |
| • Hyoid-chin distance <3 fingerbreadths                 |  |
| • Mallampati Classes III & IV                           |  |
| • Pathology around upper airway (peritonsillar abscess) |  |
| • Limited range of motion                               |  |

### Regional vs General Anesthesia in Difficult Airway Patients

Consider Regional	Do Not Consider Regional
Superficial surgery	Invasive surgery
Minimal sedation needed	Significant sedation required
Anesthetic may be provided with local	Extensive local will be required or risk of intravascular injection is high
Good airway access	Poor airway access
Surgery may be stopped at any time	Surgery cannot be stopped after start

Source: Adapted from Barash PG, et al. *Clinical Anesthesia*, 5th ed. Lippincott Williams & Wilkins, Philadelphia, 2005. With permission.

### Airway Devices

- Oral and nasal airways
  - Typically inserted secondary to loss of upper airway muscle tone in anesthetized patients → usually caused by tongue or epiglottis falling against posterior pharyngeal wall
  - Length of nasal airway estimated by measuring from nares to meatus of ear
  - Use caution with insertion in pts on anticoagulation or with basilar skull fractures
- Mask airway
  - Facilitates O<sub>2</sub> delivery (denitrogenation) as well as anesthetic gas using airtight seal
  - Hold mask with left hand while right hand generates positive-pressure ventilation → (use < 20 cm H<sub>2</sub>O to avoid gastric inflation)
  - One-handed technique
    - Fit snugly around bridge of nose to below bottom lip
    - Downward pressure with left thumb & index finger, middle & ring finger; grasp the mandible while pinky finger is placed under angle of jaw to thrust anteriorly
  - Two-handed technique
    - Used in difficult ventilatory situations
    - Bilateral thumbs hold mask down while fingertips displace jaw anteriorly
- Edentulous patients may be a challenge to ventilate (difficult to create a mask seal) → consider leaving dentures in place, oral airway, buccal cavity gauze packing

- **Difficult mask ventilation: maneuvers to maintain airway patency**
  - Call for additional help (have someone else squeeze bag)
  - Insert oral and or nasal airways
  - Extend neck & rotate head
  - Perform jaw thrust

#### Independent Risk Factors for Difficult Mask Ventilation

- Presence of a beard
- Body mass index >26 kg/m<sup>2</sup>
- Lack of teeth
- Age >55
- History of snoring

Source: Langeron, O. Prediction of difficult mask ventilation. *Anesthesiology* 2000; 92:1229.

- Supraglottic airways (laryngeal mask airways)
  - Insertion technique:
    - Patient placed in sniffing position
    - Deflated LMA cuff is lubricated & inserted blindly to hypopharynx
    - Cuff is inflated to create a seal around entrance to larynx  
(*Tip rests over upper esophageal sphincter, cuff upper border against base of tongue, sides lying over pyriform fossae*)
  - Indications
    - Alternative to endotracheal intubation (not as a replacement) or mask ventilation
    - Rescue device in expected/unexpected difficult airway
    - Conduit for intubating stylet, flexible FOB, or small diameter ET
  - Contraindications: Pharyngeal pathology, obstruction, high aspiration risk, low pulmonary compliance (need peak inspiratory pressures >20 cm H<sub>2</sub>O), long surgeries
  - Disadvantages: do not protect the airway, can become dislodged

#### Laryngeal Mask Airway Models

Type	Description	Advantage
Disposable LMA	Most commonly used. Adults: Size #3–5	Alternative to ET intubation, useful in unexpectedly difficult airways
Flexible LMA	Thin-walled, small-diameter, wire-reinforced barrel that can be positioned out of midline	Kink-resistant
ProSeal LMA	Includes a gastric drain, posterior cuff to allow positive-pressure ventilation with 40 cm H <sub>2</sub> O	Allows positive-pressure ventilation, protection from aspiration
Fastrach LMA	Cuff, epiglottic elevating bar, airway tube, handle, flexible ETT	Allows blind intubation in difficult airways ± fiberoptic

- Endotracheal tubes
  - Used to deliver anesthetic gas directly to trachea & provide controlled ventilation
  - Modified for a variety of specialized applications: Flexible, spiral-wound, wire-reinforced (armored), rubber, microlaryngeal, oral/nasal RAE (preformed), double-lumen tubes
  - Airflow resistance depends on tube diameter, curvature, length
  - All endotracheal tubes have an imprinted line that is opaque on radiographs

#### Oral Tracheal Tube Sizing

Age	Internal Diameter (mm)	Tube Length at Lip (cm)
Full-term infant	3.5	12
Child	4 + age/4	14 + age/2
Adult: female	7.0–7.5	20
male	7.5–8.5	22

- Rigid laryngoscopes: Used to examine larynx & facilitate tracheal intubation
  - Macintosh blade (curved): Tip inserted into vallecula; use size 3 blade for most adults
  - Miller blade (straight): Tip inserted beneath laryngeal surface of epiglottis; use size 2 blade for most adults
  - Modified laryngoscopes: Wu, Bullard, & Glidescope for use in difficult airways

- Flexible fiberoptic bronchoscopes
  - Indications: Potentially difficult laryngoscopy/mask ventilation, unstable cervical spines, poor cervical range of motion, TMJ dysfunction, congenital/acquired upper airway anomalies
- Light wand
  - Malleable stylet with light emanating from distal tip, over which ETT is inserted
  - Dim lights in OR & advanced wand blindly
    - Glow in lateral neck → tip in piriform fossa
    - Glow in the anterior neck → correctly positioned in trachea
    - Glow diminishes significantly → tip likely in esophagus
- Retrograde tracheal intubation
  - Performed in awake & spontaneously ventilating pts
  - Puncture cricothyroid membrane with 18-gauge needle
  - Introduce guidewire & advanced cephalad (80 cm, 0.025 in.)
  - Visual wire with direct laryngoscopy & guide ETT through vocal cords
- Airway bougie
  - Solid or hollow, semimalleable stylets usually passed blindly into trachea
  - ETT is threaded over bougie into trachea; can feel "clicking" as passes over tracheal rings
  - May have internal lumen to allow for insufflation of O<sub>2</sub> & detection of CO<sub>2</sub>

### Required Equipment for Intubation

O<sub>2</sub>, positive-pressure ventilation source (ventilator) & backups (bag-valve-mask/E-cylinder)

Face masks

Oropharyngeal & nasopharyngeal airways

Tracheal tubes & stylets

Syringe (10 mL) for inflation of tracheal tube cuff

Suction

Laryngoscope handles

Laryngoscope blades (Mac & Miller)

Pillow, towel, blanket for pt positioning

Stethoscope

Capnograph or end-tidal CO<sub>2</sub> detector

### Airway Management: Orotracheal Intubation

- Elevate height of bed to laryngoscopist's xiphoid process
- Place patient in *sniffing position*: neck flexion, head extension; aligns oral, pharyngeal, & laryngeal axes to provide the straightest view from lips to glottis
- Preoxygenate with 100% O<sub>2</sub>
- Induce anesthesia
- Tape pt's eyes shut to prevent corneal abrasions
- Hold laryngoscope in left hand, scissoring mouth with right thumb & index finger
  - Insert laryngoscope in right side of mouth, sweeping tongue to left
  - Advance until glottis appears in view
  - Do not use laryngoscope as a lever in a pivoting maneuver (instead lift "up and away")
- Using the right hand, pass the tip of the ETT through vocal cords under direct visualization
- Inflate ETT cuff with least amount of air necessary to create seal during positive-pressure ventilation
- Confirm correct placement of ETT with (1) Chest auscultation, (2) ETCO<sub>2</sub>, (3) ETT condensation, (4) palpation of ETT cuff in sternal notch
 

*Earliest manifestation of bronchial intubation is ↑ peak pressure (right mainstem bronchus common)*
- **Rapid sequence intubation**
  - Indication: pts at ↑ risk for aspiration (full stomach, pregnant, GERD, morbidly obese, bowel obstruction, delayed gastric emptying, pain, diabetic gastroparesis)
  - Use rapid paralyzing agent: succinylcholine (1–1.5 mg/kg) or rocuronium (0.6–1.2 mg/kg)
  - Place cricoid pressure (Sellick maneuver) as pt is induced
    - Protect from regurgitation of gastric contents to oropharynx
    - Help visualize vocal cords during laryngoscopy

- Intubate pt once paralytic takes effect (30–60 sec); do **not** ventilate pt during this time
- Proper cricoid pressure should be performed with “BURP” technique:
  - Displace larynx (**B**)ackward, (**U**)pward, (**R**)ight, with (**P**)ressure
- **“Modified” rapid sequence intubation**
- A variety of modifications to standard rapid sequence induction, no standard definition
- May include standard RSI with use of nondepolarizing agent (pts with ↑ K<sup>+</sup>) and establishment of mask airway prior to initiation of paralysis with succinylcholine

### Airway Management: Nasotracheal Intubation

- Indications: intraoral, facial/mandibular procedures
- Contraindications: basilar skull fractures, nasal fractures or polyps, underlying coagulopathies
- Preparation: anesthetize & vasoconstrict mucosa with lidocaine/phenylephrine mix or cocaine → select nares that pt can breath through most easily
- Lubricated ETT is advanced perpendicular to face below inferior turbinate via selected nares → direct bevel laterally away from turbinates
- Advance ETT until able to visualize tip in oropharynx under direct laryngoscopy → use Magill forceps with right hand to advance/direct through vocal cords

### Airway Management: Awake Flexible Fiberoptic Intubation

- **Equipment:** Ovassapian/Williams/Luomanen airway, topical anesthetics, vasoconstrictors, antisialagogues, suction, fiberoptic scope with lubricated ETT
- **Indications:** Cervical spine pathology, obesity, head & neck tumors, hx of a difficult airway
- **Premedication:** Sedation (midazolam, fentanyl, dexmedetomidine, ketamine)
- **Technique:**
  1. **Take time to topicalize airway** (key to success; see table below)
  2. Place special oral airway or grab tongue with gauze
  3. Keep fiberoptic scope in midline while advancing until epiglottis appears
  4. Advance scope beneath epiglottis using antero/retroflexion as needed
  5. Once vocal cords are visualized, advanced scope into trachea
  6. Stabilized scope while ETT is advanced off scope into trachea → if resistance is encountered, rotate ETT tube 90 degrees
  7. After insertion, visualize carina with scope to avoid endobronchial intubation

### Nerve Blocks to Anesthetize the Airway

#### Topical anesthesia for tongue/oropharynx

- Cetacaine spray (tetracaine/benzocaine combination)
  - benzocaine toxicity occurs at ≈100 mg; can lead to methemoglobinemia (tx with methylene blue)
- Viscous lidocaine: 2–4 mL, swish & swallow
- Nebulized lidocaine: 4%, 4 mL for 5–10 min (or atomizer)
- Lidocaine jelly: 2% on tongue blade, peaks in 5–10 min

#### Superior laryngeal nerve block (sensory innervation to epiglottis, arytenoids, & vocal cords)

- Laterally displace hyoid bone toward block side, direct 22-gauge needle to lateral portion of hyoid bone
- Withdraw slightly & walk off bone inferiorly (below each greater cornu)
- Advance through thyrohyoid membrane (may feel loss of resistance)
- Aspirate & inject 2 mL of 2% lidocaine superficial & deep to membrane

#### Transtracheal block (recurrent laryngeal nerve)

- Penetrate cricothyroid membrane with a 22-gauge plastic tip catheter and 10 mL syringe
- After aspirating air, remove needle & attach a syringe with 4 mL of 4% lidocaine
- Inject at end of expiration to anesthetize glottis & upper trachea

#### Recurrent laryngeal nerve block

- Aim for ipsilateral lesser cornu of thyroid cartilage in tracheoesophageal groove
- Insert needle perpendicular to pt directing it medially, contacting lesser cornu of thyroid cartilage
- Once reached, withdraw needle slightly & inject

#### Glossopharyngeal nerve block (posterior third of tongue)

- Inject 2 mL 1%–2% lidocaine in glossopharyngeal arch



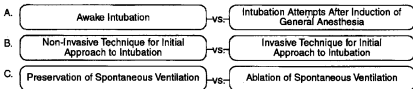
**Figure 4-1 ASA difficult airway algorithm.** (Note: 30% of anesthesia related deaths stem from issues of airway management.) (Reproduced with permission from The American Society of Anesthesiologists.)

AIRWAY 4-6

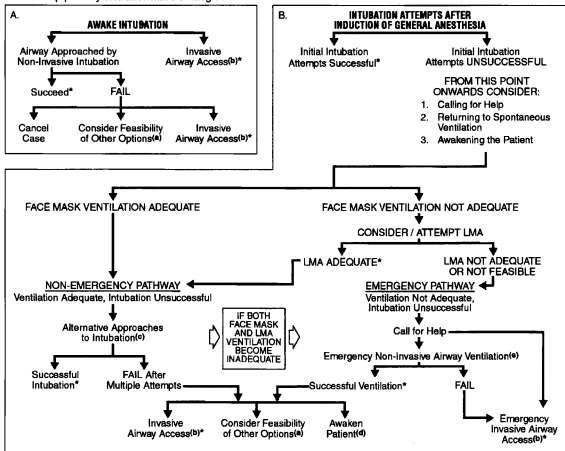


### DIFFICULT AIRWAY ALGORITHM

- Assess the likelihood and clinical impact of basic management problems:
  - Difficult Ventilation
  - Difficult Intubation
  - Difficulty with Patient Cooperation or Consent
  - Difficult Tracheostomy
- Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management
- Consider the relative merits and feasibility of basic management choices:



4. Develop primary and alternative strategies:



\* Confirm ventilation, tracheal intubation, or LMA placement with exhaled CO<sub>2</sub>

a. Other options include (but are not limited to): surgery utilizing face mask or LMA anesthesia, local anesthesia infiltration or regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.

b. Invasive airway access includes surgical or percutaneous tracheostomy or cricothyrotomy.

c. Alternative non-invasive approaches to difficult intubation include (but are not limited to): use of different laryngoscope blades, LMA as an intubation conduit (with or without fiberoptic guidance), fiberoptic intubation, intubating stylet or tube changer, light wand, retrograde intubation, and blind oral or nasal intubation.

d. Consider re-preparation of the patient for awake intubation or canceling surgery.

e. Options for emergency non-invasive airway ventilation include (but are not limited to): rigid bronchoscope, esophageal-tracheal combi-tube ventilation, or transtracheal jet ventilation.

Practical Approach to Unanticipated Difficult Airway	
<b>Plan A</b>	<ul style="list-style-type: none"> <li>Standard laryngoscopy with blade of choice</li> <li>If unable to intubate → make 2nd attempt with a different blade</li> <li>Make no more than 2 attempts (avoid ↑ risk of oral bleeding, secretions &amp; edema)</li> </ul>
<b>Plan B</b>	<ul style="list-style-type: none"> <li>Direct laryngoscopy &amp; insertion of bougie or intubating catheter</li> <li>Confirm placement by (1) using hand on anterior neck to palpate catheter advancement through glottis; (2) after this, 40 cm of catheter should reach carina &amp; provide resistance (no resistance will be encountered if in esophagus); (3) if using intubating catheter, may attach to ETCO<sub>2</sub> monitor</li> </ul>
<b>Plan C</b>	<ul style="list-style-type: none"> <li>Insertion of LMA (disposable, Fastrach™, Proseal™)</li> <li>5.0 or 6.0 ETT will fit through disposable LMA (± fiberoptic assistance)</li> </ul>
<b>Plan D</b>	<ul style="list-style-type: none"> <li>Terminate anesthetic &amp; awaken patient</li> <li>Perform awake fiberoptic intubation</li> <li>Perform surgical airway (i.e., tracheostomy)</li> </ul>

## Tracheal Procedures

- **Indications:** Emergency tracheal access when an airway cannot be secured via nasal/oral route
- **Percutaneous transtracheal jet ventilation**
  - Simple & relatively safe means to sustain a patient during a critical situation
  - Attach 12, 14, or 16-gauge IV catheter to 10-mL syringe partially filled with saline
  - Advance needle through cricothyroid membrane with constant aspiration until you get air
  - Advance angiocatheter; disconnect syringe, attach oxygen source
    - High-pressure O<sub>2</sub> (25–30 psi), insufflation of 1–2 sec, 12/min with 16-gauge needle → will deliver approximately 400–700 mL
    - Low-pressure O<sub>2</sub> (bag-valve-mask 6 psi, common gas outlet 20 psi)
- **Cricothyroidotomy**
  - Contraindications: Patients <6 yr/o (upper part of trachea not fully developed) → incision through cricothyroid membrane ↑ risk of subglottic stenosis
  - Sterilize skin
  - Identify cricothyroid membrane
  - Transverse incision with #11 blade ≈ 1 cm on each side of midline
  - Turn blade 90 degrees to create space to pass ETT
  - Insert ETT caudally, inflate cuff, confirm breaths sounds

## Techniques of Extubation

- Extubation performed when pt either deeply anesthetized (stage 3) or awake (stage 1)
  - Extubation during light anesthesia (stage 2) may → laryngospasm/airway compromise
- Patient's airway should be aggressively suctioned while on 100% O<sub>2</sub> prior to extubation
- Prior to extubation, pt should be awake, following commands, neuromuscular blockade reversed
- Untape ETT, deflate cuff, remove ETT while providing small amount of positive pressure
  - Remove secretions at distal end of ETT
- Place mask on pt with 100% O<sub>2</sub> while verifying spontaneous & adequate ventilation
- Consider using 1.5 mg/kg of IV lidocaine 1–2 min before manipulation of airway & extubation
  - Blunt airway reflexes
- Deep extubation
  - Indicated to prevent ↑ BP, ICP, or IOP, bronchospasm (in asthmatics)
  - Contraindicated in pts at ↑ risk for aspiration or who may have a difficult airway

## Complications of Laryngoscopy and Intubation

- **General complications**
  - Physiologic stimulation, hypercarbia, hypoxia, dental damage (#1 cause of malpractice claims)
  - Airway trauma, vocal cord paralysis, arytenoid dislocation, ulceration/edema of glottic mucosa
  - Tube malfunction and/or malposition
- **Specific complications**
  - **Postintubation croup** in children secondary to tracheal/laryngeal edema
  - **Recurrent laryngeal nerve damage** from ETT cuff compression → vocal cord paralysis
  - **Laryngospasm** from stimulation of superior laryngeal nerve
    - Involuntary/uncontrolled muscular contraction of laryngeal cords
    - Caused by pharyngeal secretions or direct stimulation of ETT during extubation
    - Treat with (1) gentle positive pressure ventilation, (2) succinylcholine (0.25–1 mg/kg to relax laryngeal muscles)
  - **Negative-pressure pulmonary edema**
    - Can occur during strong inspiratory effort caused by large negative intrathoracic pressure gradient against closed vocal cords
    - Prevention: place bite block prior to emergence
    - Treatment: maintain airway, provide O<sub>2</sub>, consider PEEP/reintubation

# ANESTHESIA TECHNIQUES

BENJAMIN UNGER

## INTRODUCTION

- Anesthetic technique based on surgical requirements, pt comorbidities, & psychological state
- Communication with surgical team & pt essential in determining optimal plan

## PATIENT INTERVIEW AND EVALUATION

- Discuss anesthetic options with pt
- Patient may have had experience with different techniques in past
- Answer all questions: positive experience can reduce preoperative anxiety

## PREMEDICATION

- Goals: ↓ anxiety, provide analgesia for regional techniques/placement of invasive monitors/IV access, ↓ secretions (oral surgery/fiberoptic intubation), ↓ likelihood/risk of aspiration, control heart rate/bp
- Oral meds usually given 60–90 min and IM meds 30–60 min before arrival in OR

Drug Class/ Drug	Adult Dose (mg)	Onset/Peak (min)	Notes
<b>Benzodiazepines</b> —anxiolysis, sedation, & amnesia (no guarantee against recall, no analgesia)			
Diazepam (PO)	5–20	30–60 in adults, 15–30 in children	Crosses placenta; highly protein-bound (↑ potency in pt with ↓ albumin)
Lorazepam (IM)	3–7	30–40	Among benzos, lorazepam has most delayed onset & longest action (may cause prolonged sedation)
Lorazepam (IV)	Titrate 1–2.5 mg doses		
Midazolam (IM)	0.05–0.1 mg/kg	5–10; 30–60	Rapid onset & short duration; given within 1 hr of surgery
Midazolam (IV)	Titrate 1–2.5 mg	1–2	
<b>Barbituates</b> —main advantage = cost; primarily induces sedation; no analgesia, may cause disinhibition; little cardiorespiratory depression at usual premed doses			
Secobarbital (PO, IM)	50–200	60–90	Sedation for 4 hr; performance may be impaired for 10–22 hr
Pentobarbital (PO, IM)	50–200		Prolonged action; not appropriate for short procedures
<b>Opioids</b> —treat pain assoc. with preop experience (regional anesthesia, central lines) provide little anxiolysis, may cause dysphoria; consider supplemental O <sub>2</sub>			
Morphine (IM, IV)	5–15	15–30; 45–90	Lasts 4 hr
Meperidine (IM)	50–150	Unpredictable	Lasts 2–4 hr
<b>Antihistamines</b>			
Diphenhydramine (PO, IM)	25–75		Sedation; can use with cimetidine & steroids to protect against histamine release from allergic rxns
<b>Anticholinergics</b> —useful for drying oral secretions (oral surgery/fiberoptic intubation)			
Atropine (IM)	0.3–0.6		
Scopolamine (IM)	0.3–0.6		
Glycopyrrolate (IM)	0.1–0.3		Least sedating agent (does not cross blood–brain barrier)
<b>H<sub>2</sub> antagonists</b>			
Cimetidine (PO, IM, IV)	300		
Ranitidine (PO)	50–200		
Famotidine (PO)	20–40		
<b>Antacids</b>			

(continued)

Drug Class/ Drug	Adult Dose (mg)	Onset/Peak (min)	Notes
Sodium citrate (PO)	10–20 mL		
<b>Gastric motility stimulator</b>			
Metoclopramide (PO, IM, IV)	5–20		
<b>Antiemetics</b>			
Ondansetron (IV)	4–8 mcg/kg		
Granisetron (IV)	3 mcg/kg		

Source: Adapted from Moyers JR, Vincent CM. "Chapter 21. Preoperative Medication." In: Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 4th ed. Lippincott Williams & Wilkins: Philadelphia, PA: 2001:551–565.

<b>Pediatric Premedication Dosing</b>				
Medication	Route	Dose (mg/kg except where noted)	Onset/ Duration (min)	Notes
Midazolam	PO/PR	0.25–0.75 (max 20 mg)	20–30; 90	
	Nasal drop/ spray	0.2–0.5	10–20	May be preferred route for infants
	IV	0.5–5 yr: 0.05–0.1 >5 yr: 0.025–0.5	2–3; 45–60	
Diazepam	PO	0.2–0.3	60–90	Reliable GI absorption; prolonged action
Ketamine	PO	3–8	20–25	
	IM	4–5	5; 45	
	IM	2–3 (using high conc. 50 mg/mL mixed with 0.1 mg/kg midazolam)		This combo may prolong recovery
Clonidine	PO	2–4 mcg/kg	>90 min	May require supplemental O <sub>2</sub> ; no amnesia & may ↑ MAC
Fentanyl lollipop	PO transmucosal	10–20 mcg/kg		Not as effective as midazolam; can cause facial pruritus, respiratory depression, & PONV

Source: Adapted from Bozkurt P. Premedication of the pediatric patient—anesthesia for the uncooperative child. *Curr Opin Anaesthesiol* 2007;20:211–215.

## ANESTHESIA TECHNIQUES

- Monitored anesthesia care (MAC): Anxiolysis, sedation, analgesia & monitoring by anesthesia personnel for rapid changes in pt status & anesthesia state/requirements
- General anesthesia: Pt unresponsive to significant stimulation; often requires airway, ventilatory and/or cardiovascular support
- Neuraxial techniques: Spinal/epidural alone or combined with above techniques for intraop & postop analgesia to chest, abdomen, & lower extremity
- Peripheral nerve block: Minimal physiological effects make these techniques useful

## MONITORED ANESTHESIA CARE VS GENERAL ANESTHESIA:

### ASA DEFINITIONS

- MAC: Anesthesia service that involves varying depths of sedation, analgesia, & anxiolysis but, most importantly, requires that provider is "prepared & qualified to convert to general anesthesia when necessary."
- General anesthesia: state when "patient loses consciousness & the ability to respond purposefully . . . irrespective of whether airway instrumentation is required"

Continuum of Depth of Sedation (ASA Definition)				
	Minimal Sedation (Anxiolysis)	Moderate Sedation/ Analgesia ("Conscious Sedation")	Deep Sedation/ Analgesia	General Anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful** response to verbal or tactile stimulation	Purposeful** response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular Function	Unaffected	Usually maintained	Usually maintained	May be impaired

\*\*Reflex withdrawal from a painful stimulus is **NOT** considered a purposeful response.

Commonly Used Drugs for Conscious Sedation				
Drug Name	Induction Bolus Dose	Maintenance Infusion Rate	Maintenance Intermittent Boluses	Notes
<b>Benzodiazepines</b>				
Midazolam	1–5 mg		1–2 mg	Rapid onset & short duration; often given alone or as anxiolytic adjunct to narcotic boluses, remifentanyl infusion and/or propofol infusion
<b>Opioid analgesics</b>				
Alfentanil	0.2–0.5 mg	0.5–2 mg/hr	0.2–0.5 mg	
Fentanyl	25–50 mcg		25–50 mcg	
Remifentanyl		0.025–0.1 mcg/kg/min	25 mcg	Avoid large boluses (risk of chest wall rigidity); ↓ dose when given with midazolam or propofol
<b>Hypnotics</b>				
Propofol	0.25–0.5 mg/kg	2–4 mg/kg/hr (30–70 mcg/kg/min)	0.3–0.5 mg/kg (300–500 mcg/kg)	Easily titratable, rapid recovery, antiemetic effects; pain on injection
Dexmedetomidine	1 mcg/kg over 10 min	0.2–0.7 mcg/kg/hr	–	Some analgesia with little respiratory depression; bradycardia & hypotension common side effects; may have prolonged sedation; small doses of midazolam & fentanyl initially may help reduce the initial bolus & prolonged sedation
Ketamine	0.1 mg/kg	2–4 mcg/kg/min	–	Maintenance of cardiovascular system & respiratory drive make ketamine appealing; avoid in pts with CAD, uncontrolled HTN, CHF and arterial aneurysms

Source: Adapted from Hillier SC. "Chapter 47. Monitored Anesthesia Care." In Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia*, 4th ed. Lippincott Williams & Wilkins: Philadelphia, PA; 2001:1247.

**FLUMAZENIL** (for Antagonism of Benzodiazepine Effects)

- Initial recommended dose = 0.2 mg
- If desired level of consciousness not achieved in 45 sec, repeat 0.2-mg dose
- 0.2-mg dose may need to be repeated q60sec to max of 1 mg
- Note: Be aware of potential for re sedation

**INHALATIONAL INDUCTION**

- Allows induction without IV access, as IV placement can be anxiety-provoking
- Onset of anesthesia faster in children than in adults  
(ratio of alveolar ventilation to FRC is in inverse proportion to body size)

**Pediatric Technique**

- For infants & children who tolerate a mask:
  - Start with 70% nitrous oxide in mask on pt
  - Introduce volatile agent only after 3 to 5 min of N<sub>2</sub>O/O<sub>2</sub>
  - Cut back N<sub>2</sub>O and increase O<sub>2</sub> percentage as potent agent is added
- For anxious children—rapid induction (in as few as 4 breaths):
  - Often requires involvement of multiple personnel and/or parents
  - Prime circuit with 70% N<sub>2</sub>O, O<sub>2</sub> & 8% sevoflurane
  - Place mask firmly on pt while monitoring airway throughout
  - With loss of consciousness, increase percentage of O<sub>2</sub> & decrease N<sub>2</sub>O
  - Decrease sevoflurane conc over next few min as agent equilibrates
  - Support ventilation as needed
  - Place IV (often with an assistant to assure appropriate attention to airway)

**Adult Inhalational Induction**

- Consider for adults in whom IV placement is extremely anxiety-provoking/difficult
- Disadvantages: May cause ↑ cough, hiccups, & possibly ↑ risk of nausea/vomiting
- Prime circuit with 8% sevoflurane & 70% N<sub>2</sub>O  
(usually requires 3 fill/empty cycles of an occluded anesthesia circuit)
- Instruct pt to exhale completely & then inhale from mask to vital capacity & hold
- If still conscious & unable to hold breath any longer, instruct pt to take additional deep breaths

**INTRAMUSCULAR INDUCTION**

- May be useful technique for:
  - Uncooperative pts in whom IV placement/inhalation induction impossible
  - Loss of control of pt and/or airway during attempted inhalation induction
  - Agitation/disinhibition with premedication
  - Need for rapid sequence induction without venous access
- Typical IM dosing:
  - Ketamine 6.5–10 mg/kg, 10% solution
  - Atropine 0.02 mg/kg, to reduce secretions
  - Succinylcholine 3–4 mg/kg, included for rapid sequence induction

*Note: Atropine & succinylcholine may be combined in same syringe; administer midazolam after IV placement to prevent ketamine emergence delirium*

**RECTAL INDUCTION****Characteristics**

- Convenient for healthy children old enough to have separation anxiety but still not mature enough to cooperate (8 mo to 5 yr/o)
- Parents & child familiar with rectal route for other medications (acetaminophen)
- Avoids needle for IM/IV induction & struggle involved with inhalational induction

**Technique**

- Cut 14-Fr suction catheter to 10 cm & lubricate
- Place catheter in pt's rectum & administer medication through syringe
- Follow medication with air bolus to purge remaining drug from catheter lumen
- Instruct parent/caregiver to hold buttocks together for at least 2 min
- Anticipate defecation & provide caregiver with waterproof mat
- Harmless hiccuping may occur
- Constant monitoring by anesthesia personnel required throughout
- Pt should be taken to procedure area as soon as sufficient sedation achieved
- Maintain primary attention on supporting pt's airway

Typical Agents and Dosing for Rectal Induction			
Medication	Dose (mg/kg)	Onset (min)	Comments
Thiopental (10%)	40	5-15	Hiccough common
Methohexital (1-10%)	25-30	5-15	Hiccough common; contraindicated in pts with seizure risk; may sting on injection
Ketamine (5%)	6-10	7-15	Causes catecholamine release which ↑ intraocular & intracranial pressure; excess salivation can be treated with atropine; potential dysphoria treated with benzodiazepine

Source: Adapted from Wetzel RC, Maxwell ZG. "Chapter 79. Anesthesia for Children;" *Principles and Practice of Anesthesiology* 2nd ed. Mosby: St. Louis, MO: 1998. 2094-2097.

Stages of Anesthesia		
Stage I	Amnesia	Time from induction of anesthesia to loss of consciousness
Stage II	Excitatory period	Irregular breathing, ↑ risk of laryngospasm, emesis, & arrhythmias
Stage III	Surgical anesthesia	Constricted pupils, regular breathing, no movement
Stage IV	Overdose	Hypotension, apnea, dilated/nonreactive pupils

### COMPONENTS OF ANESTHESIA

- An anesthetic may contain any or all of the following components: **anxiolysis, analgesia, hypnosis, amnesia, paralysis**
- Inhalational & IV agents provide anxiolysis & hypnosis, little or no analgesia (except for ketamine)
- Narcotics provide analgesia, little or no hypnosis/sedation

### "BALANCED ANESTHETIC" TECHNIQUE

- Concept of using paralysis & controlled ventilation to achieve optimal surgical conditions with ↓ doses of other anesthetic agents  
*A "balance" of virtues of different agents allows less of each to be used*
- Allows for faster emergence & less risk of cardiovascular collapse
- Paralysis may increase risk of intraoperative awareness

### MONITORING OF NEUROMUSCULAR BLOCKADE/PARALYSIS

#### Technique

Peripheral nerve stimulator (PNS) electrically stimulates motor nerve adductor pollicis (ulnar n.), obicularis oculi (facial n.), posterior tibial n., peroneal n.

#### Train of Four

- Four stimuli given at a frequency of 2 Hz every 5 sec
- Potentially eliciting 4 twitches (T1-T4)
  - TOF ratio T4:T1 indicates degree of neuromuscular block
  - Nondepolarizing agents:  
 Produce progressive reduction in magnitude of T1-T4;  
 number of elicited twitches indicates degree of blockade with recovery, twitches appear in reverse order
  - Depolarizing agents (succinylcholine):  
 Produce equal but reduced twitches (no fade)

#### Tetanic Stimulation

- Tetanic stimulation:** Concept that acetylcholine is depleted by successive stimulations; 50 Hz for 5 sec produces detectable fade in muscle contraction
- Extent of fade related to neuromuscular block
  - No fade = no neuromuscular block
  - Sustained response to tetanus present when TOF ratio is >0.7

**Double-Burst Stimulation**

- Two bursts of three stimuli at 50 Hz with each triple burst separated by 750 ms
- Decrease in 2nd response indicates residual block
- Ratio is related to TOF ratio but easier to interpret reliably

**Posttetanic Count**

- 50-Hz tetanic stimulus given for 5 sec, followed by stimulus at 1.0 Hz 3 sec later
- No. of responses detectable predicts time for spontaneous recovery
- Fade response appears earlier than train of four
- Can be used under deep paralysis to estimate time to recovery

**Phase II Blockade with Succinylcholine**

- Postjunctional membranes repolarized, but still not responding to acetylcholine
- Resembles blockade by nondepolarizing agents (get TOF fade, tetanic stim)
- Mechanism unknown, occurs when succinylcholine dose exceeds 3–5 mg/kg IV
- Reversal agents (neostigmine) may antagonize phase II blockade

Clinical Assessment of Blockade	
Twitch Response	Clinical Correlate
95% suppression of single twitch at 0.15–0.1 Hz	Adequate intubating conditions
90% suppression of single twitch; train-of-four count of one twitch	Surgical relaxation with nitrous oxide–opioid anesthesia
75% suppression of single twitch; train-of-four count of three twitches	Adequate relaxation with inhalation agents
25% suppression of single twitch	Decreased vital capacity
Train-of-four ratio >0.75; sustained tetanus at 50 Hz for 5 sec	Head lift for 5 sec; vital capacity = 15–20 mL/kg; inspiratory force = –25 cm H <sub>2</sub> O; effective cough
Train-of-four ratio >0.9	Sit up unassisted; intact carotid body response to hypoxemia; normal pharyngeal function
Train-of-four ratio = 1.0	Normal expiratory flow rate, vital capacity, and inspiratory force. Diplopia resolves

Source: From Dunn P., et al. *Clinical Procedures of the Massachusetts General Hospital*, 7th ed. Lippincott Williams & Wilkins: Philadelphia, PA: 2006. With permission.

**AWARENESS**

- Complication where pt regains consciousness during general anesthetic & can recall events afterwards
- Pts experience ranges from benign recall of conversation to posttraumatic stress disorder (PTSD) involving disturbed sleep, nightmares, flashbacks, & general anxiety
- Negative psychological consequences can last for years after the event
- If awareness occurs, pts often respond favorably to a complete explanation, apology, & reassurance that they are not crazy

**Frequency of Awareness** (from prospective study of 11,785 general anesthetics)

- 0.15% of all cases
- 0.18% with paralysis
- 0.10% without paralysis

**Patient Populations at Increased Risk of Awareness**

- Trauma victims: 11–43%
- Cardiac surgery: 1.1–1.5%
- Obstetric cases under general anesthesia: 0.4%
- History of substance abuse
- Previous episode of intraoperative awareness
- History of difficult intubation or anticipated difficult intubation
- Chronic pain pts using high doses of opioids
- ASA physical status IV or V
- Patients with limited hemodynamic reserve

(From Sandin RH, Enlund G, Samuelsson P., et al. Awareness during anaesthesia: A prospective case study. *The Lancet* 355. 2000;707–711)



## Guidelines for Prevention and Management of Intraoperative Awareness

### Prevention

- Check delivery of anesthetic agents to pt
- Consider premedication with amnesics
- Give adequate dose of induction agents
- Avoid muscle paralysis unless it is needed, even then avoid total paralysis
- Supplement N<sub>2</sub>O & opioid anesthesia with  $\geq 0.6$  MAC of volatile agent
- Administer  $\geq 0.8$  MAC when volatile agents are used alone
- Use amnesics when light anesthesia is only regimen tolerated by pt
- Inform pt about possibility of awareness
- Consider brain function monitoring

### Management

- Perform detailed interview with patient
  - Verify patient's account*
  - Sympathize & apologize*
  - Explain what happened*
  - Reassure about nonrepetition in future*
- Offer psychological support
- Record interview in patient's chart
- Inform patient's surgeon, nurse, & hospital lawyer
- Visit patient daily during hospital stay & keep in contact by telephone after
- Do not delay referral to a psychologist or psychiatrist

From Ghoneim MM, Weiskopf RB. Awareness during anesthesia. *Anesthesiology* 2000;92:597-602. With permission.

## BRAIN FUNCTION MONITORING, DEPTH OF ANESTHESIA, AND AWARENESS

- Brain function monitors analyze EEG signals & translate them into a number between 0 and 100 that corresponds to anesthetic depth
- Two devices currently available (BIS from Aspect, SEDLine from Hospira)
- ASA position: Brain function monitoring *not* routinely indicated & decision to use should be made on a case-by-case basis by individual practitioner
- Unclear whether brain function monitoring reduces incidence of awareness (Avidan MS, Zhang L, Burnside BA., et al. Anesthesia awareness and the bispectral index *N Engl J Med* 2008;358:1097-1108.)

### Interpretation of Brain Function Monitor Number During GA

>60	Increased risk of awareness during GA
40-60	Appropriate anesthetic depth
<40	Excessive depth of anesthesia

## BRAIN FUNCTION MONITORING & ANALGESIA

- Number correlates best with hypnotic component of anesthetic provided by benzodiazepines, propofol, & potent volatile agents
- N<sub>2</sub>O, low-dose narcotics & neuraxial/peripheral nerve blocks have little effect on the number
  - (These agents do ↓ amount of additional hypnotic needed to keep the number constant when pts are exposed noxious stimuli)*
- Ketamine confounds the number & contraindicates its use

### Potential Advantages & Disadvantages of Brain Function Monitoring

Advantages	Disadvantages
<ul style="list-style-type: none"><li>• May ↓ risk of awareness</li><li>• Prevents excessive anesthetic depth<ul style="list-style-type: none"><li>→ Faster emergence &amp; recovery</li><li>→ Reduction in drug costs</li><li>→ Possible lower long term mortality</li></ul></li></ul>	<ul style="list-style-type: none"><li>• Cost of equipment</li><li>• Provides false sense of security</li></ul>

From Sigl JC, Chamoun NG. An introduction to bispectral analysis for the electroencephalogram. *J Clin Mon Comput* 1994;392-404.

## TOTAL INTRAVENOUS ANESTHESIA (TIVA)

- TIVA anesthetics usually include hypnotic (propofol) + analgesic (remifentanyl)
- IV infusion drugs should be connected as closely as possible to pt's IV catheter (*Minimize dead space where infusion meds can accumulate*)
- TIVA may be more susceptible to dosing errors
  - Must always monitor for: IV lines that are infiltrated/kinked
  - Disconnections & dosing errors

**Advantages of TIVA over Inhalation Induction & Maintenance**

- Smooth induction with minimal coughing/hiccupping
- Easier control of anesthetic depth
- More rapid, predictable emergence
- Lower incidence of PONV
- Ideal operating conditions for neurologic surgery with reduced cerebral blood flow & cerebral metabolic rate; allows intraoperative neuromonitoring
- ↓ Organ toxicity & atmospheric pollution
- Avoids N<sub>2</sub>O side effects (expansion of closed airspaces & bone marrow suppression)

**Common Indications for TIVA**

- Anesthesia for airway endoscopies, laryngeal & tracheal surgery
- Anesthesia in remote locations or during transport
- Malignant hyperthermia-susceptible patients
- History of significant PONV

**Advantages of Continuous Infusions Compared with Intermittent Bolus Dosing**

- Avoid oscillations in drug concentration
- Minimize relative over- or underdosing
- Provide stable depth of anesthesia
- Reduce incidence of side effects (hemodynamic instability)
- Shorten recovery times
- ↓ Total drug requirements by 25–30%

**Typical Dosing Regimens for IV Agents Used for General Anesthetics**

Drug	Induction Bolus	Maintenance Infusion Rate	Maintenance Intermittent Bolus
Thiopental	5–7 mg/kg	–	–
Etomidate	0.2–0.3 mg/kg	–	–
Propofol	2–3 mg/kg	6–10 mg/kg/hr (100–180 mcg/kg/min)	–
Fentanyl	50–100 mcg	0.5–4 mcg/kg/hr	25–50 mcg
Alfentanil	0.5–1.5 mg	1–3 mg/hr	0.2–0.5 mg
Remifentanyl	1–2 mcg/kg	0.1–0.25 mcg/kg/min	–
Sufentanil	0.2 mcg/kg	0.2–0.4 mcg/kg/hr	–
Ketamine	0.1–0.2 mg/kg	5–10 mcg/kg/min	–

From Urman RD, Shapiro FE. "Chapter 9. Anesthetic Agents. Which One?" *Manual of Office-Based Anesthesia Procedures*. Lippincott Williams & Wilkins: Philadelphia, PA: 2007:63.

**Titration of Maintenance Infusions**

- Titrate to anticipated intensity of observed responses to surgical stimulus
- Drug requirements are highest during endotracheal intubation
- Requirements ↓ during surgical prep & draping
- Infusion rates should be ↑ a few minutes before skin incision
- Pt movement & changes in hemodynamics should guide infusions titration
- After start of surgery: If no response for 10–15 min, ↓ infusion rate by 20% if response, administer bolus & ↑ infusion rate
- Opioid should be administered to achieve analgesia
- Hypnotic should be titrated to individual requirements & surgical stimulus
- Infusion rates need to be titrated down to restore spontaneous respiration at surgery's end

**GUIDELINES FOR USING PROPOFOL****Induction of General Anesthesia**

- 2–3 mg/kg IV (reduced in pts given opioids/other premeds, aged >50)

**Maintenance of General Anesthesia**

- 80–150 mcg/kg/min IV combined with N<sub>2</sub>O or an opiate
- 120–200 mcg/kg/min IV if sole agent
- Consider reducing dose after 2 hr (propofol accumulates)

- Turn off infusion 5–10 min prior to desired time of emergence (can give 1–2-mL bouss as needed to keep pt asleep until emergence)

### Sedation

- 10–50 mcg/kg/min IV

## EXTUBATION & EMERGENCE

### Common Extubation Criteria

- Regular respiratory rate
- Stable SpO<sub>2</sub>
- Adequate paralysis reversal (sustained head/leg lift for 5 sec)
- Tidal volumes >4 mL/kg
- Return of consciousness (following commands)
- Stable end-tidal CO<sub>2</sub> at physiologic levels

### Indications for Continued Postop Intubation

- Epiglottitis
- Localized upper airway edema secondary to surgery or trauma
- Surgery causing injury to recurrent laryngeal nerves
- Upper airway edema from massive intraoperative volume infusion (especially combined with prolonged Trendelenburg or prone positioning)
- Unstable hemodynamics or continued bleeding

### Deep Extubation

- Indications: Asthma, ↑ risk of intracranial bleeding, delicate cosmetic sutures, compromised ocular globe, intravitreal gas
- Contraindications: Full stomach, obstructive sleep apnea, difficult airway
- Advantages: ↓ Coughing & bucking  
↓ Strain on incision sutures  
↓ Risk of ↑ intracranial and/or intraocular pressure
- Disadvantages: Loss of stimulation leading to apnea  
Laryngospasm  
Aspiration

### Postop Respiratory Complications after GA (from 24,157 cases)

Complication	OR	PACU	OR + PACU	% of All Respiratory Problems
Respiratory insufficiency	19	48	67	60
Respiratory obstruction	14	9	23	20
Laryngospasm	15	7	22	20
Total	48	64	112	100

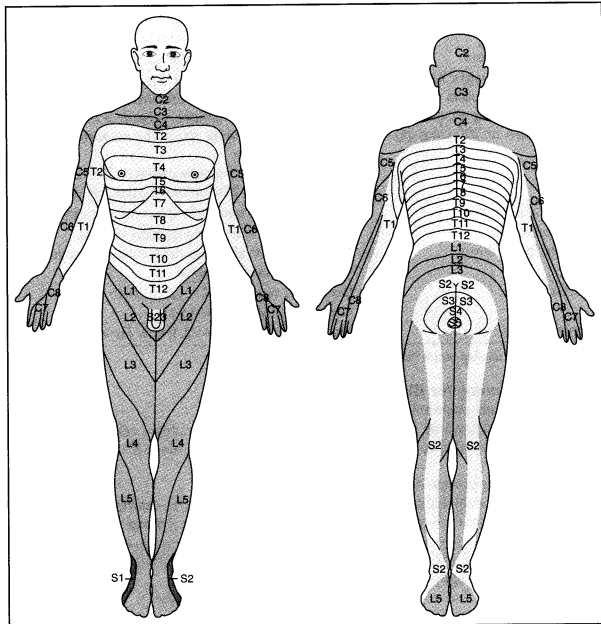
Source: Adapted from Lee PJ, MacLennan A, Naughton NN., et al. An analysis of reintubations from a quality assurance database of 152,000 cases. *J Clin Anes* 15(8):575–581.

## SPINAL/EPIDURAL ANESTHESIA

### Neuraxial Anatomy

- Spinal cord extends from base of skull → L1–L2 in adults/L3 in infants
- Dural sac extends from base of skull → S2 in adults/S3–S4 in infants
- Anatomic landmarks: Tip of scapula (T8)  
Iliac crest (L4)  
Sacral cornu (S5)
- Thoracic spinous processes—angled downward (in relation to vertebral bodies)
- Lumbar spinous processes—angled horizontally
- Spinal column curved like an “S”: Thoracic kyphosis at T4; lumbar lordosis at L3
- **Midline approach**—order of tissues encountered  
skin → subcutaneous tissue → supraspinous & interspinous ligaments → ligamentum flavum → epidural space → dura mater → subdural space → arachnoid mater → subarachnoid space
- **Paramedian approach** (needle insertion 1–2 cm lateral to midline)  
Bypasses supraspinous & interspinous ligaments and spinous processes; useful for narrowed intervertebral spaces, calcified ligaments, thoracic epidural (angled, overlapping spinous processes)

**Figure 6-1 Dermatome map.** (From Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 4th ed.: Lippincott Williams & Wilkins: Philadelphia, PA: 692. With permission.)



### Physiologic Effects

- Neurologic: Order of nerve blockade  
Small sympathetic C fibers → small sensory A $\delta$  fibers (pain & temp) → large sensory (A $\beta$ ) fibers (proprioception & touch) → large motor fibers (A $\alpha$ )  
Blockade of preganglionic sympathetic fibers exceeds sensory block (spinal > epidural) (often by up to two dermatomes, with degree of blockade determined by block height)

- Cardiovascular  
Sympathectomy (spinal > epidural) → loss of vascular tone → hypotension & reflex tachycardia  
Supra-T4 levels block cardioaccelerator fibers (T1-T4) → paradoxical bradycardia → decreased CO & further hypotension; large volumes of local anesthetic used for epidurals → higher systemic absorption → direct cardiac depressant effects
- Pulmonary  
Impaired cough reflex, high blockade ↓ use of accessory resp muscles (intercostals) → use caution in pts with limited pulmonary reserve  
Inspiratory fx preserved (unless respiratory centers (C3-C5) blocked)  
PFT changes: ↓ or unchanged VC, ↓ ERV, ↓ expiratory flow rate
- GI  
Sympathectomy → hyperperistalsis (get unopposed parasympathetics) → N/V
- GU  
Sacral blockade → atonic bladder (consider catheterization); renal blood flow usually maintained

### Contraindications to Placing a Neuraxial Block

- Absolute  
Refusal/inability to cooperate, local infection, coagulopathy, severe hypovolemia, sepsis, severe cardiac disease (aortic/mitral stenosis), ↑ ICP
- Relative  
Prior back surgery, neurologic disease (MS), infection distal to needle insertion site

### Summary of ASRA Regional Anesthesia & Anticoagulation Guidelines

Medication Class	Recommendation
Aspirin, other NSAIDs	No contraindications
Clopidogrel	Wait 7 d before needle insertion
Ticlopidine	Wait 14 d before needle insertion
GP IIb/IIIa inhibitors	Wait 8–48 hr before needle insertion
Low-molecular-weight heparin (High-dose enoxaparin 1 mg/kg q12h or 1.5 mg/kg qd; dalteparin 120 U/kg q2h or 200 U/kg qd)	Wait 10–12 hr (for normal dose) or 24 hr (for high dose) before needle insertion Postop bid dosing: Remove catheter 2 hr before 1st dose, which should be no earlier than 24 hr postop. Postop qd dosing: Remove catheter 10–12 hr after last dose & 2 hr before subsequent dose
Heparin SQ >4 days	Assess platelet count prior to removing catheter
Heparin intraop (vascular procedures)	Delay heparin 1 hr after insertion, remove catheter 2–4 hr after last dose; reheparinization OK after catheter removal
Warfarin	Document normal INR <1.5 prior to needle insertion & catheter removal
Thrombolytics	No specified time interval; follow fibrinogen level

Source: Adapted from *Reg Anesth Pain Med* 2003;28:172–197.

### Patient Positioning for Block Placement

- Goal for optimal positioning: widen intervertebral spaces  
→ Knees flexed towards abdomen, chin flexed to chest, shoulders relaxed
- *Sitting position*—easier to identify midline, can create saddle block if using hyperbaric solutions
- *Lateral decubitus position*—use if patient unable to sit  
→ Can preferentially block 1 side (if using hypo- or hyperbaric solutions)
- *Prone jackknife position*—good for perirectal surgery (if using hypobaric solution)

### Complications of Neuraxial Anesthesia

#### Common to both spinal and epidural anesthesia

- Backache
- Pruritus
- Hypotension
- Urinary retention
- Nerve injury
- Infection
- Hematoma

#### Other spinal complications

- **Transient neurologic symptoms (TNS)**—more common with ambulatory procedures, lithotomy position, lidocaine spinals. Symptoms: Delayed onset of radicular pain in lower back, buttocks, posterior thighs (can last up to 7 d)

### Complications of Neuraxial Anesthesia (Continued)

- **Cauda equina syndrome**—occurs with repeated admin of conc. local anesthetic. Symptoms: Bowel/bladder incontinence and/or neurologic impairment; seek immediate neurosurgical consult
- **Postspinal headache**—(see postdural puncture headache below)
- **High/total spinal**—supracervical blockade can cause cardiovascular collapse, apnea, loss of consciousness; supportive treatment/intubation may be necessary

#### Other epidural complications

- **Postdural puncture headache (PDPH)**—from inadvertent dural puncture (wet tap); usually self-limited (<7 d). Initial management: hydration, caffeine (500 mg), NSAIDs, abdominal binders. Epidural blood patches >90% effective for persistent PDPH
- **Spinal cord injury**—can occur if wet tap occurs at level above where spinal cord ends
- **Local anesthetic toxicity**—dizziness, tinnitus, CNS excitation, seizures, cardiac arrest can occur from systemic absorption of local anesthetic. Treatment is supportive. Consider 20% intralipid emulsions for refractory cardiac arrest

## SPINAL ANESTHESIA

- Rapid & reliable onset of lower body anesthesia by injecting local anesthetic into the intrathecal space
  - Blocks spinal cord fibers and nerve rootlets
  - Usually single-shot, although continuous catheters can be used
- Consider volume loading (500–1000 mL fluid) to avoid effects of rapid sympathectomy
- Needles:
  - Small gauge (>24 G), pencil point needles (Sprotte, Whitacre) reduce risk of PDPH → Often require introducer (19 G) to penetrate superficial tissue
  - Large gauge (<22 G), cutting (Quincke, Greene) needles → Used in difficult spinal to penetrate fibrotic, calcified ligaments
- Introduce needle (midline or paramedian technique) at L2–L5 interspaces until dural “pop” felt or if CSF flows freely when stylet removed
- Taylor approach: Spinal performed at L5–S1 by inserting needle 1 cm medial & caudal to ipsilateral posterior superior iliac spine & directing it cephalomedially toward the midline

### Factors Affecting Anesthetic Spread In Intrathecal Space

<b>Baricity</b>	Local solution density in relation to CSF; mixing medication with dextrose or sterile water results in hyperbaric or hypobaric solutions (respectively) <ul style="list-style-type: none"> <li>• Isobaric (density = CSF)—get block at level where medication is injected</li> <li>• Hyperbaric (density &gt;CSF)—get spread of medication with gravity in intrathecal space</li> <li>• Hypobaric (density &lt;CSF)—get spread of medication against gravity in intrathecal space</li> </ul>
<b>Patient position</b>	Gravity can help spread of medication within intrathecal space when hypo- or hyperbaric solutions used
<b>Spinal curvature</b>	Thoracic kyphosis at T4 prevents migration of spinal toward cervical region
<b>Other factors:</b> Dose, volume, temperature of medication injected, age, increased abdominal pressure, pregnancy, direction of needle bevel	
<b>No effect:</b> Weight, height, gender, barbotage	

- Duration of spinal anesthesia:
  - Depends on type & dose of local anesthetic used
  - Duration can be prolonged with vasoconstrictors (phenylephrine/epinephrine)

### Characteristics of Local Anesthetic for Spinal Anesthesia

Local Anesthetic	Concentration (%)	Duration of block (min)	
		Plain	With Vasoconstrictors
Procaine	10	30–50	50–75
Lidocaine	1–2	45–60	75–90
Mepivacaine	2	50–70	80–120
Bupivacaine	0.5–0.75	90–120	140
Tetracaine	0.5	90–150	180–300

## Level of Sensory Block Needed for Surgical Procedures

Sensory Level	Type of Surgery	Local Anesthetic and Dose
T4 (nipple)	Upper abdominal surgery C-section	Tetracaine, bupivacaine, or ropivacaine 8–16 mg
T6-T7 (xiphoid)	Lower abdominal surgery Appendectomy Herniorrhaphy	Lidocaine 75–100 mg, bupivacaine or ropivacaine 10–14 mg
T10 (umbilicus)	Hip surgery TURP Vaginal delivery	Lidocaine 50–75 mg, tetracaine 6–10 mg, bupivacaine or ropivacaine 8–12 mg
L1 (inguinal ligament)	Lower extremity	Tetracaine, bupivacaine, or ropivacaine, 6 mg
L2-L3 (knee)	Foot surgery	Tetracaine, bupivacaine, or ropivacaine, 6 mg
S2-S5	Hemorrhoidectomy	Lidocaine 30–50 mg

Source: Table adapted from Stoelting RK, Miller RD. *Basics of Anesthesia*, 4th ed. Churchill Livingstone; New York, NY 2000.

## EPIDURAL ANESTHESIA

- Slower onset, more controlled segmental spread of anesthesia using larger quantities of medication (roughly 10× more than spinal doses) injected into epidural space
- Usually a continuous catheter technique; can target select dermatomes (unlike spinal)
  - Thoracic epidurals → thoracic & upper abdominal surgeries
  - Lumbar epidurals → labor analgesia, lower abdominal, pelvic, lower extremity surgeries

### Identifying the Epidural Space

#### Loss-of-resistance (LOR) technique

- Engage epidural needle in ligament
- Attach low friction syringe (filled with air/saline) to epidural needle
- Apply constant/intermittent pressure to syringe as needle is slowly advanced
- When needle passes through ligamentum flavum (into epidural space), plunger will be easily advanced (indicating a loss of resistance)

#### Hanging-drop technique

- Drop of fluid placed on hub of epidural needle (once engaged in ligamentum flavum)
- Advance needle until fluid sucked into needle hub (indicates epidural space)

- Catheter placement & epidural space verification:
  - Thread epidural catheter 3–5 cm into epidural space
  - Aspirate catheter to assess for intravascular/intrathecal insertion (look for CSF/blood)
  - Consider epidural test dose (3 mL of 1.5% lidocaine with epinephrine 1:200,000) → Shows if catheter intrathecal (dense spinal) or intravascular (tachycardia, tinnitus)
- Medications
  - Surgical anesthesia: high local conc. (2% lidocaine, 0.5% bupivacaine)
  - Postop pain/labor analgesia: dilute local conc. + opioid (0.1% bupivacaine + 0.005% fentanyl)
    - (combo provides synergy, and reduces side effects (motor block, pruritis))
  - Adjuvants can supplement block (clonidine, epinephrine, phenylephrine)

### Characteristics of Local Anesthetics for Epidural Anesthesia

Local Anesthetic	Concentration (%)	Onset (min)	Duration (min)
Chloroprocaine	2–3	3–10	30–90
Lidocaine	1–2	5–15	60–120
Bupivacaine	0.25–0.5	10–20	120–240
Ropivacaine	0.2–0.5	10–20	120–240

Source: From Stoelting RK, Miller RD. *Basics of Anesthesia*, 4th ed. Churchill Livingstone; New York, NY: 2000.

- Factors affecting quality of epidural block
  - Volume injected, vasoconstrictors, site of injection, & parturients
  - Sodium bicarbonate can hasten block onset (↑ nonionized local → easier neuronal diffusion)
    - 1 mEq for each 10 mL lidocaine/chloroprocaine
    - 0.1 mEq for each 10 mL of bupivacaine (to avoid precipitation)
  - Patient position has no effect (unlike spinal anesthesia)
- Management
  - Continuous, bolus, or patient controlled epidural analgesia (PCEA) techniques
  - Continuous infusion rate depends on pt characteristics & type of solution used (continuous infusions often run at 4–10 mL/hr with a bolus dose every 5–15 min)
- Epidural troubleshooting
  - One-sided or patchy block—provide medication bolus, pull back catheter, or replace catheter
  - Unable to thread catheter—verify epidural space with LOR, then advance needle 1 mm & retry
  - Unable to remove catheter—change pt position (flexing, extending, rotating spine); try again later

Doses of Epidural Opioids			
Drug	Dose (mg)	Onset (min)	Duration (hr)
Alfentanil	2	5	1
Sufentanil	0.005–0.010	3–5	2–4
Fentanyl	0.05–0.10	5–20	3–5
Methadone	5–8	10–20	6–8
Hydromorphone	1	15–20	7–15
Meperidine	30–100	5–10	4–20
Morphine	3–5	30–60	12–24

### COMBINED SPINAL–EPIDURAL (CSE) TECHNIQUE

- Advantages
  - Combines a rapid-onset block (spinal) with ability to provide continuous management (epidural)
- Equipment
  - Epidural tray with specially made Tuohy needle (has back hole for spinal needle insertion); alternatively can use regular Tuohy needle with appropriately sized spinal needle
- Dose of spinal medication
  - Surgical anesthesia: Normal dose (see table above)
  - Labor analgesia: Low-dose opioid + local anesthetic (fentanyl 25 mcg + bupivacaine 2.5 mg)
- Technique
  - Proceed as if placing an epidural
  - Place spinal needle through epidural needle past dura (once epidural space identified)
  - Once CSF is obtained, inject medication, remove spinal needle, and thread epidural catheter
- Disadvantages
  - Inability to test dose epidural catheter (do not know if epidural will work after spinal wears off)
  - Slightly ↑ incidence of pruritus, respiratory depression, or transient fetal bradycardia

### CAUDAL ANESTHESIA

- Epidural anesthesia performed at sacral level close to where dural sac ends
- Indications
  - Commonly used in children for low superficial abdominal, perineal, or sacral anesthesia
  - Can be used for 2nd stage of labor, perineal, or sacral anesthesia in adults
  - More difficult in adults due to obscure anatomic landmarks
- Anatomy
  - Sacral hiatus—posterior opening to sacral canal at S5 level (entrance identified by sacral cornua). Sacral hiatus can also be identified as third point of an equilateral triangle using posterior superior iliac spines as the two other points
  - Sacrococcygeal membrane—equivalent of ligamentum flavum (overlies entrance to sacral hiatus)



- **Positioning:** Lateral or prone
- **Technique**
  - Insert needle between sacral cornu at 45-degree angle until slight ↓ in resistance encountered (signifying penetration of sacrococcygeal membrane)
  - Redirect needle parallel to patient & insert another 1–1.5 cm
  - Syringe should be aspirated for CSF/blood & test dose should be given
  - Catheter may be threaded (similar to an epidural)
- **Medications**
  - Pediatric dose: 0.5–1 mL/kg of 0.125–0.25% bupivacaine +/- epinephrine
  - Adult dose: 15–20 mL of local anesthetic
- **Complications:** Similar to those for an epidural

## PERIPHERAL NERVE BLOCKS

### Introduction

- Peripheral nerve blocks rely on local anesthetics injected around specific nerves/nerve bundles to prevent sensory transmission back to spinal cord/CNS
- Uses include surgical anesthesia +/- general anesthesia, pain management, or postoperative analgesia

### Preparation and Materials

1. Standard patient monitors (SpO<sub>2</sub>, ECG, blood pressure cuff)
2. Sedative medications & oxygen
3. Sterile tray and gloves
4. Block needle—short, B-bevel (blunt), beaded-tip, or insulated needle
5. Infusion catheter and appropriately sized block needle (if performing continuous nerve block)
6. Local anesthetic
7. Method of nerve localization (i.e., nerve stimulator, ultrasound machine, etc.)
8. Emergency airway equipment and intubation medications\*

### Nerve Localization Techniques

Paresthesia	Block needle used to elicit paresthesia when it contacts nerve. Injection should cause transient enhanced paresthesia; intense searing pain indicates intraneural injection (stop immediately, withdraw needle, reassess)
Nerve stimulation	Insulated needle connected to nerve stimulator elicits muscle twitches in target nerve's pattern of innervation. Dial current down slowly from >1 mA to <0.5 mA while retaining muscle twitch
Ultrasound-guided	Ultrasound probe used to visualize target nerve, block needle, & local anesthetic
Infiltration/field blocks	Local anesthetic injected in close proximity to nerve to be blocked based on its constant relationship with anatomic landmarks

- **Contraindications**—similar to those for neuraxial blocks
- **Complications** common to all nerve blocks:
  - Nerve injury, local anesthetic toxicity, infection, hematomas
  - Sterile technique should be used for any block to reduce risk of infection
  - Aspirate from syringe every 5–10 mL for blood to avoid intravascular injection of local anesthetic

## SUPERFICIAL AND DEEP CERVICAL PLEXUS BLOCKS

- **Anatomy**
  - C1–C4 ventral rami (which divide into superficial & deep branches)
    - Superficial branch → four main nerves (provides cutaneous sensation of neck from jaw line to T2)
    - Deep branch → ansa cervicalis = loop formed by C1–C3 nerves (provide sensory & motor function to deeper structures of neck, including phrenic nerve, strap & prevertebral muscles)
- **Indications**
  - Neck surgery including lymph node dissection, tracheostomy, carotid endarterectomy, thyroid surgery

- **Superficial Cervical Plexus Block**
  - Insert block needle at midpoint of posterior border of sternocleidomastoid
  - Inject 15–20 mL local anesthetic in a fan-like manner in cranial & caudad direction
  - Complications: Trapezius muscle paralysis can occur from blockade of CN XI
- **Deep Cervical Plexus Block**
  - Draw a line from mastoid process to transverse process of C6 (Chassaignac's tubercle)
  - Draw a second parallel line that is 1 cm inferior to it & mark off points that are 2, 4, & 6 cm from mastoid process, which correspond to C2, C3, & C4 transverse processes
  - At each point insert block needle at a slightly caudad angle until transverse process is met
  - Withdraw needle slightly once bone is contacted and inject 5 mL; repeat at other levels
  - Alternatively, can make one injection at C4 with caudad pressure held to facilitate cephalad spread
  - Complications/side effects: Horner's syndrome, phrenic & superior laryngeal nerve block, seizure, intrathecal/epidural injection, injection into vertebral artery

## INTERCOSTAL NERVE BLOCKS

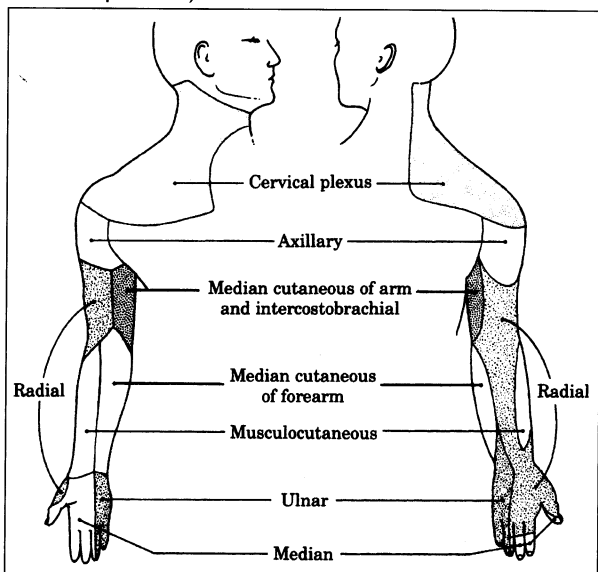
- **Anatomy**
  - Intercostal nerves arise from ventral rami of T1–T11
  - Each nerve sends off 5 branches, including gray and white communicantes, dorsal, lateral, and anterior cutaneous branches
  - Nerves run along inferior aspect of ribs with intercostal artery & vein
  - Anterior & lateral cutaneous branches readily blocked at posterior angle of rib just lateral to sacrospinalis muscle group
  - Block can be performed at midaxillary line (may miss lateral cutaneous branch)
- **Indications**
  - Does not provide adequate anesthesia for surgery (except for very superficial chest wall procedures)
  - Provides supplementary analgesia for rib fractures, thoracic procedures, mastectomy, upper abdominal procedures
- **Technique**
  - Positioning: Prone, sitting, or lateral decubitus
  - Palpate inferior edge of rib to be blocked at its posterior angle (6–8 cm lateral to midline)
  - Insert needle at angle 20 degrees cephalad until needle contacts inferior portion of rib
  - Redirect slightly until needle slides underneath rib
  - Advance needle another 3 mm (a fascial pop can sometimes be felt)
  - After negative aspiration, inject 3–5 mL of local anesthetic
- **Complications**
  - Pneumothorax—patients with limited pulmonary reserve are a relative contraindication
  - Local anesthetic toxicity—risk greatly ↑ as number of levels blocked ↑

## BRACHIAL PLEXUS BLOCKS

- Brachial plexus: Nerve roots from C5–T1—Trunks → Divisions → Cords → Branches
- Can block brachial plexus by injecting 25–40 mL of local anesthetic in certain areas along its path (see table, Brachial Plexus Blocks, page 6-8)
- Can also perform individual nerve blocks for selective blockade or as rescue blocks (inject 3–5 mL local)
  - **Radial**
    - Elbow: Needle inserted lateral to biceps tendon until it makes contact with lateral epicondyle
    - Wrist: Local anesthetic injected in a ring subcutaneously starting from radial artery pulsation anteriorly to extensor carpi radialis posteriorly at level of radial styloid
    - Muscle twitch/innervation: Extensor muscles of forearm → wrist extension
  - **Median**
    - Elbow: Needle inserted medial to brachial artery pulsation 1–2 cm proximal to elbow crease
    - Wrist: Needle inserted lateral to palmaris longus tendon & medial to flexor carpi radialis until piercing the deep fascia

- Muscle twitch/innervation: Forearm flexors, 1st/2nd hand lumbricals and thenar eminence → wrist flexion, thumb opposition
- Ulnar
  - Elbow: Needle inserted in ulnar groove just proximal to medial epicondyle
  - Wrist: Needle inserted between flexor carpi ulnaris tendon & ulnar artery at level of styloid process
  - Muscle twitch/innervation: Majority intrinsic hand muscles → hand flexion
- Musculocutaneous
  - Inject local anesthetic into substance of coracobrachialis ;
  - Muscle twitch/innervation: Coracobrachialis, brachialis and biceps brachii → elbow flexion/supination

**Figure 6-2 Upper extremity innervation.** (From Dunn PD, et al. *Clinical Anesthesia Procedures of the MGH*, 7th ed. Lippincott Williams & Wilkins: Philadelphia, PA: 79-280. With permission.)



REGIONAL 6-8

Brachial Plexus Blocks				
Block	Indications	Technique, Landmarks and Needle Insertion	Muscle Twitch	Comments
Interscalene	Shoulder or upper arm surgery	Level of cricoid cartilage (C6) in interscalene groove between anterior & middle scalene muscles	Deltoid, triceps, biceps, or pectoralis; diaphragm (phrenic) → needle too anterior; trapezius (CN XI) → needle too posterior	Complications & side effects are similar to deep cervical plexus block. Pneumothorax also possible if block too low. This block misses lower roots of brachial plexus in ulnar distribution

**Brachial Plexus Blocks (Continued)**

Block	Indications	Technique, Landmarks and Needle Insertion	Muscle Twitch	Comments
<b>Supraclavicular</b>	All types of upper extremity surgery except shoulder	<i>Classic approach:</i> 1 cm superior to midpoint of clavicle at an angle parallel to pts head & neck <i>"Plumb bob" approach:</i> Intersection of lateral border of sternocleidomastoid muscle & clavicle at angle perpendicular to skin	Any muscle twitch in the hand	Pneumothorax can occur because of close proximity of pleural dome. Phrenic nerve block & Horner's syndrome are common side effects
<b>Infraclavicular</b>	Elbow, forearm, wrist, hand surgery	<i>Coracoid approach (Raj technique):</i> 2 cm medial & inferior to coracoid process at an angle perpendicular to skin	Any muscle twitch in the hand	Risk of pneumothorax is low (pleura does not typically extend far laterally)
<b>Axillary</b>	Procedures below elbow	Perivascular injection of local anesthetic around axillary artery in axilla using a nerve stimulation or transarterial approach. Location of nerves in relation to artery: Radial (posterior), ulnar (inferior), median (superior)	Targeted toward individual nerves to be blocked: Radial = wrist extension, median = wrist flexion, ulnar = hand closure/thumb adduction	Complications associated with arterial puncture; not suitable for shoulder surgery. Will need to block musculocutaneous nerve if lateral forearm coverage is desired

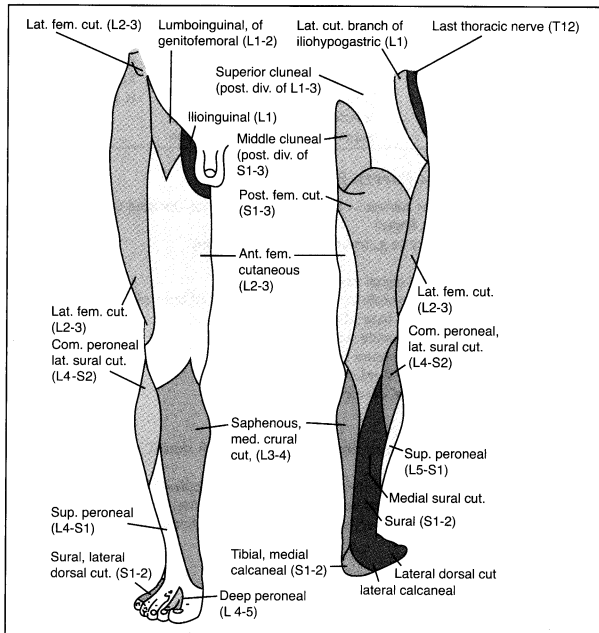
**DIGITAL NERVE BLOCK (UPPER EXTREMITY)**

- Anatomy: Median & ulnar nerves → common digital nerves (located on bilateral ventrolateral aspect of each finger)
- Indications: Finger surgery (e.g., trauma, distal amputation)
- Techniques
  1. Block of volar & dorsal digital nerves
    - Pronate hand, insert needle at dorsolateral aspect of base of finger
    - Aim anteriorly toward base of phalanx
    - Advance until contact made with phalanx
    - Withdraw needle from bone contact
    - Aspirate, then inject 2–3 mL of local (plain)
    - Repeat on each side of base of finger
  2. Transthecal digital block
    - Supinate hand
    - Insert needle at 45-degree angle into flexor tendon sheath, at level of distal palmar crease
    - Aspirate, then inject 2–3 mL of local (plain) while applying proximal pressure
- Local anesthetics: Any plain local anesthetic (no epinephrine)
- Complications: Intravascular injection, hematoma, digit ischemia, nerve injury, and infection

## LOWER EXTREMITY BLOCKS

- Lower extremity innervated by lumbar (L1-L4) & sacral plexuses (L4-S3)
- Lumbar plexus: Gives rise to the iliohypogastric, ilioinguinal, genitofemoral, femoral, obturator, & lateral femoral cutaneous nerves
  - With the exception of the femoral nerve, individual nerve blocks are commonly performed by infiltration without nerve stimulation
  - The **psoas compartment/lumbar plexus block** achieves blockade of the entire lumbar plexus. Complications from block can include epidural, subdural, or intrathecal injection, retroperitoneal hematoma, and/or visceral injury
  - The **3-in-1 block** relies on spread of a large volume of local anesthetic (30–40 mL) to achieve blockade of the femoral, obturator, and lateral femoral cutaneous nerves
- Sacral plexus: Gives rise to sciatic nerve
  - Sciatic nerve divides into tibial & common peroneal nerve at popliteal fossa
  - Tibial nerve gives rise to posterior tibial nerve & contributes to sural nerve
  - Common peroneal divides deep into the superficial & deep peroneal nerves, contributes to sural nerve
- Combination blocks
  - The 3-in-1 + sciatic or lumbar plexus + sciatic blocks can provide anesthesia to essentially the entire lower extremity (3-in-1 + sciatic may miss the most proximal and medial portion of the upper leg)
  - The knee joint is innervated by multiple nerves (femoral—anterior, obturator—multiple nerves: Femoral (anterior), obturator (medial), lat femoral cutaneous (lateral), and sciatic (posterior) → a combination block would be needed to provide complete anesthesia for surgery
  - Advantages over neuraxial blockade: unilateral limb anesthesia, ↓ risk of sympathectomy (hypotension, urinary retention), & avoidance of neuraxial hematomas in pts on anticoagulation
  - Disadvantage: Because anesthesia to the entire lower extremity usually requires >1 block, neuraxial anesthesia is a practical alternative

**Figure 6-3 Cutaneous innervation of the lower extremity. Adapted from Gray H. Anatomy of the Human Body. Lea & Febiger: Philadelphia, PA: 1918.**



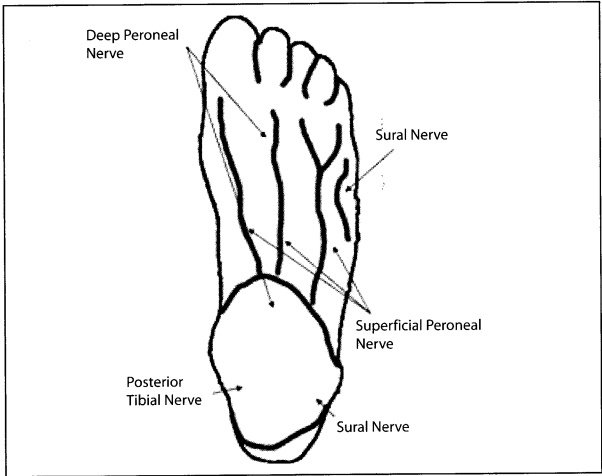
## Lumbar Plexus Blocks

Block	Indications	Technique, Landmarks, and Needle Insertion	Comments
<b>Ilioinguinal/ iliohypogastric</b>	Inguinal and genital procedures	Insert needle 3 cm medial & inferior to anterior superior iliac spine at an angle advancing back to contact iliac spine. Inject 10 mL of solution while withdrawing needle	Use in conjunction with genitofemoral block for inguinal herniorrhaphy
<b>Genitofemoral</b>	Inguinal and genital procedures	<i>Genital branch</i> —Inject 2–3 mL just lateral to pubic tubercle <i>Femoral branch</i> —Inject 5 mL subcutaneously below inguinal ligament lateral to femoral artery	Because genital branch lies within inguinal canal, block is often supplemented by surgeon
<b>Lateral femoral cutaneous</b>	Superficial lateral anterior thigh procedures including skin graft	Insert needle perpendicular to skin 2 cm medial & caudal to anterior superior iliac spine until pop is felt through fascia lata	Commonly performed as part of a 3-in-1 block
<b>Obturator</b>	Medial thigh procedures, relief of adductor muscle spasm, prevention of obturator reflex during TURBT of lateral wall	Needle inserted 1.5 cm lateral & caudal to pubic tubercle until it contacts pubic ramus. Walk needle off pubic ramus in lateral/caudal direction & advance another 3 cm	Commonly performed as part of a 3-in-1 block to achieve anesthesia/analgesia for knee, hip, or medial thigh procedures; obturator reflex not suppressed with spinal for TURBT (can lead to sudden leg adduction and bladder wall perforation from resectoscope)
<b>Femoral</b>	Anterior thigh procedures; analgesia for femoral shaft fx and knee procedures	Lateral to femoral artery at inguinal crease; nerve stimulator can be used to elicit a patellar/quadriceps twitch; sartorius twitch → needle too medial & superficial	Most common block performed for total knee arthroplasty for postop analgesia; Keep finger on femoral pulse to prevent arterial puncture from occurring
<b>3-in-1 block (femoral obturator, lateral femoral cutaneous)</b>	Anterior thigh procedures; analgesia for femoral shaft fx and knee procedures	Performed similar to femoral nerve block with needle inserted at a more cephalad 45-degree angle to skin. Pressure held inferior & medial to injection site to facilitate spread along fascia cephalad & laterally	Spread of anesthetic can be unreliable leading to increased incidence of block failure (esp obturator); extends the coverage area of the femoral nerve block in the upper leg; can be performed with a sciatic block to obtain whole leg anesthesia
<b>Psoas compartment/ lumbar plexus</b>	Anterior thigh procedures; postop analgesia for hip and knee procedures	Lateral decubitus or prone position—4 cm lateral to midline on a line drawn connecting top of iliac crests; if transverse process contacted, walk needle off in a cephalad direction	Muscle twitch—quadriceps; stimulate only to 0.5 mAmp (<0.5 mAmp may cause epidural). Can be performed with a sciatic block to obtain whole leg anesthesia

## Lower Extremity Blocks

Block	Indications	Technique, Landmarks, and Needle Insertion	Muscle Twitch	Comments
<b>Sciatic</b>	Lower extremity surgery, commonly used in combination with other blocks	<i>Classic approach</i> (lateral decubitus position)—5 cm inferior to midpoint of line drawn from posterior iliac spine to greater trochanter of femur <i>Anterior approach</i> (supine position)—8 cm caudad from perpendicular line drawn from midpoint of line connecting anterior superior iliac spine to pubic tubercle at an angle perpendicular to skin	Hamstrings, calf, foot, or toes; for anterior approach: only, hamstrings may be directly stimulated → redirect slightly medial or lateral; patellar (femoral) → needle too shallow	Anterior approach is a deep block & requires a needle length of approximately 150 mm
<b>Popliteal</b>	Surgery distal to the knee (calf, ankle, and foot). Often combined with a saphenous block for ankle and foot surgery	<i>Classic intertendinous approach</i> (prone position)—6 cm superior & 1 cm lateral to midpoint of line drawn from biceps femoris (lateral) to semitendinosus (medial) tendons in popliteal crease <i>Saphenous block</i> performed by injecting ring of anesthetic subcutaneously around medial aspect of tibial tuberosity	Calf, foot, or toes	Sciatic nerve divides into tibial & common peroneal nerves at this level. Performing block too closely to popliteal crease may lead to incomplete block of either nerve
<b>Ankle</b>	Foot Surgery	Infiltration block of five individual nerves at cephalad level of the malleoli (use 3–5 mL for each injection): <i>Deep peroneal</i> —lateral to tibialis anterior pulse between extensor hallucis longus & tibialis anterior tendons <i>Superficial peroneal</i> —infiltrate anesthetic subcutaneously from deep peroneal needle insertion site towards lateral malleolus <i>Saphenous</i> —infiltrate anesthetic subcutaneously from deep peroneal needle insertion site towards medial malleolus <i>Posterior tibial</i> —insert needle anteriorly towards medial malleolus, just medial to the achilles tendon <i>Sural</i> —insert needle anteriorly towards lateral malleolus, just lateral to Achilles tendon	Not used	4 of 5 nerves blocked are terminal branches of sciatic nerve (saphenous is femoral)

Figure 6-4 Innervation of the foot/ankle for ankle block. (Image courtesy of J. Ehrenfeld)



### INTRAVENOUS REGIONAL ANESTHESIA (BIER BLOCK)

- **Indications**
  - Short surgical procedures of extremities less than 1–1.5 hours (e.g., carpal tunnel release)
- **Materials**
  - Double tourniquet
  - Esmarch bandage
  - Small (22–24)-gauge IV in arm to be blocked (to be removed following the block)
  - Additional IV access on nonoperative arm (for administration of other medications)
  - 40–50 mL of plain 0.5% lidocaine
- **Technique**
  - Elevate arm above body & exsanguinate arm (distal → proximal) using an Esmarch bandage
  - Inflate proximal cuff 100 mm Hg higher than systolic (or roughly 300 mm Hg)
  - Remove bandage, inject 40–50 mL of plain 0.5% lidocaine through the small-gauge IV & remove IV
  - Encourage surgeons to start operating as soon as possible
  - If tourniquet pain occurs, inflate distal cuff & then release proximal cuff; this will provide an additional 20–30 min of anesthesia. Inform surgeons of limited surgical time left
- **Complications/problems**
  - Local anesthetic toxicity can occur if tourniquet is faulty/deflated too early
  - If surgery finishes in <20 min, keep tourniquet up for another 10 min; alternatively, release & reinflate tourniquet after 10 sec
  - Be prepared to conduct a general anesthetic/MAC in the event surgery is prolonged



# PERIOPERATIVE MONITORING

FRANCIS X. DILLON

## PULSE OXIMETRY

**Basis:** Two wavelengths of light absorbed differently by Hb & Hb-O<sub>2</sub>  
Pulsating arteriolar flow subtracted from nonpulsatile venous blood → gives arterial saturation

**Hb-O<sub>2</sub> Dissociation Curve:** Relates % of Hb saturation to PaO<sub>2</sub> (Fig. 7-1)

Right shift: Peripheral offloading of O<sub>2</sub> enhanced. Causes:

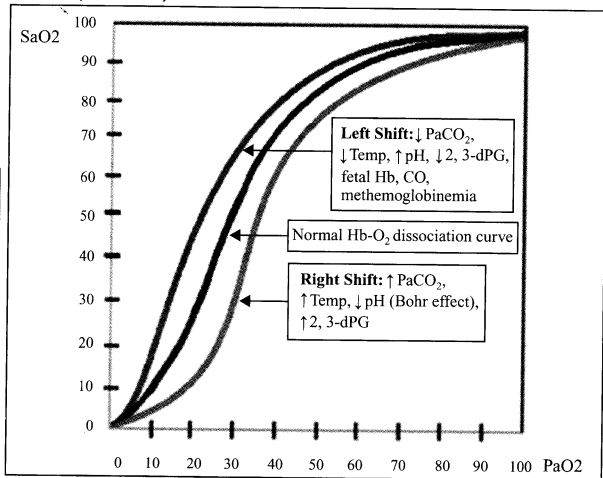
• ↑ Temp, ↑ H<sup>+</sup> (Bohr effect), ↑ CO<sub>2</sub>, ↑ 2,3-diphosphoglycerate, adult Hb

Left shift: Higher affinity Hb-O<sub>2</sub> & offloading of O<sub>2</sub> inhibited. Causes:

• ↓ Temp, alkalosis (Bohr effect), ↓ CO<sub>2</sub>, ↓ 2,3-diphosphoglycerate, fetal Hb

• Carbon monoxide, methemoglobinemia

**Figure 7-1** The hemoglobin-oxygen (Hb-O<sub>2</sub>) dissociation curve (Courtesy of S. Shah, MD, with permission.)



MONITORS 7-1

### Information in Pulse Oximeter Waveform

- Pulsatile waves match HR
- Dicrotic notch represents closure of aortic valve (damped if cold, atherosclerotic, or on pressors)
- Envelope height varies with respiration if hypovolemic
- SpO<sub>2</sub> of 90% = PaO<sub>2</sub> 60 mm Hg; SpO<sub>2</sub> of 98% = PaO<sub>2</sub> 90 mm Hg (SpO<sub>2</sub> readings <90 are inaccurate)

### Indications for Pulse Oximetry Monitoring

- Sedation for endoscopy, ECT or TEE
- Weaning of low-flow O<sub>2</sub>/ventilator support
- Monitoring during general anesthesia & in the PACU
- Monitoring following neuraxial blocks with opioids
- Sleep study documentation of apnea-hypopnea index (OSA)
- Ward observation following surgery in patients with OSA
- Ambulatory monitoring for pulmonary HTN or CHF

Hazards of Pulse Oximetry			
Hazard	Predisposing Condition	Hazard	Predisposing Condition
Finger or ear necrosis	Hypothermia	Finger or ear necrosis	Tape/wrapping too tightly
	Ischemia		Edema
	↓ Cardiac output		Prolonged monitoring
	Use of pressors		Immaturity
	Ground fault/ electrical failure		Prematurity
	MRI induced currents		Sepsis

Artifact	SpO <sub>2</sub> reading	Remedy/Notes
Low perfusion (hypothermia, pressors, atherosclerosis)	Lowers	Need to restore perfusion & pulsatile flow; can passively warm (do not actively warm)
Movement	Raises	Move probe to ear
Nail polish	Lowers	Remove polish or turn probe 90°; black, purple, blue colors worst
Vasopressors	Lowers	Consider dobutamine (↑ extremity perfusion)
Carboxyhemoglobin (CO exposure)	Raises	↑ FiO <sub>2</sub> ; Hyperbaric O <sub>2</sub> ; use orange color co-oximetry
Methemoglobinemia (benzocaine)	Lowers	Administer methylene blue dye
Hemoglobinopathies	Lowers	Supportive care; transfusion
Anemia (severe)	Raises	Consider transfusion; in anemic pts, 100% SpO <sub>2</sub> may still mean significantly ↓ O <sub>2</sub> delivery
Acidosis	Lowers	Correct; shifts HbO <sub>2</sub> curve to right
Alkalosis	Lowers	Correct; shifts HbO <sub>2</sub> curve to left
Cardiopulmonary bypass	Lowers/obliterates	Restore pulsatile flow following CPB

## NONINVASIVE BLOOD PRESSURE MONITORING (OSCILLOMETRIC SPHYGMOMANOMETRY)

**Basis:** Air cuff inflated around extremity, transducer reads oscillations from systolic pulsation

- Inflation pressure raised above systolic & then deflated until oscillations appear
- Maximal amplitude of oscillations = systolic; min. amplitude of oscillations = diastolic
- Waveforms analyzed by software, not displayed (each manufacturer has own algorithm)

### Causes of Artifacts

- Movement, obesity, poor perfusion, hypotension, extreme hypertension, bradycardia
- Wrong cuff size: Too small cuff gives ↑ pressure reading
- Irregular pulse: A. fib, PVCs, *pulsus bisferiens* (hypertrophic cardiomyopathy), *pulsus alternans* (pericardial effusion), *pulsus paradoxus* (tamponade), *pulsus parvus et tardus* (aortic stenosis)

### Complications

- Skin necrosis, soft tissue injury, phlebitis, neuropathy (peroneal, radial), compartment syndrome (biceps), petechiae, bruising, IV line infiltration/occlusion, interference with pulse oximetry

## TEMPERATURE MONITORING

**Basis:** Thermistors (resistors with resistance inversely proportional to temperature) quantify temperature

### Causes of Operative Temperature Loss

- Anesthesia factors:
  - ↓ Hypothalamic set point (around 34.5°C)
  - ↓ Heat generation (anesthesia → metabolic slowdown)
  - ↓ Muscle thermogenesis from muscle relaxation
  - Ventilation causes heat & moisture losses
- OR heat losses:
  - Loss from convection, radiation, evaporation
  - Loss from conduction (pt in contact with cold, wet surfaces)
  - Irrigation of cavities (e.g., peritoneum) & viscera (e.g., bladder)

### Complications of Hyperthermia

- More rapid and less predictable drug clearance
- Cellular & metabolic derangements (hypercarbia, acidosis)
- CNS more vulnerable to injury when warm as opposed to cool

### Complications of Hypothermia (below 36°C)

- ↑ Length of stay, ↑ wound infections, coag. problems, ↑ shivering, ↑ dysrhythmias

**Artifacts:** Related to probe placement (core temperature vs peripheral)

**Hazards:** Trauma (from probe placement), infection, shock (from grounded equipment)

**Placement:** "Core temp" (esophageal, PA) more meaningful than "shell temp" (rectum, axilla)

### Malignant Hyperthermia

- Temp ↑ of 0.5°C in 15 min is significant & should raise concern
- Early dantrolene administration only effective therapy (See Appendix D)

## ARTERIAL BLOOD PRESSURE (ABP) MONITORING

**Basics:** Circulatory tree—intra-arterial catheter—fluid—transducer mechanical system

- Modeled by differential equation: includes natural frequency & damping ratio
  - Damping ratio < 1, underdamped, "ringing" waveform results
  - Damping ratio = 1, critically damped, no ringing & fidelity preserved
  - Damping ratio > 1, overdamped, poor fidelity, lose high-freq info (i.e., dicrotic notch)
- Longer & thinner tubing, ↑ viscosity, kinks & bends: all cause ↑ resistance & damping
- Arterial waveform has higher freq. info. closer to aorta (↑ damping along distal arteries)
  - Central (e.g., femoral) A-line better than radial for conveying high-freq info
- Mechanics of A-line transducer system:
  - Short, rigid tubing with incompressible fluid (saline) typically used (↓ damping effect)
  - Strain gauge attached to a diaphragm changes resistance with tension (pressure)
  - Wheatstone bridge "amplifies" change in resistance & is calibrated
  - High-frequency of mech. system → greater measurement accuracy
  - Dense fluid raises frequency of system → greater accuracy (saline better than air; blood better than saline)
- Sources of signal degradation
  - Excess tubing length, too thin tubing caliber, excess fittings (stopcocks), large air bubbles (slow frequency response & increase damping ratio)

### SAFELY FLUSHING AN ARTERIAL LINE

1. Turn stopcock (up usually) so transducer is opened to air
2. Flush Luer-Lock to clear it of blood (hold gauze by stopcock to catch blood)
3. Turn stopcock so handle is horizontal (i.e., in starting position)
4. In short bursts of 2 sec or less, pull rubber line or squeeze valve to flush line back toward pt
5. Ensure that entire line is eventually cleared of blood (no bubbles present)
  - If line is flushed continuously (not in 2 sec bursts), the artery may fill with saline & flow retrograde (transducer pressure head is 300 mmHg) to aortic arch & embolize (saline/air) to common carotid & brain

### HAZARDS OF ARTERIAL LINE MONITORING

- Embolization: From flush mechanism; limit flush < 2 sec to prevent retrograde flow
- Thrombosis: Risk ↑ with duration of placement; catastrophic if end-artery (brachial)
- Nerve injury: Nerves closely related to arteries anatomically
- Vascular injury: AV fistula, hematoma
- Limb loss: Inadvertent drug injection (i.e. thiopental, phenergan) → severe vascular injury → amputation

### Indications for Arterial Blood Pressure Monitoring

- Surgical need for tight BP control (neurosurgical/vascular procedures)
- Measurement of mean arterial pressure crucial to derive cerebral perfusion pressure or coronary perfusion pressure
- Measurement of oxygenation critical (e.g., pulmonary surgery with lung isolation, cardiac surgery)
- Severe or labile hypertension
- Hypotension anticipated (sepsis, cardiogenic shock, hypovolemia)
- Massive blood loss & need for transfusion anticipated
- Need for frequent arterial blood sampling
  - Diagnosis & treatment of acidosis/alkalosis
  - Frequent lab values needed (blood glucose, K<sup>+</sup>, hemoglobin)

### Indications for Arterial Blood Pressure Monitoring (continued)

- NIBP ineffective/impractical
- Obese patients where NIBP cuff pressures are unreliable
- Lengthy cases where NIBP cuff trauma may be significant
- Cases where arm positioning make cuff potentially traumatic (thoracotomy)
- NIBP cuff subject to compression/motion artifact (cases with arms tucked)

Site of Arterial Cannulation	Disadvantage
Superficial temporal	Retrograde cerebral embolization possible
Radial	May be small, tortuous, or insufficiently anastomosed with ulnar artery
Ulnar	May be small, tortuous, or insufficiently anastomosed with radial artery
Brachial	An end-artery: Risk of limb thrombosis; remove as soon as possible
Femoral	Atheromatous, deep, prone to infection & AV fistula formation
Dorsalis pedis	Small & odd angle: Difficult to cannulate; waveforms often damped
Posterior tibialis	Tortuous & odd angle: Difficult to cannulate; waveforms often damped

### CENTRAL VENOUS LINES & CENTRAL VENOUS PRESSURE (CVP) MONITORING

**Uses:** Monitoring (CVP & central blood O<sub>2</sub> saturation—surrogate for SvO<sub>2</sub>)  
Central drug/fluid/blood product administration (may be safer route for vasoactives)  
Renal replacement therapy (CVVH/HD) & temporary pacemaker lead placement

#### Hazards of Central Line Placement

- Pneumothorax, hemothorax, thrombus, thromboembolus
- Arterial puncture, hematoma, AV fistula formation
- Nerve injury
- Infection including systemic sepsis, septic thrombus, endocarditis
- Knotting or breakage of catheter, erosion through vessel, endocardium, myocardium
- Thoracic duct injury, chylothorax
- Microshock, dysrhythmias

#### Artifacts

- Height: Increase in a-line reading 12" drop in transducer height = 22.4 mmHg
- Positioning: (e.g., jackknife) may affect CVP
- Abdominal compression/retraction may impede CVP & blood return
- Insufflation of CO<sub>2</sub> in abdomen (laparoscopy) may artifactually raise CVP
- Rapid infusion of IVF may artifactually raise CVP
- AV fistula in the arm may cause artifactually high CVP

### Indications for CVP Monitoring

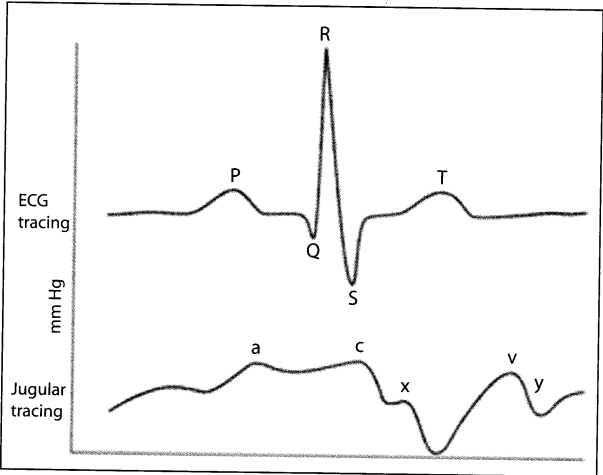
- Monitoring central pressures (RA & CVP)
- Monitoring RV overload
- Treating RV infarction
- Vasoactive/chemo infusions (avoid venous & soft tissue ischemia/injury)
- Rapid infusion of fluid/transfusion
- Use of transvenous pacing electrodes or passage of a PA catheter

### NORMAL CVP & CORRESPONDING ECG WAVEFORM (Fig. 7-2)

Feature of the CVP Waveform		
Waveform Feature	Results From	Remark
"a" wave	Venous distention from RA contraction	Biggest pulsation visible, especially during inspiration
"c" wave	Bulging of tricuspid valve into RA during RV isovolumic systole	Called the "c" wave because it coincides with (& is accentuated by) carotid pulse
"x" descent	(1) Atrial relaxation & (2) caudal displacement of tricuspid valve during RV systole	Occurs during systole, accentuated in constrictive pericarditis

Feature of the CVP Waveform (continued)		
Waveform Feature	Results From	Remark
"v" wave	Increasing volume of blood in the RA during ventricular systole when the TV is closed	Late systolic, accentuated with tricuspid regurgitation
"y" descent	Opening of tricuspid valve & subsequent rapid inflow of blood into RV	Rapid & deep with TR; slow with TS or atrial myxoma, both of which cause slowing of outflow from RA

Figure 7-2 CVP waveform placed in synchrony with the ECG waveform.



CVP Waveform Abnormalities					
<b>Abnormality</b>	Absent "a" wave	Large "a" wave	Cannon "a" wave (Occasional large pulsations at irregular intervals)	Ventricularized "c-v" waves (Resemble cannon "a" waves but are frequent, and at regular intervals)	"M" or "W" waves (diminished x and prominent y descents)
<b>Cause</b>	No atrial contraction	Dyssynergic atrial contraction or obstruction of tricuspid valve	Atrial contraction against a closed tricuspid valve	Reflux of blood into right atrium in systole	Rapid flow out of atrium in diastole
<b>Associated with</b>	Atrial fibrillation	1st-degree AV block severe, tricuspid stenosis	PVC, paced rhythm, junctional rhythm	Tricuspid regurgitation	Constrictive pericarditis

## MEASUREMENT OF CARDIAC OUTPUT

- Fick equation:  $CO = VO_2 \div (CaO_2 - CvO_2)$  where:
  - $VO_2$  = oxygen consumption = uptake of  $O_2$  from inspired gases in L/min
  - $CaO_2$  = content of  $O_2$  in arterial blood in liters  $O_2$ /L blood
  - $CvO_2$  = content of  $O_2$  in venous blood in liters  $O_2$ /L blood
- Mixed venous  $O_2$  saturation ( $SvO_2$ ):
  - Gives an indication of tissue oxygenation
  - Normal  $SvO_2$  is 75% (range 60%–80%); higher under anesthesia, up to 90%
  - $CaO_2$  (in mL/L blood) =  $[13.4 \times \text{Hb conc. (in mg/dL)} \times SaO_2 / 100] + [0.031 \times PaO_2 \text{ (in mmHg)}]$
  - $CvO_2$  (in mL/L blood) =  $[13.4 \times \text{Hb conc. (in mg/dL)} \times SvO_2 / 100] + [0.031 \times PvO_2 \text{ (in mmHg)}]$

Causes of Low Mixed Venous		Causes of High Mixed Venous	
Low $O_2$ Delivery	$\uparrow O_2$ Use	Low $O_2$ Use	Other
<ul style="list-style-type: none"> <li>Hypoxia</li> <li>Anemia</li> <li><math>\downarrow</math> Cardiac output</li> <li>Alkalosis</li> <li>Methemoglobinemia</li> </ul>	<ul style="list-style-type: none"> <li>Fever</li> <li><math>\uparrow</math> Metabolic states</li> <li>Shivering</li> </ul>	<ul style="list-style-type: none"> <li>CN poisoning</li> <li>Hypothermia</li> </ul>	<ul style="list-style-type: none"> <li>Impaired <math>O_2</math> tissue delivery (sepsis, burns)</li> <li>Mitral regurg.</li> </ul>

Note: If PA catheter is wedged (in contact with arterial blood), mixed venous will be high.

- Thermodilution technique
  - Ensure tip of catheter is in main pulmonary artery
  - Quickly inject 10 mL of cold saline or 5% D5W into CVP port
  - Thermistor monitors temp change & reports area under curve

Cardiac Output Erroneously $\downarrow$	Cardiac Output Erroneously $\uparrow$
<ul style="list-style-type: none"> <li>Large volume injected</li> <li>R-to-L shunt</li> <li>Catheter tip too proximal</li> </ul>	<ul style="list-style-type: none"> <li>Small volume injected</li> <li>L-to-R shunt</li> <li>Wedged catheter</li> <li>Tricuspid regurgitation</li> </ul>

## CONSIDERATIONS FOR PA THERMODILUTION CATHETER INSERTION & MONITORING

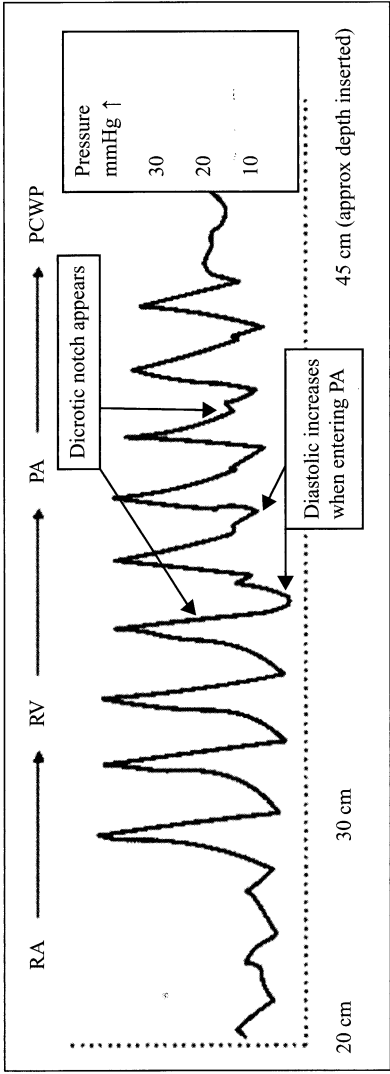
- Decision for insertion: Many question utility of PA measurement
- Risk–benefit analysis critical: Must know what you are seeking to measure
  - Filling pressures intraoperatively**  $\rightarrow$  to determine exact amount of preload needed
    - Useful in COPD (sensitive to excess fluid), large 3rd-space loss, pulm HTN, RV or LV failure
    - PA looks at LV filling pressures (in addition to RV filling pressures, via CVP)
  - SVR**
    - Liver transplantation where liver dz has caused chronic shunting, SVR  $\downarrow$
    - Sepsis, SVR may be too  $\downarrow$  for adequate global perfusion
    - Free flaps/plastic surgery where peripheral cold may make SVR  $\uparrow$
    - Pressors/inotropes infused where SVR may become too  $\uparrow$
    - CHF where SVR may be relatively  $\uparrow$
  - Cardiac output** (cardiac, CHF, or large fluid cases)
    - Frank–Starling curve (C.O. as a function of preload) shows if pt is in CHF or hypovolemic
      - Plot PA diastolic pressure (on x-axis) vs C.O. (y-axis) on graph paper
    - Titrate dopamine vs dobutamine vs amrinone to improve C.O.
    - Provide real mixed venous  $O_2$  blood sample for assessing  $O_2$  saturation & delivery
- Insertion site (see table below)
  - Consider passing a PA sheath with obturator (plug) or CVP inserted lumen
    - Allows later optional PA use if circumstances warrant it (e.g., oliguria, hypotension)

### Percutaneous Access Sites for CVP or PA Catheter Placement

Vein Location	Peripheral Arm (brachial or cephalic)	External Jugular (EJV)	Internal Jugular (IJV)	Subclavian	Femoral
Advantages	Easy to access; good for long-term access (PICC)	Easy to access	Easy to access	Most comfortable, best for long-term placement, pt may ambulate easily	No risk of hemothorax; easy to access during CPR
Disadvantages	Catheter may not pass through small vessel; may not give good CVP waveform	Catheter may not pass owing to angle of EJV as it joins the SCV; hard to do venipuncture into the EJV; may not give good CVP waveform	Near trachea and carotid; difficult to dress with bandage, uncomfortable for long-term access and hygiene	Clavicle may block access; subclavian artery near; pneumothorax or hemothorax possible	Risks of infection & thrombosis ↑; pt must be immobilized
Infection rate	↓	↓	↔	↓	↑
Pneumothorax risk	Impossible	↓ But possible	↓ But possible	Possible Obtain radiograph following placement	Impossible
Lymphatic injury possible	No	No	No	Yes; thoracic duct may be lacerated if access L. subclavian vein	No
Thrombosis risk	↓	↓	↔	↔	↑

- Tips for PA catheter placement (see Chapter 11, on anesthesia procedures)
  - Calibrate carefully ("zeroing"): Decisions regarding LV function depend on a few mm Hg!
  - Check balloon & valve to ensure balloon holds air (1 cm) when valve is closed
  - Pass to 20-cm depth & assure CVP waveform before inflating (1 mL only) balloon
  - Be sure pt is flat, not with head down, when "floating" PA catheter
  - Know & anticipate waveforms & locations of distal tip
    1. ≈ 20 cm is CVP, may inflate balloon with 1 cm air
    2. ≈ 30 cm is RV, pressure much greater, no dichrotic notch
    3. ≈ 40 is PA, diastolic c several mmHg ↑ & notch appears
    4. ≈ 45 (near PCWP) some damping & ↑ in pressure will occur:
      - a. Let balloon tip occlude while you very slowly advance catheter
      - b. When you get wedge waveform (see Fig. 7-3), disconnect breathing circuit
      - c. Read mean PCWP (approximately equivalent to LVEDP; 4–15 mmHg normal)
      - d. Reconnect circuit, deflate balloon, observe PA waveform return
      - e. Withdraw catheter 1–2 cm for safety & do not reinflate *in situ*
      - f. If you want another PCWP, safest option is to withdraw to 20 cm & refloat
- **PA perforation:** Likely when *no wedge pattern evident after deep insertion*
  1. Circumstances that predispose to PA perforation: Papillary muscle ischemia, mitral stenosis or regurgitation, pulm HTN or intrapulmonary shunting, LV failure  
Beware: After seeing *no definitive wedge pattern, repeated attempts to advance catheter may perforate PA*
  2. Many times PA catheter will give a good wedge pattern (the waveform continues to look like PA even though catheter is in PA & ought to have wedged)  
Beware: *Coiling or false negative wedging may occur, predisposing to PA rupture*

Figure 7-3 Typical waveform progression of PA catheter floating through the cardiac chambers (Adapted from Mathews, L. Internet Scientific Publications: Sugar Land, TX: 2007. with permission.)





**Common Reasons for PA Catheterization**

- Assess RV pressure
- Diagnose & treat pulm HTN
- Assess hypotension: (Sepsis vs cardiogenic shock on basis of CO & SVR)
- Manage severe organ dysfunction/severe sepsis
- Administer fluids judiciously (COPD/pulm HTN/burns/renal failure)
- Diagnose & treat intraoperative oliguria
- Manage MI with shock
- Construct a Starling curve of CO vs preload
- Manage valvular heart dz with appropriate preload & afterload adjustment
- Assess vasoactive therapy by serial CO & SVR measurements
- Manage pts with cerebral vasospasm after SAH (may involve induced hypertension, hemodilution, & hypervolemia under PA catheter monitoring)
- Manage cardiac patients after heart surgery

**Possible Contraindications to PA Catheterization**

Previous/planned pneumonectomy (consequence of PA rupture would likely be fatal)	Existing pacemaker lead
Tricuspid prosthesis, pulmonic prosthesis Tricuspid stenosis, pulmonic stenosis	Warfarin, heparin, antiplatelet therapy (e.g., clopidogrel or high dose aspirin)
R. atrial or ventricular masses; documented mural thrombi or valvular growth	Endocarditis
Cyanotic heart dz or R-to-L shunt	Floating during CPB
Latex allergy (if PA components are latex)	Tight AS (until chest is open, as floating → A. fib → loss of atrial kick → CV collapse)
Recent bifascicular block or LBBB Severe ventricular dysrhythmias	Mitral regurgitation, which may make wedging impossible (predisposes to rupture)
Heparin-coated catheter in HIT <sup>+</sup> patient	Atrial dysrhythmias (consider echo to rule out atrial thrombus)

**NORMAL PA WAVEFORMS AS CATHETER IS PASSED FROM CVP (RA) TO RV TO PA TO PCWP****Hemodynamic Variables and Waveforms of the PA Catheter****Distance (cm) Passed from Catheter Tip →**

PA catheter passage benchmark	0	20 or more	30 or more	40 or more	45 or more
Typical waveform	Damped CVP	Characteristic, undamped, CVP with a, c, x, v, y waves	RV wave with no dichrotic notch; low diastolic essentially equal to RA	PA wave with dichrotic notch and diastolic elevated over RVD value	Damped, decreased "wedge" waveform
Typical pressures (mm Hg)	0 [Catheter must read zero at LA level]	CVP <sub>m</sub> = RAM = 2–6	RVS = 15–25 RVD = 0–8 RVM = 5–14	PAS = 15–25 PAD = 8–15 PAM = 10–20	PAOP = PCWP ≈ LAM = 6–12
Typical difficulties encountered at this stage of passage	Tip may bend or reflex on itself, causing very high pressures eventually equilibrating with the pressure	Tip may traverse RA & continue down IVC (rather than entering the RA), giving CVP waveform well beyond 20 cm	Catheter may coil in RV; giving an RV waveform well beyond 40 cm, beware knotting may occur in this setting	Beware of pulmonary artery rupture! Mitral insufficiency or intra-pulmonary shunting may make it impossible to obtain a "wedge"	Beware of pulmonary infarction! Catheter should not be left wedged; deflate balloon & restore PA waveform

(continued)

## Hemodynamic Variables and Waveforms of the PA Catheter (Continued)

Distance (cm) Passed from Catheter Tip →

PA catheter passage benchmark	0	20 or more	30 or more	40 or more	45 or more
Suggested remedy	Deflate, withdraw, refloat	Deflate, withdraw, refloat; consider fluoroscopic guidance	Deflate, withdraw, refloat; consider fluoroscopic guidance	Leave catheter in PA position if you believe it has been inserted far enough; use pulmonary diastolic as surrogate for PAOP	Deflate & withdraw PA catheter several cm if waveform remains or becomes wedged; be sure PA waveform returns

## “V” WAVES IN A WEDGE TRACING

Usu. sign of severe mitral regurgitation (get transmission of large pressure waves from LV, through incompetent mitral valve, into LA, and pulmonary vessels)

Complications of PA Catheter Placement		
Complication	Consider	Remedy
Ventricular dysrhythmias	Dysrhythmias may occur on PA catheter placement, usually self-limited	Xylocaine 50–100 mg IV during passage; check Mg, K, levels & pH
Pulmonary embolus	↑ Risk when PA catheters used compared with CVP	Take PA catheter out when no longer needed; heparin, aspirin, other anticoagulation for thromboprophylaxis as indicated
Catheter will not wedge; gives unchanged PA waveform even though inserted beyond 45–50 cm	Consider catheter coiling in RV; make sure pt is supine; consider possibility of intrapulmonary shunting, congestion, or MR (all of which may obliterate wedge waveform even though catheter is properly placed)	Deflate, reinsert with pt supine; consider chest radiograph or using fluoroscopy; consider assistance from a colleague
During insertion, pressure climbs monotonically until off scale	Catheter may be catching on a central vein & kinking back on itself, blocking the lumen	Consider deflating, reinserting while pt is supine; consider radiography or fluoroscopy
Good CVP waveform, but catheter shows no RV waveform even though deeply inserted (30 cm)	Catheter may have traversed RA & be going down IVC; catheter may be having trouble getting through a small or stenotic tricuspid valve & kinking or coiling in RA	Consider deflating, reinserting while pt is supine; consider radiography or fluoroscopy; make sure balloon is not inflated with more than 1 mL of air (may prevent passage through valve)
Catheter will not pass after 20–25 cm, feels stuck or constrained; gives CVP waveform that is damped/goes off scale	Catheter may be going up arm or back up the IJV into the intracranial venous system	Deflate, withdraw carefully, reinsert with head or arm repositioned, or insert catheter into another vein
Catheter gives good RV waveform but will not pass into PA at or beyond expected length (40–45 cm)	RV may be abnormally large & catheter may not have reached pulmonic valve pulmonic valve may be small or stenotic	Advance a few more cm carefully; do not overinflate balloon; consider fluoroscopic or radiographic guidance

(continued)

### Complications of PA Catheter Placement (Continued)

Complication	Consider	Remedy
Catheter wedges (morphology of waveform changes & envelope is smaller) but not convincingly	RV may be large & PA catheter may be coiling in RV; undiagnosed intracardiac R-to-L shunt should be considered Consider intrapulmonary shunting, MS or MR; not uncommon in pts with systemic shunting (chronic liver failure)	Consider stopping attempts to wedge & try following PAD in lieu of PCWP
Catheter performs perfectly before & after wedging & deflation, but over time, appears to have wedged again on its own without being advanced manually	Catheter may have warmed & floated out distally (with balloon uninflated), thereby wedging tip into smaller pulmonary arteriole ( <i>This is a setup for PA rupture!</i> )	<b>DO NOT</b> reinflate balloon! Withdraw the catheter to 20 cm & if CVP waveform is good, refloat catheter
PA waveform morphology cyclically changes or envelope varies with PEEP or during the respiratory cycle	Likely hypovolemia; When airway pressure ↑, PA compression occurs → waveform morphology & magnitude may change	Administer fluid, follow PAD, & observe waveform
Massive hemoptysis observed in tracheal tube or severe shock develops suddenly	<b>PA rupture</b>	Do not manipulate catheter; resuscitate as a team: Call for help (thoracic/cardiac surgeon); extubate & insert double lumen tube; stop bleeding from affected lung by inserting Fogarty catheter into lumen from which bleeding is seen; watch for compliance changes & obtain radiograph if shock occurs without hemoptysis; CPB may be needed during PA repair
PA catheter is fixed & cannot be withdrawn	Either knotting has occurred, some mechanical catch has occurred with the sheath, or catheter has been inadvertently sutured in place	Don't pull harder—this can be very hazardous. Consult interventional radiologist or surgeon for diagnosis & treatment
PCWP was damped appropriately when wedged, but with posteroinferior ischemic changes on ECG, large waves are seen in wedge position	Consider ischemia in posterior papillary muscle of mitral valve; this may have led to large "v" waves as the ischemic muscle caused sudden regurgitation in the formerly sound valve	Treat underlying ischemia or infarction as it becomes feasible; afterload reduction, aortic balloon counterpulsation, inotropic support, & diuresis may be necessary

Normal Values for Hemodynamic Variables			
Variable	Value		Units
HR	60–100		beats/min
SBP	90–140		mmHg
DBP	60–90		mmHg
MAP	70–105		mmHg
RAP (= CVP)	2–6		mmHg
RVSP	15–25	:	mmHg
RVDP	0–8		mmHg
RVMP	5–14		mmHg
PASP	15–25	:	mmHg
PADP	8–15		mmHg
PAMP	10–20		mmHg
PAOP = PCWP	6–12		mmHg
PCWP = LAP			

Summary of Derived Hemodynamic Variables			
Variable	Formula	Approximate Value	Units
CO	$CO = SV \times HR$	4.0–8.0	L/min
SV	$SV = CO \div HR$	60–100	mL/beat
SVI	$SVI = CI \div HR$	33–47	mL/m <sup>2</sup> -beat
BSA	$BSA = W^{0.425} \times H^{0.725} \times 0.007184$ (formula of Dubois and Dubois)	1.73	BSA in m <sup>2</sup> ; W in kg; H in cm
CI	$CI = CO \div BSA$	2.31–4.62	L/min
SVR	$SVR = (80 \times [MAP - CVP]) \div CO$	900–1500	dyne-sec/cm <sup>5</sup>
SVRI	$SVRI = (80 \times [MAP - CVP]) \div CI$	1600–2400	dyne-sec/cm <sup>5</sup> -m <sup>2</sup>
PVR	$PVR = 80 \times (MPAP - PAWP) \div CO$	<250	dyne-sec/cm <sup>5</sup>
PVRI	$PVRI = 80 \times (MPAP - PAWP) \div CI$	255–285	dyne-sec/cm <sup>5</sup> -m <sup>2</sup>

Summary of Advanced Derived Hemodynamic Values			
Variable	Formula	Approximate Value	Units
LV stroke work = LVSW	$SV \times (MAP - PAWP) \times 0.0136$	58–104	g-m/beat
LV stroke work index = LVSWI	$SVI \times (MAP - PAWP) \times 0.0136$	50–62	g-m/m <sup>2</sup> -beat
RVSW	$SV \times (MPAP - RAP) \times 0.0136$	8–16	g-m/beat
RVSWI	$SVI \times (MPAP - RAP) \times 0.0136$	5–10	g-m/m <sup>2</sup> -beat
Coronary perfusion pressure (CPP)	DBP – PAWP	60–80	Mm Hg
RVEDV	SV/EF	100–160	mL
RVESV	EDV – SV	50–100	mL
RVEF	SV/EDV	40–60	Percent

# MECHANICAL VENTILATION

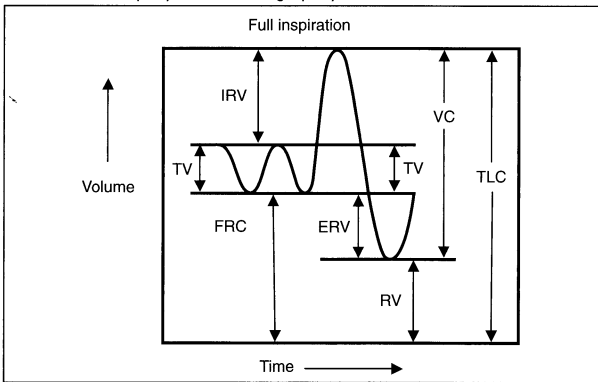
FRANCIS X. DILLON

## INTRODUCTION AND OVERVIEW

### Common ventilation modes

- Volume control (VC): A predetermined volume is delivered in a given interval
- Pressure control (PC): A predetermined pressure is delivered in a given interval

**Figure 8-1 Lung volume subdivisions:** FRC = functional reserve capacity; IRV = inspiratory reserve volume; TV = tidal volume; ERV = expiratory reserve volume; RV = residual volume; VC = vital capacity; TLC = total lung capacity.



## ADVANTAGES OF SPONTANEOUS VS POSITIVE-PRESSURE VENTILATION

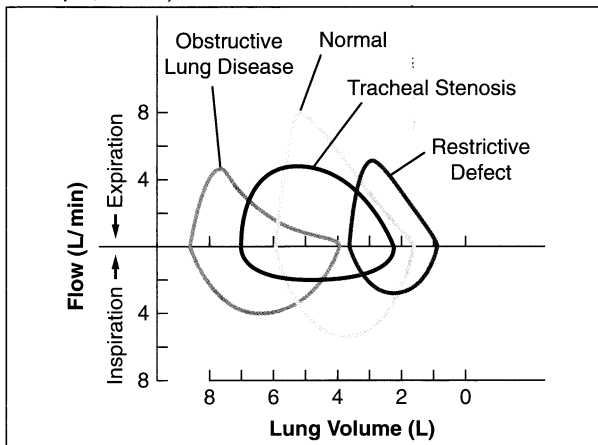
- If coughing is impossible (pt intubated, sedated, anesthetized), lungs may become atelectatic
  1. Occurs within minutes & secretions may accumulate
  2. Presence of ETT or tracheostomy obliterates intrinsic PEEP  
(pts cannot cough effectively → predisposes to atelectasis, secretion retention)
  3. Cumulative incidence of ventilator associated pneumonia (VAP) is approximately 1–3% per day of intubation
- PEEP, suctioning, & alveolar recruitment maneuvers counteract atelectasis
- Extubation as soon as feasible allows cough & normal respirations to occur
- Use of supplemental oxygen:
  1.  $FiO_2 \leq 50\%$  → usually nontoxic; pure  $O_2$  must be given for >16 hr for toxicity
  2. Using supplemental  $O_2$  postop may prevent wound infections
  3. COPD pts may tolerate supplemental  $O_2$  without becoming apneic

### Low-Flow Supplemental O<sub>2</sub>: Typical Means of Administration

Method of Low-Flow O <sub>2</sub> Administration	Device	O <sub>2</sub> Flow (L/min)	FI <sub>O<sub>2</sub></sub>	Capnography Feasible	Humidified	Complications	Comment
Nasal cannula O <sub>2</sub>	Nasal cannula attached to standardized flowmeter	0 1 2 3 4 5 6 7	0.21 0.25 0.29 0.33 0.37 0.41 0.45 0.49	Yes with specially designed cannulas; attach to sampling port of capnograph	Yes	Dried nasopharynx; swallowing of air; bleeding, sinusitis	Pts may mouth-breathe or have nasal constriction, sinusitis, packing, nasogastric tubes, etc. (add 4% FI <sub>O<sub>2</sub></sub> for each L inc of O <sub>2</sub> )
Venturi face mask	Venturi diluters attached to tubing	10+	0.30-0.50	No	Yes		Fixed FI <sub>O<sub>2</sub></sub> diluters entrain room air
Simple face mask		10+	0.55	No	No	Drying of mucosa	
Nonbreather face mask with reservoir bag		10+	0.80	No	No	Drying of mucosa	
Ambu (bag-valve-mask)	Has 15-mm adapter to attach standard mask or ET tube	10+	1.0	No	No	Spontaneous breaths difficult to see in bag	Spontaneous breathing or breaths from caretakers; PEEP feasible
Oxygen tent		10-15	0.50	Yes	Yes	Combustion risk	Used in pediatrics

## FLOW-VOLUME LOOPS

Figure 8-2 Normal and Abnormal Flow Volume Loops (Reproduced with permission from Barash PG. *Clinical Anesthesia*, 6th ed. Lippincott Williams & Wilkins: Philadelphia, PA: 2009.)



## MECHANICAL VENTILATION: PROTECTIVE STRATEGIES

### Current Strategies to Prevent Volutrauma, Barotrauma, Atelectrauma, Tracheal Ischemia & O<sub>2</sub> Toxicity

- Alveolar overdistention (volutrauma), rather than excessively ↑ airway pressure (barotrauma), may be more injurious to lung
- Smaller tidal volumes (6 mL/kg) are recommended with greater RR
- Higher PaCO<sub>2</sub> levels allowed (permissive hypercapnia) in treating acute lung injury & ARDS

### Specific Settings

- TV 6 mL/kg (prevents volutrauma: trauma from overdistention of alveoli)
- Plateau pressure <30 cm H<sub>2</sub>O (prevents barotrauma: Trauma from excessive pressure)
- PEEP >6–10 cm H<sub>2</sub>O (prevents atelectatic trauma: Repeated alveolar closure at end-expiration)
- FiO<sub>2</sub> <50% to prevent O<sub>2</sub> toxicity

### Effects of Positive End-Expiratory Pressure (PEEP)

Pulmonary System	
Advantages	Remarks
Improves hypoxemia	Hypoxemia caused by ARDS, pneumonia, pulm. edema, drowning, atelectasis
Opens airways throughout resp. cycle	By recruiting alveoli & larger lung segments
↑ FRC	Further improving oxygenation
Improves pulmonary compliance	Further ↓ risk of barotrauma & hypotension
↓ Inspiratory work of breathing	Further improving oxygenation, esp. effective if pt is trapping air (auto-PEEP)
Prevents derecruitment	Protects against atelectasis esp during long surgeries
Treats auto-PEEP (airway trapping 2 <sup>o</sup> ↓ expiratory phase in asthma/COPD)	Diagnose this by looking at flow-time curve on ventilator (flow not returning to zero before inspiration = auto-PEEP)
Prevents atelectatic trauma (repetitive closure of alveoli at end-expiration)	Atelectatic trauma correlates with ↑ serum cytokines & markers of lung injury
Allows to ↓ FiO <sub>2</sub>	May ↓ risk of O <sub>2</sub> toxicity

(continued)

**Effects of Positive End-Expiratory Pressure (PEEP) (Continued)**
**Pulmonary System**

<i>Disadvantages</i>	<i>Remarks</i>
May cause barotrauma	Types of barotrauma include pneumoperitoneum, pneumomediastinum, pneumopericardium, pneumothorax, subcutaneous emphysema, tension pneumothorax, shock
May enlarge pneumothorax	Contraindicated even if a small pneumothorax is present without chest catheter or tube
May worsen V/Q mismatching	Esp by enlarging West Zone 1 (apex of lung) where alveolar pressure > arterial pressure > venous pressure
↑ Dead-space ventilation ( $V_D/V_T$ )	Adds work without $O_2$ for pt
May worsen bronchopleural fistula leak	Contraindicated in bronchopleural fistula
May cause significant leak following tracheal/pulm surgery (pneumonectomy)	Contraindicated in recent tracheal surgery, lobectomy, or pneumonectomy unless chest tube present
May lead to ↑ IV administration	

**Cardiovascular System**

<i>Advantages</i>	<i>Remarks</i>
If successful, improves $AaDO_2$ & $O_2$ delivery to tissues & myocardium	
Treats airway obstruction (OSA)	OSA associated with dysrhythmias, myocardial infarction, death
↓ LV afterload	

<i>Disadvantages</i>	<i>Remarks</i>
↓ Left & right ventricular preload	Primary mechanism for reducing C.O.
↓ Venous return	Primary mechanism for reducing C.O.
↑ CVP	Makes hard to assess fluid & preload status with CVP
↑ RV afterload	Worsens RV failure in certain individuals
↓ Ventricular compliance	Causes interventricular septum to bulge into LV (↓ C.O.)
Worsens hypotension in hypovolemia	As in hemorrhage, dehydration, sepsis

**Central Nervous System**

<i>Advantages</i>	<i>Remarks</i>
↑ $O_2$ delivery to CNS	If indeed PEEP is effective at treating hypoxemia
Treats OSA	OSA greatly ↑ risk of stroke
<i>Disadvantages</i>	<i>Remarks</i>
↑ Intracranial pressure	By ↑ CVP & by directly ↑ CSF pressure
↓ Cerebral perfusion pressure	By ↓ MAP & ↑ CVP
↑ Risk of embolization into left circulation	May ↑ risk of opening an occult probe-patent foramen ovale (cause sudden R to L shunt, hypoxemia, embolization)

**Hepatic, Renal, Neuroendocrine Systems**

<i>Advantages</i>	<i>Remarks</i>
↓ Renal & hepatic blood flow (↓ C.O.)	May affect clearance of drugs
<i>Disadvantages</i>	<i>Remarks</i>
↑ ADH secretion	Various effects but largely helps retain free water (preload)



## Overview of Modes of Ventilation

### Simple modes for use in OR

Mode of Ventilation	Advantage	Disadvantage	NMB Needed or Feasible?	Triggered by Breath?	Synchronized to Breath?	Spontaneous Breathing Allowed?
Volume control (VC)	Simple, delivers fixed TV & RR	May give excessive pressure if compliance decreases	Feasible	No: Breaths timed by ventilator	No	Yes but pt may hyperventilate
Pressure control (PC)	Simple, delivers fixed plateau pressure & RR	May give inadequate volume if compliance decreases	Feasible if ventilator is time-cycled	No	No	Yes but pt may hyperventilate

### Simple modes for longer-term support in ICU

Assist-control (AC)	Delivers fixed minimum TV or pressure with each triggered breath	Each breath triggered means a ventilator breath delivered	Feasible & may be helpful to pt	Yes	Yes	Yes, but because every breath is supported by the ventilator, hyperventilation may occur
Intermittent mandatory ventilation (IMV)	Like AC but also allows periods of spontaneous ventilation	Spontaneous breathing periods not well tolerated without PS	No if spontaneous respirations desired	No	No	Yes

### Modes used for weaning in ICU or PACU

Pressure support (PS)	Augments spontaneous breathing; good for weaning	Sub-optimal if no reliable respiratory drive	Neither	Yes	Yes	Yes, it must occur or there will be no PS
Spontaneous intermittent mandatory ventilation (SIMV)	Synchronized to breath	None	Feasible—but without spontaneous RR, this is just AC	No	Yes	Yes

### Modes used to treat severe hypoxemia or lung injury in ICU

Inverse ratio (IR): I:E ratio inverted, $\geq 1$	For severe hypoxemia; may be used in either PC or VC setting	Poorly tolerated in awake pts; barotrauma possible if they cough	May be needed if heavy sedation is insufficient	No	No	No
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**Overview of Modes of Ventilation (Continued)**

Mode of Ventilation	Advantage	Disadvantage	NMB Needed or Feasible?	Triggered by Breath?	Synchronized to Breath?	Spontaneous Breathing Allowed?
Pressure-regulated volume control (PRVC)	Can set min RR, target TV, upper insp pressure limit; ventilator adjusts pressure from breath to breath according to compliance; less barotrauma	Longer inspiratory time may lead to more auto-PEEP	Feasible but not beneficial if there is no spontaneous breathing	Yes	Yes	Yes
Airway pressure-release ventilation (APRV)	Allows spontaneous respiration and reduced risk of barotrauma	As in PC, TV may vary depending on compliance	Neither	No	No	Yes: this mode is like CPAP at a high level, allowing spontaneous breaths with intermittent release of CPAP
Continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP)	↑ Oxygenation without active support; demonstrates weaning	Requires good respiratory drive and effort	Neither	No	No	Yes. PEEP and CPAP are similar; CPAP occurs during spontaneous breathing; PEEP occurs during positive pressure ventilation
Bilevel positive airway pressure (BIPAP)	Allows spontaneous respiration in hypoxemic pts	Lower mean airway pressure than APR; less barotrauma	Neither	No	Yes	Yes: This is like APRV but with a longer expiratory phase
High-frequency ventilation (HFV)	Different types used for treating hypoxia and preserving CO	Not widely available or commonly used				

PRVC, HFOV, PRVC, & APRV ventilation, allow significant ↑ in avg. airway pressure (recruit more alveoli & improve AaDO<sub>2</sub>) & allow pt to breathe spontaneously; advantage = less sedation & muscle relaxation required

## BiPAP vs CPAP

- CPAP = delivery of continuous positive air pressure throughout respiratory cycle
- BiPAP = two levels of positive air pressure, inspiratory & expiratory  
(expiratory pressure is lower to facilitate exhalation)

## DISCONTINUING MECHANICAL VENTILATION

### SIMV Plus PS is a Common Weaning Mode in Many ICUs

- Start with full support (IMV = 10 or so), plus PS 10–15 cm H<sub>2</sub>O, PEEP 5–10
- Decrease by 1–2 breaths per minute till IMV = 0
- Now wean PS 1–2 cm H<sub>2</sub>O at a time until PS/PEEP is 10/5 or 5/5 cm H<sub>2</sub>O or lower (for more deconditioned pts, you may need to go as low as PS/PEEP = 2/5)
- At the same time gradually reduce FiO<sub>2</sub> according to SaO<sub>2</sub> or PaO<sub>2</sub> (more sensitive to oxygenation changes)

Criteria for Extubation Used in Many ICUs	
Criterion	Remarks
Five-second head lift (correlates with NIP of 50 cm H <sub>2</sub> O): best indicator of upper airway strength	Conscious head lift to command, not from coughing, is optimal
PaO <sub>2</sub> /FiO <sub>2</sub> > 120 or equivalently, PaO <sub>2</sub> > 60 on FiO <sub>2</sub> of 0.50	O <sub>2</sub> requirement should be 50% or lower before extubation is considered; FiO <sub>2</sub> 40% is better
PEEP support < 6 cm with adequate oxygenation	Sudden withdrawal of PEEP may cause fall in PaO <sub>2</sub>
CNS intact (GCS ≥ 12)	See GCS Scale, Chapter 20
Ability to maintain PaCO <sub>2</sub> < 50 mm Hg and 7.30 < pH < 7.50	Sepsis, hyperthermia, hyperalimentation, ↑ CO <sub>2</sub> production; these causes may be addressed; metabolic alkalosis from GI losses or diuretics may cause CO <sub>2</sub> retention; treat alkalosis with NaCl & KCl in IV fluid or other measures
Ability to maintain spontaneous respiration without tiring	Extubation of pts from PS of ≥ 5 cm H <sub>2</sub> O may cause them to tire after extubation
Ability to clear secretions without plugging bronchioles	Assess secretions prior to extubation, remember anticholinergic side effects of opioids
No overt risk of pulm aspiration (no ongoing bowel obstruction or high NG/OG output)	Aspiration may occur with intact airway reflexes if bowel obstruction is severe or GI bleeding is brisk
Adequate Hb, cardiac output, & cardiovascular stability	Hg ≥ 10 allows adequate O <sub>2</sub> carrying capacity (for AaDO <sub>2</sub> ) & viscosity (for maintaining SVR & therefore BP)
Pain is controlled adequately to allow spontaneous breathing without excessive splinting	Consider intercostal, intervertebral, epidural, or intrapleural local anesthetics or subarachnoid block to allow deep breathing and cough

## CAPNOGRAPHY

CO<sub>2</sub> identified in expired gases by various spectrographic properties (using either IR, Raman, or mass spectroscopy)

### Uses and Indications for Capnography

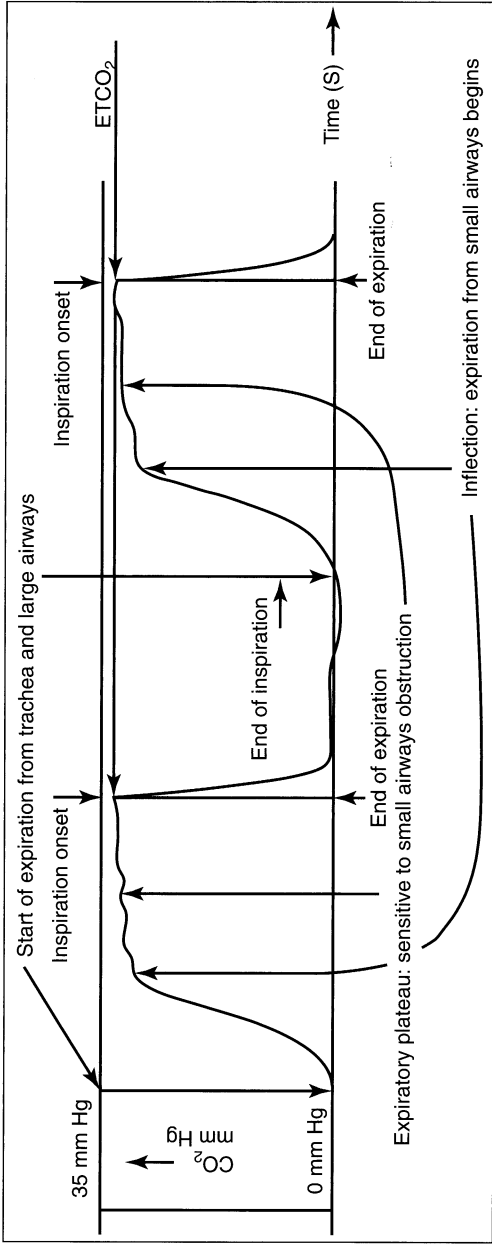
- Detection of esophageal intubation
- Detection of disconnection from breathing circuit, inadvertent extubation, or insufficient ventilation
- Detection of hyperventilation
- Detection of high metabolic rates: (i.e., Malignant hyperthermia or sepsis)
- Detection of low metabolic rates: (i.e., Hypothermia, lowered cardiac output states)
- Detection of pulmonary embolism
- Detection of bronchospasm/small airway obstruction
- Calculation of V<sub>d</sub>/V<sub>t</sub> or dead space ventilation (need PaCO<sub>2</sub> & ET-CO<sub>2</sub>)  
$$V_D/V_T = [(PaCO_2 - P_{ET}CO_2)/PaCO_2]; \text{ normal } V_d/V_t = 0.30$$

**Hazards of capnography are few in number, may be of great magnitude when:**

- Old in-line IR detectors can heat up, causing facial thermal injury
- May fail to detect disconnection if machine disabled by secretions

CAPNOGRAPHS

Figure 8-3 Normal capnograph (normal expired CO<sub>2</sub> waveform). (Courtesy of Prof. David Sainsbury, University of Adelaide, Australia.)



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# FLUIDS, ELECTROLYTES, & TRANSFUSION THERAPY

ARANYA BAGCHI

## FLUID MANAGEMENT

### Fluid Compartments

- Total body water (TBW)  $\approx$  60% body wt in males, 50% in females (TBW inversely proportional to amount of adipose tissue in body)
- Intracellular fluid (ICF)  $\approx$  2/3 and extracellular fluid (ECF)  $\approx$  1/3 of TBW
  - Interstitial fluid volume (IFV  $\approx$  2/3 of ECF)
  - Plasma volume (PV  $\approx$  1/3 of ECF)

### Assessing Volume Status

- Hypovolemia may include:
  - Volume depletion (loss of Na & water from ECF)
  - Dehydration (loss of water from ICF)
- Accurate assessment of volume status is challenging; entire clinical picture must be considered
- A systematic approach to estimating blood volume (see Figure 9-1 below)

### Volume Deficits (also see Chapter 22)

- ICF deficits (free water losses): Insensible losses from skin & resp system, free water loss by kidneys (diabetes insipidus, central or nephrogenic)
  - Characterized by cellular dehydration,  $\uparrow$  plasma osmolality & Na
  - Won't present as acute circulatory collapse; gradually correct with free water
- ECF deficits: Blood loss, GI losses (vomiting, diarrhea) & distributional changes (ascites, burns, "3rd spacing")
  - Labs:  $\rightarrow$   $\uparrow$  BUN/Cr ratio,  $\downarrow$  urinary Na excretion ( $<20$  mEq/L) & conc urine
  - Can cause rapid circulatory instability; treat expeditiously with isotonic crystalloids, colloids, or blood

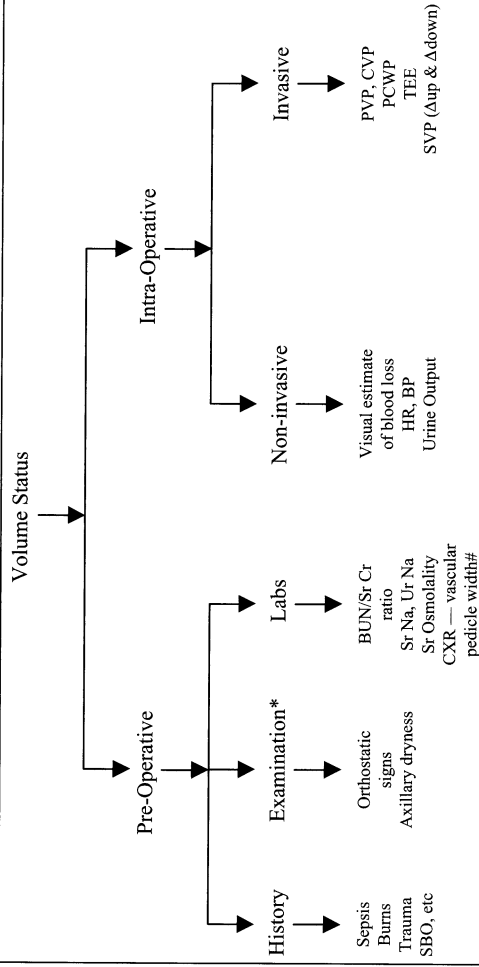
### Volume Replacement

- Expected plasma volume (PV) increment:  
 $\Delta PV = \text{volume infused} \times (\text{baseline PV}/V_d \text{ of infused fluid})$
- Traditional approaches to fluid replacement (using isotonic crystalloids):  
**Fluid replacement = maintenance + deficit + 3rd space + losses**
  - Maintenance (mL/h): 4-2-1 formula (4 mL/kg/hr for 1st 10 kg; 2 mL/kg/hr for next 10 kg; 1 mL/kg/hr beyond that)
  - Deficit (mL): Maintenance  $\times$  hours NPO; 50% given in 1st hr & 25% in 2nd and 3rd hr
  - Third space (mL/hr): Depending on magnitude of surgical insult—4 mL/kg/hr (mild), 6 mL/kg/hr (moderate), 8 mL/kg/hr (severe)
  - Losses: Blood loss replaced with  $\times 3$  crystalloid, or  $\times 1$  colloid; other losses (ascitic fluid) replaced according to estimated electrolyte composition
- Traditional approach criticized
  - Leads to overresuscitation; studies suggest more restrictive approach to fluid replacement  $\rightarrow$  assoc with improved outcomes (*Ann Surg* 2003;238: 641)
  - Not tailored to dynamic response of individual pt to stress of surgery
  - Underestimates crystalloid amount required to replace blood losses  
animal studies suggest ratios of 4:1 to 5:1 needed (*Arch Surg*. 1969;98:281)

### Crystalloids & Colloids

- No evidence for superiority of either colloids or crystalloids (may be some advantages in using each in specific subgroups of pts)
- IV fluids can have proinflammatory properties (*Resuscitation* 2004;60:91) (LR, NS, & hetastarch  $\rightarrow$  shown to  $\uparrow$  neutrophil activation; blood, 7.5% hypertonic saline, & albumin did not have this effect)

Figure 9-1 Assessment of volume status.



SBO, sm bowel obstruction; PVP, peripheral venous press.; TEE, transesophageal echo; SVP, systolic press. Variation on a-line trace in mechanically ventilated pts.

\*Only orthostatic signs (postural Δ in HR > 30 bpm, postural dizziness) & axillary dryness are predictive of hypovolemia due to blood loss. (JAMA 1999; 281:1122)

#Vascular pedicle width on CXR correlates well with volume status (Chest 2002; 121:942)

Compositions of Crystalloids & Colloids					
Fluid	Na (mEq/mL)	K (mEq/mL)	Osm (mOsm/L)	pH	Other
Extracellular fluid	140	4.5	290	7.4	Cl = 108 Lactate = 5 mEq/L
D5W	0	0	252	4.5	Glucose 50 g/L
0.9 NS	154	0	308	6.0	Cl = 154
LR	130	4	273	6.5	Ca = 3 mEq/L Lactate = 28 mEq/L HCO <sub>3</sub> = 27mEq/L Cl = 110
5% Albumin	145 ± 15	<2.5	330	7.4	COP = 32–35 mm Hg
6% Hetastarch	154	0	310	5.9	COP = 30 mm Hg
D5¼ NS	39	0	320	4.2	Cl = 39
Plasmalyte	140	5	294	7.4	Cl = 98
3% NS	513	0	1025	5.6	Cl = 513

Source: From Kaye AD, Kucera JJ, Intravascular fluid & electrolyte physiology. In *Miller's Anesthesia*, 6th ed. Elsevier: Philadelphia, PA:2005.

Colloid Osmotic Pressure (COP) = net osmotic pressure exerted by plasma proteins

#### Daily Adult Electrolyte Requirements

Electrolyte	Daily Requirements
Na	60–150 mEq
K	70–150 mEq
Cl	60–150 mEq
Mg	8–24 mEq
Phos	7–10 mmol/1000 kcal

#### Advantages/Disadvantages of Colloids & Crystalloids

Solution	Advantages	Disadvantages
<b>Colloid (albumin, hetastrach, dextran)</b>	<ul style="list-style-type: none"> <li>↓ Infused volume</li> <li>↑ Time in plasma volume (<i>maintenance of oncotic pressure</i>)</li> <li>↓ Peripheral &amp; cerebral edema</li> <li>maintains plasma osmolality</li> <li>↑ O<sub>2</sub> delivery</li> <li>↓ Inflammatory response</li> </ul>	<ul style="list-style-type: none"> <li>↑ Cost</li> <li>Coagulopathy</li> <li>Pulmonary edema (<i>in capillary leak states</i>)</li> <li>↓ GFR (<i>renal tubule obstruction</i>)</li> <li>Allergic reactions possible (<i>dextran &gt; hetastrach &gt; albumin</i>)</li> </ul>
<b>Crystalloid</b>	<ul style="list-style-type: none"> <li>↓ Cost</li> <li>↑ Urinary flow</li> <li>Replaces IFV</li> </ul>	<ul style="list-style-type: none"> <li>Hemodynamic improvement transient</li> <li>Peripheral edema (<i>protein dilution</i>)</li> <li>Pulmonary edema (<i>fluid overload</i>)</li> <li>Dilutional coagulopathy</li> </ul>

Source: From Prough DS, et al. Acid-base, fluids & electrolytes. In Barash PG, Cullen BF, Stoelting RK, eds., *Clinical Anesthesia*, 5th ed. Lippincott Williams & Wilkins: Philadelphia, PA:2005.

#### Notes on Specific Fluids

- **5% Dextrose**—used to replace free water; isotonic to plasma but rapidly becomes free water (dextrose is metabolized); used to treat dehydration losses; limited intra-operative use
- **Lactated Ringer's (LR)**—most widely used solution; lactate metabolized in liver to CO<sub>2</sub> & water; **unsuitable** for pts with **end-stage liver dz**; mixing LR with PRBCs → leads to clotting (2<sup>o</sup> to LR's calcium content)
- **0.9% Saline** or "normal" saline (NS)—widely used in OR; useful in neurosurgery because of its osmolality; widely used in pts with renal failure; administration of large volumes of NS → lead to *hyperchloremic acidosis*
- **Hypertonic Saline (3% & 7.5%)**—2 well-defined uses: (1) intravascular volume expansion in hypovolemic shock, (2) reduce cerebral volume & ICP
- **Albumin**—5% & 25% conc. available; circulatory half-life normally 16 hr (as short as 2–3 hr in pathophysiologic conditions); made from pooled human blood; minimal/no risk of transmitting infections; 5% albumin = isotonic with plasma; concerns that albumin ↑ mortality are unfounded (SAFE study. *N Engl J Med* 2004;350:2247)
- **Hydroxyethyl starch (hetastarch)**—high-molecular-weight synthetic colloid, ↑ plasma COP up to 2 d; available as 6% solution in NS or LR; renally excreted; side



effects → elevates serum amylase, anaphylactoid rxns, coagulopathy (inhibits platelets); to minimize plt inhibition → restrict dose to 20 mL/kg/day

- **Dextran**—dextran 40 & dextran 70 (numbers refer to average molecular mass of solution); side effects → anaphylactoid rxn, ↑ bleeding time, interference with blood cross-matching; rare cases of noncardiogenic pulmonary edema; renal obstruction/acute renal failure

Dextran 40 → used in vascular surgery to prevent thrombosis;  
6% dextran 70 → used for same indications as 5% albumin

## APPROACH TO ACID BASE ANALYSIS

Check arterial pH

pH < 7.40 (acidic)

- $PCO_2 > 40$  respiratory acidosis
  - Hypoventilation (e.g., overdose)
  - Obstruction (COPD)
  - Decr respiratory drive (EtOH, drugs)
  - Neuromuscular disease
- $PCO_2 < 40$  metabolic acidosis
  - Check gap:  $Na - (bicarb + Cl)$ 
    - Normal gap ( $12 \pm 2$ )
    - Decreased bicarb
      - Diarrhea
      - Renal tubular acidosis
    - Increased gap ( $> 12$ )
      - Methanol
      - Uremia
      - DKA (diabetic ketoacidosis)
      - Paraldehyde
      - INH (isoniazid)
      - Lactic acidosis
      - Ethylene glycol (antifreeze)
      - Salicylates

pH > 7.40 (alkalemic)

- $PCO_2 < 40$  respiratory alkalosis
  - Hyperventilation
- $PCO_2 > 40$  metabolic alkalosis
  - Vomiting
  - Diuretics
  - Antacid abuse
  - Increased aldosterone

### Primary Acid/Base Disorders

1° Disorder	Problem	pH	PaCO <sub>2</sub>	HCO <sub>3</sub>
Metabolic acidosis	Gain of H <sup>+</sup> or loss of HCO <sub>3</sub>	↓	↓	↓
Metabolic alkalosis	Gain of HCO <sub>3</sub> or loss of H <sup>+</sup>	↑	↑	↑
Resp. acidosis	Hypoventilation	↓	↑	↑
Resp. alkalosis	Hyperventilation	↑	↓	↓

### Acid/Base: Rules of Compensation

1° Disorder	Formula
Metabolic acidosis	↓ PaCO <sub>2</sub> = $1.25 \times \Delta HCO_3$ (also, PaCO <sub>2</sub> = last two digits of pH)
Metabolic alkalosis	↑ PaCO <sub>2</sub> = $0.75 \times \Delta HCO_3$
Acute Resp. acidosis	↑ HCO <sub>3</sub> = $0.1 \times \Delta PaCO_2$ (also, ↓ pH = $0.008 \times \Delta PaCO_2$ )
Chronic Resp. acidosis	↑ HCO <sub>3</sub> = $0.4 \times \Delta PaCO_2$ (also, ↓ pH = $0.003 \times \Delta PaCO_2$ )
Acute resp alkalosis	↓ HCO <sub>3</sub> = $0.2 \times \Delta PaCO_2$
Chronic resp alkalosis	↓ HCO <sub>3</sub> = $0.4 \times \Delta PaCO_2$

Source: Adapted from *Pocket Medicine*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2008, chapters 1-4.

## ELECTROLYTES

**Hyponatremia** (Na<sup>+</sup> < 135 mEq/L)

- *Etiology* (see Figure 9-2 below)
- *Symptoms*—nausea, vomiting, weakness, muscle cramps, visual disturbances, ↓ level of consciousness, agitation, seizures, coma

Cerebral edema → when  $\text{Na}^+ < 123 \text{ mEq/L}$

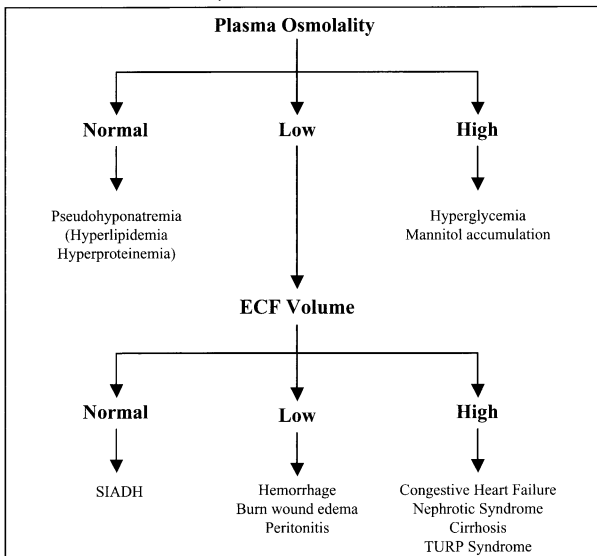
Cardiac symptoms → when  $\text{Na}^+ < 100 \text{ mEq/L}$

- **Treatment**—mild hyponatremia → free water restriction ± loop diuretics  
severe hyponatremia with neurologic symptoms → 3% hypertonic saline  
 $\text{Na}^+$  dose (mEq) =  $[\text{IBW (kg)} \times (140 \text{ Na}^+) (\text{mEq/L})] \times 0.6 \times (0.85 \text{ in women})$   
correction rate → do not exceed 0.6–1 mEq/L/hr or 8 mEq/L over 24 hr  
(too rapid correction → central pontine myelinolysis)
- **SIADH treatment**
  - Free water restriction; demeclocycline used for chronic cases
  - Can use loop diuretic + fluid replacement with hypertonic saline

### Guide to Using Hypertonic Saline

1. Calc Na deficit that needs to be repleted to achieve Na of 120
2. Calc no. liters of 3% saline (513 mEq of Na/L) needed to replete Na deficit
3. Calc rate of infusion to achieve replacement at 0.5 mEq/L/hr

**Figure 9-2 Etiology of hyponatremia.** (Modified from Braunwald E, Fauci AS, Kasper DL, and Hauser SL, eds. *Harrison's Principles of Internal Medicine*, 15th ed. New York: McGraw-Hill, 2001.)



Fluids 9-5

### Hypernatremia ( $\text{Na}^+ > 145 \text{ mEq/L}$ )

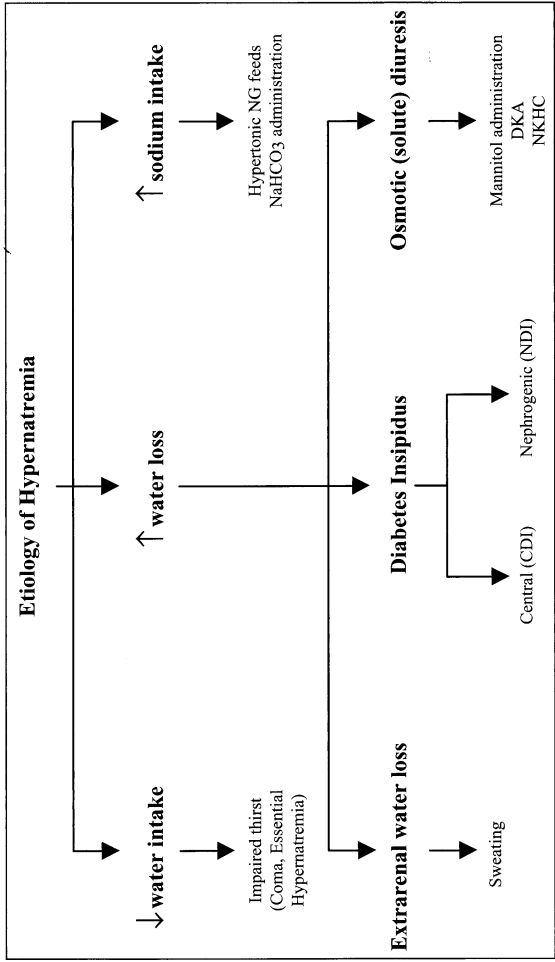
- **Etiology** (see Fig. 9-3, below)
- **Symptoms**—thirst, lethargy, mental status changes → coma/convulsions
  - Slowly developing hypernatremia = usually well tolerated
  - Acute severe hypernatremia → cellular dehydration → brain shrinkage → meningeal vessels tear → intracranial hemorrhage
- **Treatment**—restore normal osmolality & volume by correcting water deficit  
water can be replaced PO (safest) or by IV infusion of free water or hypotonic crystalloids

$$\text{Free water deficit (L)} = \left[ \frac{[\text{Na}^+] - 140}{140} \right] \times \text{TBW (L)}$$

lower plasma Na by 0.5 mEq/L/hr; no more than 12 mEq/L/24 hr  
(too rapid correction → acute brain swelling)

- Central diabetes insipidus—treated with intranasal DDAVP
- Nephrogenic diabetes insipidus—may be reversible if cause identified
- Symptomatic polyuria—treated with Na restriction & thiazide diuretics

Figure 9-3 Etiology of hypernatremia. DKA, diabetic ketoacidosis; NKHC, nonketotic hyperosmolar coma.



NKHC: non-ketotic hyperosmolar coma; NDI: nephrogenic diabetes insipidus

## Hypokalemia ( $K^+ < 3.5$ mEq/L)

Major Causes of Hypokalemia	
Mechanisms	Causes
Inadequate intake	Alcoholism, hyperaldosteronism, starvation, anorexia nervosa
Renal losses	Diuretics, chronic metabolic alkalosis, $\downarrow Mg^{2+}$ , renal tubular acidosis
GI losses	Vomiting, diarrhea, villous adenoma
ECF to ICF shift of $K^+$	$\beta_2$ agonists, insulin, acute alk, vit B <sub>12</sub> therapy, lithium overdose

Source: From Kaye AD & Kucera IJ. intravascular fluid & electrolyte physiology. In *Miller's Anesthesia*, 6th ed. Elsevier: Philadelphia, PA: 2005.

- **Symptoms**—fatigue, muscle cramps, progressive weakness leading to paralysis;  $\uparrow$  risk of arrhythmias; can enhance digitalis toxicity; cause hepatic encephalopathy
- **ECG**—U waves, ventricular ectopy,  $\pm$  prolonged QT
- **Treatment**—IV  $K^+$  (up to 40 mEq/L via peripheral IV; 100 mEq/L via central line) infusion at 20 mEq/hr unless paralysis/ventricular arrhythmias present; oral supplementation rarely practical in OR/ICU  
Treat underlying cause; avoid dextrose solutions ( $\uparrow$  insulin  $\rightarrow$  further  $\downarrow K^+$ )

## Hyperkalemia ( $K^+ > 5.5$ mEq/L)

- **Etiology**—see table below.

Etiology of Hyperkalemia	
Mechanisms	Causes
Pseudohyperkalemia	Sample lysis, marked leukocytosis or megakaryocytosis
Altered internal $K^+$ balance	Acidosis, hypoaldosteronism, malignant hyperthermia
Altered external $K^+$ balance	renal failure, drugs (ACEi, ARB, NSAIDs, $K^+$ -sparing diuretics)
Miscellaneous iatrogenic	Sux-induced hyperkalemia, ischemia-reperfusion,

Source: From Kaye AD, Kucera IJ. intravascular fluid & electrolyte physiology. In *Miller's Anesthesia*, 6th ed. Elsevier: Philadelphia, PA: 2005.

- **Symptoms**—cardiac toxicity most serious
- **ECG changes**—peaked T waves  $\rightarrow$  prolonged PR interval & QRS duration  $\rightarrow$  loss of P waves  $\rightarrow$  widening of QRS complex  $\rightarrow$  sine waves (merged QRS & T waves)  $\rightarrow$  V fib/asystole
- **Treatment**—hemodialysis for pt with renal failure & life-threatening hyperkalemia

Treatment of Hyperkalemia			
Intervention	Dose	Onset	Comment
Calcium gluconate Calcium chloride	1–2 amps IV	Few min	Transient effect Stabilizes cell membrane
Insulin	Reg. insulin 10 U IV 1–2 amps D50W	15–30 min	Transient effect Drives $K^+$ into cells
Bicarbonate ( $NaHCO_3$ )	1–3 amps IV	15–30 min	Transient effect Drives $K^+$ into cells in exchange for $H^+$
$\beta_2$ agonists	Albuterol 10–20 mg inh. or 0.5 mg IV	30–90 min	Transient effect Drives $K^+$ into cells
Kayexalate	30–90 g PO/PR	1–2 hr	$\downarrow$ total body $K^+$ Exchanges $Na^+$ for $K^+$ in gut
Diuretics	Furosemide $\geq 40$ mg IV	30 min	$\downarrow$ total body $K^+$
Hemodialysis	For renal failure/life-threatening $\uparrow K^+$		$\downarrow$ total body $K^+$

Source: Adapted from Sabatine MS, ed. *Pocket Medicine*, 3rd ed. Lippincott Williams & Wilkins: Philadelphia, PA: 2008.

## Hypocalcemia ( $Ca^{2+} < 8.4$ mg/dL)

- $Ca^{2+}$  levels must be corrected for serum albumin conc (or use ionized calcium)

$$\text{Corrected } [Ca^{2+}] = [Ca^{2+}] + \{0.8 \times (4.0 - [\text{albumin}])\}$$

- **Etiology**—hypoparathyroidism, pseudohypoparathyroidism, hypomagnesemia, **low vitamin D** levels, hyperphosphatemia (seen in tumor lysis syndrome or rhabdomyolysis), presence of calcium chelating agents
  - Common OR causes: (1) hyperventilation, (2) blood transfusion  $>1.5$  mL/kg/min
- **Symptoms**—acute hypocalcemia  $\rightarrow$   $\uparrow$  nerve/muscle excitability  $\rightarrow$  paresthesias/tetany (Chvostek & Trousseau signs), laryngospasm, hypotension, & dysrhythmias
- **Treatment**—treat hypomagnesemia (if present) first
  - $\text{Ca}^{2+}$  gluconate infusion (2 g in 50–100 mL saline) over 10–15 min  $\rightarrow$  followed by calcium chloride or calcium gluconate infusion (0.5–1.5 mg/kg/hr of **elemental** calcium)
  - 1 g of calcium gluconate = 93 mg elemental calcium;
  - 1 g calcium chloride = 272 mg elemental calcium)

### Hypercalcemia ( $\text{Ca}^{2+} >10.3$ mg/dL)

- **Etiology**— $1^{\circ}$  hyperparathyroidism, malignancy, vit D or A intoxication, immobilization, drugs (thiazides)
- **Symptoms**—mild to moderate hypercalcemia often asymptomatic osteopenia with pathologic fractures, nephrolithiasis, GI symptoms, neurologic symptoms (weakness, confusion, stupor, coma)
  - “stones, bones, abdominal groans and psychic overtones”
- **Treatment**— $1^{\text{st}}$  line treatment = correction of hypovolemia with normal saline
  - Biphosphonates, calcitonin &  $\text{Ca}^{2+}$   $\downarrow$  agents (mithramycin & glucocorticoids)
  - Pts with muscle weakness  $\rightarrow$  receive  $\downarrow$  doses of muscle relaxants

### Hypomagnesemia ( $\text{Mg}^{2+} <1.3$ mEq/L)

- **Etiology**—nutritional (inadequate intake, TPN, chronic alcoholism),  $\uparrow$  renal excretion (hypercalcemia, osmotic diuresis), drugs (diuretics, aminoglycosides, amphotericin B); common in critically ill pts
- **Symptoms**—resp muscle weakness, arrhythmias (torsades de pointes)
- **Treatment**—1–2 g of  $\text{MgSO}_4$  in 15 min, followed by 24-hr infusion (6 g in 1 L)  $\downarrow$  doses & frequent monitoring for pts with renal insufficiency

### Hypermagnesemia ( $\text{Mg}^{2+} >2.5$ mEq/L)

- **Etiology**—usually iatrogenic, seen during treatment of preeclampsia, pts taking  $\text{Mg}^{2+}$  containing antacids/laxatives; renal insufficiency  $\uparrow$  risk
- **Symptoms**—neuromuscular abnl (prolonged PR,  $\uparrow$  QRS) (5–10 mEq/L) lowered DTRs (10 mEq/L) lethargy, weakness & respiratory failure (10–15 mEq/L) hypotension, bradycardia & cardiac arrest ( $>20$ –25 mEq/L)
- **Treatment**—IV calcium (1–2 g calcium gluconate over 10 min); mech ventilation for resp failure; temporary pacing for significant bradyarrhythmias; dialysis may be required if renal insufficiency present

### Hypophosphatemia ( $\text{PO}_4^{3-} <2.8$ mg/dL)

- **Etiology**—malabsorption (vit D deficiency, chronic alcoholism),  $\uparrow$  renal excretion (hyperparathyroidism, osmotic diuresis, post renal transplant) transcellular shifts (insulin admin., resp. alkalosis, malnutrition treatment)
- **Symptoms**—muscular abnl (weakness, impaired diaphragmatic fx, rhabdomyolysis) neurologic abnl (paresthesias, dysarthria, confusion, seizures, coma) hematologic abnl (hemolysis & platelet dysfx)
- **Treatment**—IV phosphate for severe/acute dz
  - Na- or K-phos 0.08–0.16 mmol/kg in 500 mL 0.45% saline over 6 hr
  - Serum phosphate, calcium, & potassium monitored q8hr
  - Stop IV repletion when oral therapy possible
  - Must avoid hyperphosphatemia (can lead to hypocalcemia)

### Hyperphosphatemia ( $\text{PO}_4^{3-} >4.5$ mg/dL)

- **Etiology**—usu.  $2^{\circ}$  to  $\downarrow$  renal excretion (renal failure, hypoparathyroidism, biphosphonate therapy); transcellular shifts (rhabdomyolysis, hemolysis, tumor lysis syndrome) &  $\uparrow$  intake (vit D intoxication, phosphorus cathartics)
- **Symptoms**—attributable to hypocalcemia (see above), & metastatic calcification of soft tissues (when calcium–phosphorus product  $>70$ )
- **Treatment**—correct renal insuff, dialysis for pts with renal failure, phosphorus-binding antacids

## TRANSFUSION THERAPY

### Blood Typing Tests

- ABO incompatibility → most common reason for transfusion reactions (>99%)
- **ABO, Rh typing**
  - 0.2% chance of transfusion reaction after this test
  - Rh-neg pts produce anti-Rh Ab's *only* after being exposed to Rh antigen
- **T/S (type & screen)**
  - Recipient's plasma (may contain antibodies) + stock RBC soln (known ag's) → watch for rxn (testing recipient for presence of Ab's)
  - 0.06% chance of transfusion reaction after this test
- **T/C (type & cross)**
  - Recipient's plasma (may contain antibodies) + donor RBC's → watch for agglutination (suggests incompatibility)
  - Can detect M, N, P, Lewis, Rh, Kell, Kidd, Duffy antibodies
  - 0.05% chance of transfusion reaction after this test

### Emergency Transfusion

- Give **type-specific** or **type O** blood
- Rh-negative pts should receive anti-Rh globulin (if given Rh positive blood)
- After 8–10 units type O whole blood, **do not** switch to type-specific blood (A, B, or AB): Hemolytic rxn possible (due to anti-A & anti-B Ab's in type O transfused blood)

Recipient's Blood			Reactions With Donor's RBCs			
ABO Antigens	ABO Antibodies	ABO Blood Type	Donor is type O	Donor is Type A	Donor is Type B	Donor is Type AB
–	Anti-A Anti-B	O	C	I	I	I
A	Anti-B	A	C	C	I	I
B	Anti-A	B	C	I	C	I
A & B	–	AB	C	C	C	C

C, compatible; I, incompatible

## COMMON BLOOD PRODUCTS

### Red Blood Cells

1 unit ≈ 300 mL: 180 mL RBC, 130 mL storage solution (Hct ≈ 55%); ↑ pt's Hct 3%/unit

*Indications* (practice guidelines, few RCTs)

- Acutely ill, hospitalized pts
- Age <40, Hct <24
- Age 40–60, Hct <27
- Age 60–70, Hct <30
- Acute bleed: Generally not needed for <6% Hct drop
- Chronic anemia without significant underlying cardiovascular disease, Hct <21

### Packed Red Blood Cells (PRBC)

- Used to ↑ O<sub>2</sub>-carrying capacity of blood
- 1 unit of PRBCs can ↑ hematocrit by 2–3%; PRBCs must be ABO- & Rh-compatible
- *Leukocyte reduced* → to prevent nonhemolytic febrile transfusion rxns
- *Washed* → prevent allergic transfusion rxn mediated by recipient Ab's
- Pts without cardiac pathology, transfusion generally not required at hct's >21% (The TRIC Study, *NESM* 1999;340:409–417.)

### Equation for Arterial O<sub>2</sub> Content in Blood

$$(\text{Hemoglobin} \times 1.36) \times \text{SpO}_2 + \text{PaO}_2 \times (0.003)$$

Normal hemoglobin adult range: Male = 13–18 g/dL; female = 12–16 g/dL

Normal PaO<sub>2</sub> (arterial O<sub>2</sub> partial press) = 80–100

*Significance:* 1. As hemoglobin drops, O<sub>2</sub> content drops

2. Hb makes largest contribution to O<sub>2</sub> content in blood

→ Transfusion may be of greater benefit than slight rise in PaO<sub>2</sub> for chronically hypoxic pts

Available Blood Components				
Component	Content	Indications	Volume	Shelf Life
RBCs Whole	RBCs and WBCs, platelet debris, plasma, fibrinogen	Red cell volume and plasma volume replacement	450 ± 50 mL	Heparin 48 hrs ADSOL 42 days ACD 21 days CPD 28 days CPDA-1 35 days
Packed RBCs	RBCs, WBCs, plasma, platelet debris	Red cell volume replacement	200 mL	same as whole blood
Frozen RBCs	No plasma, minimal WBCs & platelet debris	Red cell volume replacement in special circumstances	160–190 mL	Frozen: 3 years Thawed: 24 hours
Platelets	Platelets, low WBCs, some plasma	Platelet count less than 50,000-100,000, clinical signs of dilutional thrombocytopenia and/or platelet dysfunction	30–50 mL/unit	Pheresis: 24 hours Room temperature: 5 days, Frozen with DMSO: 3 years
Fresh frozen plasma	Plasma proteins, all coagulation factors	Bleeding from factor deficiencies, anti-thrombin III deficiency, massive transfusions, coumadin reversal	200–250 mL	Thawed: 6–24 hours Frozen: 1 year
Cryoprecipitate	Factors VIII, XIII, fibrinogen, fibronectin, von Willebrand's Factor	Hemophilia A, von Willebrand's disease, fibrinogen deficiency	25 mL/unit	Thawed: 4–6 hours Frozen: 1 year
Factor VIII concentrate	Factor VIII, fibrinogen, von-Willebrand's Factor	Hemophilia A (classic hemophilia)	Lyophilized (requires reconstitution)	2–8°C: 1 year Room temp: 3 months
Factor IX concentrates (Konyne, Proplex)	Factor II, VII IX, X	Hemophilia B (Christmas disease)	Lyophilized (requires reconstitution)	2–8°C: 1 year Room temp: 1 month
Albumin 25% (5%)	Albumin	Volume expansion, maintenance of intravascular oncotic pressure	250 or 500 mL, (50 mL)	3–5 years
Plasma protein fraction	Albumin, alpha globulin, beta globulin	Volume expansion, maintenance of intravascular oncotic pressure	250 mL	3–5 years

Source: From Ritter DF, Sarsnic MA. Transfusion therapy, part I. *Prog Anesthesiol* 1989; 3:1–14.

### Whole Blood

- Largely replaced by component therapy (more efficient use)
- Exceptions include complex pediatric cardiac surgery & military hospitals in war zones (*J Trauma* 2006;60[6]: S59)

### Platelets

- A single 6-pack of platelets ≈ 300 mL; usually ↑ platelet count by ≈ 30,000
- Pooled & single-donor units have equal hemostatic effectiveness
- Stored at room temp for up to 5 d (↑ risk of bacterial infection after 5 d)

- Contains all plasma coagulation factors (except factor V & VIII → found in FFP)
- No need for ABO compatibility
  - Rh-negative women of childbearing age should receive Rh-negative platelets

Suggested Platelet Transfusion Thresholds	
<10,000	Prophylaxis (based on studies in cancer pts <i>N Engl J Med</i> 337[26]:1870–1875)
<20,000	Any bleeding or pre-procedure; pts with concurrent coagulation disorder/infection
<50,000	Major bleeding or during surgery; prior to CNS or major eye surgery; bleeding with trauma; pretracheal bleeding; after prolonged cardio-pulmonary bypass

Given platelets for ITP with life-threatening bleeding from GI/GU tracts or from CNS; mucous membrane bleeding usually precedes fatal hemorrhage

**Contraindications:** TTP/HUS & HIT

**Complications**

- Survival duration (normal half-life =  $3 \pm 0.2$  d): sepsis, splenomegaly, ITP, TTP, HUS, DIC, AIDS, or drugs (heparin, vancomycin, quinidine, penicillins, cephalosporins, sulfa)

**Platelet refractoriness**

- **Definition**—platelets  $\uparrow < 7000/\mu\text{L}$  when measured 15–60 min after 2 separate platelet transfusions
- **Causes**—non-immune (drugs, infection, splenomegaly)  
immune: Anti-HLA or antiplatelet Ab's
- **Treatment**—request ABO-matched platelets, check posttransfusion increment check HLA percent reactive antibodies (**PRA**), perform HLA typing, & consult transfusion medicine; if diffuse mucosal bleeding → consider aminocaproic acid (Amicar)

### Fresh Frozen Plasma (FFP)

- Contains all plasma coagulation factors
- Duration of effect  $< 7$  hr (half-life of factor VII = 7 hr)
- Used to restore clotting factors in setting of
  - Massive transfusion ( $> 1$  blood volume in 24 hr)
  - Liver disease (often at dose of 10–15 mL/kg)
  - Urgent reversal of warfarin-induced anticoagulation (5–8 mL/kg)
- FFP must be ABO-compatible; volume expansion is not an appropriate use of FFP

Indications for FFP Transfusion	
INR $> 2.0$	Prophylactic transfusion prior to invasive procedures or for actively bleeding pts
INR 1.5–2.0	FFP may be of value in actively bleeding pts; uncertain benefit as pre-procedure prophylaxis; unlikely to correct INR value without massive no. of units of FFP ( <i>Transfusion</i> 2006;46:1279)
INR $< 1.5$	FFP not indicated

**Complications**

- Volume overload, transfusion-related acute lung injury (TRALI)

**Contraindications**

- Known anaphylactoid reactions to plasma products (pts with anti-IgA antibodies)

### Cryoprecipitate

- Contains vWF, factor VIII, fibrinogen, factor XIII; usual dose = 8–10 units
- ABO compatibility preferred, not required

**Indications**

- Hypofibrinogenemia, von Willebrand dz (unresponsive to DDAVP) & hemophilia

**Contraindications**

- Pts with hypofibrinogenemia ( $< 100$  mg/dL) from generalized coagulopathies may have other defects in addition to fibrinogen, should receive FFP instead

### Albumin

- 5% = isooncotic; 25% = hyperoncotic; 12.5 g total albumin (in 5% & 25% preps)

**Indications**

- Support of shock, major burn pts
- Cirrhosis, spontaneous bacterial peritonitis (SBP) or after large-volume paracentesis
- Large study of ICU pts ( $n = 6997$ ) showed no advantage to albumin compared with saline for initial volume support (*N Engl J Med* 350;2247–2256)



### Factor VII (NovoSeven, eptacog alfa)

- Used in uncontrollable bleeding in surgical and hemophilia patients
- Initiates coagulation in only those sites where tissue factor (TF) is also present (TF is exposed to the blood in vessel injury)
- Increased risk of DVT, PE, MI
- May improve outcomes in acute intracerebral hemorrhage

### DDAVP

- Release of endothelial stores of factor VIII and increases VIII:vWF
- Useful in von Willebrand's dz (type 1 & 2a) and some cases of hemophilia A

### Complications of Massive Transfusion

- $\uparrow K^+$ ,  $\downarrow Ca^{2+}$  (citrate preservative binds  $Ca^{2+}$ )
- Dilutional thrombocytopenia (may need FFP & platelets)
- Metabolic alkalosis (due to citrate forming  $HCO_3^-$ )
- Hypothermia

$$\text{Estimated allowable blood loss} = EBV \times (H_{\text{initial}} - H_{\text{low}}) / H_{\text{initial}}$$

$H_{\text{initial}}$  = initial Hct

$H_{\text{low}}$  = final lowest acceptable Hct

$$\text{Estimated blood volume (EBV)} = \text{weight (kg)} \times \text{average blood volume}$$

### Average Blood Volume

Premature neonates	95 mL/kg
Full-term neonates	85 mL/kg
Infants	80 mL/kg
Adult men	75 mL/kg
Adult women	65 mL/kg

### Estimating Blood Loss in

#### Surgical Sponges

Sponge Type	Fluid Capacity
4 × 4 sponge	10 mL
Ray-tech sponge	10–20 mL
Lap sponge	100 mL

### Infectious Complications

Nucleic acid technology (NAT) for blood product screening

→ significantly  $\downarrow$  incidence of transfusion-related hepatitis & HIV

### Transfusion Complications: Estimated Risk

Noninfectious	Risk (per unit)	Infectious	Risk (per unit)
Febrile	1:100	CMV	Common
Allergic	1:100	Hepatitis B	1:220,000
Delayed hemolytic	1:1,000	Hepatitis C	1:1,600,000
Acute hemolytic	<1:100,000	HIV	1:1,800,000
Fatal hemolytic	<1:250,000	Bacteria (PRBCs)	1:500,000
TRALI	1:5,000	Bacteria (platelets)	1:12,000

(N Engl J Med 1999;340:438; JAMA 2003;289–959)

- **Hepatitis B:**  $\approx$  35% of infected individuals demonstrate acute dz  
 $\approx$  1–10% become chronically infected
- **Hepatitis C:** Up to 85% of infected pts suffer chronic infection  
 $\rightarrow$  20% develop cirrhosis, 1–5% hepatocellular carcinoma
- **Bacterial infx:** Most common causes of transfusion related deaths  
(1 in 2000 platelet recipients gets an infection  $\rightarrow$  10–25% of these develop severe sepsis; mortality for transfusion-assoc sepsis  $\approx$  60%)
- **Other infx:** viral (cytomegalovirus, West Nile virus), protozoan (malaria, toxoplasmosis), bacterial (Lyme) and prion (Creutz-Jakob): dz

### Coagulopathic Complications

Typically seen in setting of massive blood transfusions

- **Dilutional thrombocytopenia**  $\rightarrow$  treat with platelets if microvascular bleeding occurs
- **Disseminated intravascular coagulation (DIC)** (see below, page 9-20)
- **Low factor V & VIII levels**  $\rightarrow$  decrease to 15% & 30% of normal values, respectively, in stored blood; contribute to inadequate hemostasis after massive transfusion; give FFP in setting of bleeding with prolonged APTT & normal platelet count

### Transfusion Reactions

- **Acute hemolytic transfusion reaction**
  - Due to ABO or major antigen incompatibility
  - Usually due to clerical errors, incidence of 1:250,000 transfusions
  - Symptoms: Chills, fever, chest, flank pain  $\rightarrow$  often masked by anesthesia; may only see hypotension, unexplained bleeding, & hemoglobinuria

#### Treatment of a Suspected Hemolytic Transfusion Reaction

1. Stop transfusion
2. Treat hypotension with fluids and/or vasopressors
3. Maintain urine output (75–100 mL/hr) with fluids, mannitol, & furosemide
4. Alkalinize urine (give 40–70 mEq bicarb per 70-kg body wt) to prevent precipitation
5. Send unused blood & fresh pt sample to blood bank (for recrossmatch)
6. Send blood sample to lab for free Hb, haptoglobin, Coombs test, DIC screen
7. Consider corticosteroids

- **Nonhemolytic transfusion reactions**
  - Etiology: Usually febrile or allergic in nature; caused by antibodies against donor WBCs or plasma proteins
  - Signs: Fever, hives, tachycardia & mild hypotension
  - Treatment: Rule out hemolytic transfusion rxn & bacterial contamination symptomatic treatment/support
  - Prevention: Leukocyte-reduced PRBCs & washed PRBCs may  $\downarrow$  incidence

Diagnosis and Management of Transfusion Reactions		Symptoms/Signs	Cause	Treatment
<b>Type</b>	<b>Notes</b>			
<b>Febrile nonhemolytic transfusion reaction</b>	<ul style="list-style-type: none"> <li>• Most common (1:200–500)</li> <li>• 15% will have a 2nd reaction</li> </ul>	Fever (1°C above pretransfusion), chills, ± mild dyspnea within 1–6 hr after transfusion	RBCs: Class I HLA Ab against donor leukocytes Plts: Storage-dependent cytokines	<ul style="list-style-type: none"> <li>• Stop transfusion &amp; rule out hemolytic reaction, severe infection</li> <li>• Give antipyretics, IM meperidine in pts with chills and rigors</li> <li>• Use leukoreduced blood products in the future</li> </ul>
<b>Simple allergic reaction</b>	<ul style="list-style-type: none"> <li>• 1:333–500</li> </ul>	Hives ± itching	Transfused allergens in plasma cause mast cell degranulation	<ul style="list-style-type: none"> <li>• Pause transfusion</li> <li>• If only hives ± itching, may continue same unit</li> <li>• Give antihistamines if pt symptomatic</li> <li>• Unlikely to recur: in future, consider premedication with antihistamines and use washed cells if repeated reactions</li> </ul>
<b>Transfusion-related acute lung injury (TRALI)</b>	<ul style="list-style-type: none"> <li>• 1:5,000</li> <li>• 1–6 hr after onset of transfusion</li> <li>• CVP is normal</li> <li>• Looks like ARDS</li> <li>• Death ~10%</li> </ul>	Acute respiratory distress, hypoxemia, hypotension, fever, pulmonary edema	Donor antibodies agglutinate host neutrophils to cause lung injury	<ul style="list-style-type: none"> <li>• Stop transfusion</li> <li>• ABCs, O<sub>2</sub>, mechanical ventilation, diuresis, ? steroids</li> <li>• If recovers, not at increased risk for recurrent episodes following transfusions from other donors</li> </ul>
<b>Acute hemolytic transfusion reaction</b>	<ul style="list-style-type: none"> <li>• 1:15,000, fatal in 1:250,000 to 1:600,000</li> <li>• Results in DIC, shock, acute renal failure</li> </ul>	Fever, chills, N/V, pink plasma, flank pain, pink, red or brown urine, or any combination of the above symptoms	Destruction of donor RBC by preformed recipient Abs. Usually secondary to ABO incompatibility	<ul style="list-style-type: none"> <li>• Stop transfusion, leave IV attached for treatment</li> <li>• Start NS at 100–200 mL/hr</li> <li>• Lasix 40–80 mg IV initially, then titrate to UO &gt; 100 mL/hr for 24 hr</li> <li>• From other arm obtain direct antiglobulin test (will be positive), CBC, lytes, new blood bank sample</li> <li>• May require pressors; watch for hyperkalemia (</li> </ul>

<b>Anaphylactic transfusion reaction</b>	<ul style="list-style-type: none"> <li>• 1:20,000 to 1:50,000</li> <li>• Rapid onset</li> </ul>	Rapid anaphylaxis including hypotension angioedema, respiratory distress	Due to presence of specific anti-IgA Abs in small subset of IgA deficient pts	<ul style="list-style-type: none"> <li>• Immediately stop transfusion</li> <li>• ABCs, vasopressors may be necessary</li> <li>• Epinephrine 0.3 mL of 1:1000 solution SQ</li> <li>• Methylprednisolone</li> <li>• Prevent by using IgA-deficient blood ultra-washed or deglycerolized RBCs</li> </ul>
<b>Sepsis</b>	<ul style="list-style-type: none"> <li>• 1:500,000 for PRBCs</li> <li>• 1:12,000 for platelets</li> </ul>	High fevers, rigors, nausea without diarrhea and hypotension	Due to bacterial contamination of product (longer storage increases risk)	<ul style="list-style-type: none"> <li>• Stop transfusion</li> <li>• Send bag, tubing, remaining product to Blood Transfusion Service</li> <li>• Draw blood cultures</li> <li>• Start broad-spectrum antibiotics</li> </ul>
<b>Delayed hemolytic transfusion reaction</b>	<ul style="list-style-type: none"> <li>• 1:2000</li> <li>• Seen after multiple transfusions, transplantation, pregnancy</li> <li>• 2–10 d posttransfusion</li> </ul>	Slow Hct drop, slight fever, increase in unconjugated bilirubin, spherocytes	Amnestic Ab response from reexposure to foreign red cell Ag including Rh antigens	<ul style="list-style-type: none"> <li>• None in absence of brisk hemolysis</li> <li>• Inform patient and Blood Transfusion Service so that future transfusions avoid implicated antigens</li> </ul>
<b>Post transfusion purpura</b>	<ul style="list-style-type: none"> <li>• Uncommon</li> <li>• Mostly in multiparous women</li> <li>• 5–10 days after transfusion of platelet-containing products</li> </ul>	Severe thrombocytopenia lasting days to weeks	Amnestic antibody response from reexposure to PIA-1 antigen	<ul style="list-style-type: none"> <li>• Preferred therapy is IVIG in high doses 1.0 g/kg/d × 2 d</li> <li>• Only washed cells or PIA-1-negative cells in future in consultation with Blood Transfusion Service</li> </ul>
<b>Transfusion-associated graft-versus-host disease (GVHD)</b>	<ul style="list-style-type: none"> <li>• Rare and almost always fatal</li> <li>• Occurs in patients with immunodeficiency or in cases of homozygous host and heterozygous donor</li> <li>• Develops 4d–1mo after transfusion</li> <li>• Not induced by FFP, cryo, or deglycerolized red cells</li> </ul>	Fever, rash (maculopapular), RUQ pain, >LFTs, diarrhea anorexia pancytopenia Dx: 1. Biopsy 2. HLA typing of circulating lymphocytes	Allogeneic attack of host tissue by activated donor lymphocytes Difference from post-BMT GVHD is high incidence of pancytopenia Most die from infection	<ul style="list-style-type: none"> <li>• No real therapy</li> <li>• Majority of cases (&gt;90%) are fatal</li> <li>• Anecdotal success with several agents (<i>Br J Haematol</i> 117[2]:275)</li> <li>• Prevent by using irradiated products</li> </ul>

## Transfusion-Related Acute Lung Injury (TRALI)

- Noncardiogenic pulmonary edema occurring within 4 hr of blood product (most commonly FFP administration)
- Mechanism—rxn between donor anti-HLA or antileukocyte Ab's & recipient leukocytes
- Treatment—stop transfusion, supportive care
- Outcomes—mortality ≈ 5–10%, most pts recover within 96 hr

## Metabolic Complications

- Citrate intoxication—uncommon unless blood transfused >150 mL/70 kg/min
  - Hypothermia, liver dz, liver transplantation, & hyperventilation ↑ risk
  - Monitor ionized calcium during rapid transfusions
  - Treat hypocalcemia with Ca gluconate (30 mg/kg) or Ca carbonate (10 mg/kg)
- Hyperkalemia—unlikely at transfusion rates <120 mL/min
  - Rarely of clinical significance

## Disorders of Coagulation

Extrinsic vs intrinsic coagulation pathways

- Accessory (intrinsic) pathway: Factors VIII, IX, XI, XII
- Extrinsic pathway: Factors III, VII
- Common pathway: Factors V, X, thrombin (2), fibrin (1)

Primary Hemostasis	Secondary Hemostasis
<ul style="list-style-type: none"><li>• Constriction of injured vessels</li><li>• Exposure of subendothelial collagen</li><li>• Adhesion &amp; aggregation of blood platelets on damaged surface</li><li>• Formation of 1° hemostatic plug</li></ul>	<ul style="list-style-type: none"><li>• Formation of thrombin catalyzed by surface of activated platelets</li><li>• Formation of thrombin via activation of factor VII by tissue factor</li><li>• Conversion of fibrinogen → fibrin catalyzed by thrombin</li><li>• Formation of fibrin clot &amp; its stabilization</li></ul>

## Coagulation Studies

Thorough history = best tool to detect presence of a coagulation disorder

- *Prothrombin Time (PT)*—measure of extrinsic coag pathway (factors II, V, VII, X)
  - Sensitive to factor VII deficiency
  - International normalized ratio (INR) standardizes PT values to allow interlab comparison
  - Normal PT values ≈ 11–13.2 sec
- *Partial Thromboplastin Time (PTT)*—test of intrinsic coag pathway (factors VIII–XII)
  - Elevated in pts on heparin & pts with other circulating anticoagulants (factor VII antibodies, lupus anticoagulant)
  - Normal PTT values ≈ 25–37 sec

Screening Test Abnormalities in Inherited and Acquired Coagulopathies			
PT	PTT	Inherited	Acquired
↑	↔	Factor VII deficiency	Vit. K deficiency; Liver dz; factor VII inhibitors
↔	↑	Hemophilias, vWD	Factor inhibitors; antiphospholipid Ab
↑	↑	Deficiency: fibrinogen, factor II, factor V	DIC; liver dz; inhibition of fibrinogen, factors II, V or X

Source: Adapted from *Pocket Medicine*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

- *Activated clotting time (ACT)*—modified clotting time of whole blood
  - Accessory (intrinsic) pathway activated by adding kaolin or diatomaceous earth
  - Normal ACT ≈ 110–130 sec.
  - Can be performed in OR (point-of-care test)
- *Bleeding time*—crude assay of platelet function; poorly reproducible, rarely used
- *Fibrinogen*—normal level ≈ 170–410 mg/dL
  - May be depleted in massive hemorrhage or DIC
  - An acute phase reactant; can be elevated following trauma or inflammation
  - Goal fibrinogen level >100 mg/dL for pts with severe bleeding/massive transfusion
- *Fibrin degradation products (FDP)*—made by action of plasmin on fibrinogen
  - ↑ In DIC, 1° fibrinolysis, & severe liver dz (due to impaired clearance)
  - Influence clotting by interfering with fibrin monomer polymerization & by impairing platelet function
- *D-dimer*—specific fragment produced when plasmin digests cross-linked fibrin
  - ↑ In DIC, pulmonary embolism & in the immediate postop period

- Thromboelastogram (TEG)
- Viscoelastic test that characterizes formation & strength of blood clot over entire period of clotting & fibrinolysis
- Assesses coagulation system, platelet function, & fibrinolysis

#### Effects of Some Commonly Used Agents on Coagulation Parameters

Agent	Bleeding Time	Prothrombin Time	Activated Thromboplastin Time	Activated Clotting Time	Time to Peak Effect	Time to Normal Hemostasis Post-therapy	Comments
Aspirin	↑↑↑	-	-	-	Hours	1 week	Platelet function not accurately predicted by bleeding time
Other NSAIDs	↑↑↑	-	-	-	Hours	3-5 days	Platelet function not accurately predicted by bleeding time
Heparin, regular intravenous	↑	↑	↑↑↑	↑↑↑	Minutes	4-6 hours	Monitor activated clotting time or activated thromboplastin time
Heparin, regular subcutaneous	↑	↑	↑↑	↑↑	1 hour	4-6 hours	Activated thromboplastin time may remain normal: Monitor anti-Xa activity
Heparin, low molecular weight Subcutaneous	-	-	-/↑	-/↑	12 hours	1-2 days	Activated thromboplastin time may remain normal: Monitor anti-Xa activity ...
Thrombolytic agents	↑↑↑	↑	↑	-	Minutes	1-2 days	Frequently administered along with intravenous heparin

↑: Clinically insignificant increase; ↑↑: Possibly clinically significant increase; ↑↑↑: Clinically significant increase

## Characteristics of Anticoagulants

	Warfarin	Unfractionated Heparin	Low-Molecular-Weight Heparin	Factor Xa Inhibitors	Direct Thrombin Inhibitors
No. Cascade targets	Many	Many	Few	Few	Few
Activity specificity	Nonspecific	Nonspecific	Specific	Specific	Specific
No. daily doses	1	2-3	1-2	1	1-2
Route	PO	IV, SC	SC	SC, PO	IV, PO, SC
Monitoring	INR	aPTT, plt count	Plt count, anti-Xa	None	aPTT, liver fx
Variability in response	High	High	Low	None	Low
Risk of HIT	None	2-5%	1-2%	None	None
Other notes	Inhibits factors II, VII, IX, X, protein C	Binds antithrombin III			

Source: Adapted from Nutescu EA, et al. *Cleve Clin J Med* 2005;72(Suppl 1):S2-S6.

## Properties and Antidotes for Anticoagulants & Fibrinolytics

Anticoag.	t <sub>1/2</sub>	Labs	Rx for overdose w/ serious bleeding*
UFH	60-90' RES	↑ PTT	Protamine IV 1 mg/100 U unfractionated heparin (UFH) (max 50 mg). For infusions, dose to reverse = 2× rate of UFH given per hr.
Bivalirudin	25', K	↑ PTT	Dialysis
Lepirudin	80', K	↑ PTT	Dialysis
Argatroban	45', L	↑ PTT	? Dialysis
Enoxaparin	8°, K	(anti-Xa)	? Protamine (reversal incomplete)
Fondaparinux	24°, K	(anti-Xa)	? Dialysis
Warfarin	36°, L	↑ PT	<i>No bleeding:</i> INR > 5: vit. K 1-5 mg PO (superior to SC, IV at 24°; 2.5 mg for INR 6-10; 5 mg for INR > 10 (Archives 2003;163:2469) <i>Bleeding:</i> vit. K 10 mg IV + FFP 2-4 units IV q 6-8°
Fibrinolytic	20-90' LK	↓ fbgm ↑ FDP	Cryoprecipitate, FFP, ± aminocaproic acid

\*Initial step should be immediate d/c of anticoag. Decision to dialyze should take into account time for anticoag. to be metabolized (noting renal/liver insufficiency) w/o dialysis vs. potential sequelae of bleeding while waiting. K, kidney; L, liver; RES, reticuloendothelial system.

## Platelet Inhibitors

### Aspirin

- Irreversibly inactivates cyclooxygenase (COX) enzyme
- Get suppression of prostaglandin & thromboxane production

### Ibuprofen

- NSAID that reversibly inhibits cyclooxygenase

### Clopidogrel

- Irreversible blockade of adenosine diphosphate (ADP) receptor on platelet cell membranes

### Abciximab

- Platelet aggregation inhibitor (inhibits glycoprotein IIb/IIIa)

### Dipyridamole

- Inhibits platelet adhesion

## Bleeding Disorders (Coagulopathies)

- *Classic hemophilia (hemophilia A, factor VIII deficiency)*

- X-linked recessive trait, 1:5,000 live male births
- Prolonged PTT but normal PT & normal platelet function
- Bleeding episodes related to level of factor VIII activity
  - < 1% spontaneous bleeds
  - 1–5% bleeding after minor trauma
  - >5% infrequent bleeding
- Treatment: Factor VIII replacement (cryoprecipitate, lyophilized or recombinant factor VIII)
- Activity levels of 20–40% recommended prior to surgery
- Half-life of factor VIII ≈ 8–12 hr
- 20% of pts will eventually develop factor VIII antibodies
  - Treated with high-dose factor VIII, activated factor IX, or plasmapheresis
- High incidence of hepatitis & HIV (given exposure to blood products)
- *Christmas disease (hemophilia B, factor IX deficiency)*
- Sex linked, occurring almost exclusively in males, incidence 1:100,000
- Presentation similar to hemophilia A
- Treatment: Factor IX concentrates, rFVIIIa or FFP
- For surgical hemostasis → factor IX activity levels of 50–80% required
- Half-life of factor IX ≈ 24 hr
- *von Willebrand disease (vWD)*
- Abnormalities of von Willebrand factor (vWF)
  - Glycoprotein produced by megakaryocytes & endothelial cells
  - vWF stabilizes factor VIII & forms cross-links between platelets & endothelial cells
- vWD is classified as type 1 (classic), type 2 (variant) & type 3 (severe)
  - Type 1 vWD = autosomal dominant inherited
  - Most common inherited bleeding disorder, prevalence = 1%
  - Pts present with variable bleeding tendency; epistaxis often presenting feature
  - Most common laboratory finding = prolonged bleeding time
  - Treatment: Desmopressin (0.2 mcg/kg in 50 mL saline over 30 min) or cryoprecipitate; desmopressin has half-life ≈ 8–12 hr

### Heparin-Induced Thrombocytopenia (HIT)

Overview of Heparin-Induced Thrombocytopenia (HIT)		
Feature	Type I	Type II
Mechanism	Direct effect of heparin	Immune (Ab)-mediated
Incidence	20%	1–3%
Onset	After 1–4 d of heparin	After 4–10 d; can occur early (<24 hr) if prior exposure in last 100 d; can occur after heparin stopped
Platelet nadir	>100,000/ $\mu$ L	30–70,000/ $\mu$ L or 50% decrease from baseline
Sequelae	None	Thrombotic events (HITT) in 30–50% Rare hemorrhagic problems
Management	Continue heparin; observe	<b>Stop heparin</b> ; start alternative anticoagulation therapy

Source: Adapted from *Pocket Medicine*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

- Type II HIT = immune-mediated thrombocytopenia triggered by IgG antibodies against heparin platelet factor 4 (PF4) complexes (PF4 antibodies)
  - Bound antibody → stimulates platelet activation → thrombocytopenia, platelet aggregation, & thrombosis
  - Many more pts develop antibody than the syndrome
  - 50% of cardiac surgery pts exposed to heparin developed PF 4 Ab's
    - Only 1% go on to clinical HIT
    - Risk reduced with the use of low-molecular-weight heparin
    - Risk eliminated with use of fondaparinux or direct thrombin inhibitors (lepirudin, argatroban)
- Type II HIT treatment: Stop all heparin exposure, including heparin flushes
  - Start alternative anticoagulation
    - Argatroban (1–2 mcg/kg/min)
    - Lepirudin (0.4 mg/kg bolus, then infuse at 0.15 mg/kg)
  - In absence of alternate anticoagulation, 40% of pts will develop thrombotic complications, with resultant amputation in 10–20% & death in 30–50%



- **Oral anticoagulation:** Do not start until platelet count is  $>100,000/\mu\text{L}$
- Warfarin should overlap direct thrombin inhibitors (as warfarin reduces protein C levels before prothrombin, causing transient hypercoagulable state)
- Optimal duration of therapy unknown, consider  $>6$  weeks

### Disseminated Intravascular Coagulation (DIC)

Consequence of abnormal, diffuse activation of the coagulation & fibrinolytic systems

Causes of DIC	
Acute	Chronic
<ul style="list-style-type: none"> <li>• Sepsis, ARDS</li> <li>• Shock</li> <li>• Trauma</li> <li>• Obstetric (e.g., amniotic fluid embolism)</li> <li>• Hemolytic transfusion reaction</li> <li>• Extensive burns</li> </ul>	<ul style="list-style-type: none"> <li>• Malignancy</li> <li>• Liver disease</li> <li>• Retained dead fetus</li> <li>• Intra-aortic balloon pump</li> <li>• Peritoneovenous shunt</li> <li>• Aortic dissection/aneurysm</li> </ul>

- **Pathogenesis**
  - Excessive deposition of fibrin throughout microvasculature & consumption of coagulation factors
  - Widespread platelet activation & fibrinolysis
- **Clinical features**
  - Petechiae, ecchymoses, oozing from surgical sites
  - Diffuse thrombosis  $\rightarrow$  life-threatening ischemia of vital organs
- **Laboratory features**
  - Elevated D-dimers, PT, & PTT levels
  - Serial measurements reveal a falling fibrinogen level & platelet count
  - FDPs elevated (but nonspecific)
  - Peripheral blood smears  $\rightarrow$  schistocytes (from microvascular RBC trauma)
- **Treatment**
  - Recognition & treatment of underlying cause of DIC
  - FFP or cryoprecipitate to keep fibrinogen  $>50$  mg/dL & replace clotting factors
  - Platelets should be kept  $>25,000$ – $50,000/\mu\text{L}$
  - Consider heparin for pts with predominantly thrombotic DIC
  - Inhibitors of fibrinolysis (aminocaproic acid, aprotinin) **not** recommended

### Vitamin K Deficiency:

- Vitamin K is needed by the liver to make prothrombin (factor II); factors VII, IX, X; protein C; protein S. Deficiency can lead to coagulopathy and  $\uparrow$  PT/INR.
- Treatment: Vitamin K 2.5–10 mg SC/IM/PO or 1–10 mg IV at  $\leq 1$  mg/min

### Sickle Cell Disease (also see Chapter 29, "Chronic Pain")

- Abnl hemoglobin (HbS) results in sickling  $\rightarrow$  chronic hemolysis, vasoocclusive crises
- End-organ effects: Renal & pulm. infarction, liver cirrhosis, CVAs, bone ischemia

### Perioperative Management of Sickle Cell Disease

- Ensure adequate hydration
- Avoid factors that cause sickling (hypoxia, hypothermia, dehydration, acidosis, polycythemia, infection)
- Consider preop simple transfusion to HCT  $\approx 30\%$
- Consider exchange transfusion to keep HbS  $<40\%$  (can also  $\downarrow$  blood viscosity)

# COMMON INTRAOPERATIVE PROBLEMS

RANDY FAYNE

**HYPOXEMIA:** PaO<sub>2</sub> <60 mm Hg or SpO<sub>2</sub> <90%

## Differential Diagnosis

- Ventilation/perfusion ( $\dot{V}/\dot{Q}$ ) mismatch
  - Most common pathophysiologic cause of hypoxia
  - Results from decreases in alveolar ventilation with respect to lung perfusion
  - Examples: Hypoventilation/shunt/diffusion defect (pneumonia, pneumonitis, pulmonary edema), pulmonary/fat emboli
- Improper endotracheal tube placement
  - Endobronchial, esophageal, oropharyngeal
- Oxygen supply inadequacies
  - Equipment failure, high altitude
- Alveolar hypoventilation ( $\downarrow$  PaO<sub>2</sub> with  $\uparrow$  PaCO<sub>2</sub>)
  - COPD, inadequate ventilator settings, asthma, bronchitis, bronchospasm
  - Drug overdose (benzodiazepines, narcotics, muscle relaxants)
  - Neuromuscular abnormality (myasthenia gravis, Guillain-Barré, polio)
- Anatomic right  $\rightarrow$  left cardiac shunt
- Intrapulmonary shunting
  - $\downarrow$  Ventilation in perfused lung regions
    - $\rightarrow$  Shunting of venous blood into arterial system without being oxygenated
  - O<sub>2</sub> therapy unable to improve PaO<sub>2</sub>
- Diffusion abnormality
  - Impaired transfer of O<sub>2</sub> from alveoli across the capillary membrane
  - Sarcoidosis, interstitial lung disease
- $\downarrow$  O<sub>2</sub> hemoglobin carrying capacity (leftward shift of hemoglobin dissociation curve)
  - Hypothermia/alkalosis/hypocarbia/CO poisoning

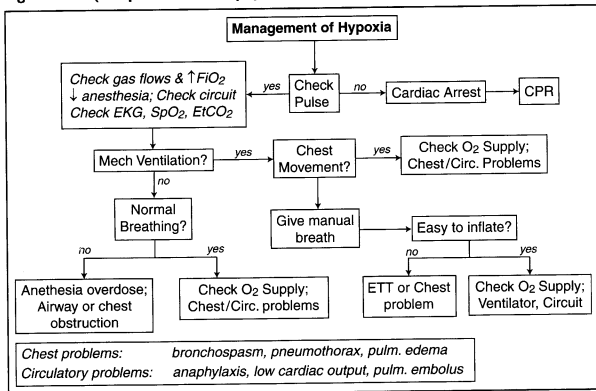
## Investigations

- Check pulse oximeter waveform & probe placement
- Auscultate lungs
- Inspect endotracheal tube & circuit
- Bag ventilate to observe chest excursion
- Draw arterial blood gas

## Treatment Options

- Place patient on 100% FiO<sub>2</sub>
- Ensure ventilator settings are appropriate
- Consider bronchoscopy
- Consider bag ventilation
- Treat underlying problem

**Figure 10-1 (Adapted from Murphy, Fale. Pocket Reference to Anaesthesia, 2nd ed.)**



## BRONCHOSPASM

### Causes

- Preexisting reactive airway disease (asthma)
- Manipulation of upper airway (oral endoscopy)
- ETT with inadequate anesthesia
- ETT causing carinal or bronchial stimulation (endobronchial intubation)
- Excessive histamine release (morphine, atracurium) or  $\beta$ -blockade
- Anaphylaxis
- Pulmonary edema

### Investigations

- Examine ETT for patency (secretions, kinks) & proper position
- Examine for wheezing, air movement
- Capnograph  $\rightarrow$  shows expiratory upsloping
- High peak airway pressures, hypoxia & hypercobia
- **Rule out:** Pneumothorax, PE, & pulmonary edema

### Management

- $\uparrow$  FiO<sub>2</sub>
- $\uparrow$  Anesthetic depth (inhalational agents are bronchodilators)
- $\uparrow$  Expiratory time,  $\downarrow$  RR  $\rightarrow$  helps  $\downarrow$  gas trapping
- Give nebulized albuterol via ETT (not effective in severe bronchospasm/lost airway)
- Epinephrine IV/SC (esp for anaphylaxis)  $\rightarrow$  titrate to effect
- Aminophylline (2nd line treatment—6 mg/kg bolus, then 0.5 mg/kg/hr)
- Hydrocortisone (long term)

**HYPOTENSION:** MAP  $<$ 60 mm Hg or 20–25% reduction from baseline

### Differential Diagnosis

- Decreased preload
  - $\downarrow$  Blood volume (hemorrhage, inadequate fluid replacement, third spacing)
  - $\downarrow$  Venous return (change in pt position, i.e., Trendelenburg)
  - Pericardial tamponade, pneumothorax, surgical compression of venous structures, pneumoperitoneum from laparoscopy, excessive PEEP
- Decreased afterload
  - Sepsis, vasodilating drugs (anesthetics), anaphylactic rxn, neurologic injury
- Decreased contractility
  - MI, arrhythmias, CHF, anesthetic effect, electrolyte imbalances

### Investigations

- Examine BP cuff for fit
- Examine preoperative BP trends
- Calculate fluid balance (including blood loss)
- Ensure IV site intact & not infiltrated
- Examine arterial line waveform for respiratory variation

### Treatment Options

- Administer fluid bolus
- $\downarrow$  Anesthetic agents
- Administer vasopressors (phenylephrine 40–100 mcg/ephedrine 5–10 mg)
- Administer other vasoactives/inotropes (norepinephrine, dobutamine, milrinone, dopamine)
- Consider invasive monitoring (CVP, arterial line, PA catheter, Echo)

**HYPERTENSION:** BP  $>$ 140/90 mm Hg or MAP  $>$ 20–25% baseline value

### Differential Diagnosis

- Primary HTN
  - HTN with no known cause (70–95% of hypertension)
- Secondary HTN
  - Pain/surgical stimuli (inadequate anesthesia, tourniquet pain), ETT stimulation, bladder distention
  - Hypercarbia, hypoxia, hypervolemia, hyperthermia
  - Intracranial pathology ( $\uparrow$  ICP, herniation, hemorrhage)
  - Endocrine problems (pheochromocytoma, Cushing syndrome, hyperthyroidism, hyperparathyroidism)
  - EtOH withdrawal
  - Malignant hyperthermia
  - Inadvertent vasoactive drug administration
  - Antihypertensive medication withdrawal

- Consider timing of HTN with case events:
  - HTN prior to induction
  - Withdrawal from antihypertensive medications, essential hypertension, pain
  - HTN postinduction
  - Laryngoscopy effect, improper ETT placement, hypercarbia from esophageal intubation, misplacement of gastrostomy tube into trachea, pain, hypoxia
  - HTN during the case
  - Inadequate pain control, hypercarbia, pneumoperitoneum, fluid overload, drugs (vasopressors), bladder distention

### Investigations/Treatment Options

- Examine BP cuff size & placement, arterial line waveform
- Review anesthetic/surgical events of the case
- Check for hypoxia/hypercarbia
- Check vaporizer agent level
- Administer antihypertensives (beta blockers/vasodilators)

**HYPERCARBIA:** ↑ CO<sub>2</sub> levels (as measured by blood gas or/end-tidal gas analysis) (normal values 38–42 mm Hg)

### Differential Diagnosis

- ↑ CO<sub>2</sub> production
  - Malignant hyperthermia
  - Sepsis
  - Fever/shivering
  - Thyrotoxicosis
- ↓ CO<sub>2</sub> elimination
  - Reduced minute ventilation
    - Altered lung mechanics (atelectasis, pneumoperitoneum with CO<sub>2</sub>, surgical retractors preventing lung expansion)
    - Airway obstruction (secretions, mucous plugging)
    - Inadequate ventilator settings (↓ volumes, ↓ fresh gas flows)
    - Oversedation
  - Increased dead space
    - ETT malfunction (kinks, endobronchial intubation)
    - Exhausted CO<sub>2</sub> absorber
  - Drug effects (muscle relaxants/narcotics/benzodiazepines)
- Consider timing of ↑ CO<sub>2</sub> with case events:
  - ↑ CO<sub>2</sub> at the start of a case
    - Improper ETT placement, inadequate ventilator settings, oversedation of spontaneously breathing pt
  - ↑ CO<sub>2</sub> postinduction/during case
    - MH, neuroleptic malignant syndrome (NMS), improper vent settings, thyrotoxicosis, release of tourniquet, exhausted CO<sub>2</sub> absorber
  - ↑ CO<sub>2</sub> during emergence
    - Inadequate reversal of muscle relaxants, residual narcotic/anesthetic effects, neurologic causes, electrolyte disturbances, hypoglycemia

### Investigations/Treatment Options

- Examine pulse oximeter
- Ensure appropriate ventilator settings
- Examine CO<sub>2</sub> absorber for exhaustion
- Consider ABG
- If spontaneously breathing: Assist breathing, lighten sedation
- If mechanically ventilated: Increase minute ventilation

**HYPOCARBIA:** ↓ CO<sub>2</sub> levels (as measured by blood gas or/end-tidal gas analysis)

### Differential Diagnosis

- Hyperventilation
- ↓ Metabolic rate (hypothermia, hypothyroidism)
- Pulmonary embolism
- Air embolus
- Cardiac arrest (hypoperfusion)
- ETT dislodgement/circuit disconnect

### Investigations/Treatment Options

- Check breathing circuit
- Check blood pressure, heart rate, SpO<sub>2</sub>
- Check/modify ventilator settings
- Treat underlying cause

### ↑ PEAK AIRWAY PRESSURES

#### Differential Diagnosis

- Circuit problem (stuck valve, PEEP valve on wrong, kinked hose)
- ETT problem (kinked/bitten, plugged with mucus, bad positioning)
- Drug induced (opiate chest wall rigidity, inadequate paralysis/anesthesia, MH)
- ↓ Pulmonary compliance (asthma, insufflation, pneumothorax, aspiration)

#### Treatment

- Check tubes, hand ventilate, 100% FiO<sub>2</sub>
- Listen to lungs, suction ETT, add bite block, consider paralysis

**OLIGURIA:** Urine production <0.5 mL/kg/hr (also see Chapter 22, Renal System)

#### Differential Diagnosis

- Prerenal: Intravascular fluid depletion
- Renal origin: Lack of renal perfusion (hypotension, cross clamping, renal artery stenosis), intrinsic renal damage (nephrotoxic drugs/vasculitis)
- Postrenal: Ureteral obstruction/disruption, obstruction of Foley catheter

#### Investigations/Treatment Options

- Examine vital sign monitors to establish hemodynamic stability
- Examine/irrigate Foley catheter for obstruction/improper placement
- Review possible nephrotoxic drugs & withdraw
- Examine fluid administration/blood loss/surgical manipulation
- Consider fluid challenge to treat prerenal oliguria
- Treat underlying cause

### MYOCARDIAL ISCHEMIA/INFARCTION

Damage to heart muscle from imbalance between myocardial O<sub>2</sub> supply & demand

#### Etiology

- Atherosclerosis (accounts for 90% of MIs)
- Coronary aneurysms
- Coronary artery spasm
- O<sub>2</sub> demand outweighs supply (e.g., aortic stenosis)
- Blood viscosity changes (polycythemia)
- Embolic sources (endocarditic vegetations)

#### Investigations

- Lead II—best for arrhythmia detection (RCA association & nodal system)
- Lead V5—best for ischemia detection (LAD & anterior/lateral areas of heart)
- Both lead II & V5 will detect >90% of ischemic events
- ST-segment depression  $\geq 0.1$  mV  
(usually subendocardial pattern → due to partially obstructed coronary)
- ST-segment elevation  $\geq 0.2$  mV  
(usually transmural pattern → due to thrombosed coronary)
- T-wave inversions & Q waves
- Dysrhythmias
- Hypotension
- TEE (most sensitive method for determining early ischemia)
- CK, CK-MB, troponins, cardiac consult (for possible coronary intervention)

#### Treatment Options

Goal: Maintain acceptable balance of myocardial O<sub>2</sub> supply & demand

- (note: If ↑ afterload, preload, contractility, & heart rate → ↑ myocardial O<sub>2</sub> demand)
- Maintain BP within 20% of preoperative levels
- Confirm correct placement of ECG leads, consider 5- or 12-lead ECG
- Notify surgeon of ischemia & coordinate completion of surgical procedure
- Place patient on 100% FiO<sub>2</sub> & ensure adequate ventilation
- Consider reducing anesthetic agents
- Consider  $\beta$ -blocker administration if tachycardic
- Evaluate BP stability & consider invasive monitoring (arterial line/CVP/PA)
- If hypotensive with ischemic ECG changes  
  ↑ BP with pressors to ↑ myocardial perfusion pressure
- Consider fluid therapy & inotropic agents to support myocardial contractility

- Consider anticoagulation (aspirin, heparin)
- Obtain intraoperative cardiology consult to coordinate care

## **MALIGNANT HYPERTHERMIA (SEE APPENDIX D)**

- Definition: Inherited syndrome of skeletal hypermetabolism after exposure to a triggering agent
- Mechanism: ↓ reuptake of ionized  $Ca^{2+}$  by sarcoplasmic reticulum  
→ intracellular  $Ca^{2+}$  accumulation/potential of muscle contraction  
→ ↑ Aerobic/anaerobic metabolism
- Triggering drugs: Succinylcholine, potent volatile agents (sevoflurane, desflurane, isoflurane)
- Nontriggering drugs:  $N_2O$ , narcotics, local anesthetics, nondepolarizing muscle relaxants (cisatracurium, vecuronium, rocuronium), IV induction agents (propofol, ketamine, etomidate, barbiturates)
- Prevention: For MH-susceptible patients, use a "clean" machine (remove vaporizers, change  $CO_2$  absorber, flush with high flow  $O_2$  for 20 min)

### **Differential Diagnosis**

- Neuroleptic malignant syndrome
- Thyrotoxicosis
- MAOI reactions
- Pheochromocytoma
- Inaccurate end-tidal  $CO_2$  monitoring

### **Clinical Presentation/Investigation**

- Can occur anytime during an anesthetic & postoperatively (up to 24 hr)
- Early signs: ↑ end tidal  $CO_2$  levels despite adjustment of ventilation, tachycardia
- Late signs: ↑ temp, rhabdomyolysis & myoglobinuria, metabolic & respiratory acidosis, rigidity, dysrhythmias, HTN, cardiac arrest, masseter spasm, hypoxemia, hyperkalemia
- Lab testing: ABG (check for acidemia, elevated CK, myoglobinuria, elevated  $K^+$ )  
↑ difference in mixed venous  $CO_2$  & arterial  $CO_2$

### **Treatment**

- Call for help & notify surgeon
- Discontinue triggering agents
- Hyperventilate with 100%  $FiO_2$
- Administer dantrolene (2.5 mg/kg IV)  
→ Repeat dantrolene until MH controlled (up to 10 mg/kg IV)  
→ May need to administer for up to 72 hr after episode
- Monitor ABG, vital signs, serum CK
- Treat acidemia with sodium bicarbonate
- Cool patient with IV fluids, cold water lavage in stomach & bladder to temp  $<38^\circ C$
- Treat arrhythmias & promote renal function with fluids/mannitol/furosemide
- Contact Malignant Hyperthermia Hotline as needed: 800-644-9737

**BRADYCARDIA:** Heart rate  $<60$  bpm

### **Differential Diagnosis**

- Altered impulse formation (↑ vagal tone or ↓ SA node automaticity)
- Pharmacologic agents ( $\beta$ -blockers, Ca-channel blockers, cholinergics, narcotics, anticholinesterases,  $\alpha_2$ -agonists)
- Pathologic causes (hypothermia, hypothyroidism, sick sinus syndrome, hypoxemia)
- Myocardial ischemia
- Surgical/anesthesia stimuli (traction on eye, neuraxial anesthesia, laryngoscopy)
- Reflex bradycardia

### **Investigations/Treatment**

- Confirm correct ECG lead placement
- Check vital signs for hemodynamic stability  
→ If stable, consider anticholinergics/ephedrine  
→ If unstable, ↑  $FiO_2$  to 100%, abort anesthetic, administer epinephrine/atropine/CPR, consider placement of pacing device
- Treat underlying cause

**TACHYCARDIA:** Heart rate  $>100$  bpm

Differential depends on presence/absence of hyper/hypotension

### **Differential Diagnosis:**

- Tachycardia + Hypertension
- Pain/light anesthesia/anxiety

- Hypovolemia, hypercapnia, hypoxia, acidosis
- Drugs: Vagolytic drugs (pancuronium, meperidine), ketamine, ephedrine, epinephrine, anticholinergic drugs (atropine/glycopyrrolate), desflurane, isoflurane, beta agonists, vasodilators → reflexive tachycardia (hydralazine), caffeine
- Electrolyte abnormalities: Hypomagnesemia, hypokalemia, hypoglycemia
- Myocardial ischemia
- Endocrine abnormalities: Pheochromocytoma, hyperthyroidism, carcinoid, adrenal crisis

### **Tachycardia + Hypotension**

- Anemia
- Congestive heart failure
- Valvular heart disease
- Pneumothorax
- Immune mediated problems (anaphylaxis, transfusion rxns)
- Myocardial ischemia
- Sepsis
- Pulmonary embolism

### **Treatment Options**

- Ensure adequate oxygenation and ventilation
- Verify ECG leads placement
- Assess BP & prepare to treat depending on scenario
- Consider arterial line placement
- Assess volume status if hypotension exists and treat accordingly
- Assess depth of anesthesia
- Treatment underlying cause

### **DELAYED EMERGENCE**

#### **Differential Diagnosis**

- Residual drug effects (volatile agents, narcotics, muscle relaxants)
- Neurologic complications (seizure with postictal state, CVA, infection, tumor effect)
- Metabolic (electrolyte abnormalities, hypoglycemia, hyperglycemia, adrenal failure)
- Respiratory failure (due to hypercarbia/hypoxia)
- Cardiovascular collapse
- Hypothermia
- Sepsis

#### **Investigations/Treatment Options**

- Ensure muscle relaxants have been reversed
- Ensure hypoxia & hypercarbia do not exist
- Check glucose/electrolytes & replace accordingly
- Consider neurologic imaging
- Supportive care

### **ANAPHYLAXIS: Severe type 1 hypersensitivity allergic reaction (IgE)**

#### **Differential Diagnosis**

- Anaphylactoid—not IgE-mediated, no prior sensitization to antigen required
- Vasovagal reactions generalized urticaria/angioedema, asthma exacerbations
- Myocardial infarction, stroke

#### **Clinical Manifestations**

- Cardiovascular collapse, tachycardia, dysrhythmias
- Bronchospasm, pulmonary & laryngeal edema, hypoxemia
- Rash, skin flushing, peripheral/facial edema

#### **Treatment Options**

- Remove stimulus (if known)
- Oxygen, consider intubation
- Give volume if hypotensive
- Hydrocortisone 250 mg to 1.0 g IV or methylprednisolone 1 to 2 g IV
- Epinephrine 20- to 100-mcg IV bolus followed by infusion if necessary (can give 0.5 to 1.0 mg IV for cardiovascular collapse)
- Diphenhydramine 50 mg IV/ranitidine 50 mg IV
- Norepinephrine 4–8 mcg/min
- Sodium bicarbonate 0.5–1 mEq/kg for persistent acidosis
- Consider intubation (if pt not intubated)
- Evaluate airway for edema prior to extubation

### **Prevention**

- Premedicate with diphenhydramine (H<sub>1</sub> blocker), ranitidine (H<sub>2</sub> blocker), prednisone

## **LATEX ALLERGY**

### **Incidence/Risk Factors**

- Pts with spina bifida & congenital genitourinary abnormalities
- Health-care workers (housekeepers, lab workers, dentists, nurses, physicians)
- Rubber industry workers
- Atopic patients (asthma, rhinitis, eczema)
- Pts having undergone multiple procedures

### **Mechanism**

- IgE-mediated immune response

### **Preop Eval**

- No routine diagnostic testing indicated (RAST & skin tests used occasionally)

### **Equipment/Drug Considerations**

- Routine preop administration of H<sub>1</sub> & H<sub>2</sub> blockers **not** usually recommended

### **Anesthetic Considerations:**

- Avoid products that may contain latex  
(gloves, tourniquets, blood pressure cuffs, face masks, ETT tubes, PA catheters, IV tubing with latex injection ports, rubber stoppers in medication vials)
- Notify entire OR team (nurses, surgeon) & place large sign on OR door

### **Treatment**

- Latex reaction may present as anaphylaxis (>20 min after exposure)
- Symptoms include hypotension, bronchospasm, rash
- Treatment similar to anaphylaxis treatment (see above)  
(remove offending agent, give 100% O<sub>2</sub>, fluid resuscitation, epinephrine, corticosteroids, diphenhydramine, amiophylline)

## **GASTRIC ACID ASPIRATION**

- Can cause chemical pneumonitis

### **Clinical Manifestations**

- Early signs: Coughing, shortness of breath, wheezing, hypoxia, & cyanosis
- Late signs: fever, metabolic acidosis, RML & RLL infiltrate on CXR

### **Management**

- If possible, place patient in head-down position
- Administer 100% O<sub>2</sub>
- Perform rigid bronchoscopy (but no lavage)
- Obtain chest x-ray
- Antibiotics (staph, pseudomonas coverage) & steroids generally not recommended



**PERIPHERAL VENOUS ACCESS****Indications**

- IV administration of drugs & fluids

**Technique**

- Apply tourniquet to extremity (proximal to access site)
  - Alternatively can use BP cuff—inflate between systolic & diastolic pressure
- Choice of vein
  - Straight vein, ideally at a bifurcation
  - Antecubital veins provide better flow than peripheral veins
    - Irritating drugs (e.g., propofol) are less painful on injection
    - Flow may become interrupted if arm is flexed (positional/emergence)
    - Accidental brachial artery puncture possible due to close proximity
  - Cannulation attempts should be from distal to proximal veins
    - Avoid infiltrate from previous attempt at proximal site
- Skin disinfection: Alcohol (enhances visibility of vein due to vasodilating effect)
- Local anesthesia: Skin infiltration with lidocaine, local anesthetic cream/tape for kids
- Vein fixation: Apply tension on skin with your nondominant hand
- Vein puncture: 20–30° angle to penetrate skin, 0–10° angle to advance catheter
- Flash in IV chamber signals needle tip in vessel (will occur before catheter is in vein)
  - Advance entire device 2–3 mm, then advance plastic catheter alone into vessel
- Remove & secure disposal of metal needle
- Fixate plastic catheter with clear adhesive tape on access site, date & time IV
- Assess catheter position by fluid challenge to test for potential infiltration

**Complications**

- Fluid/drug infiltration (signs include swelling, paraesthesia or pain)
  - Immediately disconnect IV line
  - Evaluate for possible tissue necrosis/compartment syndrome
- Intra-arterial injection
  - Immediately disconnect IV line
  - Goal: Enhance vasodilation & prevent vasoconstriction
    - Inject 10 mL saline 0.9%, 10 mL lidocaine 1% with 5000 units heparin
    - Consider stellate ganglion block, use of arterial vasodilators (Ca<sup>2+</sup> channel blockers)

**ARTERIAL ACCESS** (also see Chapter 7, on perioperative monitoring)**Indications**

- Need for continuous BP monitoring
- Surgery on pts with significant comorbidities (ASA III–V)
- Procedures with significant blood loss
- Need for frequent arterial blood gas samples

**Technique: Radial Artery**

- Allen test to assess collateral flow of ulnar artery is unreliable
- Choose nondominant pt hand unless surgical contraindication
- Fixate hand on wrist board
- Skin disinfection: Alcohol or chlorhexidine
- Pulse localization: Palpate (1–2 cm from wrist between bony head of radius & flexor carpi radialis tendon); can also use ultrasound to localize artery
- Local anesthesia: Infiltrate with lidocaine medial & lateral to the artery
- Artery fixation: Apply tension on the skin towards the periphery
- Arterial puncture: 30–45° angle to penetrate skin
- Flash in chamber signals intra-arterial location of needle tip
  - Transfixation technique:
    1. Advance entire needle 2–3 mm further
    2. Remove & secure needle for disposal
    3. Slowly withdraw plastic catheter maintaining a shallow angle to skin until pulsatile flow occurs
    4. Insert guidewire and advance catheter over-the-wire (Seldinger technique)
  - Over-the-needle technique:
    1. Advance catheter by itself once flash obtained
    2. Remove & secure needle for disposal

- Fixate plastic catheter with clear adhesive tape on access site
- Assess arterial flow by connection to transducer
- Note: If cannulation attempt unsuccessful, **do not** attempt ipsilateral ulnar artery cannulation; instead, find alternative extremity (risk of hand necrosis)

#### **Technique: Brachial Artery**

- Palpate brachial artery at ventral side of upper arm between biceps & triceps (close to antecubital fossa)
- Perform same cannulation technique described for radial artery above
- Complications include ischemia of upper extremity, brachial plexus injury

#### **Technique: Axillary Artery**

- Palpate axillary artery in groove between biceps & triceps lateral to pectoralis minor
- Perform same cannulation technique described for radial artery above
- Complications include ischemia of upper extremity, brachial plexus injury

#### **Contraindications**

- Local infection
- Diminished peripheral blood flow
- Insufficient collateral blood flow

#### **Complications**

- Bleeding, blood clot, arterial spasm or laceration, peripheral ischemia or hand necrosis

### **CENTRAL VENOUS ACCESS** (also see Chapter 7, on perioperative monitoring)

#### **Indications**

- Total parenteral nutrition (TPN)
- Administration of hyperosmolar or irritating drugs
- Administration of vasopressors
- Requirement of CVP, PA, SvO<sub>2</sub>, CO measurements
- Limited peripheral vascular access
- Hemodialysis
- Transvenous pacemaker

#### **Contraindications**

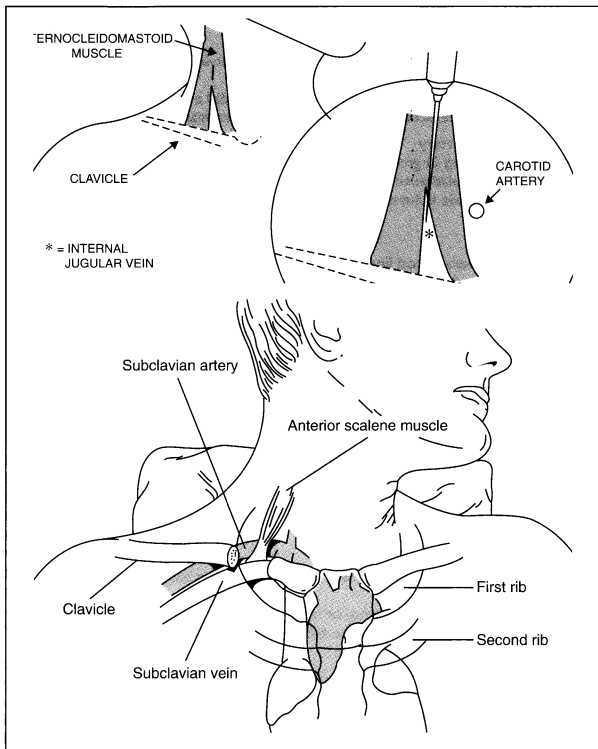
- Internal jugular vein
  - Infected site, carotid artery stenosis, ↑ ICP, access site in surgical field
- Subclavian vein
  - Infected site, contralateral pneumo- or hemothorax, contralateral thoracic intervention
  - Contralateral attempts to cannulate subclavian vein, ↓ pulm fx of contralateral lung
  - Coagulopathy, emphysema (relative)

#### **Technique: Internal Jugular Vein (IJ)**

- Positioning: Trendelenburg (provides venous distention to prevent air embolism)
- Aseptic technique: Sterile gown, face mask, gloves, skin disinfection, & whole-body drape
- Localization: Ultrasound to locate carotid artery & IJ (or palpation if US unavailable)
- Local: Infiltrate skin with local anesthetic (e.g., lidocaine) if pt awake
- Cannulation
  1. Ultrasound technique—18-ga. needle under direct visualization into vessel
  2. Palpation technique
    - Place 24-ga. finder needle 8–10 mm lateral to carotid pulse at bifurcation of medial & lateral head of sternocleidomastoid muscle with needle aimed to ipsilateral nipple and angle 30–45° to skin
    - Once venous blood aspirated, puncture IJ with 18-ga. needle at same site, angle, & depth
- Aspirate venous blood, disconnect syringe, & assess for nonarterial venous blood flow
  - Some providers will transduce catheter to ensure nonarterial placement
- Advance guidewire through 18-ga. needle (never lose control of guidewire)
- Withdraw 18-ga. needle keeping guidewire in place
  - Some providers will verify guidewire location by observation with TEE or US
- Perform 8- to 10-mm skin incision parallel to guidewire
  - Sharp side of scalpel points up toward 2 o'clock (RIJ) or 10 o'clock (LIJ)
- Insert dilator over guidewire & then remove dilator
- Insert catheter over guidewire (Seldinger technique) into vein
- Aspirate & flush all catheter lumens
- Assess for venous flow by connecting to transducer
- Obtain CXR if possible to exclude procedure-related complications (e.g., pneumothorax)

## Figure 11-1 Landmarks for internal jugular & subclavian vein catheterization

(Reproduced with permission from *Hospital Medicine: Figures 27.16 & 27.18; Lippincott*)



### Technique: Subclavian Vein

- Subclavian vein not collapsed in hypovolemic state (suspended to clavicle & pectoralis)
- Positioning: Trendelenburg (provides venous distention to prevent air embolism)
- Aseptic technique: Sterile gown, face mask, gloves, skin disinfection, & whole-body drape
- Local: Infiltrate skin with local anesthetic (e.g., lidocaine) if pt awake
- Puncture: 18-ga. needle in medioclavicular line (30–45° angle to skin) until hit clavicle
  - Once contact bone, advance needle underneath clavicle towards the sternoclavicular joint
  - Aspirate venous blood, advance guidewire
- Continue insertion of central line as described for IJ above

### Technique: Femoral Vein

- Aseptic technique: Sterile gown, face mask, gloves, skin disinfection, & whole-body drape
- Local: Infiltrate skin with local anesthetic (e.g., lidocaine) if pt awake
- Localization: Palpate femoral artery (femoral vein medial to artery)
- Puncture: 24-ga. finder needle (30–45° angle to skin) 1–2 cm below inguinal ligament
  - Aspirate venous blood, insert 18-ga. needle using same position & angle
  - Advance guidewire
- Continue insertion of central line as described for IJ above

## Complications

- Arterial cannulation, hematoma, pneumothorax (SC > IJ), chylothorax (LIJ or LSC)
- Hemothorax, infection, sepsis (Fem > SC > IJ), Thrombophlebitis (Fem > SC > IJ)
- Nerve injury (Horner syndrome, brachial plexus lesions), air embolism

## Ultrasound-Guided Central Line Placement

Insertion of central lines with US guidance may provide enhanced safety

Advantages	Disadvantages
<ul style="list-style-type: none"><li>• ↓ Complication rate in pts with predicted difficulty of line insertion</li><li>• ↓ Time &amp; ↓ attempts in pts with predicted difficulty of line insertion</li></ul>	<ul style="list-style-type: none"><li>• ↑ Time required for setup</li><li>• Potential ↑ risk of breaking sterility</li><li>• No evidence of benefit in pts where no difficulty in line placement is predicted</li></ul>

Source: Espinet A, Dunning J, Does ultrasound-guided central line insertion reduce complications and time to placement in elective patients undergoing cardiac surgery. *Interact Cardiovasc Thorac Surg* 2004;3(3):523-527.

## INSERTION OF A PULMONARY ARTERY CATHETER (PAC) (also see Chapter 7, on perioperative monitoring)

### Indications

- Management of complicated myocardial infarction (ventricular failure, cardiogenic shock)
- Assessment of resp. distress (cardiogenic vs. noncardiogenic pulm edema, 1° vs. 2° pulm HTN)
- Assessment of shock
- Assessment of fluid requirements in critically ill (hemorrhage, sepsis, acute renal failure, burns)
- Postop management of cardiac pts
- Need for heart rate pacing

### Contraindications

- Tricuspid or pulmonary valve mechanical prosthesis
- Right heart mass (thrombus and/or tumor)
- Tricuspid or pulmonary valve endocarditis

### Technique

- Central venous access as described above
- Positioning: Floating PA catheter easier in flat or slightly reverse Trendelenburg in contrast to central line placement (Trendelenburg)
- Aseptic technique: Sterile gown, face mask, gloves, skin disinfection, & whole-body drape
- PAC setup
  - Calibrate ("zero") PAC, check PAC for damage, test balloon inflation/deflation
  - Connect all lumens to stopcocks, flush to eliminate air bubbles
  - Check PAC tip frequency response by touching tip
  - PAC threaded through sterile sleeve prior to insertion into cannula
- PAC inserted percutaneously into major vein (IJ, SC, femoral) via an introducer sheath
  - RIJ: Shortest & straightest path
  - LSC: Acute angle to enter SVC (compared to RSC or LIJ)
  - Femoral veins: Distant sites, difficult if R-sided cardiac chambers enlarged (*often fluoroscopic guidance necessary*)
- Insert into introducer maintaining preformed curve (RIJ approach: Concave-cephalad)
- Once PAC enters RV, a clockwise quarter turn moves tip anteriorly (*allows easier passage into PA*)
- After inserting PAC to 20-cm mark (30-cm mark if femoral route used), inflate balloon with air (1-1.5 mL)
- **Always inflate balloon before advancing & always deflate balloon before withdrawal**
- While advancing, waveforms will be observed (distal lumen pressure monitoring):
  - RA ≈ 25 cm (RIJ)
  - RV ≈ 30 cm (↑ systolic pressure than RA, absence of dicrotic notch)
  - PA ≈ 40 cm (↑ diastolic pressure, ↓ systolic pressure)
  - PCWP ≈ 45 cm (some damping & ↓ pressure with occlusion of PA)
- Obtaining pulmonary capillary wedge pressure
  - Disconnect breathing circuit
  - Determine volume of air in balloon required to obtain a PCWP waveform (*volume < half balloon max. may indicate tip too far distal*)

- Read PCWP (correlates with LVEDP  $\approx$  4–15 mmHg is normal)
- Reconnect breathing circuit, deflate balloon, observe PA waveform return
- PA diastolic pressure usually correlates well with PCWP pressure  
(*should be used as parameter to assess left ventricular filling*)
- Withdraw PAC slightly (1–2 cm) to prevent PA rupture from distal tip migration
- Secure catheter sleeve once PCWP obtained  
(*assure PCWP pattern is reproducible before removing sterile field*)
- Troubleshooting a coiled/knotted catheter:
  - Prevention: Withdraw PAC slowly to  $\downarrow$  risk of knotting catheter upon itself
  - Use fluoroscopy if necessary to remove a knot
  - Remove PAC & introducer as one unit if unable to release a knot
- Obtain a CXR to check PAC position

#### Complication: PA Perforation

- Predisposed when **no wedge pattern evident after deep insertion**
- Circumstances that predispose to PA perforation:  
*papillary muscle ischemia, mitral stenosis or regurgitation, pulm. HTN, intrapulmonary shunting, LV failure*
- Caution if no definitive wedge pattern is observed  
(*repeated attempts to advance PAC may lead to PA perforation*)
- Coiling or actual false-negative wedging may occur & predispose to PA rupture

#### DECOMPRESSION OF A PNEUMOTHORAX (Needle Thoracostomy)

##### Indication

- Tension pneumothorax (symptoms: Hypotension,  $\downarrow$  SpO<sub>2</sub>,  $\downarrow$  breath sounds & tympanic to percussion on affected site; deviated trachea & mediastinum on CXR)

##### Technique

- Insert large bore cannula or needle into 2nd intercostal space on midclavicular line
- Release pressure in pleural cavity  
(*converts tension pneumothorax  $\rightarrow$  simple pneumothorax*)
- Subsequent chest tube insertion usually required to treat pneumothorax

##### Complications

- Lung laceration (esp. if no tension pneumothorax present)
- Reaccumulation of air in pleural space  
(*may be undetected if needle thoracostomy becomes dislodged*)

#### INSERTION OF A NASOGASTRIC TUBE (NGT)

##### Indication

- Decompression & emptying of stomach (after RSI, prior to laparoscopy, GI surgery)
- Aspiration of gastric fluid (lavage to detect intragastric blood in setting of GI bleed)
- Tube feeding
- Drug administration

##### Contraindications

- Base of skull fractures, severe facial fractures (esp to nasal bones)
- Obstructed esophagus or airway

##### Technique

- Measure tube length (tip of pt's nose to ear & down to xyphoid process)
- Lubricate end of plastic tube being inserted into anterior nares
- Advance tube through nasal cavity & into throat
- Pass pharynx rapidly with gentle continuous pressure to go into stomach  
(*if pt awake, encourage patient swallowing*)  
(*if pt asleep, consider use of laryngoscope to visualize entry into esophagus*)
- Confirm placement by CXR (safest), aspiration or injecting air (stomach auscultation)

##### Complications

- Malplacement (endotracheal, intracranial)
- Esophageal perforation
- Pulmonary aspiration, pneumothorax
- Nose erosion/bleeding, sinusitis, sore throat

# ACUTE PAIN MANAGEMENT

NALINI VADIVELU • CHRISTIAN WHITNEY

## Normal Pain Mechanisms & Pain Pathways

- Pain pathways from periphery: Start in peripheral nociceptors → end in brain
- Nociceptors present in skin, mucosa, muscles, & joints
  - Stimulated by mechanical, chemical, thermal stimuli
  - Inflammatory agents (bradykinins, cytokines, prostanooids) can sensitize nociceptors
- **Somatic pain** = musculoskeletal pain; arranged in dermatomes
- **Visceral pain** = pain from organs (bladder, bowel, ovaries)

## Neuropathic Pain

Definition: Pain caused by a lesion/dysfunction of the nervous system

- Due to pathological change in pain pathways (peripheral or central)
- Subtypes: Inflammatory or noninflammatory
  - Inflammatory pain includes cancer pain, complex regional pain syndrome, herpes zoster neuritis (shingles)
  - Noninflammatory pain includes postherpetic neuralgia, stump pain, trigeminal neuralgia
- Usually develops after partial injury
- Uninjured 1° sensory neurons can change phenotype

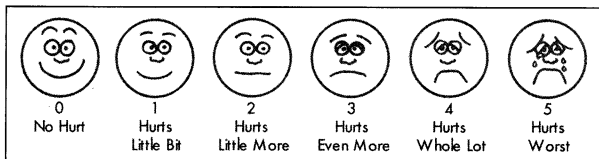
## Pain Assessment

- Pain description: Throbbing, burning, dull, aching, crushing, shooting
- Timing: When pain started, duration, onset (sudden/gradual), exacerbating or relieving factors, diurnal variation, accompanying signs, frequency
- Localization and radiation of pain

## Pain Measurement

- **Numerical rating scale:** Pt marks 1–10 line intensity of pain (0 = no pain, 10 = worst pain imaginable)
- **“Faces” rating scale:** Mostly utilized with children <3 yr of age; also adult pts with language barriers, cognitive impairment
- **Multidimensional pain scales:** Assess effect of pain on mood & daily function; Brief Pain Inventory (BPI) is one such pain scale

**Figure 12-1 Wong-Baker FACES Pain Rating Scale (From Hockenberry MJ, Wilson D: Wong's Essentials of Pediatric Nursing, 8th ed., St. Louis, 2009, Mosby. Used with permission. Copyright Mosby.)**



## ACUTE PAIN MANAGEMENT

Acute pain = pain that occurs as direct result of tissue damage, <3 months in duration

- Can occur as a result of trauma, surgical insult, labor, infection, or inflammation
- Acute postoperative pain → inflammation is a major cause

Adverse Physiologic Effects of Uncontrolled Acute Pain	
<b>Cardiac</b>	HTN, tachycardia, dysrhythmias, MI
<b>Pulmonary</b>	Atelectasis, V/Q mismatch, pneumonia
<b>Endocrine</b>	Protein catabolism, hyperglycemia, fluid retention
<b>Immune</b>	Immune function impairment
<b>Coagulation</b>	Hypercoagulation, ↑ platelet adhesion
<b>GI</b>	Ileus
<b>GU</b>	Urinary retention

## Treatment

- Includes opioid & nonopioid meds
- Opioids commonly used—can be given parenterally, helpful immediately postop

## Opioid Rotation

- Definition: Using different opioids interchangeably & via different routes
- Presence of major organ dz (liver/kidney dysfx) can markedly ↓ dose required for adequate clinical response
- Lack of complete cross tolerance between different opioids  
→ Essential to use lower than equianalgesic doses
- Oral & parenteral doses often dissimilar (due to pharmacokinetic differences)
- Opioid-assoc itching: Dilute naloxone & Benadryl → can both be used
  - Naloxone 400 mcg in 1 L of LR or NS given at 75 mL/hr
  - Avoid Benadryl in advanced age

### Changing From One Opioid to Another (example)

1. Calculate total 24-hr opioid dose (Morphine 15 mg q4h = 90 mg in 24 hr)
2. Find new opioid on equianalgesic table (Hydromorphone 7.5 mg = 30 mg morphine)
3. Solve equation for new dose (morphine)
4. Divide new 24-hr dose by # doses/day (90 mg/30 mg = x/7.5 → 22.5 mg)
5. ↓ Calc dose by 25–50% (22.5 mg/4 doses = 5.7 mg q6h)
6. Titrate to clinical effect (4 mg q6h)

### Equianalgesic Opioid Dosage Table for a 70-Kg Adult

Drug	Duration	Oral	Parenteral	Half-life
Codeine	4–6 hr	200 mg	120 mg IM	3 hr
Morphine	3–6 hr	30–60 mg	10 mg IM/IV	1.5–2 hr
Hydromorphone	4–5 hr	7.5 mg	1.5 mg IM/IV	2–3 hr
Meperidine	2–4 hr	300 mg	75 mg IM/IV	3–4 hr
Fentanyl	1–2 hr	N/A	0.1 mg IM/IV	1.5–6 hr
Oxycodone	4–6 hr	20 mg	N/A	N/A
Methadone	4–6 hr	10–20 mg	10 mg IM/IV	15–40 hr
Oxymorphone	3–6 hr	N/A	1 mg IM/IV	
Levorphanol	6–8 hr	4 mg	2 mg IM/IV	
Hydrocodone	3–4 hr	30 mg	N/A	
Propoxyphene HCl (Darvon)	3–4 hr	130 mg	N/A	12 hr
Propoxyphene napsylate (Darvon-N)	3–4 hr	200 mg	N/A	12 hr

Fentanyl patch (duragesic)  
12/25/50/75/100 mcg q72hr

Indication: Chronic pain that requires continuous opioids;  
use only in pts already on opioid therapy  
Equivalence: 25 mcg q72h = 50 mg oral morphine q24h

### Common Oral Opioids

Drug	Dose
Codeine/acetaminophen*	Adults: 1–2 tab q4–6h
Tylenol #2 (15 mg/300 mg)	
Tylenol #3 (30 mg/300 mg)	
Tylenol #5 (60 mg/300 mg)	
Hydrocodone/acetaminophen*	Based on hydrocodone content
Norco (5, 7.5, 10 mg/325 mg)	Adult (5 mg) 1–2 tab q4–6h
Vicodin (5/500, 7.5/750)	Adult (7.5, 10 mg) 1 tab q4–6h
Methadone	Adult: 2.5–10 mg PO q3–4h
Dolophine (5, 10 mg)	
Oxycodone/acetaminophen*	Adults 1–2 tabs PO q4–6h
Percocet (2.5, 5, 7.5, 10/325; 7.5/500; 10/650)	
Oxycodone (OxyContin)	5–30 mg PO q4hr prn
Oxycodone extended release	10–160 mg PO q12h
Tramadol (Ultram)	50–100 mg PO q4–6h

\*Max acetaminophen dose = 4 g/day in adults. Large percentage of population lack enzyme to converting codeine → morphine (accounts for variability to analgesic effects)

Source: Adapted from Ezekiel MR. *Handbook of Anesthesiology*, 2008 ed. Indianapolis, IN: CCS Publishing, 2008.

Opioid Side Effects	Treatments
Nausea/vomiting	Switch/wean agent, ondansetron, phenergan, prochlorperazine, haloperidol, metoclopramide
Allergic reaction	Switch agent, diphenhydramine or other antihistamines
Respiratory depression	Support airway, ↓ dose, consider naloxone
Pruritus	Switch agent, nalbuphine (antihistamines not very effective)
Delirium	Switch agent, ↓ dose, haloperidol, olanzapine
Constipation	Laxatives (senna, lactulose) + stool softeners (Colace)
Sedation	↓ Dose, hold anxiolytics, consider CNS stimulants if persistent

Common Non-narcotic Adjuvants			
	Route	Dose	Side Effects
Ketorolac	IV/IM	30 mg q6h (max 120 mg daily)	GI bleeding; decrease dose in renal impairment & elderly; contraindicated in active peptic ulcer dz;
Diclofenac	IV, IM, PO	50–100 mg	Leukopenia, thrombocytopenia
Ibuprofen	PO	300–800 mg	
Naproxen	PO	250–500 mg	
Gabapentin	PO	300 mg PO tid titrated to 1800 mg/d	
Ketamine	IV, IM, PO	IV: 0.2–0.8 mg/kg IM: 2–6 mg/kg PO: 6–10 mg/kg	Direct myocardial depressant Cerebral vasodilator > Sympathetic nervous system outflow (↑ HR & CO)
Clonidine	PO	0.3–0.4 mg	Bradycardia
Lorazepam	IV, IM, PO	IV/IM: 0.02–0.08 mg/kg PO: 2–3 mg	Resp depression (if given with opioids)
Ondansetron	IV	IV: 4 mg Peds IV: 0.05–0.075 mg/kg	Headache, dizziness, sedation, shivers, LFT elevation
Benadryl	IV, PO	IV: 10–50 mg q6–8h Peds IV: 5 mg/kg/d in 4 divided doses (max 300 mg)	Tachycardia, dizziness, seizures, urinary retention
Acetaminophen	PO, PR	0.5–2 g	Hepatotoxicity, GI upset
Celecoxib (Celebrex)	PO	100–200 mg	May ↑ risk of MI/stroke
Aspirin	PO	500–1000 mg	Reye's syndrome (do not use in children under 12 yr)

**MULTIMODAL APPROACH TO PAIN CONTROL:** (see Chapter 27, on ambulatory anesthesia)

### Analgesic Delivery Systems

#### • PO

- Not optimal for immediate postop pain (delayed time to peak effect)
- Opioids commonly combined with COX inhibitors

#### • SC/IM

- Less desirable routes (pain on injection & erratic absorption)
- Cyclical period of sedation → analgesia → inadequate analgesia common

#### • Intravenous administration

- Requires close respiratory monitoring
- Common in in PACU, ICU, & specialized units

#### • Patient-controlled analgesia (PCA)

- Allows pt to self-administer opioids with button push
- Physician specifies dose, minimum time period between doses (lockout), basal infusion rate, max delivered dose



PCA Guidelines						
Opioid	Adult Dose	Pedi Dose	Adult Lockout (min)	Pedi Lockout (min)	Adult Infusion (mg/hr)	Pedi Infusion
Morphine	1–3 mg	50–75 mcg/kg	5–20	q2–3h	0–10	15–20 mcg/kg/h
Fentanyl	0.015–0.05 mg	1 mcg/kg	3–15	q1–2h	0–0.1	2–4 mcg/kg/h
Sufentanil	0.003–0.015 mg		3–10		0–0.1	
Hydromorphone	0.1–0.3 mg	6–10 mcg/kg	10–20	q3–4h	0–0.5	3–4 mcg/kg/h
Meperidine	5–15 mg		5–15		0–30	
Methodone	10 mcg/kg	10 mcg/kg	10–15	q10–15h	20 mcg/kg	20 mcg/kg

### Neuroaxial Analgesia

- Intrathecal or epidural routes; effective for postop pain after abd, pelvic, thoracic, & orthopedic surgeries of lower extremity
- Opioid infusions often combined with local anesthetics (bupivacaine, lidocaine, ropivacaine)
- Clonidine & buprenorphine also used epidurally

### Epidural Opioids

- Site of action = pre- & postsynaptic receptors in dorsal substantia gelatinosa
- Opioid drugs enter CSF at rate dependent on physicochemical properties
  - Molecular weight, pKa, oil:water solubility
- Direct transport via spinal cord blood supply can occur
- Diffusion through dural cuff regions to spinal cord can occur
- Lipid soluble opioids (sufentanil, fentanyl) enter spinal cord faster
  - Also eliminated faster by vascular uptake, leading to a short duration of action
- Morphine = water-soluble opioid (slower action onset, longer duration of action)

### PCEA General Guidelines

Opioid	Bolus Dose (mg)	Onset (min)	Peak (min)	Duration (hr)	Infusion Rate (mg/hr)	PCEA Dose (mg)	PCEA Lockout (min)
Morphine	5	15–30	30–90	4–24	0.3–0.9	0.2–0.3	30
Fentanyl	0.05–0.1	5–10	10–20	2–3	0.025–0.05	0.02–0.03	15
Hydromorphone	0.75–1	10–15	30	8–16	0.1–0.2	0.15	30
Sufentanil	0.03–0.05	7	20–30	2–3			
Methodone	100 mcg/kg	15–20		2–3 d	2 mcg/kg/h	1 mcg/kg	15

### Epidural Local Anesthetics

Solution	Rate (mL/kg/h)
1/16% (0.0625%) Bupivacaine + 1 mcg/mL fentanyl	0.1–0.2
1/32% (0.3125%) Bupivacaine + 2 mcg/mL fentanyl + 2 mcg/mL epi	0.1–0.2
1/32% (0.3125%) Bupivacaine + 5 mcg/mL hydromorphone + 2 mcg/mL Epi	0.1–0.2
Fentanyl 2 mcg/mL + bupivacaine 1/32 % + 2 mcg/mL epinephrine	0.1–0.4
Clonidine 5 mcg/mL + ropivacaine 0.2 %	0.3–0.5 mcg/kg/h
1/5% (0.2%) Bupivacaine ± dilaudid 0.01 or 0.02 mg/mL	4–12 mL/h
1/8% (0.125%) Bupivacaine ± dilaudid 0.01 or 0.02 mg/mL	
1/16% (0.0625%) Bupivacaine ± dilaudid 0.01 or 0.02 mg/mL	

### Epidural Clonidine

- Dose = 3–5 mcg/kg; can be added to epidural mix (local or opioid)
- Prolongs duration of epidural local anesthetic by ≈ 50%
- ↓ Epidural opioid requirements ≈ 30%
- Side effects: Severe bradycardia (if used >1 mcg/kg/hr)

## Epidural Placement Levels

- Thoracotomy T4-T6
- Upper abdomen T8
- Lower abdomen T10-T12
- Lower extremity/pelvis L2-L4

## Signs of an Inadequate Epidural

- Pain at rest & with movement (pain score >5)
- Tachycardia
- ↑ Respiratory rate
- For thoracic & abd cases  
→ Inability to breathe deeply, cough, use incentive spirometer

## Testing of Epidurals

- 2% lidocaine with 1:200,000 epinephrine or 0.25% bupivacaine with 1: 200,000 epinephrine
  - Give 3 mL of either via epidural → check BP, motor block & pain relief
- May repeat 3 mL after 3–5 min
- Consider replacing epidural if no pain relief after 8 mL of test dose over 10 min

## “Splitting” Epidural with IV PCA

- Technique: Add IV PCA in addition to epidural infusion  
(*must simultaneously remove opioids from epidural infusion*)
- May be necessary for
  - Pt with opioid dependence
  - Incomplete incisional coverage from epidural
  - Upper & lower body surgeries (trauma victims)
  - Large surgical incision
  - Lower level epidural catheter placement

## Treatment of Side Effects With Epidural Analgesia

Side Effect	Treatment
Pruritus	Nalbuphine 5–10 mg IV/IM Benadryl 25–50 mg IV Naloxone 40–80 mcg IV
Nausea/Vomiting	Metoclopramide 10 mg IV Nalbuphine 10 mg IV Naloxone 40–80 mcg IV
Respiratory depression	Naloxone 0.1 mg IV

## Intrathecal Opioids

- Act on substantia gelatinosa of the spinal cord
- Potent analgesia due to central localization of receptors
- Side effects are mediated by mu receptors (in brain & brain stem)

## Intrathecal Opioids

Opioid	Dose	Duration	Indications	Side Effects
Morphine	0.2–0.4 mg	24 hr	Good spread & long duration of action	Nausea, vomiting resp depression, sedation, itching, urinary retention
Fentanyl	12.5–25 mcg	3–4 hr	For segmental spread	Nausea, resp depression
Sufentanil	5–10 mcg	2–4 hr	Very lipid soluble & has segmental spread	Nausea, resp depression
Meperidine	10 mg	3–6 hr	In addition to opioid effect has local anesthetic effect	Hypotension

## Mixed Agonists/Antagonists

- Block effects of high doses of morphine-like drugs
  - By competing with morphine-like drugs to bind to mu opioid receptor
- Produce partial agonist effects at  $\kappa$  (kappa) and/or  $\delta$  (delta) opioid receptors

<b>Mixed Narcotic Agonists/Antagonists</b>					
<b>Analgesic Drug</b>	<b>Opioid Potency</b>	<b>Respiratory Withdrawal</b>	<b>Depression</b>	<b>Side Effects</b>	<b>Dose</b>
Naloxone	None	Caused abstinence syndrome	None	No sedation	
Nalbuphine	Equal to morphine	Abrupt discontinuation can cause opioid withdrawal	Less respiratory depression than morphine	Considerable sedation; may cause psychological or physical dependence	10 mg IV q3-4hr
Buprenorphine	25-50 times more potent than morphine	Mild to moderate withdrawal effects	Ceiling in respiratory depression	Prolonged use can result in physical dependence	0.4 mg IV q4-6hr
Pentazocine	1/3 potency of morphine	Mild	Respiratory depression can be reversed by narcotic analgesics	Can cause tolerance and addiction	50 mg PO q4-6hr
Butorphanol	1/3 potency of morphine	Withdrawal effects similar to that caused by naloxone	Respiratory depression 5 times more than morphine	Sedation Nausea Unpleasant psychomimetic effects	0.5-2 mg IV q3-4hr

# PACU MANAGEMENT AND DISCHARGE

PIYUSH MATHUR

## COMMON POSTOPERATIVE PROBLEMS

### HYPOTENSION

#### Common Causes of Hypotension in PACU

- Hypovolemia
- Bleeding
- Sepsis/↓ SVR
- Cardiac arrhythmias
- Drugs/anesthesia (spinal/epidural)
- Error in measurement (inappropriate cuff size, machine malfunction)
- Pulmonary embolism
- MI/↓ myocardial contractility
- Cardiac tamponade
- Congestive heart failure
- Anaphylaxis/anaphylactoid reaction
- Pneumothorax
- Adrenal insufficiency/severe hypothyroidism

Source: Adapted from Keith, RD, et al. Cardiovascular events in postanesthesia care unit: Contribution of risk factors. *Anesthesiology* 1996;84:772.

#### Initial Diagnosis & Management

1. Examine & stabilize—check **A**irway, **B**reathing & **C**irculation
2. Fluid resuscitate—obtain adequate venous access
3. Review data—patient history, anesthesia record, surgical procedure, estimated blood loss, PACU data
4. Consider laboratory studies
  - ABG—assess oxygenation & acid-base status
  - CBC—assess hemoglobin & platelet level (also consider coagulation studies)
  - ECG—assess for arrhythmias (also consider cardiac enzymes)
  - CXR—rule out pneumothorax/hemothorax/cardiomegaly
  - Blood cultures—esp. if sepsis suspected
  - Transthoracic/transesophageal echo—assess cardiac contractility, LV/RV function, LV filling, IVC collapse, valvular abnl
5. Consider invasive monitoring—arterial BP, CVP, pulmonary artery catheter
6. Initiate pressor/inotropic support—phenylephrine, norepinephrine, dopamine
7. Obtain consultations as needed—cardiology, ICU, surgery

#### Management of Specific Conditions

##### Hypovolemia

**Diagnosis:** Tachycardia, hypotension, low CVP/PCWP, respiratory variation in arterial waveform, IVC collapse/underfilled LV on echo

**Treatment:** Fluid resuscitation, assess for causes (ongoing bleeding, diuresis, high NG output)

##### Bleeding

**Diagnosis:** Tachycardia, anemia, hypovolemia, sanguinous drain output

**Treatment:** Fluid resuscitation, blood transfusion, correct coagulopathy & thrombocytopenia, treat hypothermia, consider return to OR

##### Sepsis

**Diagnosis:** Fever, leukocytosis, tachycardia, hypovolemia, lactic acidosis

**Treatment:** Fluid resuscitation, obtain blood/specific cultures, initiate broad-spectrum antibiotics

##### Myocardial Infarction/Ischemia

**Diagnosis:** 12-lead ECG, TTE/TEE, cardiac enzymes, cardiology consult

**Treatment:** Cautious fluid resuscitation, aspirin, discuss with cardiologist & surgeon role of heparinization/cardiac cath/antiplatelet agents; consider inotropic/vasopressor/IABP support; may initiate diuresis/β-blockade once BP stabilized

##### Arrhythmias

**Diagnosis:** 12-lead ECG, cardiac enzymes, check electrolytes, ABG

**Treatment:** Treat the cause, follow ACLS protocol

- Tachyarrhythmia: Electrical/chemical cardioversion, correct electrolytes, cardiology consultation, maintenance antiarrhythmics
- Bradyarrhythmia: Atropine/epinephrine/dopamine, transcutaneous transvenous pacing, cardiology consult

## Drugs

**Treatment:** Stop the drug, administer antagonist agent (e.g., naloxone for morphine)

## Pulmonary Embolism

**Diagnosis:** ECG → sinus tach/S<sub>1</sub>Q<sub>3</sub>T<sub>3</sub>; ultrasound (US) of lower ext; D-dimer not helpful  
TTE/TEE → rule out central pulm embolism/assess RV dysfx  
V/Q scan/CT chest pulm angiogram when stable

**Treatment:** Cautious fluid resuscitation, invasive monitoring, inotropes/pressors  
Consider thromboembolectomy/catheter directed thrombolysis/anticoagulation/IVC filter placement

## Congestive Heart Failure

**Diagnosis:** Bibasilar crackles, frothy sputum on exam  
Chest x-ray → cephalization of blood vessels, pulmonary edema, ↑ cardiac shadow  
Invasive hemodynamic monitoring shows ↓ cardiac output, ↑ filling pressures

**Treatment:** Supplemental O<sub>2</sub>, diuresis, digoxin/inotropic support

## Anaphylaxis

**Diagnosis:** Tachycardia, vasodilatory shock (↓ SVR, ↑ cardiac output)  
Check serum tryptase & eosinophil count, consult allergy

**Treatment:** Remove causative agent, fluid resuscitation, diphenhydramine, steroids, epinephrine

## Pericardial Tamponade

**Causes:** Postcardiac surgery bleeding, trauma, dissecting thoracic aneurysm, procedure related (e.g., s/p CVP placement, coronary cath)

**Diagnosis:**

- Beck's triad: Hypotension, jugular venous distension, muffled heart sounds
- Pulsus paradoxus: ↓ of > 10 mmHg in systolic bp with inspiration
- ECG: Nonspecific ST-segment changes, low-voltage QRS
- Chest x-ray: Enlarged cardiac shadow
- Echo: Diagnostic & may assist in therapeutic pericardiocentesis

**Treatment:** Fluid resuscitation, pericardiocentesis, surgical repair of bleeding site

## Pneumothorax

**Diagnosis:** ↓ Breath sounds, ↓ lung markings on chest x-ray

**Treatment:** Needle decompression/chest tube placement, surgery consult

## Errors in Measurement

**Diagnosis:** Inappropriate BP cuff size, incorrect transducer level, poor arterial waveform (under/overdamped), machine malfunction

**Treatment:** Place appropriate size BP cuff, manual measurement, check a-line waveform, zero transducer at appropriate level, check equipment

## Endocrine Disorders: Adrenal Insufficiency

**Diagnosis:** ACTH stim test; random cortisol levels nonspecific & unhelpful

**Treatment:** Fluids, administer hydrocortisone, endocrinology consult

## Endocrine Disorders: Severe Hypothyroidism

**Diagnosis:** Hypothermia, bradycardia, high TSH level, low free T<sub>3</sub> & T<sub>4</sub> levels

**Treatment:** Fluid resuscitation, levothyroxine administration, endocrinology consult

## Bleeding

### Common Causes

1. Surgical bleeding
2. Coagulopathy
3. Thrombocytopenia

### Diagnosis

- Bleeding may be obvious or occult
- Important to examine surgical drains/surgical site
- Signs of hypovolemia (tachycardia, tachypnea, ↓ urine output) may suggest bleeding

### Management

- Consult surgeon, place large-bore IVs & initiate fluid resuscitation
- Send CBC, PT, PTT, INR, & fibrinogen & request blood cross-match

- Transfuse: PRBCs based on hemoglobin level, pt's condition & coexisting disease  
FFP to correct coagulopathy  
Cryoprecipitate if evidence of hypofibrinogenemia  
Platelets if level <50,000–100,000 or previous exposure to antiplatelet agents
- Consider use of recombinant factor 7 in uncontrolled, diffuse, post-op bleeding
- Assess for evidence of DIC (↓ fibrinogen, + FDP/D-dimer, ↑ PT/PTT, ↓ platelets)  
→ Occurs in mismatch transfusion, placental abruption, intrauterine fetal demise, underlying malignancy, complex infections  
→ Treat with transfusion of FFP, cryoprecipitate, & platelets
- Maintain normothermia & consider calcium administration during massive transfusion
- Alert OR personnel about need for possible take-back

## HYPERTENSION

### Common Causes of Hypertension in the PACU

- |   |   |
|---|---|
| <ul style="list-style-type: none"> <li>• Pain</li> <li>• Anxiety</li> <li>• Respiratory insufficiency (hypoxia, hypercarbia)</li> <li>• Hypothermia/shivering</li> <li>• ↑ Sympathetic activity</li> <li>• ↑ ICP</li> </ul> | <ul style="list-style-type: none"> <li>• Essential hypertension/missed medications</li> <li>• Fluid overload</li> <li>• Endocrine disease (thyroid storm) pheochromocytoma)</li> <li>• Error in measurement (inappropriate cuff size, machine malfunction)</li> </ul> |
|---|---|

### Diagnosis and Management

- Treat the underlying cause
- Resume home antihypertensives as soon as possible
- For initial management consider:
  - Labetalol 5–40 mg IV bolus q10min or
  - Hydralazine 2.5–20 mg iv bolus q10–20min
  - Lopressor 2.5–10 mg IV bolus
- For severe hypertension, consider vasodilator infusion
  - Sodium nitroprusside (0.25–10 mcg/kg/min)
  - Nitroglycerine (10–100 mcg/min)
  - Esmolol, nicardipine, cardiazem infusions may also be used

## RESPIRATORY AND AIRWAY PROBLEMS

### Common Causes of Respiratory Insufficiency in the PACU

Hypoventilation	Upper Airway Obstruction	Hypoxemia
<ul style="list-style-type: none"> <li>• Residual anesthesia</li> <li>• Residual muscle relaxant</li> <li>• Postop narcotics</li> <li>• Splinting 2° to pain</li> <li>• Tight abdominal binder</li> <li>• Obstructive sleep apnea/obesity</li> <li>• Premature infants/neonates</li> </ul>	<ul style="list-style-type: none"> <li>• Airway edema</li> <li>• Trauma</li> <li>• Vocal cord paralysis</li> <li>• Arytenoid dislocation</li> <li>• Secretions</li> <li>• Foreign body</li> <li>• Laryngospasm</li> <li>• Anxiety/Munchausen's stridor</li> </ul>	<ul style="list-style-type: none"> <li>• Atelectasis</li> <li>• Asthma/COPD exacerbation</li> <li>• CHF/fluid overload</li> <li>• Pulmonary embolism</li> <li>• ALI/ARDS</li> <li>• Aspiration</li> <li>• Pneumo/hemothorax pleural effusion</li> <li>• Diaphragmatic injury/paralysis</li> <li>• Pneumonia</li> </ul>

Source: Adapted from Keith RD, et al. Critical Respiratory events in the postanesthesia care unit: patient, surgical, and anesthetic factors. *Anesthesiology* 1994;81:410.

### Respiratory Insufficiency: Diagnosis & Management

1. Assess **A**irway, **B**reathing, **C**irculation
2. ↑ Delivered FiO<sub>2</sub>, ↑ flow rate & consider nonrebreather or shovel mask
3. Consider jaw thrust/chin lift, placement of oral/nasal airway
4. Consider positive-pressure ventilation with bag-valve mask
5. Consider intubation vs noninvasive ventilation (CPAP/BiPAP)
6. Review pt history, OR & postop course, fluid status, & medications administered
7. Consider ABG, chest x-ray (rule out pneumothorax/pulmonary edema)

## Respiratory Insufficiency: Management of Specific Conditions

Hypoventilation

**Diagnosis:** Hypoventilation/inadequate ventilation for sufficient gas exchange  
↑ PaCO<sub>2</sub> & respiratory acidosis

Treatment of Hypoventilation	
Suspected Cause	Therapy
Residual inhalational/IV anesthesia	Arouse patient, be supportive
Residual muscle relaxant	Administer anticholinesterase inhibitor (neostigmine)
Postop narcotic administration	Administer naloxone
Splinting 2° to pain	Initiate pain control, consider PCA/regional analgesia
Tight abdominal binder	Release binder, consult surgeon
Obstructive sleep apnea/obesity	Reposition patient, consider BiPAP
Premature infants/neonates	Supplemental O <sub>2</sub> , consider acetaminophen/regional analgesia instead of narcotics

Fluid Overload/Pulmonary Edema

**Diagnosis:** Hypoxemia on ABG, high CVP/PCWP  
Chest x-ray: ↑ Pulmonary vasculature, ↑ interstitial/alveolar fluid, pleural effusion, fluid in fissure

**Treatment:** Stop IV fluids; administer diuretics (furosemide 20–100 mg IV)  
Provide supplemental O<sub>2</sub>; consider noninvasive mechanical ventilation

Atelectasis

**Diagnosis:** ↓ Breath sounds, opacification on chest x-ray

**Treatment:** Incentive spirometry; inhaled N-acetylcysteine to loosen secretions; reposition patient; CPAP/BiPAP; bronchoscopy to remove impacted secretions; chest physiotherapy; positive-pressure ventilation with PEEP

Asthma/COPD Exacerbation

**Diagnosis:** Wheezing on auscultation

**Treatment:** Albuterol/atrovent; steroids (methylprednisone 125 mg IV); cromolyn sodium; CPAP/BiPAP; severe bronchospasm may require intubation  
Aminophylline (6 mg/kg IV load, followed by infusion 0.5–1 mg/kg/hr)

Pulmonary Embolism

**Diagnosis:** ECG → sinus tach/S<sub>1</sub>Q<sub>3</sub>T<sub>3</sub>; US of lower ext.; D-dimer not helpful  
TTE/TEE → rule out central pulm embolism/assess RV dysfx  
CT chest/<sup>V</sup>/Q scan/pulm angiogram when stable

**Treatment:** Cautious fluid resuscitation, invasive monitoring, inotropes/pressors, consider thromboembolctomy/catheter-directed thrombolysis/anticoagulation/IVC filter placement

ALI (Acute Lung Injury)/ARDS (Acute Respiratory Distress Syndrome)

**Diagnosis:** ARDS = acute respiratory failure without evidence of left heart failure  
Bilateral infiltrates on chest x-ray, PaO<sub>2</sub>/FiO<sub>2</sub> ratio <200  
ALI = (ARDS features + PaO<sub>2</sub>/FiO<sub>2</sub> ratio <300)

**Treatment:** Treat the underlying cause & maintain lung protective ventilation

(See ARDS section in Chapter 17, on anesthesia for thoracic surgery)

Aspiration

**Diagnosis:** Chest x-ray may reveal foreign body, infiltrates, atelectasis or collapse

**Treatment:** Supportive care for small aspirations (no respiratory compromise)  
Large aspirations: rapid sequence intubation, gastric decompression, mechanical ventilation with high PEEP, bronchoscopy to remove large foreign bodies; prophylactic antibiotics & steroids are ineffective; bronchoalveolar lavage & routine suctioning should not be performed  
Prevent recurrence: elevate head of bed, avoid sedation, place NG tube

Upper Airway Obstruction/Stridor

**Causes:** Airway edema/trauma, vocal cord paralysis, arytenoid dislocation, secretions, foreign body

**Treatment:** Racemic epinephrine, dexamethasone, humidified air, heliox  
Treat secretions with suctioning & admin of glycopyrrolate (0.2 mg IV)  
Severe edema/trauma may necessitate reintubation  
Obtain ENT consult for vocal cord paralysis/arytenoid dislocation/removal of foreign body

### Pneumothorax/Hemothorax/Pleural Effusion

**Diagnosis:** Chest x-ray diagnostic most of the time

**Treatment:** Needle decompression of chest (2nd intercostal space in midclavicular line), chest tube decompression  
Exploratory thoracotomy for large hemothorax/ongoing bleeding

### Diaphragmatic Injury/Paralysis

**Diagnosis:** Elevation of hemidiaphragm on chest x-ray

**Treatment:** Paralysis 2° to regional blocks are usually temporary  
Supportive treatment with ↑ FiO<sub>2</sub>, reassurance, noninvasive ventilation  
Large diaphragmatic injury may require surgical repair

### Pneumonia

**Diagnosis:** Fever, cough, leukocytosis, new infiltrate on chest x-ray  
Obtain respiratory secretions & blood cultures

**Treatment:** Broad spectrum antibiotics

### Laryngospasm

**Diagnosis:** Laryngospasm = involuntary contraction & closure of vocal cords  
Presence of inspiratory stridor, ↑ inspiratory effort, poor air exchange  
→ leading to ↓ SpO<sub>2</sub>, pulmonary edema, cardiac arrest  
Risk factors: Young age, upper respiratory infection, GERD, obesity, ENT surgery, obstructive sleep apnea

**Treatment:** Cessation of stimulation, positive pressure ventilation with 100% O<sub>2</sub> if positive pressure ventilation is ineffective  
1. Consider inducing anesthesia with propofol  
2. Consider inducing muscle relaxation with succinylcholine  
4% of pts with laryngospasm develop negative pressure pulmonary edema → consider mechanical ventilation with PEEP & diuresis

### Anxiety/Munchausen's Stridor

**Diagnosis:** Episodic inspiratory stridor (esp. to attract attention), nl flow volume  
Loops refractory to medical management of inspiratory stridor  
Risk factors include female, type A personality, anxiety disorder, GERD  
Fiberoptic laryngoscopy reveals posterior diamond shaped glottis

**Treatment:** Patient education, speech therapy, benzodiazepines (lorazepam 1–2 mg IV)

## NEUROLOGIC PROBLEMS

**Common problems:** Delayed awakening, emergence delirium/confusion, anxiety/panic attack, peripheral neuropathy

Delayed Awakening (see *Chapter 10 on common intraoperative problems*)

**Definition:** Delayed awakening is said to occur when a patient fails to regain appropriate level of consciousness following general anesthesia

### Causes of Delayed Awakening in PACU

Anesthesia related	Residual anesthetic Residual muscle relaxant, pseudocholinesterase deficiency Excessive narcotics
Metabolic	Hypothermia Hypoxemia Hypercarbia/hyponatremia/hypocalcemia/hypoglycemia Renal/hepatic failure
Intracranial event	Stroke/cerebrovascular accident (CVA) Seizure Intracranial hypertension

**Diagnosis:** Perform complete neuro assessment (cranial, motor, & sensory nerves)  
Review anesthetic record for drugs/doses  
Check for residual muscle relaxant with train-of-four/tetany  
Send ABG, serum sodium/calcium/glucose levels, check pt temp  
Consider application of bispectral index/EEG  
→ Low bispectral index may be suggestive of residual anesthetic  
→ EEG can assess for seizure activity  
Consider neurologic imaging to assess for stroke (noncontrast CT/MRI brain)  
Consider pseudocholinesterase deficiency (family hx, pseudocholinesterase level, dibucaine number)



**Treatment:** Consider narcotic reversal if slow respiratory rate + pinpoint pupils  
→ Administer naloxone (0.04 mg IV q2min up to 0.2 mg IV)  
Consider benzo reversal with flumazenil (0.2 mg IV q 2 min up to 1 mg IV)  
Reverse muscle relaxants, correct electrolyte abnl, rewarm pt as indicated

Stroke:

**Risk factors:** Geriatric patients, history of TIA/stroke, cardiac surgery (highest risk factor), aortic/carotid surgery, craniotomy, intraoperative hypotension, hypoxia, atrial fibrillation

**Treatment:** Consult neurology, maintain adequate SpO<sub>2</sub> & cerebral perfusion pressure

Seizure

**Causes:** Epilepsy disorder hx, preop cessation of anticonvulsants, stroke, hypoxia, hypoglycemia, hyponatremia, hypocalcemia, hypophosphatemia, trauma, hypotension, EtOH withdrawal, local anesthetic overdose

**Treatment:** Benzodiazepine (lorazepam 1–5 mg IV) for ongoing seizures;  
Provide supplemental O<sub>2</sub> & secure airway if needed;  
Consult neurology

Intracranial Hypertension

**Diagnosis:** Place intracranial pressure monitor

**Treatment:** Hyperventilate patient to lower PaCO<sub>2</sub>

Maintain CPP

Consider administration of mannitol, furosemide, 3% saline

Consider deep sedation/muscle relaxation/hypothermia in cases of refractory high ICP

Emergence Delirium/Confusion

**Risk factors:** High preop anxiety, benzodiazepine/ketamine use, age (toddlers & geriatrics), hypoxia, hypercarbia, hyponatremia, EtOH withdrawal, intubated patients, presence of Foley catheter

**Diagnosis:** Check ABG, electrolytes, consider CT scan of brain

**Treatment:** Treat underlying cause

Reorient patient & avoid aggravating factors

Haloperidol (2.5–10 mg IV)/soft limb restraints for combative pts

Anxiety/Panic Attack

**Treatment:** Reassurance, de-environmentalization, pain management helpful

Consider benzodiazepines (lorazepam 1–2 mg IV or midazolam 1–2 mg IV)

Peripheral Neuropathy (see Chapter 14, on anesthesia complications)

**Risk factors:** Excessive stretch, compression, direct trauma, positioning, diabetes, male, BMI >37 or <24, prolonged hospital stay  
Ulnar neuropathy is most common

**Treatment:** Sensory neuropathies: Usually resolve in 1–2 weeks

Motor neuropathies: Require neurologist consult, EMG, physical therapy

## PACU DISCHARGE CRITERIA

- PACU discharge criteria usually based on modified Aldrete score (*Anesthesiology* 2002;96:742)
- Clinical judgment should always supersede any score or criterion
- Postanesthesia recovery is divided into 2 phases
  - Phase 1:** Starts with pt entering PACU from OR till criteria are met for transfer to phase 2 in PACU/hospital room/ICU  
(Note: Patients are not discharged home from phase 1)
  - Phase 2:** Starts with completion of phase 1, ends with patient discharge to home

### Guidelines for Discharge From Phase 1

Mental status	Preop level of mental status; pain should be <4 out of 10 or tolerable
Vital signs	Stable & within acceptable limits
Surgical site	In appropriate condition, invasive lines & tubes patent/working
Regional anesthesia	Progressively ↓ sensory & motor block
Pts discharged from phase 1 to hospital room should have postanesthesia recovery score (modified Aldrete score) of ≥9	

## Guidelines for Discharge From Phase 2

- Redocumentation of vitals, postanesthesia recovery score
- Acceptable surgical site condition
- Adequate pain control (<3 out of 10 or tolerable)
- Ability to ambulate
- Recovery from regional anesthesia (except for peripheral nerve block)
- Discharge to a responsible adult
- Postanesthesia recovery score of  $\geq 9$
- Written & verbal instructions provided prior to discharge

### Modified Aldrete Scoring System

Activity: Able to move (voluntarily or on command)

- Four extremities 2
- Two extremities 1
- No extremities 0

Respiration

- Able to breathe deeply or cough freely 2
- Dyspnea, shallow, or limited breathing 1
- Apnea 0

Circulation

- BP  $\pm$  20 mmHg of preop level 2
- BP  $\pm$  20–50 mmHg of preop level 1
- BP  $\pm$  50 mmHg of preop level 0

Consciousness

- Fully awake 2
- Arousable on calling 1
- Unresponsive 0

Oxygen saturation

- SpO<sub>2</sub> >92% 2
- Needs supplement O<sub>2</sub> to maintain SpO<sub>2</sub> >90% 1
- SpO<sub>2</sub> <90% with oxygen 0

Source: Adapted from *J Clin Anesth* 1995;7:89–91.

### Common Discharge Issues (*Anesthesiology* 2002;96:742–752)

1. Passing of urine is not a mandatory requirement
2. Ability to drink and retain fluids is not mandatory
3. There is no minimum PACU stay period

# COMPLICATIONS OF ANESTHESIA

MICHAEL W. SANFORD • DAVID A. NAKATA

PERIOP COMP 14-1

*Complications arise from human error, equipment malfunctions, and patient comorbidities.*

## Peripheral Nerve Injury

### Classification of peripheral nerve lesions

**Neurapraxia:** No peripheral degeneration → rapid recovery

**Axonotmesis:** Associated with axonal degeneration without complete destruction → recovery slow but typically complete

**Neurotmesis:** Separation of related parts of nerve occurs → recovery is poor

### Anesthesia-related causes

*Ischemia secondary to*

- Nerve stretch: Tension within axon leads to compression of arterial & venous plexus
- Direct nerve compression: Compressive forces > mean capillary pressure (35 mm Hg) → resistance to flow & ischemia
- Both stretch & compression can act to simultaneously impact nerves (especially ulnar nerve, brachial plexus, sciatic nerve)

*Duration of ischemia*

- Unknown what peripheral nerve ischemic time → permanent damage
- Comorbidities that ↓ vascular supply may ↓ time required before damage
- Vascular tourniquets (e.g., in orthopedic surgery) shown to produce reversible nerve conduction abnormalities  
→ Tourniquet times <2 hours thought to be well tolerated  
→ Compression unlikely cause of postop neuropathy in short cases

*Other factors impacting peripheral nerves*

- Coexisting disease: Diabetes mellitus, nerve entrapment syndromes (osteo- & rheumatoid arthritis, previous trauma, tissue edema), metabolic abnormalities (malnutrition, vitamin deficiencies), drug-related (chemotherapy), hereditary neuropathies
- Coexisting issues may lead to neuropathies 2° to "double-crush syndrome:" (two separate causes of nerve injury potentiate severity of injury)

## Summary of ASA Practice Advisory: Prevention of Peripheral Neuropathy

<b>Preop assessment</b>	<ul style="list-style-type: none"><li>• Helpful to ascertain that pts can comfortably tolerate anticipated operative position</li></ul>
<b>Upper extremity positioning</b>	<ul style="list-style-type: none"><li>• Limit arm abduction to 90° in supine pts. Prone pts may comfortably tolerate arm abduction &gt;90°</li><li>• Position arms to ↓ pressure on postcondylar groove of humerus (ulnar groove); when arms tucked at side, neutral forearm position is recommended; when arms abducted on armboards, either supination or neutral forearm position acceptable</li><li>• Avoid prolonged pressure on radial nerve in spiral groove of humerus</li><li>• Extension of elbow beyond comfortable range may stretch median nerve</li></ul>
<b>Lower extremity positioning</b>	<ul style="list-style-type: none"><li>• Lithotomy positions that stretch hamstring muscle group beyond comfortable range may stretch sciatic nerve</li><li>• Avoid prolonged pressure on peroneal nerve at fibular head</li><li>• Neither extension nor flexion of hip ↑ risk of femoral neuropathy</li></ul>
<b>Protective padding</b>	<ul style="list-style-type: none"><li>• Padded armboards may ↓ risk of upper extremity neuropathy</li><li>• Chest rolls in laterally positioned pts may ↓ risk of upper extremity neuropathies</li><li>• Padding at elbow &amp; fibular head may ↓ risk of upper &amp; lower extremity neuropathies, respectively</li></ul>
<b>Equipment</b>	<ul style="list-style-type: none"><li>• Properly functioning automated BP cuffs on upper arms do not affect risk of upper extremity neuropathies</li><li>• Shoulder braces in steep head-down positions may ↑ risk of brachial plexus neuropathies</li></ul>
<b>Postop assessment</b>	<ul style="list-style-type: none"><li>• Simple postop assessment of extremity nerve function may lead to early recognition of peripheral neuropathies</li></ul>
<b>Documentation</b>	<ul style="list-style-type: none"><li>• Charting specific positioning actions during care of pts may result in improvements of care by helping practitioners focus attention on relevant aspects of patient positioning &amp; providing information that continuous improvement processes lead to refinement in patient care</li></ul>

**AIRWAY/DENTAL COMPLICATIONS****Airway Complications***Incidence*

- Unknown owing to varying significance/detection of injuries
- Minor trauma to larynx & pharynx may be as common as 6%
- Damage typically ↑ in relation to duration of intubation (many injuries result from placement of endotracheal tube)
- Many injuries occur during routine, "easy" intubations
- Delayed, chronic complications often present weeks to even months after extubation, particularly with prolonged intubations (>5 days)

*Risk Factors for Intubation Trauma*

- Difficult, traumatic, multiple attempts at intubation
- Laryngeal abnormalities (past trauma, inflammatory conditions, infection)
- Movement of endotracheal tube (tube manipulation/surgical repositioning, coughing/bucking)
- Impaired clearance of secretions
- Gastroesophageal reflux

<b>Sites of Injury</b>		
<b>Nasal</b>	<i>Nasal alar necrosis</i>	Preventable by careful (ETT) positioning
	<i>Sinusitis</i>	Incidence ↑ with duration of intubation (up to 20% with nasal intubation >5 days)
<b>Oropharyngeal</b>	<i>Contusion/lacerations of lips or pharynx</i>	Usually 2° to trauma from laryngoscope blade or ETT; rarely serious bleeding
	<i>Dental trauma</i>	Common (see discussion on next page)
<b>Laryngeal &amp; tracheal injury</b>	<i>Vocal cord/mucosal edema</i>	Most significant complication early after extubation; presents with airway obstruction <ul style="list-style-type: none"> <li>• Acute obstruction esp worrisome in children (smaller airway diameter)</li> <li>• Incidence of symptomatic laryngeal/tracheal edema up to 4% in kids</li> <li>• Tracheal erosion 2° to ETT trauma → tracheoesophageal fistula/tracheomalacia</li> </ul>
	<i>Granuloma formation</i>	More common in adults, particularly women
	<i>Vocal cord dysfunction</i>	<ul style="list-style-type: none"> <li>• May occur 2° to direct trauma, recurrent laryngeal nerve injury, arytenoid dislocation</li> <li>• Presents with partial airway obstruction/dysphonia</li> <li>• Bilateral vocal cord dysfunction presents as complete airway obstruction (requiring emergency airway)</li> <li>• May evaluate mobility with direct laryngoscopy under GA</li> </ul>
	<i>Tracheobronchial rupture</i>	<ul style="list-style-type: none"> <li>• Suspect with subcutaneous emphysema, respiratory distress, pneumomediastinum &amp; pneumothorax</li> <li>• May lead to mediastinitis &amp; death</li> </ul>
<b>Pulmonary</b>	<i>Aspiration</i>	<ul style="list-style-type: none"> <li>• Occurs up to 0.05%, usually during induction</li> <li>• Ranges from benign mild inflammation to severe inflammation, pneumonitis, asphyxiation from airway obstruction, or lethal infectious pneumonia</li> <li>• Risk factors include emergency surgery, unanticipated airway difficulty, GI obstruction</li> <li>• Prevention with nonparticulate antacids, H<sub>2</sub> receptor antagonists, or gastroprokinetic agents</li> </ul>
<b>Esophageal</b>	<i>Esophageal/pharyngeal perforation</i>	<ul style="list-style-type: none"> <li>• Often delayed presentation with mortality 25–50%</li> <li>• Associated with difficult intubation, elderly, female gender</li> <li>• Risk ↑ by insertion of devices such as TEE probes &amp; esophageal dilators</li> </ul>

- Use small ETTs with lowest possible cuff pressures (leak <30 cm in pediatric pts)
- Limit use of adjuncts (such as intubating stylets)
- Wean ventilator to minimize duration of intubation
- Treat airway infections aggressively & early
- Minimize aspiration risk (when risk factors present)
- Perform detailed assessment to prevent unanticipated airway difficulty (to ↓ chance of otherwise preventable airway injury)
- Prepare alternative plans if intubation fails
- Discuss risk of airway injury with pts preop (shown to ↓ litigation)

#### Management

- Acute airway edema/stridor: Nebulized racemic epinephrine; dexamethasone controversial.
- Prolonged intubation (>5 days): Consider laryngeal evaluation to evaluate for injury
- Chronic injury from repeated/prolonged intubation: Surgical correction may be required.
- Tracheobronchial rupture: Emergent surgical correction

#### Obtain follow-up if concerned about airway trauma

- Inform pts if airway management was difficult/nonstandard

### Dental Injuries

- Dental trauma: Most common permanent airway injury & leading source of malpractice claims (30–40%)
- Injuries: Fractured teeth, displaced restorations, subluxation, & avulsion (upper incisors most commonly affected secondary to use as fulcrum for laryngoscope)  
Deciduous tooth loss → can result in problems with permanent teeth
- Adverse outcomes → related to aspiration of teeth/restorations

#### Incidence

- Overall incidence: Reports range from 0.02–12% (75% of injuries occur during intubation)
- Injuries can occur during maintenance (poorly positioned airway, bite block, masseter spasm during wakeup)

#### Risk Factors

- Tracheal intubation; poor dentition/periodontal disease; difficult airway characteristics; past dental restoration/endodontic treatment; elderly pts; brittle enamel; loose deciduous teeth; inexperienced laryngoscopist

#### Prevention

- Detailed preop history & exam:
  - Caries/loose teeth, prostheses, past dental work
  - Assess mouth opening
  - Evaluate dentition, evidence of periodontal disease, tooth hypermobility
  - Document preexisting conditions (↓ reduces litigation if damage occurs)
- Consider tooth protection
  - Protectors (prefabricated rubber/custom-made by dentist)

#### Management

- Loosened tooth
  - Return to original position promptly; splint with tape/suture
- Displaced fragment of tooth/restoration:
  - Locate & recover all pieces; consider radiographs (chest, lateral head & neck) to exclude passage through glottis
- Avulsed tooth
  - Immediately replace tooth to original position
  - Avoid wiping or drying root surface
  - Splint temporarily with tape/suture
  - If aspiration concern prevents immediate reimplantation
    - carefully place tooth in suitable medium (saline/milk)

#### Immediate dental referral, injury documentation & discussion with pt important

- Most hospitals requires filing an incident report
- Reimbursement responsibility depends on hospital policy

### Burns

Intraoperative burns are rare; can be devastating/fatal

**Surgical Fire**

- 200 surgical fires per year in the United States
- Fire requires O<sub>2</sub>, flammable materials, & ignition source
  - O<sub>2</sub> commonly administered in OR (endotracheal, nasal cannula)
  - Flammable materials = surgical drapes, alcohol based prep solutions, plastic ETTs
  - Ignition sources = laser, electro-surgical units (ESUs), cautery
- Head & neck surgeries represent most cases involving fire in OR
  - Higher risk since nasal cannulas + laser/electrocautery → combustion
  - ETT carrying enriched O<sub>2</sub> can also ignite, leading to a "blowtorch" effect during positive pressure ventilation
- Airway fire
  - Prevention:
    - Low FiO<sub>2</sub> during lasering
    - Use of heliox
    - Use of fire resistant ETTs/wrap ETT in metal tape
    - Fill ETT cuff with saline, not air
  - Management:
    - Remove ETT/stop ventilation, discontinue O<sub>2</sub>
    - douse fire with saline/water, mask-ventilate pt
    - Perform bronchoscopy to assess airway damage

**Electrocautery/Electrosurgical Unit (ESU)**

- Current path: Electro-surgical pencil → through pt → out grounding pad
- Current density dissipated over large surface area → limits risk of burn (because of low impedance return electrode)
- ESU-associated burns:
  - Improper placement of return electrode (↓ contact surface area)
  - Fluids (blood, irrigation, skin prep) cause improper electrode contact
  - Avoid placement of return electrode over bony prominences
  - ESUs can serve as ignition source (esp if ↑ O<sub>2</sub> conc in use)

**Magnetic Resonance Imaging**

- Complications usually involve metallic objects flying into magnetic field & burns
- MRI radio frequency can cause heating of current conducting materials:
  - ECG cables & electrodes
    - Remove excess cables & avoid cable contact with skin
    - Do not loop cables, ensure ECG electrodes are firmly attached
  - Medicated patches
    - Some contain aluminized backing (can heat in MRI)
    - Avoid testosterone, nitro, nicotine, scopolamine, clonidine patches

**Perioperative Blindness****Summary of ASA Practice Advisory: Perioperative Visual Loss & Spine Surgery**

- A subset of spine surgery pts while prone have ↑ risk of perioperative visual loss including pts who are anticipated to have **substantial blood loss, or long surgeries**
- Inform high-risk pts of the small, unpredictable, risk of visual loss
- Deliberate hypotensive techniques not associated with visual loss
- Use colloids along with crystalloids to maintain volume in pts with significant blood loss
- No transfusion threshold known to eliminate risk of visual loss 2° to anemia
- High-risk pts should be positioned head level to or higher than heart
- Consider use of staged spine procedures in high-risk pts

Source: American Society for Anesthesiologists Task Force on Perioperative Blindness. Roth S, et al. Practice advisory for perioperative visual loss associated with spine surgery. *Anesthesiology* 2006;104:1319–1328.

# TRAUMA, BURN, AND CRITICAL CARE MANAGEMENT

DANIEL W. JOHNSON

ICU 15-1

## Airway Management for Trauma

### Intubation Indications

- Hypoxia, hypercarbia, trauma to the airway, severe shock, poor mental status (inability to protect airway/cooperate with procedures), severe head injury, inhalational injury

### Intubation Considerations

- Higher incidence of difficult airway with trauma
  - Facial and airway injuries
  - May need emergent surgical airway management (consider notifying surgeon)
- Intubation often occurs prior to determination of cervical spine stability
  - Must assume cervical spine instability during intubation (collar likely in place prior to airway management)
  - Mask ventilation: Chin-lift contraindicated, jaw-thrust maneuver acceptable
  - Assistant holds head firmly in neutral/stable position using two-handed manual in-line stabilization
    - Remove collar immediately following onset of neuromuscular blockade
      - Allows normal mouth opening for laryngoscopy
      - Improves ease of mask ventilation
      - Allows access to the neck (for surgical airway)
  - No evidence suggests superiority of one method of intubation over another
  - Many perform RSI followed by careful direct laryngoscopy
    - Obtain good intubating conditions in short amount of time
  - Consider awake/asleep fiberoptic intubation, induction with maintenance of spontaneous ventilation during laryngoscopy, use of a lighted stylet, early tracheostomy (especially in pts with facial/laryngeal injury)

### Induction Considerations

- Goal: Avoid hypotension (tissue at risk will tolerate ischemia poorly)
- All trauma pts considered "full stomach" (consider rapid sequence induction)
- Consider etomidate/ketamine for induction to minimize hypotension
- Patients *in extremis* may require little/no sedation for intubation
- If succinylcholine contraindicated (e.g., hyperkalemia), consider rocuronium 1.2 mg/kg IV
  - Succinylcholine **only acceptable** in acute spinal cord injury (first 24 hr)
  - After 24 hr, risk of hyperkalemic cardiac arrest rises

## Acute Spinal Cord Injury (ASCI)

### General Considerations

- Most injuries from fracture/dislocation of vertebral column
- Major causes of death in spinal cord injured patients are aspiration/shock

### Anesthesia Considerations

- Movement of patient: Must be performed carefully to maintain spine alignment
- Patient positioning: If turning prone, ensure endotracheal tube is secure
- Impairment of pulmonary mechanics: Pts with high spinal cord injuries, muscular control of ventilation may be impaired (can lead to atelectasis → decrease in FRC → V/Q mismatch)

### Significance of Spinal Cord Injury Site: (most common injury sites: C5–6, T12–L1)

C5	→ Impaired innervation of diaphragm/necessitates ventilatory support
C7	→ Reduced vital capacity/FEV <sub>1</sub> (up to 70%)
T1	→ Quadriplegia
T4	→ Bradycardia
T7	→ Reduced strength of accessory muscles of breathing
L4	→ Paraplegia

### NASCIS Protocol for Steroid Therapy

- Methylprednisolone commonly administered for blunt ASCI due to absence of other therapies
- Must be initiated within 8 hr of injury
- Methylprednisolone loading dose 30 mg/kg IV over 1 hr followed by:
  - 5.4 mg/kg/hr IV infusion for next 23 hr if started within 0–3 hr of injury (NASCIS II: *N Engl J Med* 1990;322:1405–1411)

- 5.4 mg/kg/hr IV infusion for 47 hr if started within 3–8 hr of injury (NASCIS III: JAMA 1997;277:1597–1604)
- No evidence for steroids in penetrating ASCI
- Unclear evidence in patients with documented complete thoracolumbar ASCI
- Consider administering stress ulcer & hyperglycemia prophylaxis during steroid therapy

#### Neurogenic Shock

- Triad of hypotension, bradycardia, hypothermia from functional sympathectomy (loss of vascular tone) and/or loss of cardiac inotropy/chronotropy secondary to high spinal cord injury
- More common in midthoracic injuries and higher
- In high cord injuries, loss of cardiac accelerator function & unopposed parasympathetic tone contribute to bradycardia (exacerbates impaired cardiac output)
  - Consider anticholinergics/ $\beta$ -agonists to increase heart rate
  - Consider  $\alpha$ -agonists to restore peripheral vascular tone & improve venous return

#### Spinal Shock

- Disruption of all cord function caudal to spinal cord injury
- Causes flaccid weakness/lack of spinal arc reflexes at and below injury

#### Autonomic Hyperreflexia

- Most common in injuries above T6, occurs weeks to months after the acute injury
- Results from loss of descending inhibitory impulses + sympathetic system overactivity
- Stimulation below injury level (bladder distention, surgical stimulation) causes:
  - Vasoconstriction/hypertension below injury
  - Reflex bradycardia & dysrhythmias
  - Vasodilation above injury
- Symptoms: Headaches, blurred vision, seizures, cerebral hemorrhage, pulmonary edema 2° to left heart failure, loss of consciousness, nasal stuffiness, cutaneous flushing
- Treatment:
  - Remove stimulus
  - Consider atropine for severe bradycardia
  - Hypertension treated with direct vasodilators (nitroprusside/nitroglycerin),  $\alpha$ -blockers (prazosin), ganglionic blockers (trimethaphan)
  - Consider using general anesthesia or spinal; epidural may not be as effective because of sacral sparing effect

### Intraoperative Trauma Management

Trauma Room Setup
Anesthesia machine on, fully checked & primed with 100% oxygen
OR table in correct position
Thermostat $\uparrow$ (ahead of time) to warm room
Airway equipment (including suction) available & difficult airway cart nearby
Standard monitors (including transducers)
Anesthetic & vasoactive drugs
Defibrillator available
Forced-air warming apparatus & blankets
IV tubing (must be primed); fluid warmer
Inflatable pressure bags or rapid infusion device
Supplies for large-bore IV, arterial, central access

Goal: Optimize pt stability while not delaying surgical control of bleeding clear communication must be maintained given chaotic environment

#### Maintenance of Anesthesia

- Maintenance options may include a combination of low-dose volatile agent/midazolam/fentanyl/ketamine/scopolamine (for amnesia in unstable pts)
- Volatile agent may cause hypotension in unstable pts
- Nitrous oxide often avoided due to potential pneumothorax/other air collections
- Trauma pts at high risk of intraoperative recall (consider EEG monitoring)

#### Management of Hypotension

- Ensure adequate cardiac preload (volume resuscitation)
- Vasopressors as necessary, consider other causes of shock:
  - Neurogenic shock (cord injury)
  - Cardiogenic shock (myocardial injury/existing heart disease)



- Obstructive shock (pericardial tamponade/tension pneumothorax, pulmonary emboli)
- Vasodilatory shock (sepsis/SIRS/anaphylaxis)

## Emergence/Transport

Preparation prior to transporting intubated/sedated patient to the ICU includes:

- Functioning bag valve mask + full oxygen tank
- Equipment for emergent mask ventilation/reintubation
- Emergency drugs (atropine, epinephrine, succinylcholine)
- Transport monitors (SpO<sub>2</sub>, ECG, blood pressure)
- Assistance to move the bed/gurney & secure elevators

Considerations during emergence:

- Coughing/bucking/severe hypertension may disrupt clots
- Airway may be edematous after volume resuscitation
  - Check for presence of an air leak around endotracheal tube
  - Ensure equipment for rapid reintubation available

## Fluid Resuscitation

Overall goal: Maintain perfusion to vital organs while surgeon controls hemorrhage

### Goals for Early Resuscitation of the Trauma Patient in Hemorrhagic Shock

- Maintain SBP 80–100 or MAP 60–70 (goal may be higher if ICP is elevated)
- Maintain Hb 7–9 (goal may be higher in brain injured patients/pts with comorbidities)
- Maintain normal INR & PTT
- Maintain platelets >50,000
- Maintain normal serum ionized Ca<sup>2+</sup>
- Maintain core temperature >35°C
- Prevent acidosis from worsening

Obtain adequate vascular access

Peripheral IVs (14-gauge catheters—can deliver up to 500 mL/min)

Peripheral 8.5 Fr Rapid Infusion Catheter (RIC—can deliver 850 mL/min)

9-Fr central venous catheter (can deliver 1000 mL/min)

(note: Multiport central venous catheters deliver <14 gauge IV)

Consider rapid infusion systems (Belmont/Level 1)/pressure bags

(avoid administration of IV air when giving fluids under pressure)

### Crystalloid Solutions

Advantage of crystalloid vs colloid is debatable in trauma resuscitation

Administer warm lactated Ringer's while blood is obtained

(hypothermia can cause failure of coagulation cascade)

Avoid excessive administration of crystalloid solutions

(causes dilution of platelets/coagulation factors, reversal of compensatory vasoconstriction, breakage of clots due to rapid volume expansion)

Transfusion therapy (see Chapter 9, on fluids, electrolytes, and transfusion therapy)

- **Packed red blood cells (PRBCs)**
  - Uncrossmatched blood (warm type O)—use if pt has unstable hemorrhagic shock
  - Type-specific blood—substitute for type O as soon as possible (minimize exposure to anti-A & anti-B antibodies in type O blood)
  - Transfusion rate depends on bleeding rate
  - Goal Hb 7–9 (unless brain injury/comorbidities)
- **Fresh frozen plasma (FFP)**
  - Patients receiving >4 units of PRBCs may require FFP
  - Consider transfusing 1 unit of FFP for every 4–6 units of PRBCs (exact ratio controversial)—also use INR/PTT/clinical picture as guide
  - ABO compatibility required, Rh compatibility not required
- **Platelets**
  - Platelet count >50,000 generally desirable
  - ABO compatibility not required, Rh compatibility not required
- **Adverse effects of massive transfusion**
  - Calcium depletion 2° to citrate chelation
  - Transfusion reaction (given cumulative risk of clerical errors)
  - Hyperkalemia (secondary to hemolysis in stored blood)
  - Transfusion-related immunomodulation (TRIM)
    - Transfusion-related acute lung injury (TRALI)
    - Volume overload/congestive heart failure (CHF)

## Monitoring During Fluid Resuscitation

- Arterial waveform variation with positive-pressure ventilation may be a marker of fluid responsiveness
- Urine output
- Serum markers of global tissue perfusion (base deficit, serum lactic acid, central/mixed venous O<sub>2</sub> saturation)

<b>Rhabdomyolysis</b>	
Definition	Acute disintegration of striated muscle
Causes	Trauma, crush injury, electrical shock, CPR, ischemia, arterial occlusion, compartment syndrome, DIC, burn injury, hypothermia, medications, & illicit drugs
Signs/symptoms	Acute myalgias/pigmenturia ↑ Serum ck, myoglobin, potassium, urea & phosphorus Arrhythmias (caused by excess potassium + hypocalcemia)
Consequence	Free myoglobin toxic to renal tubules → acute renal failure
Course	CK levels typically peak 2–5 days after initial insult Levels >16,000 U/L more likely to cause renal failure Hypocalcemia (from influx/deposition of Ca <sup>2+</sup> in damaged muscle) may occur
Therapy	Restore blood flow to ischemic areas IV fluids (maintain urine output 200 mL/hr until CK levels ↓) No evidence for mannitol/sodium bicarbonate Treat hypocalcemia if tetany/severe hyperkalemia develops Treat compartment syndrome if this develops Dialysis if fluid resuscitation fails to correct intractable hyperkalemia and/or acidosis

## Nutrition

Enteral feeding—preferred over parenteral nutrition (promotes maintenance of intestinal tissue)

### Total Parenteral Nutrition (TPN)

- Use only when adequate enteral feeding not possible
- Must be given centrally (due to ↑ osmolarity) via dedicated port (↓ risk of infection)
- Typical TPN formula: Carbohydrates 50–60%, proteins 15–25%, lipids 20–30%
- Insulin therapy/frequent glucose monitoring required to avoid hyperglycemia → insulin can be added to TPN solution
- Monitor labs weekly: Electrolytes, aminotransferases, alkaline phosphatase, bilirubin, triglycerides, cholesterol, prealbumin and transferrin
- Complications
  - Infection/sepsis, excessive CO<sub>2</sub> production, hepatic steatosis, hyperglycemia, hyperlipidemia, impaired immune function, electrolyte derangements, muscle weakness
- Intraoperative management
  - TPN should not be abruptly stopped without replacing carbohydrate source (can result in hypoglycemia)

### Necrotizing Fasciitis/Myonecrosis

Definitions	Deep infection that involves fascia & subcutaneous tissue; myonecrosis indicates muscle involvement
Signs/symptoms	Cellulitis, ↑ temp, lethargy, subcutaneous tissue has hard, “wooden” feel, pain out of proportion to exam; gas gangrene = severe fulminant clostridial myonecrosis (may also see crepitus)
Pathology	Group A streptococcal species, <i>Staph aureus</i> , anaerobic streptococcal species & bowel flora
Course	Infections can spread rapidly/cause systemic toxicity; mortality is high
Treatment	Early surgical debridement & broad-spectrum antibiotics

## Glycemic Control in ICU Patients

Some evidence that insulin infusions for aggressive glucose maintenance improves outcome  
 → Risk of tight glycemic control = hypoglycemia (may outweigh benefit)

## Burn Management

### Pathophysiology

- Skin destruction → impairs heat regulation, fluid/electrolyte maintenance, microbial protection
- Circulating mediators trigger systemic inflammatory response
  - Hypermetabolism, immune suppression & alteration in cell membrane permeability
  - Massive fluid shifts from vascular compartment to burned tissue
  - Edema occurs in burned tissue as well as in unaffected tissues
  - Fluid loss from intravascular space can cause hypovolemic shock

### Initial Evaluation and Management

Depth of burn injury—1st, 2nd, & 3rd degree classification system replaced by:

- Partial-thickness burn: Destruction of epidermis & portion of dermis (blanches with pressure & still has pain sensation)
  - Superficial partial thickness → confined to upper third of dermis
  - Mid partial thickness → involves middle third of dermis
  - Deep partial thickness → leaves only a portion of dermis viable
- Full-thickness burn: No dermis is viable (will not blanch with pressure & is insensitive to pain)

### Total body surface area (TBSA)

- “Rule of nines” estimates surface area burned in adult patients
  - Head & each upper extremity represent = 9% TBSA
  - Anterior trunk, posterior trunk, & each lower extremity = 18% TBSA
  - Less accurate in children, due to different bodily proportions (see diagram)

### Airway and Respiratory Management

Deliver maximal  $\text{FiO}_2$  during initial resuscitation major burn victims

#### Inspiration of hot gases

→ Can cause direct airway damage/obstruction from edema

#### Tracheal intubation usually indicated

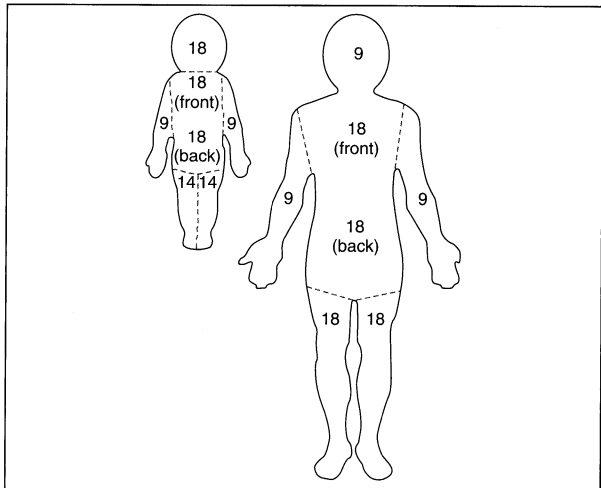
→ Perform before airway edema renders this impossible

#### Suspect inhalation injury when head/neck involved

(singd nasal hairs, swelling of nose, mouth, lips, throat; cough productive of soot)

Chest wall expansion may be ↓ in major chest burns → consider emergency escharotomies

Figure 15-1 Rule of nines (Courtesy of J. Ehrenfeld, MD)



**Cardiovascular and Fluid Management**

- Parkland formula: 4 mL of lactated Ringer's per kg per % TBSA burn in initial 24 hr
  - Half of calculated fluid given during 1st 8 hr postburn, rest over next 16 hr (example: 70-kg man with 60% TBSA burn needs:  $4 \times 70 \times 60 = 16,800$  mL; give 8400 mL of LR during 0–8 hr after burn, 8400 mL during 8–24 hr)
  - Patient's daily maintenance fluid should be given concurrently
- Cardiac output reduced immediately postburn ( $\downarrow$  circulating vol + direct myocardial depression)
  - 3–5 days postburn, hypermetabolic state  $\rightarrow$   $\uparrow$  cardiac output; ( $2\text{--}3 \times$  normal),  $\downarrow$  SVR

**Intraoperative Management: General Considerations**

**Airway management:** Often challenging  $\rightarrow$  from edema/deformity of normal anatomy  
 NG tube should be placed early (pts often develop ileus after burn)

**Monitors:** Must have temp monitoring, consider CVP/PA line

**Resuscitation:** Correct acid-base/electrolyte abnl, address coagulopathies; expect large volume blood loss during excision & grafting (order adequate colloid/blood products in advance) (obtain IV access for large volume resuscitation)

**Warming modalities:** Cover patient,  $\uparrow$  OR temp, warm all fluids, use warming blankets; heat often lost during pt transport to & from OR

**Anesthesia for Burn Surgery**

**Relaxants:** Depolarizing agent succinylcholine is dangerous after initial 12–24 hr (potential for profound hyperkalemia & subsequent cardiac arrest)

**Induction:** Burn pts exhibit  $\downarrow$  response nondepolarizing agents, need  $\uparrow$  dose Consider ketamine & etomidate in pts with uncertain CV/volume status

**Maintenance:** After early course (during which  $\uparrow$  FiO<sub>2</sub> desirable), nitrous oxide may be added

**Analgesia:**  $\uparrow$  Opioid requirements due to tolerance &  $\uparrow$  in volume of distribution

<b>Bioterrorism and Chemical Warfare Agents</b>			
<b>Potential Agent</b>	<b>Effect</b>	<b>Treatment</b>	<b>Staff Protection</b>
Botulinum	Paralysis (symmetric descending weakness)	Trivalent botulism antitoxin or botulism immune globulin	Universal precautions
Nerve agents (Sarin, VX)	Cholinergic crisis	Atropine repeated q5–10min and pralidoxime followed by infusion	Level C chemical protection suits & filtration breathing apparatus
Hydrogen cyanide	Inhibits aerobic respiration; metabolic acidosis	Na thiosulfate, Na nitrite, hydroxycobalamin	Level C chemical protection suits & filtration breathing apparatus
<i>Bacillus anthracis</i>	Mediastinitis, meningitis, multiorgan failure	Ciprofloxacin, doxycycline, penicillin, and streptomycin	Isolation, vaccination
Variola virus (smallpox)	Rash, pneumonia	Cidofovir	Isolation, vaccination with vaccinia immunoglobulin

**Systemic Inflammatory Response Syndrome (SIRS)/Sepsis**

SIRS: Common causes include surgery, major trauma, pancreatitis/transfusion reaction

<b>Criteria for SIRS (two or more must be present)</b>		
Fever $>38^\circ$	OR	Hypothermia $<36^\circ$
Tachypnea $>20$ breaths/min		
Hypocarbica with PaCO <sub>2</sub> $<32$ mmHg (with spontaneous breathing)	OR	Requirement for mechanical ventilation
Heart rate $>90$		
Leukocytosis $>12K$	OR	Leukopenia $<4K$ OR greater than 10% band forms

**Criteria for Sepsis**

Sepsis	SIRS + infection
Severe sepsis	Sepsis + hypotension/hypoperfusion & acute organ dysfunction
Septic shock	Severe sepsis + hypotension that does not respond to fluids
Refractory septic shock	Hypotension persists >1 hr despite fluid resuscitation

**Acute Physiology and Chronic Health Evaluation (APACHE) II Score**

Most commonly used ICU illness scoring system: ↑ Score = ↑ disease severity/risk of death

Useful in initial evaluation of patients with severe sepsis

Calculated based on: Temp, MAP, heart rate, respiratory rate, P(A-a)O<sub>2</sub> or PaO<sub>2</sub> (depending on FiO<sub>2</sub>), arterial pH, sodium, potassium, creatinine, hematocrit, WBC count, GCS score, HCO<sub>3</sub>, age, and chronic health status

Several web-based calculators available (<http://www.sfar.org/scores2/apache22.html>)

**Associated Problems in Sepsis**

Shock (from vasodilation, intravascular depletion, myocardial depression)

Respiratory failure (from endothelial injury → alveolar leak & impaired oxygenation)

Acute lung injury (ALI)/Acute respiratory distress syndrome (ARDS)

Renal failure/metabolic acidosis

Disseminated intravascular coagulopathy (DIC)

Multiple organ dysfunction syndrome

**Early Goal-Directed Therapy (EGDT)** (*N Engl J Med* 2001;345:1368–1377)

Strategy to ↓ mortality in pts with severe sepsis/septic shock

(Overall goal: Optimize parameters that match oxygen demand & oxygen delivery)

Specific goals include:

1. CVP 8–12 mm Hg (maintained by crystalloid boluses)
2. MAP 65–90 mmHg (maintained by vasopressors or vasodilators)
3. Urine output >0.5 mL/kg/hr
4. Maintain central venous oxygen saturation >70%

**Steroids in Sepsis**

- Hydrocortisone—no improved survival/reversal of shock in pts with septic shock either overall or in pts who did not have a response to corticotropin  
→ Hydrocortisone hastened reversal of shock in pts in whom shock was reversed (*N Engl J Med* 2008;358(2):111–124)
- Earlier study showed pts in septic shock may benefit from hydrocortisone 50 mg IV q6hr & fludrocortisone 50 mcg enterally daily (*JAMA* 2002;288:862–871)
- Subsequent large trials have not confirmed this benefit  
→ Recommend: Consider steroids for refractory septic shock not responsive to vasopressors

**Markers of Resuscitation in Sepsis**

Continuous central or mixed venous oxygen saturation monitoring

Inadequate tissue perfusion → rise in oxygen extraction

→ Lower than normal O<sub>2</sub> level in blood returning to right heart

Lactic acid level or base deficit

**Recombinant Activated Protein C (Xigris)**

- May ↓ mortality in pts with severe sepsis at high risk of death (↑ APACHE II score)
- Common adverse reaction = bleeding
- Dosing = 24 mcg/kg/h IV continuous infusion for 96 hr
- Contraindications: Active bleeding, hemorrhagic stroke within 3 mo, severe head trauma/intracranial surgery/intraspinal surgery within 2 mo, intracranial neoplasm or mass lesion, evidence of cerebral herniation, presence of an epidural or spinal catheter, or trauma with risk of life-threatening bleeding

Surviving sepsis campaign: Intl. Guidelines for Management of Severe Sepsis & Septic Shock, 2008. (Tables reproduced with permission from *Crit Care Med* 2008;36(1):296–327.)

**Initial Resuscitation and Infection Issues**

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline.

- Indicates a strong recommendation or “we recommend”
- Indicates a weak recommendation, or “we suggest”

*Initial resuscitation (first 6 hours)*

- Begin resuscitation immediately in patients with hypotension or elevated serum lactate >4 mmol/L; do not delay pending ICU admission (1C)
- Resuscitation goals: (1C)
  - CVP 8–12 mm Hg<sup>a</sup>
  - Mean arterial pressure ≥65 mm Hg
  - Urine output ≥0.5 mL·kg<sup>-1</sup>·hr<sup>-1</sup>
  - Central venous (superior vena cava) oxygen saturation ≥70%, or mixed venous ≥65%
- If venous oxygen saturation target is not achieved (2C)
  - Consider further fluid
  - Transfuse packed red blood cells if required to hematocrit of ≥30% and/or
  - Start dobutamine infusion maximum 20 μg·kg<sup>-1</sup>·min<sup>-1</sup>

*Diagnosis*

- Obtain appropriate cultures before starting antibiotics provided this does not significantly delay antimicrobial administration (1C)
  - Obtain two or more BCs
  - One or more BCs should be percutaneous
  - One BC from each vascular access device in place >48 hrs
  - Culture other sites as clinically indicated
- Perform imaging studies promptly to confirm and sample any source of infection; if safe to do so (1C)

*Antibiotic therapy*

- Begin intravenous antibiotics as early as possible and always within the first hour of recognizing severe sepsis (1D) and septic shock (1B)
- Broad-spectrum: One or more agents active against likely bacterial/fungal pathogens and with good penetration into presumed source (1B)
- Reassess antimicrobial regimen daily to optimize efficacy, prevent resistance, avoid toxicity & minimize costs (1C)
  - Consider combination therapy in *Pseudomonas* infections (2D)
  - Consider combination empiric therapy in neutropenic patients (2D)
  - Combination therapy ≤3–5 days and de-escalation following susceptibilities (2D)
- Duration of therapy typically limited to 7–10 days: Longer if response slow or there are undrainable foci of infection or immunologic deficiencies (1D)
- Stop antimicrobial therapy if cause is found to be noninfectious (1D)

*Source identification and control*

- A specific anatomic site of infection should be established as rapidly as possible (1C) and within first 6 hrs of presentation (1D)
- Formally evaluate patient for a focus of infection amenable to source control measures (e.g., abscess drainage, tissue debridement) (1C)
- Implement source control measures as soon as possible following successful initial resuscitation (1C) (exception: Infected pancreatic necrosis, where surgical intervention is best delayed) (2B)
- Choose source control measure with maximum efficacy and minimal physiologic upset (1D)
- Remove intravascular access devices if potentially infected (1C)

GRADE, Grades of Recommendation, Assessment, Development and Evaluation: ICU, intensive care unit; CVP, central venous pressure; BC, blood culture.

<sup>a</sup>A higher target CVP of 12–15 mm Hg is recommended in the presence of mechanical ventilation or preexisting decreased ventricular compliance.

**Hemodynamic Support and Adjunctive Therapy**

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline.

- Indicates a strong recommendation or "we recommend"
- indicates a weak recommendation or "we suggest"

**Fluid therapy**

- Fluid-resuscitate using crystalloids or colloids (1B)
- Target a CVP of  $\geq 8$  mm Hg ( $\geq 12$  mm Hg if mechanically ventilated) (1C)
- Use a fluid challenge technique while associated with a hemodynamic improvement (1D)
- Give fluid challenges of 1000 mL of crystalloids or 300–500 mL of colloids over 30 min. More rapid and larger volumes may be required in sepsis-induced tissue hypoperfusion (1D)
- Rate of fluid administration should be reduced if cardiac filling pressures increase without concurrent hemodynamic improvement (1D)

**Vasopressors**

- Maintain MAP  $\geq 65$  mm Hg (1C)
- Norepinephrine and dopamine centrally administered are the initial vasopressors of choice (1C)
- Epinephrine, phenylephrine, or vasopressin should not be administered as the initial vasopressor in septic shock (2C). Vasopressin 0.03 units/min may be subsequently added to norepinephrine with anticipation of an effect equivalent to norepinephrine alone.
- Use epinephrine as the first alternative agent in septic shock when blood pressure is poorly responsive to norepinephrine or dopamine (2B)
- Do not use low-dose dopamine for renal protection (1A)
- In patients requiring vasopressors, insert an arterial catheter as soon as practical (1D)

**Inotropic therapy**

- Use dobutamine in patients with myocardial dysfunction as supported by elevated cardiac filling pressures and low cardiac output (1C)
- Do not increase cardiac index to predetermined supranormal levels (1B)

**Steroids**

- Consider intravenous hydrocortisone for adult septic shock when hypotension responds poorly to adequate fluid resuscitation and vasopressors (2C)
- ACTH stimulation test is not recommended to identify the subset of adults with septic shock who should receive hydrocortisone (2B)
- Hydrocortisone is preferred to dexamethasone (2B)
- Fludrocortisone (50  $\mu$ g orally once a day) may be included if an alternative to hydrocortisone is being used that lacks significant mineralocorticoid activity. Fludrocortisone if optional if hydrocortisone is used (2C)
- Steroid therapy may be weaned once vasopressors are no longer required (2D)
- Hydrocortisone dose should be  $\leq 300$  mg/day (1A)
- Do not use corticosteroids to treat sepsis in the absence of shock unless the patient's endocrine or corticosteroid history warrants it (1D)

**Recombinant human activated protein C**

- Consider rhAPC in adult patients with sepsis-induced organ dysfunction with clinical assessment of high risk of death (typically APACHE II  $\geq 25$  or multiple organ failure) if there are no contraindications (2B, 2C for postoperative patients)
- Adult patients with severe sepsis and low risk of death (typically, APACHE II  $< 20$  or one organ failure) should not receive rhAPC (1A)

**Other Supportive Therapy of Severe Sepsis**

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline.

- Indicates a strong recommendation, or "we recommend"
- Indicates a weak recommendation, or "we suggest"

**Blood product administration**

- Give red blood cells when hemoglobin decreases to  $<7.0$  g/dl ( $<70$  g/L) to target a hemoglobin of 7.0–9.0 g/dL in adults (1B). A higher hemoglobin level may be required in special circumstances (e.g., myocardial ischemia, severe hypoxemia, acute hemorrhage, cyanotic heart disease, or lactic acidosis)
- Do not use erythropoietin to treat sepsis-related anemia. Erythropoietin may be used for other accepted reasons (1B)
- Do not use fresh frozen plasma to correct laboratory clotting abnormalities unless there is bleeding or planned invasive procedures (2D)
- Do not use antithrombin therapy (1B)
- Administer platelets when (2D)  
 Counts are  $<5000/\text{mm}^3$  ( $5 \times 10^9/\text{L}$ ) regardless of bleeding  
 Counts are  $5000\text{--}30,000/\text{mm}^3$  ( $5\text{--}30 \times 10^9/\text{L}$ ) and there is significant bleeding risk.  
 Higher platelet counts ( $\geq 50,000/\text{mm}^3$  ( $50 \times 10^9/\text{L}$ )) are required for surgery or invasive procedures

**Mechanical ventilation of sepsis-induced ALI/ARDS**

- Target a tidal volume of 6mL/kg (predicted) body weight in patients with ALI/ARDS (1B)
- Target an initial upper limit plateau pressure  $\leq 30$  cm H<sub>2</sub>O. Consider chest wall compliance when assessing plateau pressure (1C)
- Allow PaCO<sub>2</sub> to increase above normal, if needed, to minimize plateau pressures and tidal volumes (1C)
- Set PEEP to avoid extensive lung collapse at end-expiration (1C)
- Consider using the prone position for ARDS patients requiring potentially injurious levels of FiO<sub>2</sub> or plateau pressure, provided they are not put at risk from positional changes (2C)
- Maintain mechanically ventilated patients in a semirecumbent position (head of the bed raised to 45°) unless contraindicated (1B), between 30° and 45° (2C)
- Noninvasive ventilation may be considered in the minority of ALI/ARDS patients with mild to moderate hypoxemic respiratory failure. The patients need to be hemodynamically stable, comfortable, easily arousable, able to protect/clear their airway and expected to recover rapidly (2B)
- Use a weaning protocol and an SBT regularly to evaluate the potential for discontinuing mechanical ventilation (1A)
- SBT options include a low level of pressure support with continuous positive airway pressure 5 cm H<sub>2</sub>O or a T piece
- Before the SBT, patients should:
  - be arousable
  - be hemodynamically stable without vasopressors
  - have no new potentially serious conditions
  - have low ventilatory and end-expiratory pressure requirement
  - require FiO<sub>2</sub> levels that can be safely delivered with a face mask or nasal cannula
- Do not use a pulmonary artery catheter for the routine monitoring of patients with ALI/ARDS (1A)
- Use a conservative fluid strategy for patients with established ALI who do not have evidence of tissue hypoperfusion (1C)

**Sedation, analgesia, and neuromuscular blockade in sepsis**

- Use sedation protocols with a sedation goal for critically ill mechanically ventilated patients (1B)
- Use either intermittent bolus sedation or continuous infusion sedation to predetermined end points (sedation scales), with daily interruption/lightening to produce awakening. Re-titrate if necessary (1B)
- Avoid neuromuscular blockers where possible. Monitor depth of block with train-of-four when using continuous infusions (1B)

(continued)



**Other Supportive Therapy of Severe Sepsis (Continued)***Glucose control*

- Use intravenous insulin to control hyperglycemia in patients with severe sepsis following stabilization in the ICU (1B)
- Aim to keep blood glucose <150 mg/dL (8.3 mmol/L) using a validated protocol for insulin dose adjustment (2C)
- Provide a glucose calorie source and monitor blood glucose values every 1–2 hrs (4 hrs when stable) in patients receiving intravenous insulin (1C)
- Interpret with caution low glucose levels obtained with point of care testing, as these techniques may overestimate arterial blood or plasma glucose values (1B)

*Renal replacement*

- Intermittent hemodialysis and CVVH are considered equivalent (2B)
- CVVH offers easier management in hemodynamically unstable patients (2D)

*Bicarbonate therapy*

- Do not use bicarbonate therapy for the purpose of improving hemodynamics or reducing vasopressor requirements when treating hypoperfusion-induced lactic acidemia with pH  $\geq 7.15$  (1B)

*Deep vein thrombosis prophylaxis*

- Use either low-dose UFH or LMWH, unless contraindicated (1A)
- Use a mechanical prophylactic device, such as compression stockings or an intermittent compression device, when heparin is contraindicated (1A)
- Use a combination of pharmacologic and mechanical therapy for patients who are at very high risk for deep vein thrombosis (2C)
- In patients at very high risk, LMWH should be used rather than UFH (2C)

*Stress ulcer prophylaxis*

- Provide stress ulcer prophylaxis using H2 blocker (1A) or proton pump inhibitor (1B). Benefits of prevention of upper gastrointestinal bleed must be weighted against the potential for development of ventilator-acquired pneumonia

*Consideration for limitation of support*

- Discuss advance care planning with patients and families. Describe likely outcomes and set realistic expectations (ID)

GRADE, Grades of Recommendation, Assessment, Development and Evaluation; ALI, acute lung injury; ARDS, acute respiratory distress syndrome; PEEP, positive end-expiratory pressure; SBT, spontaneous breathing trial; ICU, intensive care unit; CVVH, continuous veno-venous hemofiltration; UFH, unfractionated heparin; LMWH, low-molecular weight heparin.

ICU MEDICATIONS			
Drug	Class	Dose	
		Per kg	Average
<b>Pressors, Inotropes, and Chronotropes</b>			
Phenylephrine	$\alpha_1$	10–300 mcg/min	
Norepinephrine	$\alpha_1 > \beta_1$	1–40 mcg/min	
Vasopressin	$V_1$	0.01–0.1 U/min (usually <0.04)	
Epinephrine	$\alpha_1, \alpha_2, \beta_1, \beta_2$	2–20 mcg/min	
Isoproterenol	$\beta_1, \beta_2$	0.1–10 mcg/min	
Dopamine	D	0.5–2 mcg/kg/min	50–200 mcg/min
	$\beta, D$	2–10 mcg/kg/min	200–500 mcg/min
	$\alpha, \beta, D$	>10 mcg/kg/min	500–1000 mcg/min
Dobutamine	$\beta_1 > \beta_2$	2–20 mcg/kg/min	50–1000 mcg/min
Milrinone	PDE inhibitors	50 mcg/kg over 10 min then 0.375–0.75 mcg/kg/min	3–4 mg over 10 min then 20–50 mcg/min
	PDE inhibitors	0.75 mg/kg over 3 min then 5–15 mcg/kg/min	40–50 mg over 3 min then 250–900 mcg/min
Inamrinone	PDE inhibitors	0.75 mg/kg over 3 min then 5–15 mcg/kg/min	40–50 mg over 3 min then 250–900 mcg/min
<b>Vasodilators</b>			
Nitroglycerin	NO	10–1000 mcg/min	
Nitroprusside	NO	0.1–10 mcg/kg/min	5–800 mcg/min
Nesiritide	BNP	2 mcg/kg IVB then 0.01 mcg/kg/min	
Labetalol	$\alpha_1, \beta_1,$ and $\beta_2$ blocker	20 mg over 2 min then 20–80 mg q10min or 10–120 mg/h	
Fenoldopam	D	0.1–1.6 mcg/kg/min	10–120 mcg/min
Epoprostenol	Vasodilator	2–20 ng/kg/min	
Enalapril	ACE	0.625–2.5 mg over 5 min then 0.625–5 mg q6h	
Hydralazine	Vasodilator	5–20 mg q20–30min	
<b>Antiarrhythmics</b>			
Amiodarone	K (Class III)	150 mg over 10 min, then 1 mg/min $\times$ 6h, then 0.5 mg/min $\times$ 18h	
Lidocaine	Na channel (Class IB)	1–1.5 mg/kg then	100 mg then
		1–4 mg/min	1–4 mg/min
Procainamide	Na channel (Class IA)	17 mg/kg over 60 min	1 g over 60 min then
		then 1–4 mg/min	1–4 mg/min
Ibutilide	K channel (Class III)	1 mg over 10 min, may repeat $\times$ 1	
Propranolol	$\beta$ blocker	0.5–1 mg q5min then 1–10 mg/h	
Esmolol	$\beta_1 > \beta_2$ blocker	500 mcg/kg then 25–300 mcg/kg/min	20–40 mg over 1 min then 2–20 mg/min
Verapamil	CCB	2.5–5 mg over 1–2 min repeat 5–10 mg in 15–30 min prn 5–20 mg/h	
Diltiazem	CCB	0.25 mg/kg over 2 min reload 0.35 mg/kg $\times$ 1 prn then 5–15 mg/h	20 mg over 2 min reload 25 mg $\times$ 1 prn then 5–15 mg/h
Adenosine	Purinergic	6 mg rapid push if no response: 12 mg $\rightarrow$ 12–18 mg	

# ANESTHESIA FOR CARDIAC SURGERY

AMANDA RHEE • LINDA SHORE-LESSERSON

## NORMAL CARDIOVASCULAR PHYSIOLOGY

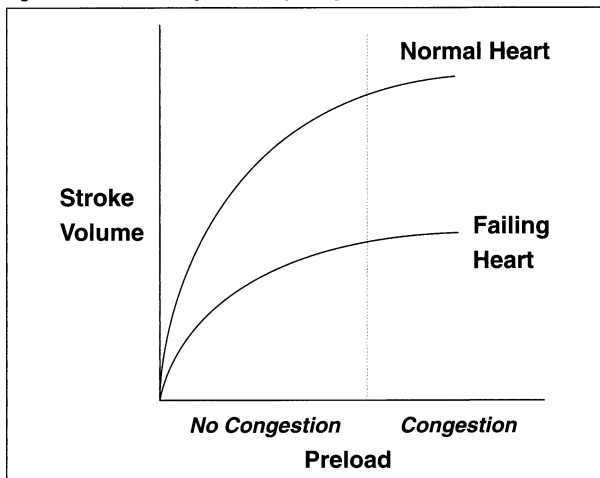
CARDIAC 16-1

Coronary Artery Anatomy			
Major Vessel	Branch 1	Branch 2	Supply
Left main coronary artery	Left anterior descending (LAD) coronary artery Circumflex artery	Septal branches	Anterior 2/3 of interventricular septum, apical anterior papillary muscle
		Diagonal branches Obtuse marginal branches	Anterior surface of left ventricle  Lateral & posterior wall of left ventricle, anterolateral papillary muscle; <i>Left dominant</i> : gives rise to posterior descending artery (PDA) which supplies inferior & posterior ventricles, posterior 1/3 of interventricular septum
Right main coronary artery	Acute marginal branches		Right ventricle
	AV nodal artery		AV node
	Usually SA nodal artery Posterior descending artery ( <i>Right dominant</i> )		SA node Inferior & posterior ventricles, posterior 1/3 of interventricular septum, posteromedial papillary muscle

### Cardiac Cycle: Definitions & Equations

- **Systole** = isovolumic ventricular contraction & ejection
- **Diastole** = isovolumic ventricular relaxation & filling
- **Cardiac output** = stroke volume × heart rate  
→ volume of blood pumped by each ventricle per minute

Figure 16-1 Frank-Starling relationship (Image Courtesy of J. Ehrenfeld)



- **Stroke volume** = amount of blood pumped out of each ventricle with each contraction
- **Cardiac reserve** = diff between cardiac output at rest & the max volume of blood the heart is capable of pumping per min.
- **Preload** = volume of blood in ventricle before systole, used to estimate left ventricular end diastolic volume (LVEDP)
- **Starling's law** = contractility depends on muscle fiber length
- **Afterload** = resistance to ejection of blood by each ventricle
- **Coronary perfusion pressure (CPP)** = aortic diastolic BP - LVEDP
- **Left ventricular wall tension** = (pressure)  $\times$  (radius/2)  $\times$  (thickness)
- **Fick eqn:**  
Cardiac output (C.O.) =  $O_2$  consumption / ([arterial  $O_2$  content] - [venous  $O_2$  content])

To Calculate Variables of Cardiovascular Function		
Variable	Formula	Normal Value With Units
Cardiac index	C.O./BSA	2.8–4.2 L/min/m <sup>2</sup>
SVR (TPR)	[(MAP - CVP) $\times$ 80]/C.O.	1200–1500 dyn*s/cm <sup>5</sup>
PVR	[(MPAP - PCWP) $\times$ 80]/C.O.	100–300 dyn*s/cm <sup>5</sup>
SV	(C.O. $\times$ 1000)/HR	60–90 mL/beat
SI	SV/BSA	20–65 mL/beat/m <sup>2</sup>
LV stroke work index	0.0136 (MAP - PCWP) $\times$ SI	46–60 g $\times$ m/beat/m <sup>2</sup>
RV stroke work index	0.0136 (MPAP - CVP) $\times$ SI	30–65 g $\times$ m/beat/m <sup>2</sup>
MAP	DBP + (SBP - DBP)/3	50–70 mmHg

### COMMON DISEASE STATES AFFECTING THE HEART

Coronary Artery Disease & Acute Coronary Syndromes	
<b>Coronary artery dz</b>	Atherosclerotic narrowing of one or more coronary arteries
<b>Ischemic heart dz</b>	Coronary blood flow does not meet requirements of myocardial $O_2$ demand
<b>Acute coronary syndrome</b>	Life-threatening conditions that result from CAD or coronary spasm (unstable angina, acute myocardial infarction, coronary emboli)
<i>Risk factors:</i>	Cigarette smoking, hypercholesterolemia, HTN, diabetes, family hx
<b>Angina pectoris</b>	Chest discomfort resulting from myocardial ischemia
<b>Stable angina</b>	Chronic angina pectoris brought on by exertion & relieved by rest (can get temporary ST seg $\downarrow$ without myocardial damage)
<b>Unstable angina</b>	$\uparrow$ Duration & frequency of angina pectoris produced by less exertion or at rest (myocardial infarction likely to result if left untreated)
<b>Variant angina</b>	Anginal discomfort at rest; results from coronary artery vasospasm (ST changes can occur, usually elevation)
<b>Non-ST-segment elevation MI (NSTEMI)</b>	Partially occlusive thrombus without myocardial necrosis
<b>ST-segment elevation MI (STEMI)</b>	Coronary thrombus completely obstructs coronary artery, causing necrosis

### Determinants of Myocardial Perfusion

**Supply:** Coronary perfusion pressure, HR, PaO<sub>2</sub>, coronary artery diameter

**Demand:** Myocardial O<sub>2</sub> consumption, HR, LV wall tension, contractility, conduction, relaxation

Treatment of Coronary Artery Disease & Acute Coronary Syndromes	
<b>Stable angina</b>	Sublingual nitroglycerin
<b>Unstable anginal/NSTEMI</b>	<b>MONA</b> —morphine, O <sub>2</sub> , nitroglycerine, ASA
	Medical therapy—nitroglycerine, $\beta$ -blockade, clopidogrel, heparin (UFH or LMWH), glycoprotein IIb/IIIa inhibitor
	Adjunctive therapy—ACE inhibitors, ARBs, HMG-CoA reductase inhibitors (statins)
	Percutaneous coronary intervention (PCI) for shock within 48 hr

## Treatment of Coronary Artery Disease & Acute Coronary Syndromes (Continued)

<b>STEMI</b>	<b>MONA</b> —morphine, <b>O</b> <sub>2</sub> , nitroglycerine, <b>ASA</b>						
	Medical therapy—thienopyridines (clopidogrel), heparin (UFH or LMWH), beta-adrenergic receptor blockers						
	Adjunctive therapy: ACE inhibitors, ARBs, HMG-CoA reductase inhibitors (statins)						
	<table border="0"> <tr> <td>If &lt; 12 hr since event:</td> <td>If &gt; 12 hr since event:</td> </tr> <tr> <td><b>Door to needle</b> (fibrinolysis with tPA) goal within 30 min</td> <td>PCI &amp; revascularization goal within 48 hr</td> </tr> <tr> <td><b>Door to balloon (PCI)</b> goal within 90 min</td> <td></td> </tr> </table>	If < 12 hr since event:	If > 12 hr since event:	<b>Door to needle</b> (fibrinolysis with tPA) goal within 30 min	PCI & revascularization goal within 48 hr	<b>Door to balloon (PCI)</b> goal within 90 min	
If < 12 hr since event:	If > 12 hr since event:						
<b>Door to needle</b> (fibrinolysis with tPA) goal within 30 min	PCI & revascularization goal within 48 hr						
<b>Door to balloon (PCI)</b> goal within 90 min							
<b>PCIs</b>	Persistent anginal episodes; significant stenosis of 1–2 coronary arteries or lower-risk pts with 3-vessel dz; favorable anatomy						
<b>CABG</b>	>50% stenosis of left main coronary artery; 2–3 vessel dz with reduced LV contractile fx or diabetes						

### Hypertension (HTN)

- Definition: >140/90 or 130/80 in high-risk pts
- Essential HTN (1° HTN)—no definable cause (95% of pts)
- 2° HTN: iatrogenic (meds), renal, aortic coarctation, pheochromocytoma, adrenocortical hormone excess, thyroid hormone abnormal, estrogen therapy, Cushing's disease
- Consequences of HTN
  - Organ damage: Ventricular hypertrophy, systolic dysfunction, CAD, stroke, abd aortic aneurysm, aortic dissection
  - Hypertensive crises: HTN encephalopathy—headache, blurred vision, confusion, somnolence, coma
- Treatment: Diuretics, sympatholytic agents ( $\beta$ -blockers/ $\alpha$ -2 agonists/ $\alpha$ -1 antagonists), vasodilators, (Ca channel blockers, ACE inhibitors, ARBs), nitrates
- Anesthetic considerations
  1. Monitoring: BP cuff versus arterial line as indicated
  2. Goal: keep BP within 20% of baseline

### Valvular Disease

#### Mitral Stenosis

Causes: Rheumatic fever, congenital stenosis

#### Pathophysiology

- $\uparrow$  LA pressure  $\rightarrow$  pulmonary edema, LV hypertrophy
- Atrial fibrillation may result from LA dilation
- Develop pulmonary HTN
- Atrial kick provides 40% of LV filling
- Stroke volume is fixed

#### Clinical feature

- High-pitched "opening snap" followed by low-frequency diastolic rumble

#### Classification

- Mild = valve area of  $\leq 2$  cm<sup>2</sup>; Critical = valve area  $\leq 1$  cm<sup>2</sup>

#### Treatment

- Medical therapy; balloon mitral valvuloplasty; open mitral commissurotomy; mitral valve replacement

#### Anesthetic management

- Maintain sinus rhythm (atrial kick provides 40% vent filling)
- Maintain preload & SV to avoid drop in SVR
- Maintain normal HR (to allow time for filling)
- Prevent  $\uparrow$  in PVR (avoid hypoxia, hypercarbia, acidosis)

#### Mitral Regurgitation

Causes: Mitral valve prolapse, ischemic heart dz, endocarditis, ruptured chordae, rheumatic heart dz, hypertrophic cardiomyopathy, LV enlargement, mitral annulus abnormal

#### Pathophysiology

- Severity determined by
  1. Systolic pressure gradient between LV and LA
  2. Systemic vascular resistance opposing forward LV blood flow
  3. Left atrial compliance
  4. Duration of regurgitation with each systole

• **Regurgitant fraction** = volume of MR/total LV stroke volume ( $>0.6$  = severe)

• **Acute MR:**  $\uparrow$  pulmonary pressure & pulmonary congestion

• **Chronic MR:**  $\uparrow$  LA size & compliance

Clinical features

• Apical holosystolic murmur radiating to axilla

Treatment

• Medical therapy; mitral valve repair/replacement

Anesthetic management

• Maintain HR normal or high

• Avoid myocardial depression

• Avoid  $\uparrow$  SVR (can worsen regurgitation)

• Initiate prophylaxis against endocarditis

• PA catheter v-waves increase as regurgitant fraction increases

Aortic Stenosis

Causes: Bicuspid AV, rheumatic fever, senile degenerative disease

Risk factors: Male gender, hypercholesterolemia, smoking

Pathophysiology

• Blood flow across valve is obstructed during systole

• Concentric LV hypertrophy

• Dependence on atrial kick to fill stiff ventricle

• Develop fixed stroke volume

• Compression of subendocardial vessels  $\rightarrow$  ischemia

Symptoms & severity

• Angina—median survival 5 years

• Syncope—median survival 3 years

• Congestive heart failure—median survival 2 years

Clinical features

• Harsh, holosystolic, crescendo-decrescendo murmur

Classification

• Mild = valve area  $<2.5$  cm<sup>2</sup>, Moderate =  $0.7$ – $1.2$  cm<sup>2</sup>, Critical  $<0.7$  cm<sup>2</sup>

Treatment

• Percutaneous balloon valvuloplasty, aortic valve replacement

Anesthetic management

• Maintain sinus rhythm (atrial kick provides 40% of preload)

• Maintain HR slow to normal (allow time for ventricular filling)

• Avoid  $\downarrow$  SVR (will  $\downarrow$  CO because of fixed SV)

$\rightarrow$  because of this, severe AS is a relative contraindication to spinal anesthesia

• Initiate prophylaxis against endocarditis

• Avoid myocardial depression as stroke volume is fixed

• Consider a-line placement for severe AS

• Consider percutaneous pacing capability in case of cardiac arrest (chest compressions usually ineffective)

Aortic Regurgitation (AR)

Causes: Leaflet abnormalities (rheumatic dz, endocarditis, bicuspid valve), dilation of aortic root (aortic aneurysm/dissection, marfan syndrome, syphilis-cystic medial necrosis)

Pathophysiology

• Acute = surgical emergency—sudden  $\uparrow$  LV diastolic pressure rise backs up to pulm circulation causing pulm congestion, acute pulm HTN, & edema

• Chronic—LV compensates with dilation & hypertrophy  $\rightarrow$  heart failure

Clinical features

• Bounding pulses

• Austin-Flint murmur—turbulent flow across mitral valve during diastole due to AR jet

Treatment

• Asymptomatic—nifedipine, ACE inhibitor, diuretics

• Symptomatic—aortic valve replacement

Anesthetic management

• Maintain sinus rhythm

• Maintain normal to high normal heart rate

• Avoid  $\uparrow$  SVR (will worsen regurgitant fraction)

• Avoid myocardial depression

• Initiate prophylaxis against endocarditis

• Consider vasodilators (nitroprusside) to  $\downarrow$  afterload

**Pulmonic Stenosis**

Causes: Congenital deformity, carcinoid heart disease

## Classification

• Mild: pressure gradient &lt;40 mmHg, Moderate 40–80 mmHg, Severe &gt;80 mmHg

## Treatment

- Balloon valvuloplasty; valve replacement

**Pulmonic Regurgitation**

Causes: Annular dilation 2° enlarged pulm artery in pulm HTN congenital/carcinoid heart dz

**Tricuspid Stenosis**

Causes: Congenital, rheumatic heart dz, right atrial tumor, endocarditis

**Tricuspid Regurgitation**

Causes: Congenital, endocarditis, carcinoid heart dz, secondary event from mitral valve or left-sided heart dz

**Hypertrophic Cardiomyopathy (HCM)**

Causes: congenital

## Clinical features

- Mitral regurgitation from SAM (systolic anterior motion of anterior mitral leaflet)
- ↑ Risk of sudden death

## Anesthetic management

- Maintain slow HR (to allow for ventricular filling)
- Maintain low state of contractility to keep chamber size full
- Maintain preload & afterload

**Idiopathic Hypertrophic Subaortic Stenosis (IHSS)**

## Pathophysiology

- LV outflow obstruction (asymmetrical hypertrophic septum interferes with LV ejection)
- LVH & RA enlargement, ↑ myocardial O<sub>2</sub> consumption  
→ subendocardial ischemia

## Anesthetic management

- Maintain sinus rhythm
- Maintain preload & afterload
- Avoid significant ↑ in HR or contractility

**Anesthetic Goals in Valvular Disease**

	Preload	Afterload	HR	SVR	Contractility
Tricuspid stenosis	High normal	High	Low	High	Maintain
Tricuspid regurg	High normal	Low	High normal	Low	Maintain
Pulm stenosis	High	High	Low	High	Maintain
Pulm insuff	High normal	Low	High normal	Low	Maintain
Mitral stenosis	High normal	High	Low	High	Maintain
Mitral regurg	High normal	Low	High normal	Low	Maintain
Aortic stenosis	High	High	Low	High	Maintain
Aortic insuff	High	Low	High normal	Low	High
CAD	Normal	High normal	Low	High	Normal
HCM	High	High	Low	High	Low
ICM	High	Low	High normal	High	High
Tamponade	High	Low	High	High	High

**Heart Blocks/Arrhythmias** (see Chapter 32, on ECG interpretation)**Bradycardias**—HR <60 bpm

*Sick Sinus Syndrome*—intrinsic SA node dysfx → inappropriate bradycardia  
(treatment: anticholinergics, beta-adrenergic agents, pacing)

Rhythms that emerge from more distal latent intrinsic pacemakers of the heart with SA node dysfunction

*Junctional escape rhythm*—narrow complex (40–60 bpm)

*Ventricular escape rhythm*—wide complex (30–40 bpm)

Conduction defects below AV node within HIS-Purkinje system

- Left bundle branch block (LBBB)
- Right bundle branch block (RBBB)
- Interventricular conduction delay

## Atrioventricular conduction system

1° AV block—PR interval increased to  $>0.2$  sec

### 2° AV block

→ **Mobitz type I** (Wenkebach)—AV delay (PR interval) gradually  $\uparrow$  with each beat until QRS is dropped after P wave

(treatment—only if symptomatic: atropine, isoproterenol, permanent pacemaker for persistent block)

→ **Mobitz type II**—sudden unpredictable dropped QRS which isn't associated with progressive PR interval prolongation

(treatment: permanent pacemaker—as this may progress to 3° AV block)

### 3° AV block—“complete heart block” or “atrioventricular dissociation”

→ no relationship between P wave & QRS

(treatment: permanent pacemaker)

### Perioperative Considerations

- Indications for temporary perioperative pacemaker insertion are same as indications outside setting of surgery
- Some cardiac surgical procedures warrant placement of temporary epicardial pacemaker leads due to nature of surgery

### Tachyarrhythmias—HR $>100$ bpm

#### Supraventricular Arrhythmias

##### Sinus Tachycardia

##### Premature Atrial Beats

(treatment—only if symptomatic:  $\beta$ -blockers)

Atrial Flutter—atria @ 180–350 bpm, ventricular rate 150 (2:1 AV block)

(treatment: unstable—electrical cardioversion;

stable— $\beta$ - & Ca-channel blockers, burst pacing)

Atrial fibrillation—atria @ 350–600 bpm, ventricular rate variable

(treatment: unstable—cardioversion & anti-coagulation;

stable—anti-coagulation, then cardioversion;  $\beta$ - & Ca-channel blockers, antiarrhythmic drugs)

Paroxysmal SVT—ventricular rate 140–250, narrow complex, hidden P's

(treatment: vagal maneuvers,  $\beta$ - & Ca-channel blockers, RFA)

##### AV reentrant tachycardia

Wolff-Parkinson-White: PR interval shortened,  $\delta$ -wave, wide QRS

(treatment: RFA,  $\beta$ - & Ca-channel blockers, avoid procainamide)

#### Ventricular Arrhythmias

##### Premature ventricular contractions—widened QRS

Couplet—two in a row, bigeminy—every other beat is PVC

Ventricular Tachycardia—three or more PVC in a row, 100–200 bpm

Sustained VT—persists for 30 s or more

Nonsustained VT—persists for  $<30$  s

(treatment: symptomatic but stable  $\rightarrow$  electrical cardioversion (200J monophasic, 100J biphasic)

asymptomatic nonsustained VT  $\rightarrow$   $\beta$ -blockers

unstable—see ACLS protocol (CPR, cardioversion, epinephrine, vasopressin, amiodarone, lidocaine)

Torsades de Pointes—polymorphic VT with QRS twisting about baseline

(treatment— $MgSO_4$  1–2 gm IV)

Ventricular Fibrillation—irregular ECG without discrete QRSs

(treatment—see ACLS protocol, CPR, cardioversion, epinephrine, vasopressin, amiodarone, lidocaine)

Asystole—no electrical activity

(treatment—see ACLS protocol, CPR, epinephrine, atropine)



**Intra-aortic Balloon Pump**

Balloon placed in descending aorta (distal to L subclavian artery proximal to renal arteries) & synchronized with ECG. Balloon *inflates* at onset of diastole, *deflates* during systole (triggered by R-wave).

Blood displacement: proximal displacement—improves coronary artery perfusion  
distal displacement—improves systemic perfusion

Goals: ↓ afterload, ↓ wall tension, LVEDP & LVEDV (↓ in myocardial O<sub>2</sub> consumption)  
↑ arterial & aortic diastolic pressures ⇒ improved coronary artery perfusion

**Indications & Contraindications for Intra-aortic Balloon Pump****Indications**

*Complications of myocardial ischemia*

- Hemodynamic: cardiogenic shock
- Mechanical: MR, VSD
- Intractable dysrhythmias
- Postinfarct angina

*Acute cardiac instability*

- Angina: Unstable, preinfarction
- Cath lab mishap: Failed PTCA
- Bridge to transplantation
- Cardiac contusion
- Septic shock

**Contraindications**

- Severe aortic insufficiency
- Inability to insert
- Irreversible cardiac dz (not transplant candidate)
- Irreversible brain damage

Source: Adapted from Barash PG. *Clinical Anesthesia*, 5th ed. Lippincott Williams & Wilkins: Philadelphia, PA: 2006.

**Pacemakers**

Pacemaker indications: Sick sinus syndrome, tachy-brady syndrome, severe heart block  
Asynchronous versus synchronous:

- **Synchronous (demand) mode**—pacer senses P wave, R wave or both; pacer is either **triggered** or **inhibited** by sensed signal
- **Asynchronous mode**—pacer fires regardless of pt's intrinsic rhythm  
→ only used in perioperative situations or if ablation has removed conduction

Biventricular pacing for congestive heart failure

- ↑ Depolarization of LV; synchronizes contraction of ventricles; ↑ cardiac fx

**Generic Pacemaker Codes**

Pacing Chamber	Sensing Chamber	Response to Sensing	Programmability	Multisite Pacing
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	I = inhibited	R = rate modulation	A = atrium
V = ventricle	V = ventricle	T = triggered		V = ventricle
D = dual (A+V)	D = dual (A+V)	D = dual (T+I)		D = dual (A+V)

**Commonly Used Pacing Mode**

Mode	Description	Function	Indication
AOO	Atrial asynchronous	A pacing	Bradydysrhythmias
VOO	Vent asynchronous	V pacing	Bradydysrhythmias
DOO	Atrial & vent. asynchronous	AV pacing	Bradydysrhythmias
VAT	Atrial sensed & vent triggered	V paced, A sensed, sensed A beat triggers V output	Complete heart block with normal SA node
VVI	Vent noncompetitive	V pacing on demand, sensed V beat inhibits paced V beat	Sinus node dysfx, chronic Afib, complete block
DDD	Universal	A paced, V paced, AV sensed, sensed A inhibits A output, sensed A triggers V output, sensed V inhibits V output	Sinus brady, sinus tachy, complete block, 2° AV block

### Anesthetic Considerations for Pacemakers

- Consider conversion to an asynchronous mode (VOO) with **magnet** or programming
- MRI can convert pacemaker to asynchronous mode
- Lithotripsy can inhibit demand pacers
- Electrocautery can inhibit demand pacers  
→ low current in short, frequent bursts or bipolar electrocautery minimizes this
- Positive-pressure ventilation can affect distance of leads from heart
- Hyper/hypokalemia can alter pacing thresholds
- Evoked potential monitoring can alter function
- When defibrillating, place paddles/pads at least 1 in. away from pacemaker
- Consider interrogating pacemaker at end of case to ensure proper functioning

### AICD (Automatic Implantable Cardioverter-Defibrillator)

Indications: Survival of sudden death episode, sustained VT, syncope from VT low EF/hypertrophic cardiomyopathy

#### Anesthetic management

- Deactivate with magnet (vendor-specific) or programming (preferred)  
→ electrocautery can interfere with proper function
- Lithotripsy should be avoided; MRI is contraindicated
- External defibrillator should be available in the OR

### Cardiopulmonary Bypass Machine

- **Basic circuit:** Blood goes from patient → venous cannula → venous reservoir → oxygenator → heat exchanger → main pump → arterial filter → arterial cannula → patient
- Other components
  - Cardiotomy suction—removes blood from field for salvage
  - LV vent—prevent LV dilation from filling by thebesian veins & bronchial arteries
  - Cardioplegia pump—administers cardioplegic solution
  - Ultrafilter—hemoconcentrates blood by removing water & electrolytes
- Cardioplegic solution—high in potassium ( $K^+$ )
  - Anterograde cardioplegia—catheter in aortic root or coronary artery os; flow into coronary arteries
  - Retrograde cardioplegia—catheter in right atrium to coronary sinus; flow into coronary veins
- Main pump:
  - Roller head**—generates nonpulsatile flow
  - Centrifugal pump**—flow is pressure-dependent, less traumatic to RBC
  - Oxygenator**—membrane oxygenator less traumatic than bubble

### Cardiopulmonary Bypass Preanesthetic Management

#### Monitoring/Access

- Arterial line: Bypass pumps usually nonpulsatile (NIBP will not work)
- CVP/PA: Place before or after induction depending on vascular access; consider PA line for complex cases, or significant myocardial disease
- TEE: Place probe after induction
- Establish large-bore IV access (18-gauge or larger)

#### Premedication

- Patients may develop myocardial ischemia with stress/anxiety → consider lorazepam 2–3 mg/midazolam 1–2 mg preoperatively
- Supplemental  $O_2$  as needed

#### Induction

- Consider high-dose narcotic induction (can minimize myocardial depression)
- Fentanyl (7–15 mcg/kg) or remifentanyl (1 mcg/kg, then 0.2–1 mcg/kg/min)  
→ may give some paralytic prior to induction (avoid chest wall rigidity)
- Consider etomidate for pts with impaired myocardial fx; otherwise  
→ propofol/thiopental
- Sevoflurane/isoflurane acceptable provided that hemodynamics (esp BP) are well controlled
- Ketamine may increase risk of myocardial ischemia
- Nitrous oxide generally avoided → risk of expansion of gaseous emboli in open heart procedures
- Paralytics: Vecuronium, cisatracurium, pancuronium (may cause tachycardia)

**Fast-Tracking in Cardiac Surgery**

- Early extubation (within 6 hr postop)  
Advantages: reduced ICU stay, lower costs
- Used for low risk cases/patients, in OPCAB, short bypass runs
- Must be planned in advance: Limit IV fluids, narcotics, keep patient warm

**Cardiopulmonary Bypass Anesthetic Considerations****Prebypass**

- Lungs down while sawing through sternum
- Consider use of antifibrinolytic agent to reduce bleeding (see table page 16-13)  
Aminocaproic acid or tranexamic acid ↓ bleeding by plasmin inhibition  
Aprotinin (no longer available; see table page 16-13)
  1. Potential ↓ bleeding, transfusions, reoperation for bleeding
  2. May be assoc with postop renal failure, ↑ mortality
- Aortic cannulation  
Lower SBP to 90-120 mm Hg before cannulation  
Complications: Aortic dissection, emboli, bleeding, hypotension
- Heparinization—prior to initiation of bypass

**Heparin**

- Mechanism: binds to antithrombin III → potentiates inhibition of factor X and thrombin
- CPB dose: 300-400 units/kg → check ACT (goal >400)
- Heparin resistance: antithrombin III deficiency, prior heparin therapy, oral contraceptive use, ↑ age
- Management of heparin resistance: Give additional dose of heparin; FFP (↑ antithrombin levels)
- Complications: Bleeding, thrombocytopenia (HIT) → heparin alternatives: Danaparoid sodium, r-hirudin, epoprostenol sodium

**Protamine**

- Mechanism: Base that ionically binds heparin (acid) by forming a stable/inactive complex
- CPB dose: 1 mg for 100 units of heparin; give small test dose first to check for allergic response (signs: Hypotension, anaphylaxis, anaphylactoid reaction, bronchospasm, pulm htn)
- Goal ACT postbypass: <120-130
- Increased risk of protamine reaction: Prior exposure, NPH/PZI insulin, rapid infusion, seafood allergy
- **Note: Never give protamine while still on bypass!**

**Sternotomy**

- Short but painful stimulus → ensure pt is adequately anesthetized, disconnect pt from ventilator to avoid pericardial/lung injury

**During Bypass**

- Ventilator turned off (don't forget to turn back on later)
- Volatile agent can be provided by perfusionist through pump
- Often administer narcotic (fentanyl) drip plus a benzodiazepine (midazolam) by either bolus dosing or infusion
- Consider insulin infusion for diabetic patients

**During Rewarming**

- When primary anesthetic is narcotic, recall occurs most often during rewarming
- Consider using small dose of scopolamine/benzodiazepines

**Alpha Stat Versus pH Stat**

**Alpha stat** arterial blood gases are **not** temperature-corrected

- During hypothermic CPB, hypothermic alkaline drift occurs
- Prioritizes maintenance of  $[\text{OH}^-]$  to  $[\text{H}^+]$  ratio both inside & outside cell
- Benefit: appears to have improved neurologic outcome
- Con: Allows leftward shift of oxyhemoglobin dissociation curve, slow cooling

**pH stat** arterial blood gases are **temperature-corrected**

- $\text{CO}_2$  added to oxygenator, which counteracts alkaline drift at lower temp. resulting in a sample which is then heated to  $37^\circ\text{C}$
- Prioritizes maintenance of extracellular pH
- Benefit: Cerebral vasodilation, faster cooling, counteracts leftward shift of oxyhemoglobin dissociation curve
- Con: ↑ Flow may carry ↑ embolic load to brain

**Which to use?** pH stat may offer protection in neonatal & infant cardiac surgery, alpha stat most commonly used today in adults

### Potential Catastrophies During Bypass

- Aortic dissection
  - Stop CPB, choose alternate cannulation site, replace/repair dissected artery
- Inadvertant carotid/innominate artery cannulation
  - Treat ensuing cerebral edema, reposition aortic cannula
- Reversed cannulation
  - Stop CPB, evacuate air, reposition cannulas, restart CPB
- Obstruction to venous return
  - Reduce pump flow, treat cause (air lock evacuation, unkink tubing)
- Massive air embolism
  - Stop CPB, place pt in Trendelenburg, remove air
- Protamine administration during CPB will result in catastrophic clotting

### Weaning off Bypass

#### Key Points

- Core temperature should be at least 36°C
- Check  $K^+$ , glucose, Hct before weaning
- Positive-pressure ventilation to evacuate air from heart, great vessels, & grafts
- Reversal of heparin anticoagulation is with 1 mg of protamine for every 100 units heparin given slowly, administration before cessation of CPB can cause a catastrophic clotting of bypass pumps.
- Check rate and rhythm → may need temporary pacing
- Consider preop and current ventricular function.
- Support SVR → goal 1000–1200 dyn\*s/cm<sup>5</sup>. Maintain cardiac output & blood pressure with vasodilators (nitroglycerine, nitroprusside) and/or vasopressors respectively and inotropes (norepinephrine, dopamine, dobutamine, epinephrine, milrinone) as needed

#### Failure to Wean From CPB

- **LV/RV failure**  
Ventilatory—"pump lung" ARDS, bronchospasm, secretions  
Preload problems  
Ischemia: Graft failure, inadequate coronary blood flow, prior MI reperfusion injury after cardioplegia aortic dissection  
Valve failure  
Pulmonary hypertension (RV failure)  
Other: Inhalational agents,  $\beta$ - & Ca-channel blockers acidosis, electrolyte abnl, hypocalcemia, hyper/hypokalemia
- **Low SVR**  
Medications: Vasodilators, inhalational agents, protamine,  
Hemodilution, hyperthermia, sepsis, anaphylaxis/anaphylactoid rxn

#### Complications of CPB

- Pulmonary: pulmonary edema—"pump lung" ARDS, heart failure, protamine, pneumothorax, hemothorax, PA rupture
- Neurologic: Focal or global ischemia
- Electrolyte/fluid imbalances
- Hematologic: Anemia, coagulopathies
- Temperature: Hypothermia

### Postop Complications

- Common reasons for pt return to the OR
  - Persistent bleeding, excessive blood loss, cardiac tamponade, unexplained poor cardiac performance
- Postoperative bleeding
  - Inadequate surgical hemostasis → return to OR coagulopathy - due to ↓ platelet count or fx, hemodilution or depletion of coagulation factors, fibrinolysis, insufficient protamine reversal, "heparin rebound"
- Pericardial tamponade
  - Suspect in postop cardiac pts with hemodynamic deterioration
  - Postop bleeding into pericardial sac → ↑ intrapericardial pressure & ↓ venous return
  - TEE findings: See systolic R atrial & diastolic R ventricular collapse
  - Stroke volume is ↓ & C.O. becomes dependent on HR
    - Compensatory tachycardia, peripheral vasoconstriction are seen

- Management: keep patient, "full, fast, tight"  
goal = maintain preload, contractility, C.O.; normal to high HR  
ventilate with ↑ rate, ↓ tidal volume, ↓ PIP, avoid PEEP  
treatment: surgical exploration, pericardiocentesis

### Transesophageal Echocardiography (TEE)

- 2D *ultrasound* + *Doppler* (pulsed-wave technology measures flow)  
Red—blood flow towards transducer  
Blue—blood flow away from transducer  
Green—rapidly accelerating or turbulent flow
- Complications: Major complications in 0.2–0.5% (*esophageal trauma, dysrhythmias, hemodynamic instability*)  
minor complications in 0.1–13% (*lip & dental injuries, hoarseness, dysphagia*)

### Cardiac Pharmacology

Heart Failure Drugs			
Name	Mechanism of Action	Dosing	Comment
Milrinone	Phosphodiesterase inhibitor	50 mg/kg over 10 minute loading dose followed by 0.375–0.75 mg/kg/min. Max 1.13 mg/kg/day	Increased inotropy and vasodilation.
Levosimendan	Calcium-sensitizing drug that stabilizes the troponin molecule in cardiac muscle	12–24 mcg/kg loading dose followed by 0.1–0.2 mcg/kg/min for 24 hr	Increased inotropy and vasodilation. May be associated with hepatic dysfunction.
Natrecor (nesiritide)	Recombinant form of human brain natriuretic peptide	2 mcg/kg loading dose followed by 0.01 mcg/kg/min for 24 to 48 hr	Venous and arterial vasodilation. Diuresis. It does not affect cardiac contractility.

### Actions of Adrenergic Agonists

Sympathomimetics	$\alpha 1$	$\alpha 2$	$\beta 1$	$\beta 2$	DA 1	Dose	Comment
Phenylephrine	+++++	?	+/-	0	0	IV push: 50–100 mcg Drip: 0.15 mcg/kg/min	Primarily vasoconstriction
Norepinephrine	+++++	+++++	+++	0	0	Drip: 0.01–0.1 mcg/kg/min	$\beta 2$ effect present but not seen clinically
Epinephrine	+++++	+++	+++++	++	0	IV push: 0.3–0.5 mg Drip: 0.01–0.3 mcg/kg/min	High doses: $\alpha$ Low doses: $\beta$
Ephedrine	++	?	+++	++	0	IV push: 5–10 mg	
Dopamine	+ to ++++	?	+++++	++	+++	Drip: 0.05–10 mcg/kg/min	DA: 0.5–4 mcg/kg/min $\beta$ : 5–10 mcg/kg/min $\alpha > 10$ mcg/kg/min
Dobutamine	0 to +	?	+++++	++	0	Drip: 2–30 mcg/kg/min	Inotropism greater than chronotropism
Dopexamine	0	0	+	+++++	+	Drip: 1 to 5 $\mu$ g/kg/min	Moderate inotropy with intense arterial vasodilation. Synthetic analog of dopamine. May protect hepatoplancnic circulation
Isoproterenol	0	0	+++++	+++++	0	IV push: 0.004 mg Drip: 0.015–0.15 mcg/kg/min	...

Source: Derived from Barash PG. *Clinical Anesthesia*, 6th ed. Lippincott Williams & Wilkins: Philadelphia, PA: 2006.

Antifibrinolytics<sup>†</sup>

Drug & Class	Mechanism	Advantages	Disadvantages	Dose
<b>e-aminocaproic acid:</b> Lysine analog	Binds to lysine binding sites on plasminogen and fibrinogen, thereby inhibiting plasminogen activation and preventing plasmin release Same as above	<ul style="list-style-type: none"> <li>• ↓ mediastinal bleeding</li> <li>• May ↓ transfusion requirements</li> </ul>	<ul style="list-style-type: none"> <li>• No effect on reop rate</li> <li>• End-organ safety not well established</li> <li>• May cause thrombosis</li> </ul>	<ul style="list-style-type: none"> <li>• Load 100–150 mg/kg followed by infusion of 10–15 mg/kg/hr</li> <li>• Load 10 g × 3: Baseline, CPB, and postprotamine</li> </ul>
<b>Tranexamic acid:</b> Lysine analog	Same as above	Ten times the potency of e-aminocaproic acid	<ul style="list-style-type: none"> <li>• Dosing rate not well standardized in literature</li> <li>• Same as above</li> </ul>	<ul style="list-style-type: none"> <li>• Low dose: Load 10–15 mg/kg followed by infusion of 1–1.5 mg/kg/hr</li> <li>• High dose: Load 100–150 mg/kg followed by infusion rate 10 mg/kg/hr</li> </ul>
<b>Aprotinin:</b> <sup>‡</sup> Nonspecific protease inhibitor (NOTE: THIS DRUG HAS BEEN REMOVED FROM THE MARKET)	Inhibits proteases kallikrein, plasmin and others leading to inhibition of intrinsic coagulation cascade, complement activation, fibrinolysis, and bradykinin formation	<ul style="list-style-type: none"> <li>• Most evidence for decreased postoperative bleeding and transfusion requirements</li> <li>• Reduces reoperation for bleeding</li> <li>• May reduce stroke rate</li> </ul>	<ul style="list-style-type: none"> <li>• May be associated with renal dysfunction and transient elevated creatinine</li> <li>• Allergy potential: IgG formation because it is derived from compound extracted from beef lung</li> <li>• Association with mortality not well defined</li> <li>• High cost</li> </ul>	<ul style="list-style-type: none"> <li>• 1-mL test dose, wait 10 min, to assess for possible anaphylactic response “high-dose regimen”</li> <li>• 2 × 10<sup>6</sup> KIU (280 mg) over 20 minutes followed by an infusion of 5 × 10<sup>5</sup> KIU (70 mg) per hour</li> <li>• Half-dose regime is half of the high dose above</li> </ul>

<sup>†</sup>Note: Administration of antifibrinolytics is contraindicated in disseminated intravascular anticoagulation (except in the context of CPB) and upper urinary tract bleeding owing to the risk of thrombosis.  
<sup>‡</sup>Aprotinin caused a higher mortality rate and a higher Cr level than aminocaproic acid or no antifibrinolytic agent. (*N Engl J Med*). Feb 21; 358(8):784–793).  
 From Shaw AD, et al. The effect of aprotinin outcome after coronary artery bypass grafting. *N Engl J Med* 2008;21: 358(8):784–793.

# ANESTHESIA FOR THORACIC SURGERY

JONATHAN M. ANAGNOSTOU

THORACIC 17-1

## PULMONARY FUNCTION TESTS (PFTs)

- Preoperative evaluation of lung resectability
  - History & physical
    - Medications
    - Smoking history (cessation counseling)
    - Exercise tolerance
  - Room air ABG
  - Spirometry (esp if  $\text{PaCO}_2 > 45$  mm Hg,  $\text{PaO}_2 < 60$  mm Hg)
  - Split-function tests (if  $\text{FVC} < 2$  L,  $\text{FEV}_1 < 50\%$  predicted)
    - Regional ventilation
    - Regional perfusion
    - Predicted postop (PPO)  $\text{FEV}_1 = \text{preop FEV}_1 \times \text{fraction functional lung tissue remaining postresection}$
    - Bronchial balloon occlusion of proposed area of resection
    - Unilateral pulmonary artery occlusion
      - Simulates postresection ventilation & stress on right heart
      - Poor prognostic signs:  $\text{PAP} > 40$  mm Hg,  $\text{PaO}_2 < 45$  mm Hg,  $\text{PaCO}_2 > 60$  mm Hg
    - Clinical judgment—diseased lung tissue may be contributing little to gas exchange
  - Predicted  $\text{FEV}_1 < 800$  mL ( $< 40\%$  predicted) implies poor survival postresection
- Risk of pulmonary complications increased with
  - Dyspnea on exertion on  $< 2$  flights stairs
  - Diffusing capacity ( $\text{DL}_{\text{CO}}$ )  $< 40\%$  predicted
- Other notable states affecting PFTs
  - Pregnancy: ↓ FRC, ↓ RV, ↓ ERV
  - Elderly: ↑ FRC, ↑ RV, ↑ lung compliance, ↓  $\text{FEV}_1$
  - Obesity: ↓ FRC, ↓ VC, ↓ ERV, ↓ IRV
  - General anesthesia: ↓ FRC

## COMMON DISEASE STATES AFFECTING THE LUNGS

### COPD

- $\text{FEV}_1/\text{FVC} < 1$
- Slowly progressive obstructive lung disease
  - Fixed obstruction with periodic exacerbations (e.g., infections)
  - Small airway obstruction
    - ↓ Forced max expiratory flow ( $\text{FEF}_{25-75\%}$ )
  - Emphysema
  - Chronic bronchitis (high incidence of cor pulmonale)
- Associated with  $> 20$  pack-yr smoking history
- Incidence  $\approx 7-8\%$  of U.S. adults
- Treatment
  - $\beta_2$ -agonists (inhaled) → albuterol/metaproterenol/salmeterol
  - Anticholinergics (inhaled) → ipratropium/glycopyrrolate/atropine
  - Steroids (inhaled or systemic)
  - Antibiotics for bronchitis/other infections
  - Supplemental oxygen (severe disease)
- Anesthetic management
  - History
    - Current status vs baseline
      - Exercise tolerance: Climbing 2 flights stairs = lower risk
    - Recent changes in meds (e.g., inhalers, steroids)
    - Recent hospitalizations, ED visits
  - Continue baseline medications
  - Pts may have erythrocytosis, pulm HTN, cor pulmonale, RV failure
  - Pulmonary consultation if poorly controlled
  - Stop smoking preop (several weeks if possible)
    - Nicotine ↑ HR & BP
    - Carbon monoxide decreases  $\text{O}_2$  delivery
    - May help ↓ wound complications
    - ↑ Chances of permanent cessation



- Adequate anesthetic depth at induction
  - Lidocaine may ↓ airway reactivity at intubation
- Consider volatile anesthetic if cardiac status allows
- Avoid nitrous oxide if bullae present
- Limit tidal volume (6 mL/kg) to minimize  $P_{aw}$  & limit PEEP (risk of bullae rupture/pneumothorax)
- Adequate expiratory time to avoid air trapping & keep low RR
- Consider deep extubation if no GERD & mask airway adequate
- COPD patients have ↑ risk of postop respiratory failure

### Asthma

- Episodic lower airway obstruction due to bronchospasm & inflammation (↓ FEV<sub>1</sub>, FEV<sub>1</sub>/FVC < 1, FEF<sub>25-75%</sub>)
  - Normal spirometry between exacerbations (may develop chronic obstruction in severe, chronic disease)
  - Inciting factors: Cold, pollen, dust, exercise, aspirin/NSAIDs, infections
- Incidence ≈ 5% of U.S. adults
- Treatment
  - β<sub>2</sub>-agonists—short-acting (inhaled) → albuterol/metaproterenol
  - Anticholinergics (inhaled) → ipratropium
  - Antileukotrienes (not useful for acute attacks) → montelukast/zileuton
  - Theophylline
  - Cromolyn (inhaled—not useful for acute attacks)
  - Steroids (inhaled or systemic)
  - Antibiotics for bronchitis or other infections
  - Supplemental oxygen (severe disease)
- Anesthetic management
  - History
    - Precipitating factors
    - Recent ER visits or hospitalizations
    - Current status vs baseline
    - Exercise tolerance
    - Climbing 2+ flights of stairs = lower risk
  - Continue preop pulmonary medications
    - Consider stress-dose steroid if chronic steroid treatment
  - Adequate anesthetic depth at induction
    - Lidocaine & propofol both ↓ airway response to intubation
  - Consider volatile anesthetic (e.g., sevoflurane = bronchodilator)
  - Intraop wheezing
    - Differential diagnosis
      1. Bronchospasm
      2. Mechanical obstruction (tube kinking)
      3. Secretions
      4. Pneumothorax
      5. Pulmonary edema
    - Management
      1. Ensure adequate depth of anesthesia
      2. Inhaled short-acting β-agonist (albuterol)
      3. Consider IV lidocaine
      4. Consider IV epinephrine
    - Consider deep extubation IF no GE reflux & mask airway adequate

### Pulmonary edema

- Etiologies
  - Cardiac
    - Myocardial dysfunction (most common)
    - Rhythm disturbances
    - Valvular decompensation
  - Noncardiac (capillary leak)
    - Sepsis/sepsis syndrome
    - Inhalation injury
    - Hypertension (severe)
    - Neurogenic
    - Negative pressure (e.g., postlaryngospasm)
- Diagnosis
  - Hypoxemia (relative or absolute)
  - Rales on auscultation

- Airway secretions ("pink froth")
- Treatment
  - Oxygen supplementation
  - Mechanical ventilation ( $\pm$  PEEP)
  - Venodilatation—nitroglycerin (if ischemia suspected)
  - Diuresis—furosemide
  - Inotrope (if low cardiac output)—dobutamine/milrinone
  - Cardioversion for dysrhythmias
  - Consider intra-aortic balloon pump (for ischemia/infarction)
- Anesthetic management
  - Invasive monitoring
    - Arterial line
    - Consider CVP or PA catheter
    - Consider transesophageal echo if available
  - Minimize myocardial depression
    - Consider etomidate for induction, opioids for maintenance
  - Avoid hypervolemia
  - Ensure inotropic support available

### ARDS (see also Chapter 15, ICU, Trauma and Burn Management)

- Diffuse, patchy pulmonary injury without cardiac failure
- Etiologies: Sepsis, aspiration, pancreatitis, pneumonia, inhalation injury, near drowning
- Leads to atelectasis,  $\downarrow$  FVC,  $\dot{V}/\dot{Q}$  mismatch
- Management
  - Treat underlying cause (e.g., infection)
  - Supplemental  $O_2$
  - Avoid hypervolemia
  - Mechanical ventilation to avoid barotrauma (*N Engl J Med* 2000;342:1301)
    - Limit  $P_{aw} \leq 30$  cm  $H_2O$
    - Tidal volume  $\leq 6$  mL/kg
    - PEEP
    - permissive hypercapnea to pH 7.25–7.3
- Anesthetic management
  - Mechanical ventilation (see above)
  - Volatile anesthetic as tolerated
  - Opioid: can improve comfort during mechanical ventilation; high dose can provide hemodynamic stability
  - Invasive monitoring
    - Arterial line
    - Consider CV or PA catheter

### Restrictive Lung Disease

- $FEV_1/FVC \approx 1$
- $\downarrow$  TLV & FRC with near normal expiratory flows
- $\downarrow$  FRC leads to rapid hypoxemia upon brief apnea
- Ventilation management
  - Use lower tidal volume:  $P_{aw}$   $\uparrow$  rapidly with larger TV
  - $\uparrow$  Ventilation rate
  - Institute PEEP
- Anesthetic management
  - Consider feasibility of regional technique
  - General anesthesia
    - Thorough preoxygenation
    - Anticipate hypoxemia on apnea at induction
    - Avoid large TV
    - Minimize residual respiratory depressants
    - Consider need for postop ventilator support

### Anesthetic Techniques—Specific Considerations

- Bleomycin related pulm fibrosis: Use  $FiO_2 \leq 40\%$  &  $\downarrow$  fluids
- Lung isolation techniques
  - Indications: facilitate surgical exposure, protect healthy lung
    - Video-assisted thoracic surgery (VATS)
    - Pulmonary resections
    - Esophagectomy
    - Thoracic aneurysm repair
    - Thoracic spine surgery
    - Protection of healthy lung from infection, hemorrhage, lavage

- Double-lumen tubes (DLTs)
  - Left more commonly used
    - May be easier to place (longer L mainstem)
    - Can be difficult to ventilate R upper lobe with R DLT
  - Tube size selection (based on height)
    - Males 39 Fr (5 ft 5 in.)–41 Fr (5 ft 10 in.)
    - Females 35 Fr (5 ft 5 in.)–39 Fr (5 ft 10 in.)
  - Typical tube depth at upper incisors (adults)
    - Males 29–31 cm, Females 27–29 cm
  - DLT insertion—see table below

### Technique Double-Lumen Tube Placement

1. Perform laryngoscopy to optimize glottic view
2. Insert bronchial cuff just below cords with tube bend pointed anteriorly (aids initial insertion)
3. Remove stylet
4. Advance DLT while rotating tube to midline (90° left for left DLT)
5. Inflate tracheal cuff & confirm tracheal placement
  - Breath sounds should be equal on both sides
  - No leak should be present around tracheal cuff
6. Inflate bronchial cuff CAREFULLY (rarely >2 mL air)
7. Recheck breath sounds
8. Selectively clamp & recheck R/L breath sounds
  - Changes may be difficult to detect if soft baseline breath sounds (e.g., COPD)
9. Malposition often missed on auscultation alone
10. Verify position with fiberoptic
  - Verify bronchial cuff down proper mainstem
    - Airway rings most prominent anteriorly
    - Identify right-upper-lobe bronchus open
    - Ensure no bronchial cuff herniation over carina upon inflation
  - DLT often migrates with lateral/neck repositioning

### Troubleshooting Double-Lumen Tubes: Both cuffs inflated & one lumen clamped

	Tracheal Side Ventilated	Bronchial Side Ventilated	Problem
Breath sounds	Clear or absent	Clear on one side, ↑ airway pressures	DLT too deep
	Absent	Both sides	DLT too shallow
	Absent or on wrong side	Wrong side only	DLT on wrong side

Source: Adapted from Dunn P. *Clinical Anesthesia Procedures of the MGH*. 7th ed. Lippincott William & Wilkins: Philadelphia, PA.

- Bronchial blockers
  - Univent tracheal tube (provides CPAP, but can't ventilate while isolating the lung)
  - Fogarty catheter (sm. size makes useful in pedi cases)
    - No lumen for deflation, CPAP, or suction
  - Wire-guided endobronchial blocker (WEB)
    - Small lumen for deflation, CPAP, or suction
    - Insertion loop: 1-time use

### Physiology of One-Lung Ventilation

- One-lung ventilation in lateral position
  - Gravity causes ↑ blood flow to dependent (bottom) lung
  - Pulmonary shunt ↓ as dependent lung is being oxygenated
- Lateral positioning with open chest
  - ↑ Blood flow to dependent lung
  - ↓ Effective compliance in dependent lung
  - ↓ FRC
  - Altered  $\dot{V}/\dot{Q}$  (↑ perfusion, ↓ ventilation-dependent lung)
    - ↓ Oxygenation, ↓ CO<sub>2</sub> elimination
- Hypoxic pulmonary vasoconstriction (HPV)
  - Vasoconstriction of pulm arteries in presence of hypoxia → redirecting blood to alveoli with higher O<sub>2</sub> tension
  - Improves  $\dot{V}/\dot{Q}$  matching → better gas exchange
  - Inhibited by vasodilators (e.g., nitroprusside, nitroglycerine)
    - alkalemia, hypocarbia, volatile anesthetics, PEEP

- Risk of hypoxemia in 1 lung ventilation
  - $\dot{V}/\dot{Q}$  impairment
  - $\downarrow$  HPV
  - Worse typically at 10–30 min (absorption atelectasis—remaining alveolar  $O_2$  depleted)

### West's Lung Zones (*J Appl Physiol* 1964;19:713)

Zone 1 (top)	<ul style="list-style-type: none"> <li>• Alveolar pressure &gt; arteriole pressure &gt; venous pressure</li> <li>• Ventilation &gt; perfusion (<math>\uparrow \dot{V}/\dot{Q}</math> mismatch)</li> <li>• <math>\uparrow</math> Dead space</li> </ul>
Zone 2 (middle)	<ul style="list-style-type: none"> <li>• Arteriole pressure &gt; alveolar pressure &gt; venous pressure</li> <li>• Ventilation = perfusion</li> </ul>
Zone 3 (bottom)	<ul style="list-style-type: none"> <li>• Arteriole pressure &gt; venous pressure &gt; alveolar pressure</li> <li>• Perfusion &gt; ventilation (<math>2^\circ</math> to gravity) (<math>\uparrow \dot{V}/\dot{Q}</math> mismatch)</li> <li>• Shunt</li> </ul>

### Anesthesia with One Lung

- Invasive monitoring
  - Arterial line—preferably dependent radial artery
  - CVP ( $\pm$  for uncomplicated VATS/wedge/lobe in healthy pt)
  - Pulmonary artery catheterization in selected pts
    - Monitor PA,  $P_{cw}$  for left heart filling pressures
    - PA catheter usually floats to the R side
    - Often not in best monitoring position (i.e., not in West zone 3)
    - Risks: dysrhythmias, PA rupture
    - No proven improvement in outcomes with routine use
- Fiberscopic verification tube/blocker placement
- Tidal volume 8–10 mL/kg
  - Smaller TV  $\uparrow$  risk atelectasis
  - Larger TV  $\uparrow$  shunting to nonventilated lung,  $\uparrow$  risk barotraumas
- $\uparrow$  Ventilation rate modestly (10%) to allow  $EtCO_2$  35 mm Hg
- Oxygen 100%
  - Maximal  $PaO_2$
  - Possible absorption atelectasis
- Limit time on 1-lung ventilation
- $\uparrow$  Incidence of hypoxemia with:
  - Right lung deflation
  - Supine position
  - Normal preop spirometry (no intrinsic PEEP)
- Management of hypoxemia
  - Recheck proper tube position with fiberscope (see below)
  - CPAP 5–10 cm  $H_2O$  to nonventilated lung
    - <5 cm  $H_2O$  generally ineffective
    - >10 cm  $H_2O$  may reinflate nonventilated lung
  - Consider PEEP to ventilated lung
    - May worsen hypoxemia via  $\uparrow$  blood flow to nonventilated lung
  - Return to 2-lung ventilation if serious irreversible hypoxemia
  - For pneumonectomy—consider early operative ligation of pulmonary artery

### Troubleshooting Double-Lumen Tubes

1. Recheck adequate balloon inflation if air leak
2. Consider returning to 2-lung ventilation (if hypoxemic/inadequate ventilation)
3. Recheck position with fiberscope
4. If large  $\uparrow P_{aw}$  or no TV
  - Consider tube too shallow (both cuffs in trachea—if ventilating tracheal lumen)
  - Consider tube too deep
    - Cuff at  $2^\circ$  carina, TV delivered to lobe
    - Risks: Barotrauma, pneumothorax
5. If right lung will not fully deflate, recheck patency of R upper lobe bronchus with fiberscope
6. Consider full repositioning
  - Insert fiberscope through bronchial lumen into airway
  - Deflate cuffs & pull tube back so bronchial cuff is below cords but above carina
  - Advance fiberscope down desired mainstem
  - Advance tube over fiberscope
  - Replace fiberscope into tracheal lumen to recheck tube position

## Anesthetic Techniques—Specific Surgical Procedures

### Mediastinoscopy

- Preop evaluation
  - Airway: Mass effects on trachea, great vessels
  - History: CV prob, stroke, SVC syndrome, Lambert-Eaton syndrome
- Complications
  - Hemorrhage, pneumothorax, chylothorax, recurrent laryngeal nerve injury, air embolization
- Anesthetic management
  - General anesthesia most common
  - Vascular access
    - Large bore IVs
    - Arterial line in left radial (right radial subject to innominate artery compression by mediastinoscope)
  - Avoid nitrous oxide
  - Muscle relaxation
    - Movement ↑ risk of surgical trauma
    - Cough/strain ↑ thoracic venous engorgement
    - Spontaneous ventilation may ↑ risk air embolism
- Postop—must check chest x-ray

### Video-assisted Thoracic Surgery (VATS)

- Preop evaluation
  - Discuss potential for open thoracotomy
- Complications
  - Bleeding, lung injury (air leak)
- Anesthetic management
  - General anesthesia most common
  - Large-bore IV (consider CVP) access
  - Arterial line
  - Lung separation (see lung isolation section above)
  - Consider slightly lower TV (e.g., 7–8 mL/kg) to ↓ mediastinal shift (improves operating conditions)
    - ↑ Ventilation rate 10% (get CO<sub>2</sub> absorption with insufflation)
    - Muscle relaxation (see “Mediastinoscopy,” above)
    - Consider neuraxial anesthetic if ↑ likelihood of conversion to open thoracotomy
- Postop—must check chest x-ray

### Pneumonectomy

- Preop evaluation
  - See “PFTs/Evaluation of Lung Resectability,” above
  - ↑ Risk of morbidity with right vs left pneumonectomy, trauma, massive hemoptysis, history of cardiac disease, >10% preop weight loss
  - Optimize treatment of existing pulmonary/cardiac disease
  - Encourage smoking cessation
- Complications
  - Bleeding, airway (stump) leak, cardiac dysrhythmias (consider role of β-blocker), cardiac herniation through pericardial defect, pulmonary edema, myocardial infarction, intracardiac shunt (can get ↑ R heart pressure, shunting via PFO)
- Anesthetic management
  - Arterial catheter
  - Central venous line
  - Consider PA catheter
    - May not easily float to nonoperative side
    - Readings may not be reliable (tip not in West zone III)
    - May interfere with surgical procedure
  - Airway
    - DLT to nonoperative side or bronchial blocker
    - Risk of intraop dislodgement with either technique
    - Risk of bronchial stump damage with manipulations
  - Muscle relaxation
  - Limit intraop fluids
  - Postop analgesia plan
    - Neuraxial opioid ± local anesthetic
    - Intercostal nerve blocks
    - Intrapleural catheter (risk of local anesthetic toxicity)
    - Systemic opioids (transition to IV PCA as tolerated)

## Mediastinal Mass Considerations

- Preop eval
  - ↑ Risk of tracheobroncheal obstruction with:
    - Orthopnea
    - Large airway compression on imaging
    - Flattened expiratory limb of flow-volume loop on PFTs
  - Eval for evidence of superior vena cava syndrome
    - Upper extremity/facial edema (may indicate airway edema)
    - Dilated upper extremity veins
    - Headache, CNS changes
    - Consider preop steroid, diuretic, elevation of head of bed
  - History syncope with position or Valsalva suggests
    - Cardiac/PA compression with hypotension
    - Critical tracheobronchial obstruction
    - Consider preop echo to eval for compression
  - Consider preop biopsy/treatment to shrink mass (if severe airway/cardiovascular compression)
- Complications
  - Acute tracheobronchial compression intraop
    - Highest risk is on transition to positive-pressure ventilation
  - Acute cardiac/PA compression with severe hypotension
  - Bleeding (esp with SVC syndrome due to venous engorgement)
- Anesthetic management
  - Arterial access preinduction
  - Large bore/central venous access
  - Consider standby cardiopulmonary bypass (femoral) if airway or cardiovascular compression by mass
  - Rigid bronchoscope available
  - If SVC syndrome:
    1. Consider lower extremity vascular access (more reliable drug/fluid delivery)
    2. Avoid jugular or subclavian lines
  - Consider spontaneously breathing fiberoptic exam/intubation if significant airway compression
  - Initiate slow, controlled induction
    1. Controlled transition from spontaneous ventilation to positive pressure
    2. Short-duration relaxant desirable to facilitate tracheal intubation
    3. If airway obstruction occurs:
      - a. Attempt lateral positioning to move mass
      - b. Resume spontaneous ventilation if possible
      - c. Attempt to pass tracheal tube beyond obstruction carefully (risk hemorrhage)
      - d. Attempt rigid bronchoscopy to open airway
      - e. Consider cardiopulmonary bypass (femoral)
    4. Smooth emergence & extubation
      - a. Cough/straining may worsen airway collapse
      - b. May ↑ bleeding (esp if SVC syndrome)

## Esophagectomy

- Preop eval
  - Nutritional status (↓ serum albumin, total protein)
  - Dysphagia (reflux, risk of chronic aspiration)
  - Prior chemo/radiation therapy
  - Risk for cardiac dysrhythmias, esp supraventricular (consider prophylactic digoxin/β-blocker)
  - Consider epidural placement
- Complications
  - Gastroesophageal reflux, esophageal leak, respiratory failure, hypotension, cardiac dysrhythmias
- Anesthetic management
  - Arterial line
  - CVP
  - Lung isolation for thoracotomy approaches
  - Avoid nitrous oxide (expands bowel gas, need high FiO<sub>2</sub> with 1-LV)
  - Consider ↓ drug dosages if pt has ↓ serum albumin

- Consider cricoid pressure at induction
  - May ↓ lower esophageal sphincter tone
  - May impede use intubation LMA
- Monitor glucose closely (esp if on TPN)
- Communicate with surgeon regarding esophageal manipulations (e.g., NG tube, esophageal bougie)
- Intraop hypotension: May be from hypovolemia, surgical compression of heart or great vessels, bleeding
- If postop mechanical ventilation planned
  - Change to standard endotracheal tube at completion of surgery

# ANESTHESIA FOR GENERAL AND ABDOMINAL SURGERY

MAGED ARGALIOUS

## Systemic Manifestations of Liver Disease

Cardiovascular	Cardiomyopathy, ↑ C.O., ↑ intravasc volume, ↓ SVR
Respiratory	Hypoxemia from intrapulmonary shunts, ↓ FRC, concurrent COPD/pneumonia, pleural effusions, resp. alkalosis
Gastrointestinal	Ascites, portal HTN, GI bleeding, ↓ gastric emptying
Renal	Renal insufficiency, hepatorenal syndrome
Hematologic	Anemia, coagulopathy, thrombocytopenia
Neurologic	Encephalopathy, neuropathy
Metabolic	Hypoglycemia, ↓ K, ↓ Na, ↓ albumin

## Child-Pugh Classification of Severity of Liver Disease

Parameter	Points Assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin mg/dL	<2	2-3	>3
Albumin g/dL	>3.5	2.8-3.5	<2.8
Prothrombin time (seconds over control)	<4	4-6	>6
INR	<1.7	1.7-2.3	>2.3
Encephalopathy	None	Grade 1-2	Grade 3-4
Grade A: Total score = 5-6	→ 1- & 2-year survival = 100% & 85%		
Grade B: Total score = 7-9	→ 1- & 2-year survival = 80% & 60%		
Grade C: Total score = 10-15	→ 1- & 2-year survival = 45% & 35%		

GENERAL 18-1

## Anesthetic Considerations in Abdominal Surgery

### Preop Evaluation

- Fluid status: Pts often hypovolemic
  - Inadequate fluid intake (fasting, anorexia)
  - Fluid loss (emesis, bowel preps, GI bleeding, fevers = insensible loss)
  - Sequestration of fluid from intravascular space (3rd spacing)

### Physical Signs of Hypovolemia

Sign	Fluid Loss (% of body weight)		
	5%	10%	15%
Sensorium	Normal	Lethargic	Obtunded
Heart rate	Normal or ↑	↑ >100 bpm	Markedly ↑ >120 bpm
Blood pressure	Normal	Mildly ↓ with resp variation	↓
Orthostatic changes in HR & BP	Absent	Present	Marked
Mucous membranes	Dry	Very dry	Parched
Urine output	Mildly ↓	↓	Markedly ↓

### Anesthetic Management

#### Technique

- Abdominal procedures usually require muscle relaxation
- Epidural analgesia may be beneficial
  - (↓ anesthetic requirements, blunt surgical stress response, ↑ postop pain relief, ↓ postop atelectasis, ↑ postop mobility)



Level of Epidural Catheter Insertion in Relation to Type of Surgery	
Surgical Location	Level of Epidural Catheter Insertion
Pancreas, spleen, esophagus, stomach, liver, gallbladder, ileal loop	T7–T10
Adrenals, small intestine, colon, kidney, ureters, uterus, ovaries, & testes	T8–L1
Prostate, urethra, & rectum	L3–L4

*Fluid Management (see Chapter 9 on Fluids, Electrolytes, and Transfusion Therapy)*

- General strategies
  - Body wt-based formulas: Rough guidelines for fluid replacement
  - Goal-directed strategies: Aimed at optimizing stroke volume, cardiac output, & tissue perfusion
  - Restrictive management: 4–8 mL/kg/hr—some evidence for ↓ postop morbidity compared with “liberal” strategies (10–15 mL/kg/hr)
- Replacement ratio: 3 mL crystalloid per 1 mL fluid loss  
1 mL colloid per 1 mL fluid loss
  - Only 1/3 of crystalloid remains intravascular, 2/3 goes into interstitium
  - Colloids remain intravascular longer than crystalloids & exert oncotic pressure
- Blood products—should be given based on clinical eval of blood loss (surgical suction canister, sponges) & lab values (hematocrit)

*Muscle Relaxation*

- Usually required for intra-abdominal procedures & abdominal closure
  - 2° to intraop bowel edema & abdominal distention
- Inhalational agents may potentiate effects of muscle relaxants
- Neuraxial blockade with local anesthetics can provide good muscle relaxation

*Use of Nitrous Oxide (N<sub>2</sub>O)*

- N<sub>2</sub>O diffuses into bowel lumen faster than nitrogen can diffuse out
- Degree of bowel distention is a function of
  - N<sub>2</sub>O conc, blood flow to the bowel, duration of admin
- Avoid N<sub>2</sub>O (relative contraindication) in bowel obstruction
  - May have large initial volume of bowel gas and/or difficult surgical closure
- Causes an obligatory reduction in FiO<sub>2</sub>
  - However, ↑ FiO<sub>2</sub> may reduce incidence of surgical wound infection
- May ↑ pulmonary artery pressure (esp in pts with pulmonary HTN)
- Possible ↑ incidence of PONV (data is mixed)

*Common Intraop Problems*

- ↓ FRC, atelectasis & hypoxemia because of
  - Surgical retraction of abd viscera to improve exposure
  - Insufflation of gas during laparoscopy
  - Trendelenburg position  
(Application of PEEP may reverse those effects)
- Hypothermia 2° to heat loss: radiation > convection > conduction > evap
  - Most heat loss occurs during 1st hr of anesthesia (1–1½°C)  
(↑ OR temp, convective warming blankets, warm IV fluids)
- Hypotension, tachycardia, & facial flushing during bowel manipulation
  - 2° to mediator release (prostaglandin F 1-α, aprostanoid)
- Opioid-induced biliary tract spasm
  - May interfere with interpretation of intraop cholangiograms  
(Reversed by naloxone, nitroglycerin, & glucagon)
- Hiccups are episodic diaphragmatic spasms relieved by
  - ↑ Anesthetic depth, ↑ neuromuscular blockade, drainage of stomach to relieve gastric distention

## Alcohol Abuse

Preop Evaluation

- Lab values denote hepatocellular dysfx
- Alcoholic cirrhosis characterized by AST/ALT ratio >2

Anesthetic Considerations

- Acute intoxication: ↓ anesthetic requirements (2° to EtOH depressant effects)
- Chronic intoxication: ↑ anesthetic requirements (2° to tolerance)
- Head & cervical spine injury must be considered in intoxicated pts

### Postop Considerations

- Unrecognized alcohol abuse may present with delirium tremens
  - Often occurs 72 hr after last drink (postop day 3)
  - Signs: Autonomic hyperactivity, tremors, hallucinations, seizures
  - Treatment: Benzodiazepines

### Multisystem Involvement in Alcoholic Abuse

Cardiovascular	Dilated cardiomyopathy, hypertension
Respiratory	COPD (20% of alcohol abuse patients)
Neurologic	Cerebellar degeneration, polyneuropathy Nutritional disorders (Wernicke-Korsakoff syndrome) Delirium tremens (alcohol withdrawal)
Gastrointestinal	Esophagitis, gastritis, pancreatitis, hepatic cirrhosis
Hematologic	Anemia, thrombocytopenia
Endocrine	↓ Gluconeogenesis (hypoglycemia), hypomagnesemia

### Anesthetic Management: Liver Surgery

#### General Considerations

- Liver resections often done for metastasis to liver or 1° hepatocellular carcinoma
- Hypoxemia → 2° hepatopulmonary shunting, atelectasis, ↓ FRC from ascites
- Prior portosystemic shunt ↑ surgical complexity & risk of surgical bleeding

#### Management of Portal Hypertension

- Pharmacologic: β-blockers
- Endoscopic: Sclerotherapy & esophageal banding for bleeding varices
- Transjugular intrahepatic portosystemic shunt (TIPS) have replaced surgical shunts, done percutaneously under fluoroscopy
- Surgery: ↑ Risk of encephalopathy, no evidence of better outcome

#### Monitoring

- A-line & CVP

#### Anesthetic Technique

- General endotracheal anesthesia
- Thoracic epidural for postop pain control (provided no coagulopathy)
- Aspiration precautions (nonparticulate antacids, rapid-sequence induction)
- Avoid N<sub>2</sub>O (risk of bowel expansion & potential ↑ pulm artery pressure)
- Avoid histamine releasing muscle relaxants (atracurium, mivacurium) to avoid further ↓ blood pressure
- Hyperdynamic circulation in pts with end-stage liver disease may require vasopressor therapy to ↑ systemic afterload
- Concomitant pulmonary HTN in pts with ESLD → avoid hypoxemia, hypercarbia, & metabolic acidosis (worsen pulmonary HTN)
- Careful NG tube placement (concern for coagulopathy + esophageal varices)
- Fluid replacement with isotonic fluids & colloids (pts have ↓ intravascular oncotic pressure)
- Prolonged hepatic “inflow” occlusion (Pringle maneuver: Occlusion of portal vein & hepatic artery) → may lead to coagulopathy & metabolic acidosis

#### Postop Care

- Bleeding: Surgical vs coagulopathy
- *Small for size syndrome* in extensive hepatic resections (remaining liver unable to support metabolic functions → ↑ lactate, ↑ liver enzymes, worsening metabolic acidosis)

### Anesthetic Management: Bariatric Surgery

#### General Considerations

- Body mass index (BMI) = body weight in kg/(height in meters)<sup>2</sup>
- Overweight = BMI >25; Obesity = BMI >30; Morbid obesity = BMI >35

#### Types of Bariatric Surgery

- Vertical band gastroplasty
  - Creation of small pouch → restricts volume of food that can be ingested
- Open Roux-en-Y gastric bypass
  - Formation of small gastric pouch anastomosed to proximal jejunum
  - Dumping synd.: ingestion of energy-dense-food → nausea, diarrhea abd pain
  - Pts at risk for Fe and B<sub>12</sub> deficiency

- Laparoscopic Roux-en-Y gastric bypass
  - Smaller incision, ↓ postop pulm complications/pain, earlier ambulation

#### Preanesthetic Considerations

- Obesity-associated comorbidities
  - HTN, hyperlipidemia, obstructive sleep apnea (OSA), GERD, type II diabetes
  - ↑ Circulating blood volume, ↑ cardiac output → ↑ in O<sub>2</sub> consumption
  - ↓ Lung compliance, ↑ ventilation/perfusion mismatch & ↓ FRC → hypoxemia
    - Long-standing hypoxemia → pulmonary HTN, & rt-sided heart failure

#### Anesthetic technique

- General endotracheal anesthesia
- Epidural analgesia for pts undergoing open Roux-en-Y bypass
  - Reduces need for systemic opioids & oversédation in pts with OSA

#### Airway Management

- Specific considerations
  - Predictors of difficult intubation: ↑ Neck circumference (>42 cm) & Mallampati score III & IV
  - Obesity = risk factor for difficult mask ventilation
  - Rapid desaturation following induction 2° to ↓ FRC, ↑ O<sub>2</sub> consumption & ↑ incidence of airway obstruction
- Management strategies
  - Preoxygenation for 3 min in a 25° head-up position
  - Consider ramped position (horizontal alignment between auditory meatus & sternal notch) to improve laryngeal view
  - Consider awake intubation if exam concerning
  - Consider aspiration precautions (antacids + rapid-sequence induction)
  - Consider use of insoluble gases (desflurane, sevoflurane)
  - Consider short-acting narcotics & sedatives (↓ risk postop resp depression)

#### Drug-Dose Adjustment in Obesity

*Some drugs have established dosing adjustments for obese pts;  
→ however, it is unknown if dosing adjustments needed for most drugs*

##### Dosage by Total Body Weight (TBW)

- Benzodiazepine (loading dose)
- Thiopental
- Propofol
- Opioids (loading dose)
- Succinylcholine

##### Dosage by Ideal Body Weight (IBW)

- Benzodiazepine (maintenance dose)
- Muscle relaxants
- Opioids (maintenance dose)

*IBW calculation:*

$$\text{Men} = 50 \text{ kg} + 2.3 \text{ kg} \times (\text{height [in]} - 60)$$

$$\text{Women} = 45.5 \text{ kg} + 2.3 \text{ kg} \times (\text{height [in]} - 60)$$

*Obese pts typically have ↑ cardiac output, ↑ volume of distribution*

#### Monitoring

- Indications for A-line: Hypoxemia, ↓ systolic fx, moderate & severely ↑ pulm artery press & inability to measure bp noninvasively
- ECG: May show RBBB 2° to pulmonary HTN
- DVT risk: ↓ Risk with pneumatic compression devices and/or SC heparin
- Equip: OR table must accommodate pt weight, capacities vary widely

#### Postop Complications

- ↑ Incidence of atelectasis & hypoxemia  
(consider use of semirecumbent position, CPAP or BIPAP)
- Negative-pressure pulmonary edema 2° to inspiration against closed glottis
- Accidental stapling of NG tube to pt stomach  
(prevent by keeping close communication with surgical team)
- DVT prophylaxis & early ambulation ↓ risk of thromboembolism

#### Anesthetic Management: Laparoscopic Surgery

##### General Considerations

- Advantages include smaller incision, ↓ surgical trauma, ↓ postop pain, ↓ pulmonary dysfx, ↓ postop ileus, faster recovery, & ↓ hospital stay
- 3 ports typically inserted into abdomen:  
(subumbilical port used for CO<sub>2</sub> insufflation to 12–15 mm Hg)

Physiologic Changes During Laparoscopy	
Physiologic Change	Mechanism
<b>Respiratory</b>	
↓ Lung compliance	Trendlenburg position, ↑ intra-abd pressure
↑ Ventilation/perfusion mismatch	↓ FRC
↑ Inspiratory pressures	Trendlenburg position, pneumoperitoneum
↑ PaCO <sub>2</sub> and ↓ pH	↓ Pulm perfusion, ↓ alveolar ventilation
<b>Cardiovascular</b>	
↑ Systemic vascular resistance, ↑ pulm vascular resistance, ↑ mean arterial pressure	Hypercapnia, ↑ intra-abd pressure, ↑ catecholamine release
↓ Venous return	Vena cava compression
↓ Cardiac output	↓ preload, ↑ afterload
<b>Neurologic</b>	
↑ Intracranial pressure	Trendlenburg position, ↑ cerebral blood flow due to hypercapnia
<b>Renal</b>	
↓ Urine output	↓ Renal blood flow; ↑ ADH secretion

#### Anesthetic Technique: Laparoscopic Surgery

- General anesthesia with endotracheal intubation and controlled ventilation
- Muscle relaxation to avoid further increase in intrathoracic pressure
- Rapid-sequence induction for antireflux procedures and patients with full stomach
- Persistent PETCO<sub>2</sub> despite adequate minute volume may signal subcutaneous emphysema
- Attenuation of hemodynamic changes to peritoneal insufflation:
  - Bradycardia → glycopyrrolate or atropine
  - Decreased C.O. & hypertension → use volume loading and/or vasopressor
  - Hypertension → use vasodilators

Causes of Hypotension During Laparoscopy	
• Reverse Trendlenburg position	• Venous gas embolism
• Bleeding & hypovolemia	• Tension pneumothorax
• High insufflation pressures	• Tension pneumoperitoneum
• Arrhythmias	• Pericardial tamponade
• Myocardial ischemia	

#### Monitoring

- Large-bore peripheral IV access (limited access to tucked arms during case)
- Orogastric tube to aspirate gas from stomach prior to trocar placement
- Acute ↑ in peak airway pressure may signal:
  - Endobronchial migration of tube (esp. with change to Trendlenburg)
  - Pneumothorax (usually accompanied by ↓ SpO<sub>2</sub>)
- Avoid ↑ peak airway pressure by using pressure-control ventilation
- Minute volume usually must be ↑ by 20% to maintain normocarbida
- Bradycardia following CO<sub>2</sub> insufflation likely vagally mediated
  - May also be 2° to hypercarbia & respiratory acidosis
- Avoid ↑ in insufflation pressure that can compromise venous return (max 12–15 mm Hg)

#### Postop Care

- Shoulder pain (suprascapular nerve irritation)—*treat with NSAIDs*
- Unrecognized intra-abdominal visceral/vascular injury
  - Progressive hypotension, ↑ abdominal girth, ↓ hematocrit
- ↑ Incidence of PONV
- Extensive subcutaneous emphysema may require mechanical ventilation

#### Large Intestinal Surgery

##### Indications

- Colon cancer, diverticulitis, ulcerative colitis, Crohn's dz, ischemic colitis, reversal of colostomy

##### Preop Evaluation

- Preop fasting + bowel prep = large fluid deficit
- Bowel obstruction can ↑ risk for gastric aspiration during induction

- Thoracic epidural analgesia (T8–12) ↓ atelectasis, ↑ early ambulation  
(*may contribute to hypotension in presence of hypovolemia*)

### Anesthetic Management: Large Intestinal Surgery

- Consider aspiration precautions if pt is obstructed
- Consider stress dose steroids if pts on preop steroids
- Fluid replacement must account for evaporative losses of exposed viscera
- Mesenteric traction syndrome: hypotension during bowel surgery from bowel-associated mediator release (vasoactive intestinal peptide)  
→ Hypovolemia, surgical bleeding, sepsis 2° to peritoneal fecal spillage

### Postop Complications

- Prokinetic agents (metoclopramide) can cause anastomotic dehiscence after colonic surgery
- Postop ileus caused by bowel manipulation, opioids, immobility, lack of enteral feeding, & bowel edema from fluid overload  
(*epidural analgesia may ↓ incidence of postop ileus*)
- Prolonged NG tube placement can → ischemic necrosis of nasal septum

### Small Intestinal Surgery

#### Indications

- Small bowel obstruction, neoplasms, intussusception, intestinal bleed, resection of carcinoid tumor, Crohn's dz

#### Carcinoid Tumors/Carcinoid Syndrome

- Carcinoid tumors typically asymptomatic  
→ May present with abd pain, diarrhea, & intermittent obstruction
- Metastatic carcinoid tumors (hepatic, pulm metastases) systemic symptoms  
→ Carcinoid syndrome: cutaneous flushing, bronchoconstriction, hypotension, diarrhea & rt-sided valvular lesions  
↑ 5-hydroxy-indole-acetic acid (>30 mg in 24-hr urine)
- Epidural analgesia may exacerbate intraop hypotension  
(*consider use of dilute local anesthetics/narcotics + volume loading*)

#### Monitoring

- Consider TEE for carcinoid (eval rt-sided heart lesions & guide fluid therapy)

#### Anesthetic Management

- Consider aspiration precautions/rapid-sequence induction for obstruction
- Carcinoid tumors
  - Avoid agents that release histamine  
(*thiopental, succinylcholine, atracurium, morphine*)
  - Octreotide (synthetic somatostatin) effective in relieving hypotension  
(*subcutaneous dose 50–500 mcg—half-life of 2.5 hr*)

#### Postop Care

- 50% of carcinoid deaths result from cardiac involvement
- Similar considerations as in large intestine surgery

### Pancreatic Surgery

#### Indications

- Pancreatic adenocarcinoma resection (Whipple: Pancreatojejunostomy with gastrojejunostomy & choledochojejunostomy)
- Treatment of complications of pancreatitis: infected pancreatic necrosis, hemorrhagic pancreatitis, drainage of pancreatic pseudocyst

#### Monitoring

- Pancreatic surgery can be assoc with significant blood loss & fluid shifts  
(*Consider A-line, CVP depending on pt comorbidities*)

#### Anesthetic Management

- Consider thoracic epidural analgesia (T6–T10) for postop pain control
- Often feeding tube tip will be adjusted by surgeon during procedure
- Pancreatic surgery for infection may be complicated by sepsis & ARDS  
*requires aggressive fluid resuscitation, vasopressor support (α-agonist, e.g., norepinephrine) & postop mechanical ventilation*

#### Postop Care

- Significant pancreatic resection → insulin insufficiency & new-onset diabetes

## Splenic Surgery

### Indications

- Splenic injury (blunt or penetrating trauma)
- Idiopathic thrombocytopenic purpura with splenic sequestration of platelets

### Preop Preparation

- Periop platelet transfusion **not** warranted (unless platelet count is  $<50,000/\mu\text{L}$  or clinical evidence of coagulopathy)

### Anesthetic Management

- Avoid drugs that interfere with platelet function (NSAIDs)

### Postop Care

- Pts should receive pneumococcal, *H. influenzae*, & meningococcal vaccines

## Hemorrhoidectomy & Drainage of Perirectal Abscess

### Anesthetic Management

- Procedures usually short, often in lithotomy/prone position
- Usually general anesthesia (consider LMA for lithotomy cases)
- Spinals may be used (hypobaric soln for prone case, hyperbaric for lithotomy)
- Deep plane of anesthesia provides sphincter relaxation

### Postop Care

- Postop pain can be severe → consider use of narcotics & NSAIDs

## Inguinal Herniorrhaphy

### Anesthetic Management

- Commonly done as an outpatient procedure
- Spermatic cord traction may initiate a vagally mediated bradycardia
  - MAC + local anesthesia most common approach
  - Spinal or general anesthesia may also be used

## Ventral Herniorrhaphy

### Preop Considerations

- Staged ventral hernia repair may ↓ incidence of postop respiratory failure (closure of large abd defects → pulm restriction)

### Monitoring

- Obtain large-bore IV access to replace evaporative fluid losses in large cases

### Anesthetic Management

- Consider epidural analgesia (T10–T12) for large ventral herniorrhaphy
- Usually done with general endotracheal anesthesia + muscle relaxation
- Smooth emergence impt. (no coughing/bucking) to avoid disruption of repair

## Appendectomy

### Preop Evaluation

- Consider preop IV hydration to replace fluid deficits (vomiting, poor intake)

### Anesthetic Management

- Performed via open or laparoscopic approach
- Consider taking aspiration precautions (rapid-sequence induction)

### Postop Care

- IV opioids usually sufficient for postop pain management

## Cholecystectomy

### Anesthetic Management

- Performed via open or laparoscopic approach
- General endotracheal anesthesia
- Opioid-induced biliary tract spasm
  - May interfere with interpretation of intraop cholangiograms
  - Can be reversed by naloxone, nitroglycerin, & glucagon
- Minimal blood loss unless abdominal vessel injury occurs

### Postop Care

- Lap cholecystectomy → less postop pain & earlier discharge (usually same day)

# ANESTHESIA FOR VASCULAR SURGERY

ROY SOTO

## Open Vascular Procedures

### Abdominal Aortic Aneurysm (AAA) Repair

- Indications: Symptomatic aneurysm, asymptomatic (if  $>5$  cm or growing  $>0.5$  cm/6 months)
- Morbidity: 5% periop MI risk; 35–40% mortality for ruptured AAA  
(risks of renal failure, ischemic colitis, spinal ischemia, & death all  $\uparrow$  if aneurysm ruptured)
- Approach: Supraceliac, suprarenal, infrarenal (depends on aneurysm extension)  
(risk of renal failure  $\uparrow$  if suprarenal clamp)
- Intraoperative derangements after x-clamping
  - $\uparrow$  In afterload ( $\uparrow$  LVEDP, LVEDV) & PCWP
  - $\uparrow$  In MAP, CVP  $\rightarrow$  HTN above the x-clamp
  - $\uparrow$  In PVR  $\rightarrow$  increased membrane permeability
  - 10–55%  $\downarrow$  in C.O. (infrarenal lowest, supraceliac highest reduction)
  - LV dilatation &  $\uparrow$  LVEDP  $\rightarrow$  subendocardial ischemia, LV failure, CHF, arrhythmias
- Potential hypoperfusion of
  - Abdominal viscera  $\rightarrow$  bowel ischemia
  - Kidneys  $\rightarrow$  renal failure ( $\uparrow$  risk with suprarenal clamp & x-clamp time  $>30$  min)
  - Extremities  $\rightarrow$  distal ischemia
  - Spinal cord  $\rightarrow$  spinal cord ischemia  $\rightarrow$  paraplegia (artery of Adamkiewicz arises from aorta: 15% originates between T5 and T8, 60% T9 and T12, 25% L1 and L2)  
(anterior spinal artery syndrome [1–40% incidence]  $\rightarrow$  supraceliac clamp  $\uparrow$  risk)
  - Release of x-clamp
    - $\downarrow$  Afterload & hypotension (due to  $\downarrow$  SVR & relative hypovolemia)
    - Return of potentially cool, acidotic blood to central circulation
    - Vasodilation: Blood from extremities releases vasodilating ischemia factors
    - Metabolic acidosis &  $\uparrow$  ETCO<sub>2</sub> &  $\downarrow$  SvO<sub>2</sub>
    - $\uparrow$  CVP from return of pooled venous blood
    - $\downarrow$  In spinal cord perfusion pressure (SCPP) 2° to  $\downarrow$  in distal aortic pressure  $\pm$   $\uparrow$  in CSF pressure 2° to brain hyperemia from x-clamp-induced HTN
- Preclamp Management
  - Preop evaluation
    - Supra- vs infraceliac
    - Coexisting diseases
    - Cardiac fx & history of CAD ( $\uparrow$  prevalence in TAAA/AAA pts)
    - Lab testing: electrolytes, BUN/Cr, Coags, CBC
  - Lines, monitors
    - 2 large-bore peripheral IVs
    - A-line: R radial for descending aortic surg, L radial for ascending aorta (TAAA); consider femoral A-line as well if planning to do bypass
    - Central line (usually an 8.5-Fr introducer) for volume infusion & CVP measurement
    - Consider PA line if suprarenal aneurysm or other significant cardiac history
    - Consider thoracic epidural
    - TEE may be helpful for early detection of cardiac ischemia (esp for high aneurysms)
    - Upper & lower body-warming blankets in place  
(lower should remain **OFF** until after reperfusion & stabilization)
    - Foley catheter: Goal urine output  $>0.5$  mL/kg/hr
    - PRBC in the OR; may also need FFP
- Management prior to clamping
  - Induction of GA: Try to maintain BPs near baseline  
(HTN can rupture aneurysm, hypotension can cause myocardial ischemia)
  - Control HR (usually with esmolol)
  - Double-lumen tube (DLT) for thoracic aneurysm  
(L-DLT may risk hemorrhage if aneurysm is eroding bronchial wall)
  - Consider deepening anesthesia prior to x-clamp to avoid HTN response
  - BP control: Nitroprusside (SNP) causes arteriolar dilation & MAP reduction; nitroglycerin (NTG) may prevent myocardial ischemia &  $\downarrow$  preload
  - Maintain relative hypovolemia to prevent HTN  $\uparrow$  inc in afterload &  $\downarrow$  risk of MI during x-clamp  
(do not overhydrate, use NTG/SNP)

- Preparation for clamp release
  - Gradually load with volume
  - Wean vasodilators & have pressors ready
  - Lighten anesthetic
- Postclamp management
  - Give fluid bolus, blood (if warranted)
  - Gradual release of clamp can ↓ hemodynamic changes
  - If severe hypotension results, reclamp & reassess
  - Pressors (phenylephrine) may be needed, although not usually given prophylactically
  - ↑ Ventilation
  - ABG before & after x-clamp removal (guide fluid & electrolyte management)
  - Monitor HCT & correct coagulopathies
  - Use standard extubation criteria (pts often stay intubated 2° large volume shifts)
- Preventing renal failure
  - Risk with supraceliac > suprarenal > infrarenal
  - Maintain renal perfusion pressure with highest possible MAP that myocardium will tolerate
  - Maintain intravascular volume
  - Consider mannitol (0.5 g/kg prior to x-clamping), furosemide, Ca<sup>2+</sup> blockers, dopamine, fenoldopam (*not proven effective*); bicarb drip
- Preventing spinal cord ischemia
  - SSEP monitoring—not useful (2/3 of cord is supplied by anterior spinal artery → motor)
  - Maintain highest MAP (distal aortic perfusion pressures) that myocardium can handle
  - Keep CSF pressures low (consider spinal fluid drain)
  - Consider shunt to maintain distal perfusion during x-clamp
  - Consider hypothermic CPB or circulatory arrest
  - Consider intrathecal papaverine
  - Consider administering steroids, barbiturates
  - Consider epidural cooling
  - Spinal cord perfusion pressure (SCPP)
    - SCPP = distal aortic pressure – (greater of spinal CSF pressure or CVP)
- If monitoring distal pressures, aim for SCPP >30 mm Hg; can drain CSF via lumbar drain, up to ~15 mL/15 min
  - (*risk of brainstem herniation with rapid or excessive CSF drainage → limit to ~75 mL*)
- Avoid excessive SNP (hypotension → ↓ perfusion, cerebral vasodilation → ↑ ICP transmitted to CSF)
- Avoid hyperglycemia (consider insulin infusion for glucose >200)
- Consider mild hypothermia (passive cooling to about 34°C)
- Other complications
  - Nerve injuries: Recurrent laryngeal nerve during thoracoabdominal repairs, brachial plexus injuries (poor pt positioning)

#### Thoracoabdominal Aortic Aneurysm (TAAA) Repair

- Management similar to AAA (see above) with following key points

#### Crawford Classification of TAAA (I–IV)

- I: Descending thoracic aortic aneurysm distal to subclavian artery
- II: Aneurysm originating at subclavian artery to distal abdominal aorta
- III: Aneurysm from mid–descending thoracic aorta to distal abdominal aorta
- IV: Abdominal aortic aneurysm (below the diaphragm)

#### Stanford Classification of TAAA (A–B)

- Type A: Intimal tear (acute) in aorta from ascending aorta to descending aorta
- Type B: Intimal tear (acute or chronic) in aorta from descending aorta down

#### Possible Associated Findings with TAAA

- Airway deviation/compression
- Tracheal deviation/compression
- Hemoptysis
- Esophageal deviation/compression
- Distortion & compression of central vasculature/anatomy
- Hemothorax & mediastinal shift
- Reduced distal perfusion

(Adapted from Dunn P. *Clinical Procedures of the MGH*. Lippincott Williams & Wilkins: Philadelphia, PA.)



### Anesthetic Management of TAAA

- A-line: Ascending aneurysm, usu. placed in L radial (innominate artery may be involved); Descending aneurysm, usu. placed in R radial (left subclavian may be clamped)
- Circ arrest: If utilized, will need to pack head in ice (cover monitors so they remain dry)
- TEE: Used intraop to detect intimal tear, coronary ostia, AI, assess embolic risk
- Neuroprotection: Thiopental 3–10 mg/kg (may offer benefit for cerebral protection)
- Partial bypass: May be used for descending aneurysms
- Ventilation: One-lung ventilation often employed
- Access: 1 large-bore peripheral IV (16- or 14-gauge) + 1 large-bore central line

### BP Control During TAAA

- If no bypass: Maintain SBP at baseline SBP +  $\frac{1}{2}$  of peak aortic x-clamp SBP
- If bypass: Maintain SBP at baseline SBP
- Can reduce proximal HTN during aortic clamp by  $\uparrow$  flow to pump &  $\downarrow$  flow to heart
- SNP should be used sparingly (or not at all) during aortic clamp (risk of  $\downarrow$  spinal cord & renal perfusion)
- $\downarrow$  Conc of volatile agent & turn off vasodilators before aortic unclamp
- Volume repletion with colloid, crystalloid, blood products before & after aortic unclamp

### Carotid Endarterectomy

- Indication: History of stroke, TIA, or significant arterial occlusion on angiography
- Morbidity: Incidence of concomitant CAD = 50%; periop mortality 1–4%
- Anesthetic techniques

#### Regional Advantages

- Pt can tell you of neurologic symptoms/deficits during surgery
- Less anesthesia required for pts with significant comorbidities
- Avoidance of coughing/bucking at case end
- Less postop hyper- & hypotension
- Potentially reduced ICU & hospital stay

#### Regional Disadvantages

- "A good general is always better than a bad regional"  
(if regional not working, pt may be uncomfortable, moving, & tachycardic)
- Some providers give "deep sedation" + regional anesthesia  
(eliminates benefit of awake detection of neurologic deficits)

#### Regional: Deep Cervical Block

- Technique: Inject anesthetic at C2, C3, C4 in line drawn between mastoid process & C6 transverse process; needle should have slight caudal angulation, contact transverse process, withdraw 2 mm & inject
- Potential complications:
  - Intravertebral artery injection
  - Horner's syndrome (sympathetic chain)
  - Hoarseness (recurrent laryngeal nerve)

#### Regional: Superficial Cervical Block

- Technique: Inject anesthetic just posterior to sternocleidomastoid (goal to spread anesthetic subcutaneously & behind SCM) at C6 level, & fanned 2–3 cm superior & inferior
- Easy technique with minimal risk & excellent efficacy

#### General Anesthesia: Advantages

- Potential for brain protection by volatile and intravenous anesthetics

#### General Anesthesia: Disadvantages

- Necessitates careful planning & drug management to avoid HTN, coughing, & bucking during emergence & extubation
- Can get hypotension (minimal surgical stim but must keep pt still)
- No proven mortality  $\downarrow$  with either technique (GA vs regional)

#### Intraoperative shunting

- Provides blood flow from common carotid artery to internal carotid artery (distal/superior to site of x-clamp)
- Indicated in pts with significant contralateral dz
- Stump pressure: Measurement of pressure distal to site of x-clamp, need to provide well-flushed A-line tubing over drape stump pressure  $<50$  mmHg = indication for shunting
- Risk of plaque dislodgement, intimal injury, & air embolus

- Hemodynamic management
  - Avoid tachycardia ( $\uparrow$  myocardial  $O_2$  demand) & hypotension ( $\downarrow$  coronary flow)
  - Maintain MAP slightly above baseline (optimizes collateral blood flow)
    - may be difficult to maintain normal MAP (minimal surgical stim)*
    - phenylephrine infusion  $\rightarrow$  ideal to maintain MAP without raising heart rate*
  - Consider nitroglycerin for reduction of BP at induction/emergence
    - esp in chronically HTN pts (may have wide swings in MAP)*
  - Consider esmolol/metoprolol to prevent tachycardia
    - intubation, reversal of neuromuscular blockade, extubation*
  - Consider A-line placement prior to induction in pts with known CAD
- Intraoperative brain monitoring has **not** been shown to improve outcomes
- CNS monitors:
  - Awake:  $\downarrow$  Cardiac morbidity & HTN, shorter ICU stay
  - EEG: May correlate with neuro changes
  - SSEPs: Sensitive, but intermittent indicator of cortical ischemia
  - Stump press poor sensitivity/specificity
  - Transcranial Doppler/brain oximetry/ $JvO_2$
- Perioperative complications
  - Brain hypoperfusion (avoid hyperglycemia)
  - Bradycardia (esp during carotid body manipulation)
    - can avoid with lidocaine infiltration by surgeon*
  - Intraoperative stroke (consider if delayed emergence/mental status change)
  - Hematoma: **Evacuate hematoma 1st, manipulate airway 2nd**
    - Diagnosis: Progressive stridor & subjective difficulty breathing; often difficult to see hematoma (dressings/patient size)
    - Treatment: **Pt back to OR stat**—if condition worsening, open wound *prior to airway manipulation*; attempts at intubation can be impossible (may result in airway swelling/bleeding, making situation worse)

#### Complications of Carotid Endarterectomy

- HTN: Damage to (or local anesthetic at) carotid sinus;  $\uparrow$  risk for neuro deficits compared to pts with normal BP (due to hyperperfusion); more likely with GA (vs regional)
- Hypotension: Removal of plaque  $\rightarrow$  inc stimulation of baroreceptors; more likely with regional
- MI: Most frequent cause of morbidity/mortality
- Stroke: Usually embolic
- Bleeding: Can lead to airway obstruction from hematoma or edema
- CNS injury: 10% pts; most common nerves—hypoglossal, vagus, recurrent laryngeal, accessory
- Carotid body damage:  $\downarrow$  Ventilatory response to hypoxemia/hypercapnia; esp. impt if 2nd side CEA

#### Endovascular Procedures

- Endovascular AAA repair
  - Monitoring for most limited to A-line (plus large-bore IV access)
  - Pressors/vasodilators usually not needed
  - Conversion to open procedure rate  $<5\%$  (should always anticipate this possibility)
  - Anesthetic options
    - General
      - Complex cases (inexperienced surgeon) or pt refuses regional/MAC
      - Always considered as backup for conversion to open procedure
    - Regional
      - Spinal: Duration of procedure usually precludes this
      - Epidural: Allows for ideal anesthesia of incision sites (bilateral femoral vascular access). But must be prepared to delay case if achieve bloody/traumatic tap or intravascular catheter
      - Regional techniques may  $\downarrow$  incidence of hypercoagulability & perioperative vessel clot formation (esp for lower extremity procedures)
    - Sedation
      - Ideal for thin pts (less dissection necessary) if surgeons apply local
      - Pt must remain still for hrs on uncomfortable fluoroscopy bed
      - Contrast induced nephropathy a concern ( $2^{\circ}$  to extensive angiography) (see below)

- Carotid stent placement
  - Requires immobile pt (minimal head/neck movement) & able to tolerate fluoroscopy table
  - Consider narcotic/ $\alpha$ -2 agonist technique (may avoid sedation-associated confusion)
- Distal angioplasty/thrombectomy
  - Pts with operative lower limb vascular dz have >50% incidence of concomitant CAD
  - Procedure times often long (on uncomfortable fluoroscopy bed) usually best to avoid long infusions/large doses of midazolam/propofol (*problem of confusion/disorientation*)
  - Always be prepared for conversion to open procedure
  - Regional techniques may ↓ incidence of hypercoagulability & perioperative vessel clot formation (esp for lower extremity procedures)

### Endovascular Safety Concerns

- Perioperative  $\beta$ -blockade: Current ACC guidelines – recommend perioperative  $\beta$ -blockade in vascular pts found to have myocardial ischemia on pre-op testing (*less strong evidence for pts with low/intermediate cardiac risk*)
- Transfusion triggers: Evidence suggests vascular pts allowed to bleed below a hemoglobin level of 10 mg/dL have ↑ incidence of periop myocardial ischemia
- Regional anesthesia & anticoagulation (see Chapter 6, on Regional Anesthesia)

### Contrast-induced Nephropathy (CIN)

- ARF after ischemia or contrast thought 2° to acute tubular necrosis from
  - Free-radical formation, which is promoted in acidic environment (e.g., renal medulla)
    - Contrast-related ↓ in renal blood flow
    - Atheroembolism
- Tips to Avoid CIN
  - Maintain plasma volume, good urine output
  - $\text{NaHCO}_3$  may be protective:  $\text{D}_5 \text{NaHCO}_3$  154 mEq/L (from pharmacy)
    - **Load:** 3 mL/kg over 1 hr, given 1 hr before contrast
    - **Maintenance:** 1 mL/kg/hr until 6 hr after procedure
    - Use 110 kg max weight for calculations
    - If bolus leads to significant HTN → stop bolus, diurese before injecting contrast, then resume infusion
  - N-acetylcysteine (free-radical scavenger)
    - 600 mg PO bid starting day before surgery and through day of surgery
- Risk Factors
  - Patient factors: Renal dz, diabetes, CHF, ↑ age, anemia, LV dysfx
  - Nonpatient factors: ↑ Osmolar or ionic contrast, contrast viscosity & volume

### Peripheral Vascular Surgery

- Preop risk: Patients often have significant comorbidities (↑ risk of associated CAD)
- Procedures: Bypass grafts (fem-pop, ilio-fem, etc.), embolectomy, pseudoaneurysm repair
- Monitoring: Invasive monitors per pt condition (hemodynamics often labile) (*place A-line in side opposite surgery*)
- Anesthetic
  - General anesthesia/regional/MAC
    - Epidural & GA → associated with comparable rates of cardiac morbidity
  - Continuous epidural/spinal
    - ↓ Incidence of postop vascular graft clotting (*Anesthesiology* 1993;79:422)
    - Continuous lumbar epidural catheter commonly used (occ spinal)
    - Awake pts can notify personnel of acute MI symptoms (chest pain)
    - Helpful for postop pain control
    - Intraop heparin after epidural placement does not ↑ risk of epidural hematoma
    - Epidural associated with ↓ incidence of reoperation for inadequate tissue perfusion (compared to GA) (*Anesthesiology* 1993;79[3]:422–434)

# ANESTHESIA FOR NEUROLOGIC SURGERY, NEURORADIOLOGY, AND ECT

JOSHUA H. ATKINS

## BASIC PRINCIPLES OF NEUROPHYSIOLOGY

### Cerebral Metabolic Rate (CMR), Cerebral Blood Flow (CBF), and Autoregulation

- Cerebral perfusion pressure (CPP) = MAP - ICP (or CVP if CVP > ICP)
  - Goal of CPP >60 mm Hg in normal pts
  - Higher pressures needed to achieve adequate tissue O<sub>2</sub> delivery in pts with existing brain pathology/elevated ICP (>70 mm Hg)
- Cerebral blood flow in healthy pts is autoregulated (MAP 50–150 mm Hg)
  - Global cerebral blood flow ~50 mL/100 g brain/min (~75% to gray matter)
  - In chronic HTN → autoregulation curve shifts to right
  - Modest hypotension may result in hypoperfusion & ischemia
- Extreme hypertension → large increases in CBF & ICP
  - Brain edema, hyperemia, & tissue injury from disruption of BBB
- Blood flow changes with cerebral metabolic rate
  - Brain O<sub>2</sub> delivery closely approximates demand (~50% of O<sub>2</sub> extracted at 1st pass)
  - Blood flow less than 15 mL/100 g/min → ischemia detectable by EEG
  - Anesthetics, temp, arterial PO<sub>2</sub> & PCO<sub>2</sub>, and pathophysiologic states influence relationship between autoregulation of CBF (see table below)
  - Displacement of brain tissue by surgical instruments impairs local perfusion
  - Global brain physiology differs from that occurring at regional/cellular level (e.g., mitochondria)

### Cerebral Blood Flow—Metabolic Rate Coupling (CBF & CMR)

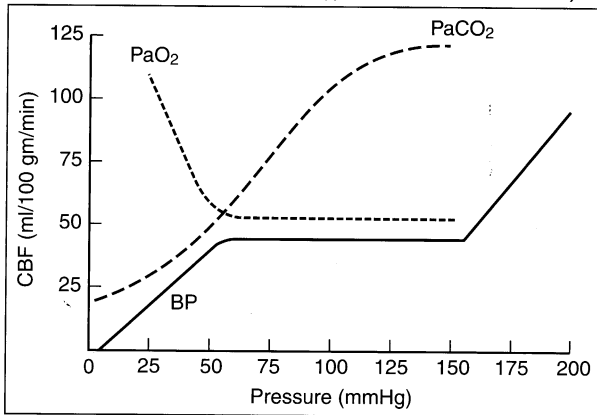
- PaCO<sub>2</sub> (normal = 20–80 mm Hg):
  - Hypercarbia: **CBF ↑, CMR ↔** Hypocarbia: **CBF ↓, CMR ↔**
  - CO<sub>2</sub> response impaired by inhaled potent agents, preserved by IV agents
  - CO<sub>2</sub> response curve flattened in presence of chronic hypercapnea
- Profound hypoxia (PaO<sub>2</sub> <50 mm Hg): **CBF ↑, CMR ↔**
- Temp: **CMR ↓ (5–7%/°C), CBF ↓**
- Potent inhalational agents: **CMR ↓, CBF ↑** (“uncouple” metabolic link to CBF)
  - Effects may be potentiated by ↓ PaCO<sub>2</sub> or IV agents
  - Agents also impair blood flow autoregulation
- Nitrous oxide: **CBF ↑, CMR ↑ (regional, not global)**
  - Effect on CMR may be attenuated by combination with other agents
- IV agents (barbiturates/propofol/etomidate/benzodiazepines)
  - CMR ↓, CBF ↓** (changes small with benzos & narcotics)
  - Generally preserve autoregulation and CO<sub>2</sub> responsiveness
  - Ketamine = unusual exception: **CBF ↑, CMR ↔** (may ↑ if used alone)
- Narcotics: Generally minimal effect on CBF and CMR
- Traumatic brain injury: heterogenous response, **CMR ↓, CBF ↑ (luxury perfusion)**
  - Luxury perfusion: CBF exceeds metabolic demand, usually after infarct
  - Basal metabolic rate: 3.5 mL O<sub>2</sub>/100 g/min
  - Avg brain: 1400 g; Avg CaO<sub>2</sub> = 20 mL O<sub>2</sub>/100 mL blood
  - Global blood flow = 50 mL/100 g/min; ischemic blood flow = 15 mL/100 g/min

### Intracranial Pressure (ICP)

ICP = cranial (closed space) pressure on brain, CSF, & blood components

- Brain components: brain mass/cells (80%); blood (10%); CSF (10%)
- Normal range: 0–10 mm Hg; CSF: 150 mL normal volume; 450 mL/d
- Increased ICP can lead to herniation & severe neurologic sequelae
  - Acute ↑ —shunting of CSF to spinal canal; ventricular compression
  - Further ↑ —compress brain tissue, mass effect, neuro deterioration
  - Severe ↑ —**Cushing's triad** (↑ BP + ↓ HR + irreg. resp.)
  - Herniation —pupil asymmetry, ocular paresis, obtundation, nausea

Figure 20-1. Relationship between cerebral blood flow in response to changes in PaCO<sub>2</sub> and PaO<sub>2</sub>. (From Dunn P. *Clinical Anesthesia Procedures of the Massachusetts General Hospital*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins 2006.)



## Management of Increased ICP

### 1. Hyperventilation

- Vasoconstriction of cerebral vasculature ↓ inflow
- ↓ Intracerebral H<sup>+</sup> conc promotes flow out of brain tissue
- Equilibration over a period of hrs will negate long-term benefits
- ↓ O<sub>2</sub> delivery > ↓ brain volume at PaCO<sub>2</sub> < 26 mm Hg
- Rapid normalization of PaCO<sub>2</sub> → brain edema & ↑ ICP

### 2. Head position

- Head-up position (15–30°) for drainage from jugular venous system  
*Often one of most effective interventions to ↓ brain volume*
- Avoid extremes of neck rotation & related venous engorgement

### 3. Direct drainage: ventriculostomy, lumbar drain

### 4. Blood pressure control: avoid hypertension (& extreme hypotension)

### 5. Pharmacologic agents

- Avoid high-dose inhaled potent agents (>0.5 MAC)
- Propofol, thiopental—↓ cerebral blood flow and CMR
- Diuretics—IV furosemide (0.1–1.0 mg/kg)  
*Potentiate effects of osmolar agents (block ion reuptake + diuresis)*

### 6. Osmotic therapy

- Relies on intact blood–brain barrier
- Mannitol (0.5–2 g/kg total dose) or hypertonic saline (HTS)
  - Comparative efficacy remains controversial
  - Hetastarch may be better choice in trauma resuscitation with hypovolemia/shock
  - Caution in setting of impaired cardiac fx (transient ↑ in extracellular volume) or unstable aneurysm
- Maintain osmolar gap (mannitol) < 20 or serum osmolarity < 320 mOsm/kg
- Mannitol—↓ viscosity & ability to scavenge radicals

## Potential Complications of Hyperosmolar Therapy

- Leakage of hyperosmolar soln into brain tissue with resulting ↑ in brain edema (late)
- Hypernatremia (with neurologic effects); hypokalemia
- Rapid overcorrection of a preexisting hyponatremia → central pontine myelinolysis
- Renal failure (from osmotic load to impaired kidneys)
- Pulmonary edema (due to intravascular fluid overload with impaired cardiac fx)
- Hypovolemia (from mannitol/furosemide diuresis + concomitant blood loss)

**Evoked Potentials****Types of potentials:**

1. EEG—depth of anesthesia, burst-suppression, isoelectric EEG, ischemia
  - Aneurysm clipping, AVM resection, carotid end-arterectomy
2. Somatosensory evoked potentials (SSEPs)—dorsal tracts & somatosensory cortex
  - Generated by peripheral nerve stimulation & central detection
  - Reliable but may not detect loss of motor function
  - Used in range of spine & intracranial procedures
  - Measurement compatible with most anesthetics  
(*Inhaled potent agents suppress more than IV agents*)
3. Brainstem auditory evoked potentials (BAEPs)—depth of anesthesia & VIII
  - Posterior fossa craniotomy, acoustic neuroma resection
  - Easy to monitor via sounds directly applied to ear
  - Difficult to suppress
4. Visual evoked potentials (VEP)—monitor visual cortex/optic nerve
  - Most sensitive to suppression, rarely used
5. Motor evoked potentials (MEP)—ventral motor pathways in the spine
  - Widely used in spine surgery, brainstem lesion resection
  - Craniotomy near motor cortex or descending tracts
  - Need to avoid long-acting muscle relaxation
6. Transcortical motor evoked potentials—motor function from cortex to endplate
  - Strict avoidance of inhalational agents
  - Etomidate/ketamine may enhance amplitude
  - Need to avoid profound muscle relaxation
7. EMG—motor potentials from spinal cord/nerves
  - Minimal effects of anesthetic agents

**Sensitivity to anesthetic agents:**

Cortical &gt; deep brain &gt; spine

Visual &gt; cortical motor &gt; deep motor &gt; spinal motor &gt; SSEPs &gt; auditory evoked

**Suppressant effects of anesthetic agents:**

1. Inhalational agents > propofol > etomidate > ketamine > opioids
2. N<sub>2</sub>O generally tolerated up to 50% when conc **STABLE** during monitoring
3. Consider TIVA (propofol/etomidate + narcotic) for easily suppressed potentials
4. Benzos in usual doses → minimal effects on monitored potentials

**EEG Frequency Ranges**

Delta rhythm (0–3 Hz)	Deep sleep, deep anesthesia, or pathologic states (e.g. brain tumors, hypoxia, metabolic encephalopathy)
Theta rhythm (4–7 Hz)	Sleep and anesthesia in adults; hyperventilation in awake children and young adults
Alpha rhythm (8–13 Hz)	Resting, awake adult with eyes closed, predominantly seen in occipital leads
Beta rhythm (>13 Hz)	Mental activity, light anesthesia

Source: From Bendo AA, Hartung J, Kass IS, Cottrell JE. Neurophysiology and neuroanesthesia. In Barash PG, Cullen BF, Stoeling RK, eds. *Clinical Anesthesia*, 2nd ed. Philadelphia: Lippincott, 1992:871–918.

**General Features**

- Mayfield pins & horseshoe headrest both commonly used
  - Pinning is highly stimulating
  - Anesthesiologist must anticipate (rather than react to) stimulation
  - Best to place invasive arterial monitoring prior to pinning
  - May inject pin sites with local anesthetic prior to pinning
  - May need to deepen anesthesia/control BP in anticipation of pinning (*may give IV lido, propofol, or nicardipine ± opioid 30 s prior*)
- Airway may be rotated away from anesthesiologist
- Soft bite block should be placed immediately after induction between molars
  - Tissue edema from tongue biting can form during surgery
  - Can prevent tube kinking from biting or positioning

- Eye protection
  - Secure taping of the closed eyelids
  - Careful application of ophthalmic ointment recommended to avoid physical & chemical injury of the cornea
- Plan early for IV access (arms out or tucked)
  - Decide early on need & site for intraop infusions
  - Infuse via visible IV (if possible) to ↓ unnoticed infiltration
- Main times of stimulation: intubation/pinning/incision/drilling/dural incision
- Avoid coughing/bucking/movement at any time
- Rapid blood loss is possible during any craniotomy
- Immediate postop assessment of neurologic function is crucial
  - Inability to perform neuro exam → immediate postop CT imaging
  - Avoid routine use of sedative medications (*confound postop assessment*)
- Opioids: Often used as part of a balanced-anesthesia technique
  - Aggressive use of narcotics may lead to delayed emergence
  - Give opioid dose early (i.e., induction, pinning, incision)
    - Fentanyl = short-acting & titratable (5–10 mcg/kg total dose)
    - Hydromorphone = longer-acting, give for postop analgesia
    - Morphine = sedating, slow emergence; avoid in intracranial proc.
      - *(morphine metabolites may accumulate in renal failure)*
    - Remifentanyl (0.1–0.5 mcg/kg/min) & sufentanil can be used as infusions
- Tight control of BP via invasive monitoring
- Fluid resuscitation
  - Normal saline is usually the crystalloid solution of choice (can ↑ CI → metabolic acidosis)
  - Avoid hypotonic or glucose containing fluids (can inc. brain swelling/injury)
  - Colloid used as indicated by clinical context
- Periop antibiotics usually indicated
- Anticonvulsant therapy
  - Indications for anticonvulsant & steroids vary with each pt
  - May potentiate neuromuscular blockers (acute) or antagonize NMB (chronic)
  - Anticonvulsant often used if contact with cortical tissue anticipated
    - IV phenytoin (dilantin) loading dose: 18 mg/kg in 250 mL NSS at 25 mg/min
      - *Caution: Rapid bolus dosing of phenytoin may cause significant cardiac arrhythmia, hypotension, & cardiovascular collapse*
    - Fosphenytoin (a phenytoin prodrug with ↓ side effects) loading dose: 15–20 phenytoin equivalent mg/kg in 250 mL NSS @ 50–100 PE mg/min
    - IV levetiracetam (keppra) loading dose: 1000 mg in 100 mL of normal saline over 15–30 min
      - Underlying renal failure, metabolic syndrome, & baseline meds should be considered prior to anticonvulsant admin
- IV dexamethasone (10-mg bolus, repeat 4 mg q 6 hr) for edema when indicated
  - Usually reserved for cases with intracranial lesion, elevated ICP, edema

#### Induction

- Controlled induction with hemodynamic stability
- Moderate hyperventilation in setting of ↑ ICP
- Combination of propofol 1–2 mg/kg, fentanyl 2–4 mcg/kg & nondepolarizing muscle relaxant is one approach
- Rapid, short-acting anti-HTN agents (esmolol, nicardipine) should be available
- Succinylcholine associated muscle fasciculation may result in transient ↑ ICP
- Coughing, bucking, or sympathetic surge during laryngoscopy with ↑ BP may cause sudden & untoward ↑ ICP (can use lidocaine 1 mg/kg to prevent)
- Physiologic manifestations of light anesthesia may be devastating in pts with intracranial aneurysm susceptible to rupture

#### Maintenance

- May use amnestic dose (0.5–1.0 MAC) of inhalational agent to ↓ brain volume
- Narcotic bolus as needed up to a predetermined estimate of total dose
- Consider TIVA if monitoring evoked potentials or if need to ↓ brain volume
  - Consider using a processed EEG monitor to guide TIVA dose
  - Common strategy
    - Propofol infusion for amnesia & brain relaxation  
(*Stop infusion once 1° resection complete to expedite emergence*)
    - Narcotic infusion for analgesia & immobility  
(*if muscle relaxation contraindicated because of monitoring*)
- Provided an appropriate anesthetic depth has been reached, HTN should be liberally treated with labetalol to decrease likelihood for emergence hypertension

- Closely monitor urine output & resuscitate with normal saline
- Monitor glucose & treat glucose >160 mg/dL with insulin
  - Hyperglycemia may predispose to or exacerbate neurologic injury
- If not contraindicated by monitoring requirements, maintain muscle relaxation to one twitch with bolus or infusion of nondepolarizing muscle relaxant
- Maintain MAP within 20% of baseline
- N<sub>2</sub>O may be used with several caveats:
  - May complicate EEG or potential monitoring if level not constant
  - May ↑ cerebral blood flow & contribute to brain swelling/↑ ICP
  - May contribute to pneumocephalus (particularly in posterior fossa proc.) or pneumothorax expansion (esp. in trauma)
  - May have deleterious effects on neuronal cells (under investigation)

#### Emergence

- BP control is critical
  - HTN episodes at emergence & postop may cause ↑ bleeding or edema
  - Treat with labetalol or nicardipine
- Prophylaxis for nausea/vomiting recommended (ondansetron)
  - Avoid promethazine, droperidol, diphenhydramine (can be sedating)
- Emergence should begin **after** Mayfield pins have been removed
- Time should be allowed (5 min) for head wrapping prior to extubation
- Extubation: Reaches baseline mental status
- Small boluses of propofol (10–40 mg) or lido (10–30 mg) can smooth emergence
- Continuous vital sign monitoring during transport

#### Special Features of Operations Involving the Posterior Fossa

- ↑ ICP commonly a concern (due to obstruction by a posterior fossa mass)
  - Consider aggressive treatment of ICP prior to induction
  - In severe cases, consider ventriculostomy under local anesthesia
  - ↑ ICP may occur if head down during positioning/surgical prep
- Prone, head-up position—concern for air embolus
- Potential for postop, compressive pneumocephalus
- Kinking of ETT with neck flexion in pins (**MUST place bite block**)
  - Postextubation macroglossia, tongue ischemia/injury
- Proximity to critical neurologic structures involving regulatory centers
  - Brainstem/medulla/pons regulatory centers
  - Bradycardia, apnea, rapid swings in BP may occur
  - Possibility for new postop deficits
- Small operative window may lead to inc. brain swelling
  - Jugular venous drainage/head position

**Sitting Craniotomy:** Lounge chair, back elevated 60°; hips & knees flexed

#### Advantages

- Operative exposure with ↓ brain swelling/volume, used for:
  - Posterior fossa lesions/cerebellar tumors
  - Pineal gland tumor resection
- Avoidance of prone position & improved ventilation dynamics

#### Disadvantages

- Risk of air embolus & paradoxical air embolus (PFO/septal defect)
  - Signs: Drop in ETCO<sub>2</sub>, hypotension, tachycardia, ↑ ETN<sub>2</sub>
  - Monitoring: precordial Doppler or continuous TEE
  - Access: Place central venous access (for air extraction)
    - May be placed via antecubital vein
    - Multiorifice catheter preferred (more effective)
- Quadriplegia from extreme neck flexion & ↓ perfusion
- Hypotension from venous pooling & head elevation may result in brain ischemia (within apparently acceptable BP limits)
  - Consider transduction of arterial BP at level of tragus (middle of ear)
  - Maintain BP ± 20% baseline MAP
  - Consider support stockings to ↓ venous pooling
- Nerve injury due to positioning (legs, arms, neck/brachial plexus)



## Awake Craniotomy

- Used for resection of tumors (motor or speech cortex) & epileptic foci
- Team approach with emphasis on communication, patience, & experience
- Critical to set pt & surgeon expectations
- Awake with variable sedation versus “asleep → awake → asleep” with LMA/ETT
  - Preference varies by center & experience
  - Simplest method is to avoid airway instrumentation
  - Only period of intense stimulation is opening/drilling/dural incision
- Patient comfort
  - Positioning with padding/pillows for optimal comfort
  - Vasoactive substances (nitroglycerin) may cause profound headache
  - Warming blanket as needed for patient comfort
  - Placement of Mayo stand over head to lift drapes off of the awake patient
- Preparation
  - IV access, monitors, & arterial line prior to blocks
  - Consider bilateral nasal trumpets (28–34 Fr) connected to O<sub>2</sub> for airway management
    - (*topical anesthetic & vasoconstrictors to nares prior to placement*)
- Consider peripheral nerve blocks
  - C.N.V<sub>1</sub>—supraorbital, supratrochlear n.
  - C.N.V<sub>2</sub>—auriculotemporal, zygomaticotemporal n.
  - Cervical branches—posterior auricular, greater & lesser occipital n.
  - Remifentanyl infusion for analgesia during block placement
  - Choice of local: Use long acting ropivacaine 0.375% with 1:200,000 epi)
  - Bupivacaine in large volume may have increased risk of cardiac toxicity
  - Allow for additional local to be infiltrated by surgeons
  - Metoprolol prior to block to blunt tachycardia
- Maintenance
  - Deeper sedation only during drilling & opening of bone flap
  - Minimize sedation just before dural incision
  - Monitor CO<sub>2</sub>—hypercarbia can contribute to brain swelling
  - Propofol/remifentanyl with spontaneous ventilation
  - Dexmedetomidine infusion is a useful adjunct
    - *Minimal resp. depression, can cause hypotension/bradycardia*
    - *Pretreat with atropine*
    - *Load 1 mcg/kg over 15 min & infuse 0.3–0.7 mcg/kg/hr*
  - Neuromonitoring
    - Map functional cortex—usually speech area
    - Requires continuous patient feedback, communication, contact

## INTRACRANIAL VASCULAR SURGERY: Aneurysm Clipping & Resection of Arteriovenous Malformations (AVMs)

### Preoperative Evaluation

- Where is the aneurysm? Has pt had subarachnoid hemorrhage (SAH)?
- Determine surgical approach & location of incision
- Need for CSF drainage (improve access/visualization)?
- Need for intraop angiography?
- Evidence of cerebral salt wasting or SIADH after SAH (electrolytes, urine output)?
- Document baseline ECG: Changes common with SAH
  - (*May be assoc. with ↓ cardiac fx—esp. Q waves*)
- In emergent surgery for hemorrhage, FFP or platelets for clinical indications
  - (*e.g., chronic anticoagulation, aspirin, plavix therapy*)

### Induction & Pinning

- Avoid profound HTN or light anesthesia
- Consider topical lidocaine prior to intubation
- Lidocaine (without epinephrine) at pinning sites prior to Mayfield application
- Potential for catastrophic bleeding; ensure large-bore IV access
- Lumbar drain may facilitate brain decompression & better surgical access
  - (*Rate & timing of CSF drainage must be coordinated with surgeon*)

### Maintenance

- BP management = critical
  - A-line mandatory; maintain ±10% baseline BP during dissection
  - If HTN → inhalationals, nicardipine, esmolol, nitroprusside, NTG
  - If hypotensive → phenylephrine, ephedrine
  - ↓ BP at surgical request during direct aneurysm manipulation
  - ↑ BP to baseline MAP during ischemic periods (temporary clipping)

- Avoid extreme hypo- or hypertension at all points
- Consider brief, deliberate hypotension during massive bleeding  
(to facilitate surgical localization & control)
- Many surgeons utilize both EEG & intraoperative cerebral angiography
- Preop placement of vascular sheath in femoral artery
- Sheath must be monitored & transduced at all times (including transport)
- Pt sedated for sheath placement
- Preop embolization of feeding vessels to AVM may be performed  
(Will limit potential for catastrophic blood loss)
- Debate over transfusion thresholds in aneurysm surgery with vasospasm
- Vasospasm usually occurs days after initial bleed
- Hemodilution (↓ blood viscosity) vs. transfusion (↑ O<sub>2</sub> delivery)
- Profound, deep anesthesia may be neuroprotective during periods of ischemia
- Strategy for periop brain protection controversial
- Burst suppression may be protective in regional ischemia
  - Can use high-dose propofol or barbiturate infusion, titrate to EEG
- High-dose sedative-hypnotics to achieve burst suppression may result in
  - Hypertriglyceridemia & metabolic acidosis (propofol)
  - Propylene glycol toxicity (etomidate)
  - Delayed emergence (all agents)
  - Hypotension (all agents)
- Data inconclusive regarding deliberate hypothermia
  - Pt populations, cooling & rewarming strategies highly variable
  - Hypothermia (35–36°C) may be an option if intraoperative ischemia is anticipated or witnessed global ischemia prior to OR
  - Rewarm prior to extubation/emergence
  - Avoid hyperthermia (↑ CMR/injury)

## VENTRICULOPERITONEAL SHUNT PLACEMENT/OMAYA RESERVOIR

**VP shunt:** Catheter in lateral ventricle to relieve ↑ ICP by continuous CSF drainage

- Proximal catheter placed into ventricle via burr hole
- Distal catheter (shunt only) placed into peritoneal cavity
- Requires GA, usually with muscle relaxation  
(Submucosal catheter must be passed from neck to abdomen)
- Minimal narcotic requirements for postop pain control
- Goal = prompt recovery of preop mental status with extubation

**Omayia reservoir:** Intraventricular catheter for CNS delivery of chemotherapeutic agents

- May be performed under local with sedation or GA
- Removal rarely requires more than local with light sedation

## EPIDURAL, SUBDURAL, & INTRACEREBRAL HEMORRHAGE (ICH)

- Wide range of etiologies for intracranial bleed
- Trauma—may present with wide range of hemorrhage
  - Consider other injuries
  - ICP often severely ↑ requiring immediate decompression
  - Blood–brain barrier often disrupted
  - Normal autoregulatory mechanisms may be dysfunctional
- Subdural—often present in older pts from vein shearing after fall
- Epidural almost always surgical emergency
- ICH may represent sentinel bleed from aneurysm, AVM, or other pathology/trauma
  - Categorized by Hunt/Hess grade
    - Grade I = asymptomatic/minimal headache; Grade V = deep coma, decerebrate rigidity
    - May have prognostic value, influences timing of surgery
- Be prepared for ongoing blood loss & need for resuscitation/transfusion
- Consider preop volume loading
- Potential for severe pre- & postop brain swelling
- Intracerebral blood = strong stimulus for vasospasm
- “Triple H” therapy (empiric institution of HTN, hypervolemia, hemodilution) may be indicated for ICH
  - Maintain MAP 30–50% above baseline
  - Maintain CVP at high normal levels
  - Raise transfusion threshold (unless ↑ cardiovascular risk)
  - Exercise caution in setting of unstable aneurysm/AVM

## TRAUMATIC BRAIN INJURY

- Unique mechanism of injury (diffuse & focal brain damage)
- Unique pathophysiology of dz
  - BBB disruption, vasoplegia, stress response
  - Associated cardiovascular instability
  - Associated pulm failure (ARDS, lung/cardiac contusion, edema)
- Often present with multiple life-threatening injuries
- ICU management crucial for good outcomes
- Glasgow Coma Scale score characterizes severity & may predict outcome
- Usually present for decompression or clot evacuation

<b>Glasgow Coma Scale (3–15 points)</b>		
<b>Best Eye Opening</b>	<b>Best Verbal Response</b>	<b>Best Motor Response</b>
Spontaneous = 4	Oriented = 5	Obeys commands = 6
To speech = 3	Confused = 4	Localizes pain = 5
To pain = 2	Inappropriate = 3	Withdrawals = 4
None = 1	Incomprehensible = 2	Flexion to pain = 3
	None = 1	Extension to pain = 2
		None = 1

**Score of  $\leq 8$  indicates coma & requires intubation**

**Age-adjusted normal scores:**

- 0–6 mo → 9
- 6–12 mo → 11
- 1–2 yr → 12
- 2–5 yr → 13
- >5 yr → 14

- ↑ ICP usually a pressing concern
  - May present to OR for emergency decompressive hemicraniectomy or clot evacuation (epidural/subdural/parenchymal hematoma)
  - Maintenance of CPP: Aggressive fluid resuscitation & BP management
  - Intractable ↑ ICP common & requires aggressive intervention
  - Regional brain tissue oximeters (e.g., Licox) of unproven clinical utility
  - No uniformly defined target CPP, individualize to clinical context (common goal >60)
- Central diabetes insipidus a common finding
  - Monitor for high-volume urine output (>300 mL/hr)
  - Measure serum/urine osmolality, serum sodium
  - May treat empirically with arginine vasopressin
- May consider institution of hypothermia for brain protection
  - Early hypothermia may be protective (developing literature)

## ELECTROCONVULSIVE THERAPY (ECT)

### Goals

- Amnesia, pt immobility, hemodynamic stability
- Provide conditions for therapeutic seizure duration (>30 s)
- Treat prolonged seizure (2–3 min) with pharmacologic agents
- Rapid recovery for discharge from recovery area

### Agents

- Many short-acting hypnotic agents have been used
  - Usually avoid benzos (raise seizure threshold)
  - Propofol (may shorten seizure duration), methohexital, thiopental, etomidate, sevoflurane, remifentanyl have all been used
  - First line: Methohexital (0.75–2 mg/kg) or etomidate (0.15–0.3 mg/kg)
- Short-acting muscle relaxation with succinylcholine (1 mg/kg) unless contraindicated
- Seizure threshold usually ↑ with number of treatments
- Propofol or benzodiazepines are reasonable choices to treat prolonged seizure

### Management

- If pt has received prior ECT treatment with adequate seizure activity
  - Consider using same anesthetic regimen (must document!)
- Preop eval as with any other general anesthetic
  - Pts with cardiac dz → the sympathetic surge (HTN/tachycardia) after seizure must be controlled

- Pts with  $\uparrow$ ICP  $\rightarrow$  may be at risk for acute decompensation (sudden  $\uparrow$  in cerebral blood flow associated with ECT)
  - Consider hyperventilation prior to stimulus
- Pts with cerebral aneurysm  $\rightarrow$  need tight BP control
- History of "awareness" with prior procedure should be fully evaluated
  - Processed EEG monitors may be useful
- Excessive anesthesia may  $\uparrow$  seizure threshold or  $\downarrow$  seizure duration
- Inadequate anesthesia may lead to awareness under anesthesia
- Monitors should include all routine monitors + end-tidal  $\text{CO}_2$  sampling
- Emergency drugs & airway equipment must be immediately available
- Preoxygenation/denitrogenation as with any general anesthetic
- Inflate BP cuff on calf of one leg **prior** to admin of succinylcholine
  - Allows monitoring of seizure activity
- **Place bite blocks between molar teeth on both sides prior to stimulation**
- Confirm loss of consciousness prior to administration of muscle relaxant
- Post-ECT confusion/memory loss/agitation is common
- Bradycardia is common during electrical stimulus
  - History of bradycardia may be (pre-) treated with glycopyrrolate
  - In pregnant patients, ECT may induce a transient fetal bradycardia
    - Fetal heart rate monitoring often used in 3rd trimester
    - ECT may induce labor & require tocolysis
    - Unless pt is in active labor, late-stage pregnancy, or has other risk factors for aspiration, intubation not typically required
- HTN/tachycardia common after ECT
  - May treat with IV labetalol, esmolol, & nicardipine
  - If pt has known profound hemodynamic response, consider pretreatment
- Some pts display profound salivation after ECT
  - Glycopyrrolate (0.1–0.2 mg) pretreatment may limit this effect
  - Use of atropine may exacerbate postictal confusion/memory loss

### Myasthenia Gravis (MG)

*Etiology:* Autoimmune antibodies against nicotinic cholinergic receptors

*Symptoms/Signs:* Laryngeal weakness  $\rightarrow$  dysphagia, dysarthria; extraocular muscle weakness  $\rightarrow$  diplopia, ptosis; skeletal muscle weakness  $\rightarrow$  worsens with activity

*Treatments:* Anticholinesterases, steroids, plasmapheresis, thymectomy

#### Risk Factors for Postoperative Respiratory Failure

Coexisting lung dz; Vital capacity  $<2.9$  L

Disease duration  $>6$  years

Pyridostigmine dose  $>750$  mg/day

Poorly controlled disease

#### Preoperative Considerations:

- Assess degree of weakness & duration of symptoms; maintain anticholinesterase therapy
- Anticholinesterase overdose  $\rightarrow$  *cholinergic crisis*  $\rightarrow$  further weakness
  - treatment: Anticholinergic administration (i.e. edrophonium)

#### Anesthetic Management:

- Minimize sedatives / respiratory depressants; consider regional
- Consider rapid sequence induction (pts at  $\uparrow$  risk of aspiration)
- Avoid muscle relaxants if possible & delay extubation if needed
- Use caution when using neostigmine ( $\uparrow$  risk of cholinergic crisis)

### Eaton-Lambert Syndrome

*Etiology:*  $\uparrow$  Release of acetylcholine due to Ca channels antibodies; assoc. with lung ca.

*Symptoms/Signs:* Proximal limb weakness, exercise improves strength,  $\downarrow$  reflexes

*Response to muscle relaxants:* Sensitive to depolarizing & non-depolarizing drugs

### Multiple Sclerosis

*Symptoms:* Visual disturbances, limb weakness, paralysis, respiratory failure, bulbar palsy

$\uparrow$  risk of aspiration +  $\downarrow$  airway reflexes = risk of postop resp. failure

Neuraxial blockade (spinal) assoc. with worsening symptoms; epidurals are *not* contraindicated

### Guillain-Barre Syndrome

*Symptoms:* ascending paralysis, may require vent. support,  $\uparrow$  aspiration risk, autonomic dysfx

Consider RSI, avoid succinylcholine, minimize muscle relaxants & opioids

### Parkinson's Disease

Loss of dopaminergic fibers  $\rightarrow$  unopposed acetylcholine activity

Avoid dopamine antagonists (haldol, promethazine, prochlorperazine, metoclopramide)

# ANESTHESIA FOR OTOLARYNGOLOGY (ENT) AND OPHTHALMOLOGY

JOSHUA H. ATKINS

## PROCEDURES IN OTOLARYNGOLOGY

### Functional Endoscopic Sinus Surgery (FESS)

- Surgery performed via nasal passages with endoscopic equipment
- May employ real-time CT guidance technology
- Usually short surgical procedure (longer for complex surgical indications)

#### Indications

- Recurrent sinusitis, abnormal transnasal breathing
- Epistaxis due to aberrant vessels/malformations (e.g., Osler–Weber–Rendu/hereditary hemorrhagic telangiectasia)
- Resection of skull base tumor
- Rhinorrhea for repair of CSF leak
- Anatomic access for resection of pituitary adenoma

#### Special Considerations

- CT guidance (head band); some centers also use intraop CT scanning
- Position: Head always rotated away from anesthesiologist, arms tucked for surgical access, ensure careful padding of ulnar n.
- EBL: Usually minimal, ↑ for skull base tumor resection/epistaxis treatment
- Hemodynamic changes assoc. with use of trans-nasal vasoactive substances
  - Oxymetazoline pledgets, lidocaine with epi, topical cocaine
  - Use intraop  $\beta$ -blockers with caution in conjunction with nasal vasoconstrictors
- Deliberate hypotension/controlled normotension in appropriate candidates
- Surgical instruments close to neural structures (cribiform plate, optic nerve)
- Pain: minimal postop narcotic needs

#### Anesthetic Management

- GA with ETT (standard or oral RAE tube), controlled ventilation preferred
  - Provides airway protection from blood & irrigation
  - Inhaled potent agent or total intravenous anesthesia (TIVA) is acceptable
- Flexible LMA or MAC anesthesia may be used in selected candidates
  - Movement during MAC or spontaneous ventilation with LMA can interfere with alignment of real-time CT guidance system
  - Surgeons need full access to nares (no trumpets, NG tubes, nasal temp probes)
  - Oral temp probe may be inaccurate owing to irrigation
  - Ophthalmic ointment to eyes
- Avoid tape across mandible (secure to L side with benzoin adhesive)
- Warming blanket & Foley catheter for longer procedures
- Periop antibiotics & dexamethasone may be indicated
- Short closure times
- Nasal packing may impair postop breathing
- Monitor for postextubation bleeding into posterior oropharynx/hypopharynx
- Thorough but gentle suctioning & passage of OG tube prior to extubation will remove accumulated surgical debris & blood
- Avoid postop CPAP/BiPAP, particularly in cases of CSF leak repair

### TIVA or Potent Agent for ENT Procedures

#### Advantages of total intravenous anesthesia

1. May ↓ bleeding in operative field
2. May ↓ operative time spent clearing field to achieve adequate visualization
3. May ↓ emergence coughing & avoid disruption of surgical homeostasis/dura repair
4. May ↓ postop nausea & vomiting if propofol used

#### Disadvantages of total intravenous anesthesia

1. Depth monitoring: Greater interindividual pharmacodynamic & pharmacokinetic variability (compared with potent agents)
2. May ↑ risk of awareness if muscle relaxation employed
3. Processed EEG monitoring difficult (interference from forehead CT image sensors)

(continued)

## TIVA or Potent Agent for ENT Procedures (Continued)

4. Undetected IV infiltration → potential for awareness & soft tissue necrosis
5. ↑ Cost of infusion medications

### One approach

- Propofol (50–150 mcg/kg/min) + remifentanyl (0.1–0.3 mcg/kg/min) infusions
- Low dose potent-agent (e.g., 0.3 MAC desflurane) as amnestic adjunct
- Agents to support BP (e.g., phenylephrine infusion) as needed
- Muscle relaxation may be indicated by surgical need/movement concerns

## CSF Leak Repair

Often performed via endoscopic sinus approach for spontaneous leak with rhinorrhea

- Usually requires intrathecal injection of fluorescein (aid in localization under FESS)
- May place lumbar drain after induction for 48- to 72-hr postop CSF drainage
- CSF opening pressure may be of prognostic utility
- Surgeon may request periop meningococcal meningitis prophylaxis (e.g., ceftriaxone)

## Microdirect/Suspension Laryngoscopy

- Performed by otolaryngologist for a range of indications
- Employs specialized laryngoscopes for exposure of anatomy/pathology
- May use robot-assisted techniques & laser devices
- Procedure is highly stimulating for relatively brief periods
- Pts often have difficult airways & significant comorbidities

### Indications

- Tumors of larynx, oral cavity, pharynx, hypopharynx
  - Biopsy, laser ablation, robotic assisted micro-resection
- Vocal cord surgery
  - Resection of vocal cord polyp
  - Vocal cord injection for cord paralysis
  - Insertion of mechanical larynx (artificial voice box)
- Tracheal stenosis—dilation/ablation of lesions
- Laser ablation/direct chemotherapy of papilloma

### Special Considerations

- Preop discussion with surgeon regarding airway management
- Potentially difficult airway
  - Prior surgery with scarring or postradiation changes (immobile larynx)
  - Supraglottic/laryngeal masses or tracheal abnormalities
  - Friable tissue → bleeding
- Positive-pressure mask ventilation may be challenging/impossible
- Airway = operative field & bed = rotated away
- Anesthetic gases may leak to environment/surgeon (open system)
- Intermittent apnea may be required for surgical access
- ETT may distort surgical anatomy & impede surgical access
- Laser ablation may be used (requires ↓ FiO<sub>2</sub>)
  - Use jet ventilation, apneic technique or laser tube
  - Fill laser tube balloons with methylene blue saline
  - Use airway fire protocol
- Surgeon may desire spontaneous ventilation (assess vocal cord movement)
- Intense but fleeting/intermittent stimulus
  - Requires constant communication between surgeon & anesthesiologist

### Anesthetic Management

- GA usually indicated (owing to intense procedure stimulus)
- Sedation & spontaneous ventilation in selected cases (with cooperative pts)
  - Requires anxiolysis & extensive topicalization with local anesthetic
- Anesthesiologist often induces GA & shares airway management with surgeon
  - Surgeon should be present prior to induction of anesthesia
- Airway management includes a variety of options
  - ETT (e.g., 5.0–6.0 mm I.D.) placed under laryngoscopy; larger if bronch planned
  - Catheter for subglottic jet ventilation placed under direct visualization (see text box, page 21–5) or jet via specialized laryngoscope
  - Intermittent apnea with mask ventilation
  - Airway device (if used) may be periodically removed for surgical access
- TIVA technique preferable to inhaled agent
  - ↓ OR contamination with inhalation gas

- More consistent depth of anesthesia
- Propofol & titratable, short-acting narcotic often used
- Muscle relaxation must be individualized for each case
  - Consider airway management, operating conditions, need for spontaneous ventilation
  - Inhalational induction may be considered

### **Medialization Thyroplasty** (Vocal Cord Medialization)

- Procedure performed to treat vocal cord paralysis/bowing
- Partial resection of thyroid cartilage & prosthesis placement

#### *Special Considerations*

- Pt cooperation = important component
- Anesthesia best provided with sedation & local injection
  - Pt able to phonate on command
  - Vocal cord movement observed under nasopharyngeal laryngoscopy
  - Surgical incision similar to thyroidectomy

## **PROCEDURES ON THE INNER EAR AND MASTOID**

### *Indications*

- Mastoidectomy/tympanoplasty for recurrent infection
- Cochlear implant for neurodegenerative hearing loss
- Myringoplasty/myringotomy tubes for infection
- Stapedectomy for conductive hearing loss/otosclerosis

### *Special Considerations*

- Multimodal prophylaxis for high-risk of nausea and vomiting (e.g., scopolamine patch, dexamethasone, and ondansetron)
- Nerve monitoring (VII/VIII); avoid deep paralysis
- N<sub>2</sub>O off before tympanic membrane closure
  - Potential for rapid expansion of airspace by N<sub>2</sub>O diffusion
- Pre- & postop communication challenges with hearing loss
  - Replace hearing aid in nonoperative ear/use hand gestures/preop coaching

### *Anesthetic Management*

- Best performed with GA & ETT (except for stapedectomy)
  - LMA can be used in selected pts
- Position: Table usually 180° away from anesthesiologist; airway covered/inaccessible
- Surgical manipulation of head must be expected during procedure
- N<sub>2</sub>O off prior to closure of tympanic membrane
- Copious local anesthetic used (lessens need for opioids)
- TIVA with propofol may reduce PONV
- Muscle relaxation frequently avoided to facilitate nerve monitoring

### **Stapedectomy**

- Usually sedation with local anesthesia (GA for selected pts)
- Sedation allows for intraop testing of hearing acuity
- Titrate meds (fentanyl, midazolam, propofol, dexmedetomidine) to allow pt cooperation
- Excessive sedation may lead to disinhibition & movement  
(precludes safe operating under the microscope)
- Some centers are investigating use of pt-controlled sedation

### **Myringotomy Tube Placement** (Placement of Ear Tubes)

- Very short procedure, usually performed in pediatric pts under mask GA
- IV access not necessary; can use IM analgesics (ketorolac & fentanyl)

### **Tonsillectomy and Adenoidectomy**

#### *Indications*

- Recurrent infection
- Obstructive sleep apnea due to hypertrophic tonsillar/adenoid tissue

#### *Special Considerations*

- Potential for difficult mask/airway—particularly in adults
- Consider oral RAE tube, secure in midline
- Procedure usually indicated owing to recurrent infection
  - May be semiurgent even in setting of active infection
- Short procedure necessitates careful use/titration of muscle relaxants
- Surgeon removal of mouth gag may result in extubation—monitor closely
- “Bring back” tonsil for bleeding common
  - Aggressive preinduction volume resuscitation (esp pediatric patients)
  - RSI or plan for potentially difficult airway (blood in airway & edema)

## Parotidectomy

- GA with ETT; consider nasal RAE on opposite side of lesion
  - Nasal tube precautions (oxymetazoline to nares, gentle dilation, tube sizing)
  - Always a risk of significant bleeding with nasal tube placement
- Facial nerve monitoring; avoid additional muscle relaxation after induction

## Uvulopalatopharyngoplasty (UVA)

- Performed for treatment of obstructive sleep apnea
- Airway management: Mask ventilation/intubation may be difficult
- Review sleep study results—apnea/hypopnea index for severity
- Consider RAMP positioning for obese patients
- Pts may require noninvasive ventilation in PACU/floor postop

## TRACHEOSTOMY

### Indications

- Ventilator-dependent resp failure
- Chronic aspiration
- Airway tumor/injury with airway compromise
- Acute stridor/bilateral vocal cord paralysis

### Special Considerations

- If already intubated: vent settings,  $O_2$  & PEEP required, intubation method & difficulty
- If not intubated: consider awake vs asleep tracheostomy
- If in resp failure/ARDS: may require special ventilator settings
  - Conventional OR ventilator limited (consider ICU vent)
  - Pt may not tolerate vent. disconnect (loss of PEEP)
- May not tolerate lowered  $FiO_2$  during electrocautery
- Considerable bleeding is rare but possible (aberrant vasculature)

### Anesthetic Management

- Awake tracheostomy (see box, page 21–5)
- GA: inhalational or TIVA; muscle relaxation may optimize surgical conditions
- Potential for ETT balloon puncture upon tracheal incision
  - Deflate ETT balloon prior to tracheal incision
  - Consider advancing ETT (balloon) prior to tracheal incision
  - Withdrawal to just above tracheotomy site under direct surgical visualization
- Do not fully extubate until tracheostomy is in place & secured
  - If tracheostomy lost, ETT can be quickly readvanced distal to tracheotomy
- Lower  $FiO_2$  (<30%) if monopolar cautery to be used after tracheotomy

### Management of Existing Tracheostomy

- Does tracheostomy have a balloon/cuff?
- Will positive-pressure vent be required? (*Limited with uncuffed tracheostomy*)
- Will unusual positioning be required?
- Is tracheostomy <7 days old?

### Management of Mature Tracheostomy (>7 days)

1. Suction existing cannula
2. Denitrogenate with 100%  $O_2$  via tracheostomy
3. Controlled inhaled induction with potent agent (e.g., sevoflurane) or IV induction tracheostomy
4. Exchange tracheal tube with a lubricated, wire-reinforced ETT that is same inner diameter or one size smaller than tracheostomy tube
5. Advance tube such that black markings are positioned at stoma & check for bilateral vent
6. Replace tube with clean tracheostomy tube at case completion

### Management of Fresh Tracheostomy

- Fresh tracheostomy (<7–10 days) requires interdisciplinary management
- Should generally not be removed outside OR (no tract)
- Fresh tracheostomy dislodgement = surgical emergency
  1. Call for surgical support & fiberoptic bronchoscope
  2. Put sterile gloves on & plug tracheostomy site with finger
  3. Do **not** attempt blind replacement of tracheostomy
    - Risk of subcutaneous placement, bleeding, & trauma
  4. Attempt mask ventilation
    - Place LMA if failed/difficult mask ventilation
  5. Attempt intubation across tracheostomy site by laryngoscopy
    - Consider fiberoptic intubation if unsuccessful
    - Advance ETT balloon past tracheotomy



- If intubation fails & ventilation is adequate, proceed to OR
  - Tracheostomy replacement via trans-LMA fiberoptic guidance may be considered in stable clinical circumstances with experienced personnel
- If above efforts fail, surgical reexploration at bedside

### Awake Tracheostomy

#### Indications

- Acute stridor/upper airway obstruction
- Severe airway trauma
- Obstructing glottic tumor
- Severe tracheomalacia

#### Blocks (see Chapter 4, airway)

- Superficial cervical plexus block
- Transtacheal block
- Superior laryngeal nerve block
- Local field block

#### Key Points

- Psychological preparation/counseling
- Pt must be able to cooperate
- Head-up position
- Supplemental O<sub>2</sub> via face mask
- Sedative meds for anxiety only
- Maintain spontaneous vent.
- May induce GA after trach in place
- Asleep with flexible LMA & spont vent may be an option

#### Adjunctive Agents

Dexmedetomidine, remifentanyl, droperidol, ketamine, midazolam

### Laser Surgery and Airway Fires

#### Airway Fire Algorithm

- Detect fire or smoke in airway
- Cease ventilation & extubate**
- Mask ventilation with O<sub>2</sub>
- Inspect airway for injury
- Reintubate
- Perform fiberoptic inspection of airway
- Consider trach if significant burn injury
- Measure carboxyhemoglobin level in blood

#### Precautions

- Goggles—including pt
- Laser sign on door
- Use of laser-resistant tube
- ↓ O<sub>2</sub> flows—FiO<sub>2</sub> < 30% optimal
- N<sub>2</sub>O may support combustion
- Surgeon communication
- Moist pledgets/packing at laser site
- Monitor laser power/duration

### Lasers (Light Amplification Stimulated Emission of Radiation)

- Types of Laser:* Nd-YAG; CO<sub>2</sub>; KTP laser; power/penetration varies
- Laser use:* Laryngeal, endometrial, endobronchial mass ablations; skin resurfacing, ophthalmologic procedures; coagulation
- Risks:* Retinal damage, airway fire, vaporized infectious/tumor particles
- Advantages:* highly focused with limited collateral tissue damage

### Introduction to Jet Ventilation

- Used frequently in complex airway surgery
- Jet ventilation catheter eliminates need for ETT
- Subglottic & supraglottic approaches (depends on procedure & anatomy)
- Safe jet ventilation requires open airway for entrained air to escape during exhalation
- Jet ventilation can achieve effective oxygenation & ventilation
  - Brief low-volume, high-frequency pulses of O<sub>2</sub> exiting the jet catheter entrain ambient air → deliver larger tidal volume
- Advanced devices (e.g., Monsoon/Mistral)
  - Set FiO<sub>2</sub>, humidity, driving pressure (DP), frequency (*f*), & inspiratory time (IT)
  - Example settings: FiO<sub>2</sub> = 100%; hum = 40%; DP = 22; psi = 120 bpm; IT = 40%
  - Peak airway pressure can be measured & alarms set

(continued)

## Introduction to Jet Ventilation (Continued)

Applications	Potential Complications	Devices
Suspension laryngoscopy	Barotrauma	Mistral
Tracheal resection	Hypercarbia	Monsoon
Limit motion in field (thoracic)	Airway desiccation	Bird
Difficult airway	Hypoxemia	Manual/Hand

### Strategies

Trans-LMA  
Supraglottic  
Sub-glottic

## Procedures in Ophthalmology

### Special Considerations

- Extremes of age (pediatrics—strabismus repair) (geriatrics—cataract surgery)
- Many ophthalmologists perform regional blocks themselves
- Complications from movement may result in blindness
- Appropriate precautions (see above) for laser surgery
- Access to airway is limited during surgery

### Special Medications in Ophthalmologic Population

- Echthiophate for glaucoma
  - Acetylcholinesterase inhibitor → prolongs action of succinylcholine
  - Systemic effects include bronchospasm, bradycardia, hypertension
- Sulfur hexafluoride gas for retinal detachment
  - Pt may have intravitreal gas bubble up to 21 days postop
  - Avoid N<sub>2</sub>O due to potential for catastrophic air expansion
- Consider avoidance of succinylcholine in selected circumstances
  - Globe injury → Increased intraocular pressure with fasciculation (*succinylcholine is **not absolutely** contraindicated*)
  - Prolonged contracture of ocular musculature after dosing may interfere with forced duction test (FDT) in strabismus surgery
- Pilocarpine & carbachol
  - Drugs that promote efflux of aqueous humor by producing miosis
  - Parasympathomimetics (cholinergic agonist)
  - Systemic effects = parasympathetic effects (bradycardia)
- Epinephrine
  - Systemic effects may lead to tachycardia/angina
- Acetazolamide
  - Carbonic anhydrase inhibitor
  - Systemic effects include metabolic acidosis, hypokalemia, ↓ ICP
- Timolol
  - β-blocker
  - Systemic effects include bradycardia, hypotension, bronchospasm
- Oral glycerol side effects: Nausea, vomiting, hyperglycemia
- Mannitol side effects: Volume overload, renal failure

### Cataract Surgery: Clear Corneal Phacoemulsification

- Pts often elderly with multiple comorbidities
- Procedures usually <1 hr
- Anesthetic goals
  - Akinesia of the eye & eyelid; adequate analgesia & pt cooperation
  - avoidance of oculocardiac reflex
- Sedation with regional block or topicalization = preferred method
  - Local infiltration with sedation
  - Regional block with local infiltration & sedation (see table below)
    - Provided by surgeon or anesthesiologist
    - Brief deepening of anesthesia facilitates block placement
    - Options include retrobulbar block; peribulbar block, sub-Tenon's block
    - Block complications: Retrobulbar hemorrhage, globe perforation, optic nerve damage, brainstem anesthesia
  - GA for selected pts (complex procedures/unable to cooperate or stay supine)

### Strabismus Surgery

- Indication: Reposition muscles to treat ocular malalignment
- Surgery almost exclusively performed in pediatric pts
- ↑ Incidence of postop nausea & vomiting

- ↑ Risk of intraop oculocardiac reflex (see box below)
- Usually performed under GA with ETT
- Nondepolarizing muscle relaxation may aid diagnostic utility of forced duction test (FDT) & surgical operating conditions

#### Other Procedures

- Repair of ruptured globe
  - Frequently emergent procedure with aspiration risk concerns (full stomach, head & associated injuries)
  - Commonly requires GA with ETT
  - Consider LMA in select circumstances (pts often have full stomach)
  - Emphasis on control of intraocular pressure (succinylcholine may ↑ IOP)
    - Avoid coughing or bucking during induction & intubation
- Intraocular surgery: Enucleation, vitrectomy, corneal transplantation, glaucoma decompression, repair retinal detachments
  - Control of eye movement & intraocular pressure critical
  - GA preferred
  - Intraocular epinephrine may be used to aid papillary dilatation
    - Monitor for systemic effects
- Detachment repair injects intraocular air or sulfur hexafluoride gas
  - Avoid N<sub>2</sub>O or discontinue well before injection
  - Avoid N<sub>2</sub>O for subsequent surgery within 3 weeks

#### Oculocardiac Reflex

**Cardiac reflex (bradycardia, sinus arrest, arrhythmia) with multiple triggers:** (1) ocular pressure, (2) ocular muscle stretch, (3) intense stimulation of empty orbit extremely common in pediatric strabismus surgery

**Mechanism:** Afferent-trigeminal  
Efferent-vagus

**Treatment/Prevention:** Anticholinergic (pre)-treatment (e.g., glyco/atropine).  
Use of regional block with local anesthetics  
Release ocular pressure/stop stimulation  
Increase anesthetic depth

#### Blocks for Ophthalmologic Surgery

- *Contraindications (regional):* uncooperative pt or pt with severe medical comorbidities that prevent positioning/immobility, trauma to eye, blindness in nonoperative eye, glaucoma, anticoagulation (relative)
- Advantages of local blocks over GA
  - Avoids complications/side effects of GA (e.g., decreased hemodynamic effects)
  - Useful for day surgery/office procedures (fast recovery)
  - Produce good eye akinesia & surgical analgesia
  - Minimal effect on IOP
- Disadvantages of local blocks:
  - Not suitable for all pts (children, language barrier, mentally handicapped)
  - Depends on skills/experience of anesthesiologist/ophthalmologist performing block
  - Not suitable for all types of surgery (open-eye surgery)
  - Complications (see below)
- Choice of technique varies with surgeon
- Most blocks currently performed by ophthalmologists

Block	Agent	Complications
Superficial application	Lidocaine 2%	Toxicity (high levels): Seizure/cardiac effects Epinephrine: Tachycardia
Subtenon's Block	Lidocaine 1–2% w/ Epinephrine (1:400,000)	Subarachnoid injection: Apnea
Peribulbar Block Retrobulbar Block (highest risk)	Bupivacaine 0.375–0.75% w/ Epinephrine (1:400,000)	Intravascular injection: Seizure Globe rupture: Proptosis/agitation Intraocular: Optic nerve damage/ blindness Chemosis Oculocardiac Reflex Vessel injury—hemorrhage/ecchymosis ↑ Intraorbital pressure/proptosis Central retinal artery occlusion Extraocular muscle injury

Peribulbar Block (25–27 gauge, 25-mm needle)

- Safer (needle inserted outside of extraocular muscle cone), but slower onset
- Primary gaze position → 2 injections above & below globe  
inject ≈ 5 mL local into superonasal orbit & ≈ 5 mL inferotemporally between lateral  $\frac{1}{3}$  & medial  $\frac{2}{3}$  of lower orbital margin

Retrobulbar Block (25–27 gauge, 3-cm needle)

- Faster onset; must anesthetize conjunctiva before needle introduction
- Insert needle halfway between lateral canthus & lateral limbus in lower conjunctiva
- Direct needle straight back until tip is beyond globe, → then direct needle toward apex of orbit to enter space behind globe between inferior & lateral rectus muscles
- Insert to depth of 25–35 cm; inject 4 mL local

Sub-Tenon's Block (25-gauge needle)

- Injection of local anesthetic directly into posterior aspect of sub-Tenon's space
- Insert needle to contact conjunctiva between eyeball & semilunaris fold (depth < 1 mm)  
Advance needle anteroposteriorly with globe directly slightly medially by needle until "click" is felt, at depth of 15–20 mm (episcleral location)
- Return globe to primary position; aspirate → inject local
- Stop at sign of chemosis (conjunctival edema) & apply ocular compression  
(Adapted from Barash PG. *Clinical Anesthesia*. 5th ed. Lippincott Williams & Wilkins: Philadelphia, PA: 2005.)

# RENAL SYSTEM AND ANESTHESIA FOR UROLOGIC SURGERY

CHRISTINE FINER

## Evaluation of Renal Function

- Urinalysis
  - Specific gravity reflects kidney's ability to concentrate; urine
  - Hematuria may occur with intrinsic renal disorders (fever, UTI, kidney stones, urologic tumors, trauma, & coagulopathy)
  - Proteinuria may occur with intrinsic renal disorders, fever, CHF, exercise
- BUN
  - Normal: 10–20 mg/dL
  - Unreliable measure of GFR; (↑ in dehydration, high-protein diet, GI bleeding, & increased catabolism)
- Creatinine
  - End product of skeletal muscle metabolism, excreted by kidneys
  - Proportional to skeletal muscle mass
  - Inversely related to GFR
  - Normal: 0.8–1.3 in men, 0.6–1.0 in women (↓ in pregnancy)
  - Less reliable measure of renal function in elderly (GFR ↓ with age but muscle mass also ↓ — can result in a normal Cr despite abnormal renal function)
- Creatinine clearance
  - GFR best indicator of renal function but difficult to measure; CrCl most reliable estimate of GFR
  - CrCl can be measured using 2- or 24-hr urine collection

Normal: 110–150 mL/min

$$\text{Estimated CrCl} = \frac{(140 - \text{age}) (\text{body weight in kg}) (0.85 \text{ in women})}{(\text{serum Cr}) (72)}$$

## Renal Failure

### Acute Renal Failure

- ↑ of Cr by ≥0.5 mg/dL or ↑ of Cr by ≥20% over 2 weeks

	Prerenal	Intrinsic	Postrenal
Urine Na <sup>+</sup>	<10 mEq/L	>20 mEq/L	>20 mEq/L
Urine osmolarity	>500	<350	<350
FE <sub>Na</sub>	<1%	>2%	>2%
BUN/Cr	>20	<10–15	<10–15
Urine Cr/Serum Cr	>40	<20	<20

### a. Prerenal

- Renal hypoperfusion resulting in ↓ GFR
- Causes
  - Hypovolemia, ↓ cardiac output, liver failure, sepsis
  - Renal vasoconstriction (ACE/COX inhibitors)

### b. Intrinsic (renal)

- Damage to renal parenchyma
- Causes
  - Acute tubular necrosis (ATN)—causes include ischemia & toxins (aminoglycosides, myoglobin, IV contrast)
  - Acute interstitial nephritis (AIN)—usually caused by drugs (NSAIDs, β-lactams, sulfonamides, rifampin)
  - Glomerulonephritis
  - DIC
  - TTP

### c. Postrenal

- Outflow obstruction (must have bilateral obstruction, unilateral obstruction if only one kidney present, kinked Foley)
- Causes
  - Nephrolithiasis, BPH, prostate cancer, neurogenic bladder

### d. Treatment

- Treat underlying disorder
- Avoid nephrotoxic drugs
- Fenoldopam & low-dose dopamine (controversial) may help prevent or treat ARF by dilating renal arteries & ↑ RBF & GFR

## Diagnostic Guide for Serum and Urine Electrolytes

Condition	Serum Values				Urine Values			
	Na <sup>+</sup> (mEq/liter)	K <sup>+</sup> (mEq/liter)	Osmolality (mosm/liter)	Bun (mg/dl)	Na <sup>+</sup> (mEq/liter)	K <sup>+</sup> (mEq/liter)	Osmolality (mosm/liter)	Urea (mg/dl)
Primary aldosteronism	140	↓	280	10	80	60-80	300-800	Low
Secondary aldosteronism	130	↓	275	15-25	<20	40-60	300-400	
Na <sup>+</sup> depletion	120-130	N or ↑	260	>30	10-20	40	600+	800-1000
Na <sup>-</sup> overload	150+	N	290+	N or ↑	100+	60	500+	300
H <sub>2</sub> O overload	120-130	↓	260	10-15	50-80	60	50-200	300
Dehydration	150	↓	300	30 or N	40	20-40	800+	800-1000
Inappropriate ADH	<125	↓	<260	<10	90	60-150	U>Posm	300
Acute tubular necrosis								
Oliguric	135	↑	N or ↑	↑↑	40+	20-40	300	300
Polyuric	135	N or ↑	275	↑	20	30	300	100-300

Source: From Link D. Fluids, electrolytes, acid-base disturbances, and diuretics. In Todres ID, Fugate JH, eds. *Critical Care of Infants and Children*. Boston: Little, Brown, 1996:410-435.

- Dialysis as indicated due to:
  - Acidosis
  - Electrolyte disturbances (hyperkalemia)
  - Intoxication (methanol, ethylene glycol)
  - Volume overload
  - Uremia

#### Chronic Renal Failure

- Either GFR  $<60$  mL/min/1.73m<sup>2</sup> or evidence of kidney damage (abnormal urinalysis, imaging or histology) for  $\geq 3$  months
- Causes:
  - Hypertension
  - Diabetes mellitus
  - Glomerulonephritis
  - Polycystic kidney disease
  - Renovascular disease

Stages of CRF		
Stage	Degree of Impairment	GFR
1	Normal	$>90$
2	Mild	60–89
3	Moderate	30–59
4	Severe	15–29
5	Renal failure	$<15$

- Treatment
  - ACE inhibitors/ARBs (valsartan) may slow progression of diabetic renal disease
  - Erythropoietin for anemia
  - Dialysis as indicated (hemodialysis/peritoneal dialysis)
  - Phosphate binders for hyperphosphatemia
  - Renal transplantation

Clinical Features of CRF	
System	Manifestations
Neurologic	Peripheral/autonomic neuropathy, encephalopathy
Cardiovascular	Hypervolemia, HTN, CHF, uremic pericarditis, pericardial effusion, accelerated atherosclerosis
Pulmonary	Pulmonary edema
Gastrointestinal	Delayed gastric emptying
Hematologic	Anemia, platelet*/leukocyte dysfunction
Metabolic	Hyperkalemia, hypermagnesemia, hyperphosphatemia, hypocalcemia, hypoalbuminemia, metabolic acidosis
Endocrine	Glucose intolerance, hypertriglyceridemia
Musculoskeletal	Osteoporosis, osteomalacia

\*Platelet dysfunction does not improve with platelet transfusion; give DDAVP or cryoprecipitate (vWF activates platelets).

Commonly Used Diuretics		
Class	Site of Action	Side Effects
Osmotic (mannitol)	Proximal convoluted tubule, loop of Henle, & collecting tubule	Hypovolemia Hyperosmolality
Loop diuretics (furosemide, bumetanide, ethacrynic acid)	Thick ascending loop	Hypokalemia Metabolic alkalosis Volume contraction Otoxicity
Thiazides (HCTZ, dyazide, metolazone)	Distal convoluted tubule	Hypokalemia Hyponatremia Metabolic alkalosis Volume contraction
Carbonic anhydrase inhibitors (acetazolamide)	Proximal convoluted tubule	Hyperchloremic metabolic acidosis
Potassium-sparing diuretics (triamterene, amiloride, aldactone)	Collecting tubule	Hyperkalemia Metabolic acidosis

## Anesthesia for Patients with Renal Disease

### Effects of Anesthesia on Renal Function

- Reversible ↓ in RBF, GFR, urine production during regional & general anesthesia can occur despite maintenance of normal BP/volume status
- RBF & GFR will usually return to normal within several hours postop

### Indirect Effects of Anesthesia

- Anesthetic agents & sympathetic blockade (during regional techniques) → Hypotension & myocardial depression → ↓ RBF & GFR
- Hydration before anesthesia may lessen hypotension (and changes in RBF)

### Direct Effects of Anesthesia

- Fluorinated agents can cause direct renal toxicity (fluoride impairs kidney's ability to concentrate urine & causes tubular necrosis)
  - Fluoride production negligible with halothane, desflurane, & isoflurane
  - Sevoflurane & enflurane release fluoride (no clinical evidence of renal damage)
- Sevoflurane reacts with carbon dioxide absorbents to form compound A (shown to cause renal damage in rat models)
  - Low fresh gas flows should be avoided with sevoflurane (use flows of  $\geq 1$  L/min)
  - Consider avoiding sevoflurane in pts with renal insufficiency (theoretical risk of nephrotoxicity)
- Common IV agents do not cause changes in GFR

### Medications to Avoid or Use with Caution in Renal Failure

- Lipid-insoluble, ionized drugs & water-soluble metabolites of hepatically metabolized drugs are renally excreted & may accumulate in renal failure
- Highly protein-bound drugs can accumulate if patient is hypoalbuminemic

<b>Opiates</b>	Morphine	Active metabolites may accumulate & have prolonged effects
	Meperidine	Active metabolites may accumulate & cause both prolonged effects & seizures
<b>Benzodiazepines</b>	Diazepam	Active metabolites may accumulate & cause sedation
<b>Muscle relaxants</b>	Pancuronium Doxacurium	Prolonged effect
	Vecuronium	Typically safe with single dose but may accumulate with repeated doses/infusion
	Succinylcholine	May be used if $K^+ < 5-5.5$ mEq/L; same amount of $K^+$ released in pts with nl renal function (0.5-1 mEq/L)
<b>Reversal agents</b>	Neostigmine Edrophonium Pyridostigmine	May have prolonged effects (however, anticholinergics may also be prolonged)
<b>Cardiovascular agents</b>	Digoxin	Levels may be ↑ due to ↓ clearance; danger for digoxin toxicity
	Nitroprusside	Accumulation of thiocyanate (neurotoxic)
	α-agonists (phenylephrine)	Constrict renal vasculature
<b>Barbiturates</b>	Thiopental Methohexital	↓ Available drug in hypoalbuminemic pts; may need smaller induction dose
<b>Antibiotics</b>	Aminoglycosides Vancomycin	Need renal dosing to avoid toxic levels

## Urologic Surgery

### Cystoscopy/Ureteroscopy/TURBT

#### General Considerations

- Indications: Need for biopsies, laser lithotripsy, extraction of stones, placement of ureteral stents
- Pts commonly elderly with comorbid medical conditions
- Irrigation fluids often used to improve visualization & for flushing
  - **Sterile water:** Hypotonic, causes hemolysis & hyponatremia when absorbed systemically; safe with electrocautery
  - **Nonelectrolyte solutions (glycine, sorbitol, mannitol):** Slightly hypotonic, can cause hyponatremia if absorbed in large volumes; safe with electrocautery
  - **Electrolyte solutions (NS, LR):** Isotonic, do not cause hemolysis when absorbed systemically; cannot be used with electrocautery



**Anesthetic Technique**

- Positioning: Lithotomy
- Usually GA, can use local/MAC/regional (T10 level necessary for instrumentation of lower GU tract), consider using LMA
- Muscle relaxation not usually necessary (consider ETT with relaxation if surgeon anticipates working near obturator nerve)
- Minimal to no postop pain; short-acting opioids (fentanyl) usually sufficient

**Complications**

- Peroneal nerve injury from lithotomy position (causes foot drop)
- Bladder perforation: Extraperitoneal perforation is more common; signs and symptoms include nausea, diaphoresis & inguinal, retropubic or lower abdominal pain

**Transurethral Resection of the Prostate (TURP)****General Considerations**

- Indications: Relief of bladder obstruction from enlarged prostate (typically BPH)
- Typically elderly pts with comorbid medical conditions
- Opening of venous sinuses may lead to absorption of large amounts of irrigation fluid (see cystoscopy, above) & can result in TURP syndrome (see below); fluid absorption dependent on duration of procedure, number of sinuses opened (related to prostate size), peripheral venous pressure, & height of irrigation fluid

**Anesthetic Technique**

- Positioning: Lithotomy
- General or regional (T10 level necessary)
  - Base choice on pts preference, coexisting diseases
  - Regional anesthesia allows for evaluation of TURP syndrome during procedure
- Muscle relaxation not required, although patient movement should be avoided (prevent further bleeding/perforation of prostate)
- Postop pain usually not significant

**Complications**

- TURP syndrome
  - Results from absorption of large volumes of irrigant fluid through venous sinuses of prostate
    - Hyponatremia & volume overload
  - Signs/symptoms: Headache, confusion, nausea/vomiting, HTN, angina, seizures, coma, cardiovascular collapse
  - May also see toxicity from absorption of irrigant solutes
  - Glycine: Can cause transient blindness, seizures
  - Ammonia: Can cause delayed awakening, encephalopathy
  - Hyperglycinemia may result in CNS toxicity & circulatory collapse
  - Treatment: Fluid restriction & diuretics to correct hyponatremia & volume overload; if pt has seizures/is comatose → consider hypertonic saline
- Bladder perforation
- Coagulopathy: Dilutional thrombocytopenia from excessive fluid absorption & DIC
- Bacteremia: Because prostate is colonized by bacteria, bacteremia may result after instrumentation
- Prophylactic antibiotics may ↓ risk of bacteremia/septicemia

**Alternatives to TURP**

- Medical management with alpha blockers
- Vaporization of prostate tissue with electrocautery/laser/thermocoagulation (avoid danger of TURP syndrome)

**Urologic Laser Surgery****General Considerations**

- Indications: Condyloma acuminatum, ureteral strictures, BPH, ureteral calculi, & superficial carcinomas of penis, ureter, bladder, or renal pelvis
- Different lasers may be used (CO<sub>2</sub>/argon/pulsed dye/Nd-YAG/KTP-532)
- Safety concerns
  - Goggles should be worn by OR personnel & patient to protect eyes from an inadvertent break in laser fiber
  - Lasers should be used intermittently & in noncontinuous mode to prevent thermal injuries
  - Special masks should be worn to prevent inhalation of active HPV particles when condyloma are being treated

**Anesthetic Technique**

- Positioning: Lithotomy
- Local with MAC, general or regional anesthesia

## Open Prostatectomy

### General Considerations

- Indications: Simple prostatectomy for BPH that cannot be resected transurethrally; radical prostatectomy for prostate cancer
- Pts often elderly with comorbid medical conditions
- Blood loss can be significant
- Retroperitoneal lymph node dissection is performed for staging purposes in prostate cancer
- Bilateral orchiectomy may be performed in symptomatic, advanced disease.

### Monitoring/Access

- Large-bore IV

### Anesthetic Technique

- Positioning: Supine
- Regional, general, or combined general/epidural
- Epidural may ↓ blood loss, improve postop pain relief, & result in recovery of bowel function more quickly
- Experienced surgeons typically able to perform procedure under general anesthesia with minimal blood loss/small incisions
- Can also be done laparoscopically (and robotically) under general anesthesia (results in less blood loss, smaller incision/less pain)
- Surgeon may ask for methylene blue/indigo carmine to assess integrity of urinary tract
  - Indigo carmine: Can cause hypertension ( $\alpha$ -agonism)
  - Methylene blue: Can cause hypotension/interfere with SpO<sub>2</sub> readings

## Cystectomy

### General Considerations

- Indications: Simple cystectomy for benign bladder disease (hemorrhagic/radiation cystitis); radical cystectomy for invasive bladder tumors
- Pts often elderly with comorbid conditions; given the association between smoking & bladder cancer, pts may be at risk for CAD & COPD
- After cystectomy, a urinary diversion must be constructed
  - Piece of ileum can be formed into an ileal conduit (brought out to the abdominal wall as a stoma)
  - Bladder suspension more involved operation (piece of bowel is formed into a pouch & connected to the urethra)
- Significant blood & fluid loss may occur

### Monitoring/Access

- Standard monitors; consider arterial line, central line given potential for large blood loss & fluid shifts
- Large-bore IV

### Anesthetic Technique

- Positioning: Supine or lithotomy
- General or combined general/epidural anesthesia

## Nephrectomy

### General Considerations

- Indications: Neoplasm, transplantation, chronic hydronephrosis, chronic infection, trauma
- Pts undergoing nephrectomy for renal cell carcinoma, will undergo preop staging to determine if tumor involves IVC or right atrium
  - Tumor may partially/completely obstruct IVC (reduces venous return & may cause hypotension); IVC may need to be clamped during resection
  - Tumor may embolize to pulmonary vasculature (signs: ↓ SpO<sub>2</sub>, hypotension, supraventricular arrhythmias)
  - Other complications: Venous air embolus, diaphragmatic injury (causing pneumothorax)
- May be performed open or laparoscopically

### Monitoring/Access

- Standard monitors; consider arterial line
- Large-bore IV (potential for significant blood loss)

### Anesthetic Technique

- Positioning: Lateral decubitus position for retroperitoneal approach/supine for trans-abdominal approach
- General anesthesia or combined general/epidural anesthesia (T7–T9 level)
- Hydration to preserve renal blood flow

# ANESTHESIA FOR ORTHOPEDIC SURGERY

ROBERT HSIUNG • PETER WU

## General vs Regional Anesthesia

- Choice based on location, surgical duration, surgeon, & patient preference
- General anesthesia (GA): Gold standard in speed & reliability
- Regional anesthesia: May ↑ analgesia & pt satisfaction, may ↓ length of stay
- Multimodal pain regimens can be used regardless of technique:  
COX inhibitors (ketorolac, celecoxib), anticonvulsants (gabapentin), intra-articular corticosteroid/local anesthetic injection, and/or opioids

GA vs Regional Simplified	
<b>Speed</b>	GA > regional
<b>Recovery</b>	Regional (peripheral nerve blocks) > GA
<b>Amnesia</b>	GA > regional unless adjuncts used
<b>Cost</b>	Variable
<b>Reliability</b>	GA > regional anesthesia
<b>Long term analgesia</b>	Regional > GA
<b>Mortality</b>	No difference in non-OB population
<b>DVT incidence</b>	GA > regional

## Positioning

- Important to prevent tissue & nerve injury, especially in pts with arthritis & spine dz
- Common peripheral nerve injuries occur with the following frequency:  
ulnar > brachial plexus > lumbosacral root > spinal cord
- Supine—most surgeries, including knee, hip, pelvis, arm, hand, foot
- Prone—spine surgery
  - Check pressure points—face/eyes, breasts/genitalia, abdomen, brachial plexus
  - Endotracheal tube dislodgement & kinking can occur
  - Stretcher should be immediately available if need to be turned back emergently
  - ↑ Risk of perioperative visual loss
    - Associated with spinal surgery, although frequency is <0.2%
    - Mechanism: unknown, may include anterior & posterior ischemic optic neuropathy (A/PION) or central retinal artery occlusion (CRAO)
    - Risk factors: prolonged surgical time (>6 hr) & blood loss (>40% EBV)
    - Avoid direct pressure on eye orbits to ↓ risk of CRAO & venous congestion
    - Deliberate hypotension has not been shown to ↑ risk
- Lateral—neck, hip surgeries
  - Need to protect pressure points & maintain neutral neck alignment
  - Axillary roll ↓ brachial plexus & vascular compression in dependent arm
- Sitting/beach chair—shoulder, clavicle surgeries
  - Provides full surgical access to the shoulder, ↑ risk of air embolism (5–25%)
- **Extremity Surgery** (see Chapter 6, on regional anesthesia)
  - Consider regional anesthesia, especially continuous catheter techniques
  - Combined general/regional may maximize speed, amnesia, & analgesia

Anesthetic Options for Extremity Surgery			
Procedure	Anesthetic Options	Positioning	Notes
<b>Upper Extremity</b>			
Shoulder surgery	<ul style="list-style-type: none"> <li>• General</li> <li>• Interscalene</li> </ul>	Beach chair, lateral	Often requires general anesthetic or heavy sedation as pt may be covered in drapes for hours
Elbow surgery	<ul style="list-style-type: none"> <li>• General</li> <li>• Supraclavicular</li> <li>• Infraclavicular</li> <li>• Axillary</li> </ul>	Supine, lateral, or prone	Consider infraclavicular catheters

(continued)

## Options for Extremity Surgery (Continued)

Procedure	Anesthetic Options	Positioning	Notes
Wrist and hand surgery	<ul style="list-style-type: none"> <li>• General</li> <li>• Axillary</li> <li>• Supraclavicular</li> <li>• Infraclavicular</li> <li>• Bier block</li> <li>• Selective nerve block</li> <li>• Digital nerve block</li> <li>• Local + MAC</li> </ul>	Supine	Typical surgeries include carpal tunnel release, distal radius ORIF, trigger finger release, & ganglion cyst excision
<b>Lower Extremity</b>			
Knee arthroscopy	<ul style="list-style-type: none"> <li>• General</li> <li>• Femoral ± sciatic</li> <li>• 3-in-1 block ± sciatic</li> <li>• Spinal</li> <li>• Epidural</li> <li>• Intra-articular local + MAC</li> </ul>	Supine	Usually ambulatory procedure; Nerve block may require supplementation with intra-articular local or IV sedation/analgesia
Total knee arthroplasty	<ul style="list-style-type: none"> <li>• General ± femoral (single shot or catheter)</li> <li>• Spinal ± femoral catheter</li> <li>• Epidural</li> <li>• Lumbar plexus + sciatic</li> <li>• 3-in-1 + sciatic</li> </ul>	Supine	Multiple combination nerve blocks possible; continuous femoral nerve catheters are common to reduce postop analgesia requirements; tourniquet use common → EBL can be >500 mL after deflation
Hip fracture	<ul style="list-style-type: none"> <li>• General ± lumbar plexus (postop analgesia)</li> <li>• Spinal</li> <li>• Epidural</li> </ul>	Supine	Mostly elderly pts with multiple medical problems; regional anesthesia may ↓ morbidity/mortality
Hip arthroplasty	<ul style="list-style-type: none"> <li>• General ± lumbar plexus (postop analgesia)</li> <li>• Spinal</li> <li>• Epidural</li> </ul>	Lateral	Risk of embolization-induced hypotension/hypoxemia/arrest during cementing; Rx = supportive resuscitation
Ankle	<ul style="list-style-type: none"> <li>• General</li> <li>• Spinal</li> <li>• Epidural</li> <li>• Sciatic ± saphenous</li> <li>• Popliteal ± saphenous</li> </ul>	Supine, lateral, or prone	Epidural may take 30+ min to set in sacral region
Foot, toe, bunion surgery	<ul style="list-style-type: none"> <li>• General</li> <li>• Ankle</li> <li>• Sciatic ± saphenous</li> <li>• Popliteal ± saphenous</li> </ul>	Supine	Except for saphenous (femoral n.), which provides sensation to medial part, foot is innervated by sciatic n.

## Spine Surgery

- Cervical spine surgery
  - Indications: Instability (trauma, tumor), arthritis/osteophytes, spinal stenosis
  - Intubation precautions
    - Consider fiberoptic intubation or techniques that avoid neck hyperextension (intubating LMA, GlideScope®)
    - All laryngoscopies cause neck extension; LMA placement may ↑ pressure on spinal cord
- Thoracic/lumbar spine surgery
  - Laminectomy/laminotomy: Excision of vertebral lamina to relieve spinal nerve pressure

- Fusion
- Instrumentation with hardware to stabilize spine until bony fusion (facilitated by inserted bone graft) can occur (6–12 months postop)
- Indications: spondylolisthesis, scoliosis, recurrent disc herniation, spinal instability (trauma)
- Often large operations with large blood loss & periop complications  
(Consider A-line, good IV access, blood & cell saver)

### Neuromonitoring (also see Chapter 20, page 20–3)

- Indication: Detection of neural pathway compromise during surgery helps guide intraop surgical decision making
- Communication between surgeon, anesthesiologist, & neurophysiologist important
- Definition: Evoked potentials are measurements of nerve conduction in response to stimulation of a neural pathway
- Technique: Waveforms evaluated for amplitude, latency, & morphology; baseline measurements performed after anesthetic stabilized (avoid boluses/rapid changes in anesthetic depth → infusions can be beneficial)
- Outcomes: No evidence of improved outcomes in lumbar decompression or lumbar fusion for degenerative spine dz according to the American Assoc. of Neurological Surgeons/Congress of Neurological Surgeons
- Types of monitoring

#### Somatosensory evoked potentials (SSEP)

- Detect injury/ischemia to dorsal columns of spinal cord
- Stimulation of common peripheral nerves (posterior tibial, median, ulnar) recorded at scalp (sensory strip)
- Rare false negatives but common false positives
- Useful in monitoring compromise of blood supply to dorsal columns (sensory tract) supplied by posterior spinal arteries
- Does not directly monitor motor tracts (anterior spinal artery)
  - motor injury possible with normal SSEPs
  - artery of Adamkiewicz supplies lower 2/3 of spinal cord via anterior spinal artery
- Results may be delayed (20 min)

#### Corticospinal motor evoked potentials (MEP)

- Used to monitor integrity of spinal cord motor tracts
- Used for aortic, intramedullary spinal cord tumor, spinal deformity, posterior fossa tumor, & intracranial aneurysms
- Useful in monitoring compromise of blood supply to anterior motor tracts supplied by anterior spinal artery
- Scalp or epidural lead placement to stimulate upper & lower extremities
- Muscle relaxants should be avoided
- Results immediate
- Safety issues: burns, movement-induced injuries, seizures, bite injuries (0.2%), contraindicated with pacemakers

#### Electromyography (EMG)

- Measures electrical activity of muscles
- Used in tethered cord release, tumor excision, acoustic neuroma resection, & facial nerve procedures
- Also performed preoperatively with nerve conduction studies to help diagnose nerve compression injuries (carpal tunnel or sciatica) or neuromuscular diseases (myasthenia gravis, muscular dystrophy, ALS)
  - Muscle relaxants should be avoided
- Factors affecting monitoring
  - Baseline measurements may be poor
  - MEPs more sensitive to anesthetics than SSEPs
    - Muscle relaxants relatively contraindicated in MEPs, but enhance SSEP signals by ↓ EMG artifact
  - ↑ Latency or ↓ amplitude may indicate neurologic dysfunction
    - Bilateral & equal changes likely 2° to temp, hypotension, or anesthetic effects
    - Unilateral changes likely 2° to ischemia/technical factors
  - Temperature (SSEP): Hypothermia: ↔ amplitude, ↑ latency  
Hyperthermia: ↓ amplitude, ↔ latency
  - Hypoxia (SSEP): ↓ Amplitude, ↑ latency may ↑ amplitude early (injury potential)
  - CO<sub>2</sub> (SSEP): PaCO<sub>2</sub> 25–50 with minimal changes  
PaCO<sub>2</sub> > 100 ↓ amplitude, ↑ latency
  - Anemia: ↓ Amplitude
  - Hypotension (SSEP): rapid BP ↓ will ↓ amplitude, ↔ latency
  - Anesthetic agent (SSEP): IV effects < volatile anesthetic effects

## Effect of IV Agents on SSEPs

↓ Amplitude	No Change	↑ Amplitude
Barbituates (↑ latency)	Propofol	Ketamine
Magnesium	Narcotics	Etomidate
α-2 antagonists	Midazolam	
	Droperidol	
	Clonidine	
	Dexmedetomidine	

Local anesthetics—inhibit SSEPs (contraindicated if monitoring)

- Volatile anesthetics: Dose-dependent ↑ latency & ↓ amplitude (N<sub>2</sub>O will depress monitoring, often more than volatiles)
- Anesthesia techniques in monitored surgeries (SSEP/MEP)
  - Halogenated anesthetics vs. TIVA
    - Propofol/narcotic (i.e. remifentani) infusions have little or no interference with neuromonitoring (SSEP and MEP) at steady state
    - Infusions can ↓ amount of volatile agent used to <0.5 MAC
  - Deliberate hypotension
    - Reducing MAP 20–30% below baseline to ↓ blood loss
    - Techniques: Deep volatile anesthesia, β-blockers, arterial or venous vasodilators
    - Risks: ↓ Perfusion & O<sub>2</sub> to vital organs (heart, brain, spinal cord)
    - Frequent monitoring of pressure points needed (↓ perfusion pressure may ↑ risk of pressure necrosis)
  - **Wakeup test**
    - Most reliable assessment of intact spine
    - Involves gentle, slow wakeup with continued analgesia coordinated with surgical team following instrumentation
    - Pt asked to follow commands upon emergence with reestablishment of general anesthesia after neuroassessment
    - Plan should be outlined with pt prior to surgery (explain possibility of intraoperative recall)
    - Common techniques for rapid awakening include TIVA (propofol, remifentani, and/or dexmedetomidine), N<sub>2</sub>O-narcotic, short-acting inhalational agent (desflurane)

## Complications in Orthopedic Surgery

- Bone cement: Methylmethacrylate bone cement
  - Polymer creates strong bond between bone & implanted hardware
  - Expands into cancellous bone when applied, can result in ↑ intramedullary pressures → displacement of intramedullary fat → **fat embolism**
  - Cement monomers can cause ↓ SVR/↓ BP if enters systemic circulation
  - Signs: Hypoxia, ↓ BP, dysrhythmias, pulmonary HTN, ↓ cardiac output
  - Therapy: High FiO<sub>2</sub>, euolemia, & hemodynamic support
- Fat embolus syndrome (FES)
  - Often occurs with long bone fractures and intramedullary reaming
  - Risk ↑ 24 hr post-injury without proper bone fixation
  - Signs: skin petechiae, rash, urine fat globules, hypotension, tachycardia, hypoxia/dyspnea, altered mental status, lung infiltrates on CXR
  - Therapy: Supportive to correct hypoxia & hemodynamic instability; intubation with PEEP for refractory hypoxemia; steroids controversial
- Management of blood loss in orthopedic surgery
  - Autologous blood donation collected several weeks prior to elective procedure
  - Preop erythropoietin
  - Tourniquet for extremity surgery
    - Application proximal to surgical site to ↓ blood flow (bloodless surgical field)
    - Complications include ischemic injury, tourniquet pain, & reperfusion injury
    - Relatively contraindicated in sickle cell pts (limb ischemia promotes sickling)
  - Acute normovolemic hemodilution—pt's blood collected at start of surgery with concomitant replacement with IV fluids; blood is returned to pt at end of surgery
  - Intraoperative blood salvage/cell saver
  - Antifibrinolytics—aminocaproic acid, tranexamic acid
  - Deliberate hypotension

### Complications of Intraoperative Tourniquet Use

<b>Ischemic injury</b>	<ul style="list-style-type: none"><li>• Prolonged inflation &gt;2 hr can cause neural/tissue ischemia with possible permanent injury</li><li>• If longer duration is needed, tourniquet should be deflated, perfusion restored, and reinflated after 10–15 min</li></ul>
<b>Tourniquet pain</b>	<ul style="list-style-type: none"><li>• Progressive hypertension occurring 30–60 min after cuff inflation, even in the setting of adequate regional anesthetic blockade (spinal, epidural, peripheral nerve block)</li><li>• Mechanism unknown; thought to be mediated through unmyelinated C fibers resistant to local anesthetic blockade</li><li>• Rx: IV analgesia frequently ineffective; vasodilators can be used to lower BP; tourniquet deflation and reinflation as above</li></ul>
<b>Reperfusion injury following tourniquet deflation</b>	<ul style="list-style-type: none"><li>• Acidic metabolites &amp; emboli generated in ischemic limb reenter systemic circulation, causing hypotension, hypoxia, hypercarbia, pulmonary HTN, embolism, and/or metabolic acidosis</li><li>• Usually transient → Rx may include fluids &amp; vasopressors; calcium &amp; bicarbonate prn for hyperkalemia &amp; severe acidosis, respectively</li></ul>

- DVT/PE
  - Prophylaxis = best prevention
  - Major cause of perioperative morbidity/mortality in orthopedic surgery
  - Risk factors: Prolonged surgery, hip/knee replacement, tourniquet use, ↓ mobility postop
    - Regional anesthesia reduces risk for DVT/PE compared to GA
    - Encourage early ambulation/physical therapy
    - Use intermittent leg-compression devices throughout perioperative period
    - Initiate low-dose anticoagulation (warfarin/LMWH) preoperatively
    - Consider prophylactic IVC filter in high-risk patients

# ANESTHESIA FOR ENDOCRINE SURGERY

MATVEY BOBYLEV

## THYROID GLAND

Physiologic State	Serum TSH	Serum Free T <sub>4</sub>	Serum T <sub>3</sub>	24-h Radioiodine Uptake
Hyperthyroidism, untreated	↓	↑	↑	↑
Hyperthyroidism, T <sub>3</sub> toxicosis	↓	Normal	↑	Normal or ↑
1° hypothyroidism, untreated	↑	↓	↓ Or normal	↓ Or normal
Hypothyroidism 2° to pituitary dz	↓ Or normal	↓	↓ Or normal	↓ Or normal
Euthyroid, on iodine	Normal	Normal	Normal	↓
Euthyroid, on exogenous thyroid hormone	Normal	Normal on T <sub>4</sub> , ↓ on T <sub>3</sub>	↑ On T <sub>3</sub> , normal on T <sub>4</sub>	↓
Euthyroid, on estrogen	Normal	Normal	↑	Normal
Euthyroid sick syndrome	Normal, ↓, or ↑	Normal or ↓	Low	Normal

## Hyperthyroidism

Causes	Clinical Features
<ul style="list-style-type: none"> <li>Grave's dz (diffuse toxic goiter)</li> <li>Toxic nodular goiter (Plummer's dz)</li> <li>Substernal &amp; multinodular goiter</li> <li>Thyroiditis</li> </ul>	<ul style="list-style-type: none"> <li>Graves' triad (visible neck mass, clinical thyrotoxicosis, exophthalmos)</li> <li>Hypermetabolism (sweating, wt loss, heat intolerance, thirst)</li> <li>Cardiovascular signs (high-output cardiac failure, CHF, peripheral edema, arrhythmias (v. tach &amp; a. fib))</li> </ul>
Diagnosis	Treatment
<ul style="list-style-type: none"> <li>History &amp; physical exam</li> <li>TFTs</li> <li>Imaging studies (MRI, US)</li> </ul>	<ul style="list-style-type: none"> <li><b>Surgical:</b> Total &amp; subtotal thyroid resection (including near-total or subtotal thyroidectomy)</li> <li><b>Nonsurgical</b> (see table below)</li> </ul>

## Medical Treatment of Hyperthyroidism

Drug	Doses	Mechanism of Action	Comments
<b>Antithyroid drugs</b> Propylthiouracil	Initial dose: 100–150 mg PO tid	Inhibit thyroid hormone synthesis; in large doses PTU blocks peripheral conversion of T <sub>4</sub> to T <sub>3</sub>	Resulting ↓ in serum T <sub>3</sub> level reported within 4–8 h after a single 200-mg PO dose of PTU
Methimazole	Initial dose: 20–40 mg/d PO Maintenance dose: 2.5–15 mg/d PO		
<b>Beta blockers</b> Propranolol	20–80 mg PO tid; 1–2 mg IV q4–8h	Ameliorate action of T <sub>3</sub> & T <sub>4</sub> in tissues, inhibit adrenergic effects	Control cardiac arrhythmias & psychomotor manifestations within minutes
Atenolol	50–100 mg/d PO		

(continued)



**Medical Treatment of Hyperthyroidism (Continued)**

Drug	Doses	Mechanism of Action	Comments
<b>Iodides</b> 10% KI Lugol's solution	Lugol soln: 3–5 gtt in water PO tid KI: 1–2 gtt in water PO bid	Blocks thyroid hormone release, inhibit T <sub>3</sub> , T <sub>4</sub> formation, ↓ thyroid gland size	Iodides reserved for treatment of thyroid storm, or for 10–14 d prior to surgery, including thyroidectomy
<b>Steroids</b> Prednisone Dexamethasone	40–60 mg PO qd for 4–6 wk 2 mg IV q6h	Inhibit conversion of T <sub>4</sub> to T <sub>3</sub> , ↓ TSH; immunosuppressive action	Can relieve symptoms of hyperthyroidism & restore serum T <sub>3</sub> conc. to normal within 7 d
<b>Radioactive iodine</b> (iodine-131)	5–15 mCi	Destruction of thyroid gland by radiation	Max effect usually seen in 3 mo

**Anesthesia for Hyperthyroidism****• Preop**

- General: Euthyroid status preferred (risk of thyroid storm), check TFTs, continue antithyroid meds & β-blockers to day of surgery
- Airway: Check for compression, tracheal deviation & substernal thyroid mass consider awake fiberoptic intubation if airway looks challenging

**• Intraop**

- General: Avoid/use sympathetic nervous system stimulants cautiously (epinephrine, ketamine, ephedrine, phenylephrine) → severe HTN/tachy
- Ensure eye protection (exophthalmos)
- Watch for signs of thyroid storm (hyperthermia, tachycardia, ↑ BP)
- Autoimmune thyrotoxicosis may be assoc with myopathies

**• Postop**

- Complications: Hormonal disturbances & airway management issues
- Thyroid storm: Life-threatening condition, can develop 6–24 h after surgery; caused by massive release of T<sub>3</sub> & T<sub>4</sub>  
Signs: Tachy, fever, confusion, vomiting, dehydration, CHF, agitation (unlike MH, not assoc with ↑ CPK, muscle rigidity or acidosis)
- Parathyroid gland damage/removal → hypocalcemia in 24–72 h postop
- Recurrent laryngeal n. damage → causes hoarseness if unilateral, stridor if bilateral diagnosis by fiberoptic laryngoscopy
- Neck hematoma → partial/complete upper airway obstruction  
treatment = prompt opening of neck wound & drainage

**Treatment of Thyroid Storm**

- Iodine: 5 drops of 10% KI PO tid or 10 drops of Lugol's PO tid; or 1 g Nal slowly by IV drip over 24 h
- Propylthiouracil: 600 mg PO given before iodine, then 400 mg q6h
- Propranolol: 40 mg PO qid; or 1 mg slowly IV q4h under close monitoring; do not exceed 1 mg/min; repeat 1-mg dose may be given after 2 min; or esmolol infusion
- IV dextrose solutions
- Correction of dehydration & electrolyte imbalance
- Cooling blanket for hyperthermia
- Antiarrhythmic drugs (e.g., Ca channel blockers, adenosine, β-blockers) if necessary for atrial fibrillation
- Treatment of underlying dz, such as infection
- Corticosteroids: Hydrocortisone 100 mg IV q8h or dexamethasone 8 mg IV qd

Hypothyroidism	
1° Causes	2° Causes
↓ Production of thyroid hormones, Hashimoto's thyroiditis (may be assoc. with DM or Addison's), congenital causes, iodine deficiency, thyroidectomy, radioactive thyroid ablation, antithyroid med overuse, amiodarone side effect	↓TSH due to isolated deficiency in TSH production by the anterior pituitary, resistance to thyroid hormones, destruction of anterior pituitary (tumor/surgery)
Clinical Features	Diagnosis
Cold intolerance, joint pains, leg swelling (due to CHF), bradycardia, depressed hypoxic & hypercapnic vent responses, abnl baroreceptor fx, hypovolemia, coarse dry skin, puffy eyes, muscle weakness, constipation, hair loss	Based on history, physical exam, TFTs
	Treatment
	Replacement therapy (levothyroxine)

### Anesthesia for Hypothyroidism

#### • Preop

- Thyroid supplements should be continued through surgery
- Delay elective surgeries in case of untreated hypothyroidism  
*(risk of cardiovascular instability & myxedema coma)*
- Subclinical hypothyroidism **not** assoc with ↑ surgical risk
- In emergency cases: consider pretreatment with IV thyroxine & steroids
- Pts usually obese, may have large tongue, short neck, delayed gastric emptying

#### • Intraop

- Hypothyroid pts sensitive to narcotics & sedatives
- Induction: maintain stable hemodynamics (consider ketamine or etomidate)
- Hypotension due to abnl baroreceptor fx, ↓ cardiac output, hypovolemia
- Hypothermia develops very fast & difficult to treat
- Metabolic disturbances common: ↓ Na & ↓ blood sugar
- Hypoventilation common (blunted response to hypoxia)
- Myxedema coma (severe form of decompensated hypothyroidism) can occur (see table below)

#### • Postop

- Hypothermia, slow drug metabolism, & resp depression may delay extubation
- Extubation should be done in awake & normothermic pt
- Regional anesthesia & ketorolac = preferable for pain control (use opioids with caution)

Myxedema Coma	
Clinical Signs	Treatment
<ul style="list-style-type: none"> <li>• Stupor/coma</li> <li>• Hypothermia</li> <li>• Bradycardia</li> <li>• CHF</li> <li>• ↓ Na</li> <li>• ↓ BP</li> <li>• Precipitated by surgery, trauma, infection, stress</li> </ul>	<ul style="list-style-type: none"> <li>• Support ventilation &amp; BP</li> <li>• IV fluids</li> <li>• Correct ↓ Na &amp; blood sugar</li> <li>• <i>Levothyroxine</i> (100–500 mcg IV slowly, then 75–100 mcg IV daily) or <i>liothyronine</i> (synthetic T3—short-acting, quick dose adjustments) at 25–50 mcg slow IV infusion, then 65–100 mcg/d divided tid/qid</li> </ul>

## PARATHYROID GLANDS

### Hyperparathyroidism

1° Causes	2° Causes
Inappropriate secretion of PTH (usually parathyroid adenoma)	↑ Secretion of PTH as response to low Ca (from other causes)
<b>Ectopic hyperparathyroidism</b> (pseudoparathyroidism) = PTH related protein (PTHrP) secretion by tumor	
Clinical Features	Diagnosis
Muscle weakness, altered mental status, coma, CHF, HTN, compression vertebral fractures, osteoporosis, kidney stones, pancreatitis & peptic ulcers	1° hyperparathyroidism: ↑ serum PTH, serum Ca >5.5 mEq/L, ionized Ca >2.5 mEq/L Imaging studies (osteoporosis, kidney stones)
Treatment	
Treatment = surgical; no effective medical treatment for 1° hyperparathyroidism (acute treatment goal = control elevated blood Ca; consider hemodialysis in severe cases)	

### Anesthesia for Hyperparathyroidism

- ECG: short PR & QT intervals, cardiac conduction disorders ( $\uparrow$  Ca levels)
- Maintain hydration & good urine output
- Consider using lower doses of nondepolarizing muscle relaxants in weak/somnolent pts

Medical Treatment of Hyperparathyroidism			
Agent	Mode of Action	Indications	Precautions
<b>Normal saline</b> 2–4 L IV daily for 1–3 d	Enhances filtration and excretion of $\text{Ca}^{2+}$	Severe $\text{Ca}^{2+} > 14$ mg/dL (3.5 mmol/L); moderate $\text{Ca}^{2+}$ with symptoms	May exacerbate CHF; lowers $\text{Ca}^{2+}$ by 1–3 mg/dL (0.25–0.75 mmol/L)
<b>Furosemide</b> 10 to 20 mg IV as necessary	Inhibits calcium resorption in distal renal tubule	Following aggressive rehydration	Loss of $\text{K}^+$ , dehydration if used before intravascular volume restored
<b>Bisphosphonates</b> —pamidronate, 60–90 mg IV over 4 h—zoledronic acid, 4 mg IV over 15 min	Inhibits osteoclast action and bone resorption	Hypercalcemia of malignancy	Nephrotoxicity, rebound $\text{Ca}^{2+}$ in hyperparathyroidism Max effects at 72 hr
<b>Calcitonin</b> 4 to 8 U per kg IM or SQ q6 hr for 24 hr	Inhibits bone resorption, augments $\text{Ca}^{2+}$ excretion	Initial treatment (after rehydration) in severe $\text{Ca}^{2+}$	Rebound $\text{Ca}^{2+}$ after 24 h, vomiting, cramps, flushing
<b>Glucocorticoids</b> —hydrocortisone, 200 mg IV daily for 3 d	Inhibits vitamin D conversion to calcitriol	Vit D intoxication, hematologic malignancies, granulomatous dz	Immune suppression, myopathy
<b>Mithramycin</b> , 25 mcg/kg/d IV over 6 hr for 3–8 doses	Cytotoxic to osteoclasts	Rarely used in severe hypercalcemia	Marrow, hepatic, renal toxicity

### Hypoparathyroidism

Causes of Hypoparathyroidism	
Absence/low PTH secretion ( $2^\circ$ to parathyroidectomy); hypoplasia/aplasia of parathyroid glands (DiGeorge syndrome); resistance of peripheral tissues to PTH action	
<b>Clinical Features</b> (results from $\downarrow$ Ca)	
<b>Neurologic:</b> Neuromuscular irritability, laryngospasm, inspiratory stridor, tetany, seizures, perioral paresthesia, dementia, <u>Chvostek's sign</u> (tap facial n. $\rightarrow$ painful facial muscle twitches) <u>Trousseau sign</u> (3-min tourniquet application $\rightarrow$ limb spasmlischemia)	<b>Cardiovascular:</b> Hypotension, prolonged QT interval, CHF <b>Musculoskeletal:</b> Muscle cramps & weakness
<b>Diagnosis</b>	
Serum Ca conc $< 4.5$ mEq/L, ionized Ca conc $< 2.0$ mEq/L, $\downarrow$ serum PTH level	
<b>Treatment</b>	
Maintain normal serum & ionized Ca level; treat acute hypocalcemia with 100–300 mg elemental Ca $\rightarrow$ 10–30 mL of 10% Ca gluconate in 150 mL D5W IV over 10 min; initial rate is 0.3–2 mg/kg/h; chronic therapy = (1) oral Ca preparations, (2) ergocalciferol, dihydrotachysterol (forms of vitamin D), (3) phosphate binders (aluminum hydroxide)	

### Anesthesia for Hypoparathyroidism

- **Preop**—serum & ionized Ca should be normalized, esp for pts with cardiac symptoms
- **Intraop**—preexisting hypocalcemia may augment neuromuscular block
  - blood products containing citrate (as well as 5% albumin) will  $\downarrow$  serum Ca level
- **Postop**—hypocalcemia may cause prolonged recovery from neuromuscular blockade

## Pheochromocytoma

- May be associated with autosomal dominant multiple endocrine neoplastic synd (MEN types 2a & b)
- Secretes epinephrine, norepinephrine, & occasionally dopamine
  - Secretion may be intermittent or continuous
  - Change in tumor blood flow, direct pressure, & meds can trigger catecholamine release

Pheochromocytoma—Causes, Features, & Treatment	
Causes	Etiology unknown: 25% familial, 75% sporadic (male:female ratio 2:1) Tumor arising from chromaffine tissue in medulla of adrenal gland & sympathetic ganglia
Clinical features	History of resistant HTN & diabetes Idiopathic dilated cardiomyopathy Hyperadrenergic attacks (self-limited episodes of headache, anxiety, diaphoresis, pallor)
Laboratory features	Measurement of fractionated metabolites of catecholamines, in 24-h urine: normetanephrine >900 mcg; metanephrine >400 mcg Clonidine suppression test: clonidine normally suppresses release of catecholamines from neurons but not from pheochromocytoma
Treatment	Surgical excision; pts should be stabilized preop with $\alpha$ - & $\beta$ -blockers, IV fluids

### Anesthesia for Pheochromocytoma

- **Preop:** Goal = control BP & restore of intravascular volume
  - Start  $\alpha$ -blockade 10–14 d prior to surgery & prior to  $\beta$ -blockade
    - If one accidentally starts  $\beta$ -blockade prior to  $\alpha$ -blockade  $\rightarrow$  severe HTN from unopposed  $\alpha$ -stimulus
  - Phenoxybenzamine =  $\alpha$ -antagonist of choice (another option is prazosin)
    - starting dose = 10 mg qd or bid, then inc dose by 10–20 mg in divided doses every 2–3 d as needed to control BP (goal final dose = 20–100 mg qd)
  - Propranolol 10 mg qid (should be initiated 3–4 d prior to the surgery)
  - Hydrate all patients with pheochromocytoma—carefully in pts with signs of CHF
  - Nitroprusside infusion (also phentolamine IV) for treatment of acute HTN crisis
  - Metyrosine—catecholamine synthesis inhibitor, sometimes used preop
- **Intraop**
  - GA vs. regional—no influence on patient outcome
  - Avoid desflurane, sympathetic stimulants (ketamine, ephedrine) & hypoventilation (cause nonneurogenic release of catecholamines), atracurium & morphine (histamine release)
  - Prepare nitroprusside & phenylephrine infusions in advance
  - A-line before induction,  $\pm$  central line (assessment of intravascular volume),  $\pm$  PA line
  - **Gentle** induction—intubation may cause massive release of catecholamines
  - Tumor manipulation—may cause massive catecholamine release  $\rightarrow$  HTN crisis
  - Suprarenal vein ligation  $\rightarrow$  acute drop in blood catecholamine level  $\rightarrow$  cause hypotension (treat with fluid administration and direct sympathomimetics)
  - Catecholamine-resistant vasoplegia: Can also use vasopressin to reverse
  - Refractory tachycardia: Treat with esmolol (25–300 mcg/kg/min)
- **Postop**
  - Maintain normal BP; in about 50% pts BP will remain elevated
  - Bilateral adrenalectomy  $\rightarrow$  steroid support may be necessary

### Diabetes

Types of Diabetes	
Diabetes Mellitus Types IA & IB	Diabetes Mellitus Type II
<ul style="list-style-type: none"> <li>• 10% of diabetics</li> <li>• Insulin-dependent</li> <li>• Develops early in life</li> <li>• <math>\downarrow</math> Familial risk</li> <li>• Type IA—antibodies to islet cells in &gt;90% cases</li> <li>• Type IB—no antibodies found</li> <li>• Insulin production virtually ceases</li> </ul>	<ul style="list-style-type: none"> <li>• 90% of diabetics</li> <li>• Non-insulin-dependent</li> <li>• Develops at older age</li> <li>• <math>\uparrow</math> Familial risk</li> <li>• Antibodies to islet cells in 0.1–1%</li> <li>• Decreased insulin production</li> </ul>

Pharmacology of Insulin			
Insulin	Onset (h)	Peak (h)	Duration (h)
Lispro, Aspart	within 15 min	1	3-4
Regular	<1	2-4	6-8
NPH	1-2	4-8	12-16
Lente	1-3	6-14	20+
Ultralente	6	14-18	18-24
Glargine	1.5	No distinct peak	24

### Anesthesia for Diabetes

#### • Preoperative

- Check type, duration and severity of diabetes—the more severe, poorly controlled and longstanding is the disease, the higher is the risk of long-term complications.
- Check current therapy for type and dose (diet, oral hypoglycemic drug or insulin)
- Morning blood sugar and HbA1c assay help to assess status of diabetic control. Creatinine level and electrolytes may reflect degree of nephropathy.
- Check for the presence of coronary artery disease, HTN, cerebrovascular disease and peripheral vascular disease, check EKG for presence of rhythm disturbances and prior MIs.
- Consider Na bicitrate and metoclopramide in patients with GERD and gastroparesis.
- Severe peripheral neuropathy may preclude use of regional anesthesia.
- Long-acting insulins should be stopped and substituted by protamine and lente insulins.
- Long-acting sulfonylurea drugs such as chlorpropamide should be stopped and substituted by short-acting agents. Metformin stopped if concern for intraop metabolic acidosis. Type-2-diabetic patients with marked hyperglycemia on oral treatment should be switched to insulin before operation

#### • Emergency surgery

- Stabilize metabolic control/volume status as much as possible (delay surgery if possible)
- Maximize glucose, electrolyte, acid-base status—insulin & glucose infusions
- Saline infusion if volume is depleted (depending on renal function & cardiac status)
- K<sup>+</sup> infusion if renal function is normal & serum K<sup>+</sup> normal or low
- Bicarbonate infusion **only** in pts with severe acidosis

#### • Intraoperative management

- Monitoring blood sugar = mandatory for all insulin dependent pts & poorly controlled pts
- Pts on NPH (neutral protamine Hagedorn) or PZI (protamine zinc insulin)
  - ↑ Risk for anaphylactic protamine reactions (2° to prior sensitization)
  - Insulin requirements in diabetics vary during surgery; must individualize

### American Diabetes Association Recommendations for Target Inpatient Blood Glucose Concentrations

Patient Population	Blood Glucose Target	Rationale
General medical/surgical	Fasting: 90-126 mg/dL Random: <180 mg/dL	Better outcomes, ↓ infection rates
Cardiac surgery	<150 mg/dL	↓ Mortality, ↓ risk of sternal wound infections
Critically ill	80-110 mg/dL	↓ Mortality, morbidity (SICU); ↓ morbidity; ↓ length of stay (MICU)
Acute neurologic disorders	<110 mg/dL	↑ Mortality if admission blood glucose >110 mg/dL

### Protocol for Perioperative Insulin Infusion

1. Mix 50 U regular insulin in 500 mL NS (1.0 U/h = 10 mL/h)
2. Initiate infusion at 0.5–1.0 U/h
3. Check blood glucose level q1–2h & adjust infusion rate as needed
4. Insulin infusion (U/h) = blood glucose (mg/dL)/150

Blood Glucose Conc	Infusion
<80 mg/dL	Turn insulin infusion off for 30 min; give 25 mL of 50% glucose; recheck level in 30 min
80–120 mg/dL	↓ Insulin infusion rate by 0.3 U/h
120–180 mg/dL	No change in insulin infusion rate
180–220 mg/dL	↑ Insulin infusion rate by 0.3 U/h
>220 mg/dL	↑ Insulin infusion rate by 0.5 U/h

### • Postop

- Treat N/V in pts with gastroparesis with metoclopramide as pts have increased risk of infection, MI, hyper/hypoglycemia, CV and renal dysfunction

### • Diabetic emergencies

- **Diabetic ketoacidosis:** usually triggered by trauma or infection in Type I DM
  - Nausea, vomiting, dehydration, polyuria, polydipsia, somnolence → coma
  - Hyperglycemia, wide anion gap metabolic acidosis, ketones in blood & urine, ↓ K<sup>+</sup>
  - Management: Place A-line, consider intubation for severe CNS depression
    - Start insulin infusion (10 U IV, then 5–10 U/h)
    - Normal saline at 5–10 ml/kg/h (fluid deficit of 3–8 L not uncommon)  
*add 5% glucose when blood sugar <250 mg/dL*
    - Replenish K (0.3–0.5 meq/kg/h)
    - Bicarbonate **not** usually required
- **Hyperosmolar, hyperglycemic, nonketotic coma** (usually type II DM)
  - Severe dehydration & assoc with acute hyperglycemia (>600 mg/dL)
  - Treatment: Correct hypovolemia & hyperglycemia
    - Fluid resuscitation with 0.45% saline
    - Give 10 U regular insulin IV stat → insulin drip (see protocol above)
- **Hypoglycemia**—result of stress, missed meal, exercise, alcohol consumption
  - Hypoglycemia is much more dangerous in unconscious pt than hyperglycemia (*safer to err on the side of hyperglycemia*)
  - Symptoms: Diaphoresis, tachy, impaired cognition, confusion, loc & seizures
  - Treatment: 50% IV glucose, initial dose 25 mL

## ADRENAL INSUFFICIENCY

Treatment of Adrenal Insufficiency: Steroid-Equivalent Doses			
Steroid	Glucocorticoid/ Anti-inflammatory Effect	Mineralocorticoid Effect	Half-Life (h)
Cortisone	100 mg	100 mg	8–12
Hydrocortisone	80 mg	80 mg	8–12
Prednisone	20 mg	100 mg	12–36
Prednisolone	20 mg	100 mg	12–36
Methylprednisolone	16 mg	No effect	12–36
Dexamethasone	2 mg	No effect	36–72

### Anesthesia for Adrenal Insufficiency

- **Preop** – administer stress dose of corticosteroid (usually 100 mg hydrocortisone IV)
- **Intraop**
  - Risk of poor fluid loading tolerance, hypoglycemia, ↑ K<sup>+</sup>, dysrhythmias
  - Unexplained hypotension (that is unresponsive to fluids & vasopressors)  
→ Treat with glucocorticoid
  - Avoid etomidate (suppresses adrenal function)
- **Postop**
  - Provide adequate corticosteroid supplementation

## Excess of Corticosteroids (Cushing's Syndrome)

### • Causes

- 1°—Adrenal adenoma/hyperplasia
  - 2°—ACTH secreting pituitary microadenoma (Cushing's dz), ACTH-secreting tumors, exogenous steroid usage
- **Clinical features:** Moon facies, buffalo hump, central obesity, hirsutism, skin atrophy, osteoporosis, easy bruising, diabetes, proximal myopathy, aseptic hip necrosis, mental status changes, pancreatitis, polyuria/polydipsia

### Anesthesia for Cushing's Syndrome

#### • **Preop:** Risk of hypokalemia & glucose intolerance (check both)

- Cushingoid pts may have HTN, CHF, fragile skin, osteoporosis
- Use stress dose steroids in case of iatrogenic Cushing's syndrome.

#### • **Intraop**

- Obese (potentially difficult airway/IV access), often HTN
- Special attention to positioning (skin breaks down easily)
- High-dose opioids may cause resp depression & difficulty with extubation

#### • **Postop course**

- Poor ventilatory performance ( $\downarrow$  FRC), poor mobilization, pressure sores

## Hyperaldosteronism (Conn's Syndrome)

### • Causes

- 1°—(Conn's syndrome) excess secretion of aldosterone by an adrenal adenoma (60%), bilateral adrenal hyperplasia (30%), carcinoma (rare)
- 2°—high plasma levels of renin & aldosterone (due to CHF/liver cirrhosis)

### • Clinical features

- Malignant HTN (centrally mediated or aldosterone-induced)
- $\downarrow$   $K^+$  often severe & may be exacerbated by diuretics  $\rightarrow$  weakness & tetany
- HTN pts often hypovolemic (hypovolemia &  $\downarrow$   $K^+$  indicate severe total  $K^+$  deficit)
- Metabolic alkalosis from  $H^+$  loss

### Anesthesia for Conn's Syndrome

#### • **Preop:** correct $\uparrow$ BP, metabolic alkalosis, hypokalemia

- Spironolactone (up to 400 mg qd) may control HTN & moderate hypovolemia/ $\downarrow$   $K^+$

#### • **Intraop:** if CHF, uncontrolled HTN, hypovolemia present $\rightarrow$ place A-line

- Surgical manipulation of adrenal may release catecholamines  $\rightarrow$  CV instability
- Give corticosteroid & mineralocorticoids in cases of bilateral adrenalectomy

#### • **Postop:** goal = maintain normal BP, electrolyte balance

- Continue corticosteroid & mineralocorticoids in cases of bilateral adrenalectomy

## POSTERIOR PITUITARY GLAND

#### • Posterior pituitary releases oxytocin & antidiuretic hormone (ADH, vasopressin)

#### • ADH stimulates kidneys to conserve water

- Low ADH  $\rightarrow$  diabetes insipidus

- High ADH  $\rightarrow$  syndrome of inappropriate antidiuretic hormone (SIADH) secretion

## Diabetes Insipidus (DI)

#### • **Causes:** Central DI—insufficient ADH by pituitary (damage from head injuries, genetic disorders, infections, vascular dz, tumors)

Nephrogenic DI—lack of kidney response to ADH (from drugs, chronic kidney dz)

#### • **Clinical features:** Thirst, polyuria (up to 20 L/d), low BP, & dehydration

#### • **Diagnosis:** Urine specific gravity of $\leq 1.005$ , urine osmolality $< 200$ mOsm/kg, random plasma osmolality $> 287$ mOsm/kg

#### • **Treatment:** SQ/nasal/PO vasopressin analogues (desmopressin), chlorpropamide, carbamazepine, thiazide diuretics

#### • **Anesthetic management**

- **Preop**—restore intravascular volume, nasal desmopressin 10 mcg bid–tid

#### • **Intraop**

- Total lack of ADH: 100 mU vasopressin before surgery followed by infusion (100–200 mU/h titrated to urine output)
- Partial ADH deficiency: No vasopressin (unless plasma osmolality  $> 290$ )

- **Postop**—continue desmopressin & monitor electrolyte balance

## Syndrome of Inappropriate Antidiuretic Hormone (SIADH) Secretion

Causes of SIADH		
Tumors	CNS Disease	Drugs
Small-cell carcinoma of the lung	Meningitis	Chlorpropamide
Prostate	Head injury	Phenytoin
Pancreas	Tumors	Clofibrate
Thymus	Subarachnoid/subdural hemorrhage	Vincristine
Lymphoma	Cerebral thrombosis/ hemorrhage	Cyclophosphamide
<b>Endocrine Disease</b>	hemorrhage	<b>Pulmonary Lesions</b>
Hypopituitarism	SLE vasculitis	Tuberculosis
Hypothyroidism	Cerebral abscess	Sarcoidosis
Hypoadrenalism	Guillain-Barré syndrome	Aspergillosis
<b>Other Causes</b>	Acute intermittent porphyria	Pneumonia
Postoperative		Lung abscess
Hepatic cirrhosis		
Alcohol withdrawal		

- **Clinical features:** ↓ Na superimposed upon symptoms of underlying pathology
- ↓ Na due to a dilutional effect, **not** Na depletion (may be no clinical symptoms)
- Symptoms: May include nausea, weakness, anorexia; Na <110 mmol/L → coma
- **Diagnosis:** Must distinguish SIADH from other causes (such as dilutional hyponatremia)
  - (causes of dilutional ↓ Na: excess infusion of dextrose/saline drips/use of diuretics)
  - Diagnosis confirmed by serum Na <130 mmol/L, plasma osmolality <270 mOsm/L, urinary Na >20 mEq/L & elevated urine osmolality
- **Treatment:** address underlying problem
  - Release of ADH (from hypophysis or tumor) cannot be suppressed by medical therapy
  - Symptomatic relief: Water intake restriction to 500–1000 mL per 24 h (plasma & urine osmolality should be measured regularly)
  - Demeclocycline: When fluid restriction is difficult
- **Anesthetic management**
  - Correct hyponatremia, monitor volume status by CVP or PA catheter
  - Monitor electrolytes (urine osmolality, plasma osmolality, serum Na) (including immediately after surgery)



# ANESTHESIA FOR OBSTETRIC AND GYNECOLOGIC SURGERY

PALOMA TOLEDO

Physiologic Changes of Pregnancy	
Metabolism and respiration	<ol style="list-style-type: none"> <li>1. Oxygen consumption ↑ by 30–60%</li> <li>2. TV ↑ 45% and RR ↑ 10%</li> <li>3. MV ↑ by 55%</li> <li>4. FRC, RV and ERV ↓ by 20% at term</li> <li>5. ↓ ERV and RV, but TLC same</li> <li>6. MV ↑ by 45% (progesterone effect)</li> <li>7. In labor, MV can ↑ to 300%</li> <li>8. <math>P_{CO_2}</math> ↓ to 28–32 mm Hg — incompletely compensated respiratory alkalosis</li> <li>9. ↓ <math>O_2</math> content (secondary to anemia)</li> </ol>
Circulation	<ol style="list-style-type: none"> <li>1. C.O. ↑'s by 40%, blood volume ↑ 35%</li> <li>2. Uterine perfusion ↑ to 700–900 mL/min (20% cardiac output)</li> <li>3. BP: Decrease in systolic and diastolic pressures, SVR ↓ 20%, HR ↑ 15%</li> <li>4. Aortocaval compression (the gravid uterus compresses the vena cava and also the aorta in the supine position) this can ↓ C.O. by 25%, increase in uterine venous pressure, uteroplacental insufficiency, and possibly fetal distress</li> <li>5. Hemodynamics postpartum: C.O. ↑ 75%, return to prelabor values by 48 h, prepregnancy values by 12–24 wk</li> <li>6. ↑ volume of distribution</li> </ol>
Hematology and coagulation	<ol style="list-style-type: none"> <li>1. Blood volume ↑ 50%, increase in plasma volume &gt; increase in RBC mass → relative anemia</li> <li>2. Plasma cholinesterase conc. ↓ by 25%</li> <li>3. Hypercoagulable state in pregnancy: ↑ platelet turnover, clotting and fibrinolysis</li> <li>4. ↑ 2,3 DPG → right shift of oxyhemoglobin curve → ↑ <math>O_2</math> delivery</li> </ol>
GI	<ol style="list-style-type: none"> <li>1. Uterine enlargement → distortion of gastroesophageal junction and distortion of pyloric angle interfere with gastric emptying</li> <li>2. ↓ In LES tone, ↑ in intragastric pressure leads to ↑ risk of aspiration</li> <li>3. Labor and narcotics ↓ gastric emptying</li> </ol>
CNS	<ol style="list-style-type: none"> <li>1. MAC ↓ by 20–40%</li> <li>2. ↓ Vasopressor response</li> <li>3. ↓ Requirement for epidural &amp; spinal local anesthetics</li> </ol>

Stages of Labor		
Stage of labor	Events	Innervation
1st	Onset of regular contractions to 10 cm cervical dilation	T10–L1
2nd	Complete dilation to delivery of infant	S2–S4
3rd	Delivery of infant to delivery of placenta	S2–S4

## Anesthesia for Labor and Delivery

- Nonpharmacologic analgesia choices: Hypnotherapy, hydrotherapy, and transcutaneous electrical nerve stimulation (TENS)
- Pharmacologic analgesia: Inhalation analgesia, parenteral opioid analgesia (fentanyl, nalbuphine), pudendal block, paracervical block, neuraxial analgesia. Of these, epidural & combined spinal-epidural analgesia are most effective

<b>Common Medications Used in Obstetric Anesthesiology for Systemic Analgesia</b>			
<b>Name</b>	<b>Class</b>	<b>Common Dosage</b>	<b>Duration</b>
Morphine	Opioid	2–4 mg IV	3–4 h
Hydromorphone	Opioid	1–2 mg IM/IV	1–2 h
Meperidine	Opioid	25–50 mg IV	2–3 h
Fentanyl	Opioid	25–50 mcg IV	30–60 min
Nalbuphine	Mixed agonist/antagonist	10 mg IV	3–6 h
Butorphanol	Mixed agonist/antagonist	1–2 mg IV	3–4 h

<b>Choices for Initiation of Labor Neuraxial Analgesia</b>		
	<b>Advantages</b>	<b>Disadvantages</b>
Combined spinal epidural (CSE)	<ol style="list-style-type: none"> <li>1. Rapid onset compared with epidural, important for multiparous women in active labor</li> <li>2. Minimal motor blockade</li> <li>3. Improved overall maternal satisfaction compared with epidural analgesia</li> <li>4. Lower probability of failed epidural catheters</li> </ol>	<ol style="list-style-type: none"> <li>1. Cannot diagnose a non-functional catheter until spinal recedes</li> <li>2. Increased pruritus compared with epidural analgesia</li> </ol>
Epidural	<ol style="list-style-type: none"> <li>1. Ability to slowly titrate level</li> <li>2. Ability to diagnose a functional catheter after placement (important for parturients with bad airways, morbid obesity)</li> </ol>	<ol style="list-style-type: none"> <li>1. Slower onset than CSE</li> <li>2. High concentrations of local anesthetic may lead to motor blockade</li> </ol>
Continuous spinal catheter	<ol style="list-style-type: none"> <li>1. Ability to titrate level</li> <li>2. Rapid onset</li> </ol>	Increased risk of postdural puncture headache compared to CSE or epidural

Source: Adapted from *Lancet* 1995;345:1413–1416 & *Anesthesiology* 2001;95:913–920.

<b>Anesthesia for Cesarean Delivery</b>		
<b>Situation</b>	<b>Choices for Anesthesia</b>	<b>Advantages</b>
Urgent cesarean delivery	<ol style="list-style-type: none"> <li>1. Spinal/CSE</li> <li>2. Epidural</li> <li>3. Extending anesthesia with indwelling epidural catheter or spinal catheter</li> <li>4. GA</li> </ol>	For urgent cesarean deliveries: <ul style="list-style-type: none"> <li>• If patient has an epidural or spinal catheter in situ, it may be dosed for surgery</li> <li>• If the patient does not have an indwelling catheter, a spinal may be performed unless there is a contraindication to regional anesthesia, in which case a general anesthetic should be performed</li> </ul>
Emergency cesarean delivery	<ol style="list-style-type: none"> <li>1. Spinal</li> <li>2. Extending anesthesia with indwelling epidural or spinal catheter</li> <li>3. GA</li> <li>4. Local anesthesia block by surgeon (in case of inability to perform #1–3)</li> </ol>	In emergency cesarean deliveries, decision regarding mode of anesthesia depends on maternal & fetal status: <ul style="list-style-type: none"> <li>• If there is an indwelling epidural catheter, it may be dosed with a fast-onset local anesthetic such as chloroprocaine</li> <li>• If there is severe fetal distress and there is not an indwelling epidural catheter, there may not be time to perform a regional technique; therefore general anesthesia should be performed</li> <li>• If patient and fetus are stable, a regional technique should be performed</li> </ul>

## Contraindications to Regional Anesthesia

Absolute	Patient refusal Severe coagulopathy Sepsis or infection at puncture site Severe hypovolemia Increased intracranial pressure
Relative	Valvular cardiac lesions (severe AS or MS) Thrombocytopenia (see below)
Controversial	Previous back surgery Thrombocytopenia (thresholds vary by institution: <i>Beilin</i> in a retrospective review found no incidence of neurologic complications in women with platelet counts of 69,000–80,000 at time of neuraxial analgesia placement ( <i>Anesth Analg</i> 1997;85:385–388))

## Morbidity and Mortality of GA in Obstetric Population

1. Anesthesia-related complications are 6th leading cause of pregnancy-related deaths in U.S.
2. Incidence of failed intubation is 1:280 (8× higher than nonpregnant population)
3. 73% of anesthesia-related maternal deaths occurred during general anesthesia
4. Majority of women who died from complications of general anesthesia primarily died from airway management problems, including aspiration, induction, or intubation problems; inadequate ventilation; and respiratory failure

Source: Adapted from *Anesthesiology* 1997;86:277–284.

## General Anesthesia: Anesthetic Implications of Maternal Physiologic Changes

### Endotracheal intubation

- Smaller endotracheal tubes required (may have airway edema)
- Increased risk of trauma with nasotracheal intubation
- Increased risk of failed intubation

### Maternal oxygenation

- Increased physiologic shunt when supine
- Increased rate of denitrogenation
- Increased rate of decline of PaO<sub>2</sub> during apnea

### Maternal ventilation

- Increased minute ventilation required

From: Shnider SM, Levinson G. Anesthesia for Cesarean Section. In *Anesthesia for Obstetrics* (3rd ed; Shnider SM, Levinson G eds.) 1993:211–246.

## Common Medications Used in Neuraxial Analgesia & Anesthesia

Scenario	Common Regimens
Labor analgesia initiated by CSE technique	<ul style="list-style-type: none"><li>• 25 mcg fentanyl or 10 mcg sufentanil</li><li>• 2.5 mg bupivacaine with 15 mcg fentanyl</li></ul>
Labor analgesia initiated with epidural technique	Common initial epidural dosing regimens include local anesthetic ± fentanyl 50–100 mcg: <ul style="list-style-type: none"><li>• Bupivacaine 0.125–0.25% (15 mL)</li><li>• Ropivacaine 0.1–0.2% (6–10 mL)</li><li>• Lidocaine 1% (6–10 mL)</li></ul>
Common test dose regimens	<ul style="list-style-type: none"><li>• Lidocaine 1.5% + epi 1:200,000 (3 mL)</li><li>• Fentanyl 100 mcg</li><li>• Air (1 mL) while monitoring precordial Doppler</li><li>• 3% 2-chloroprocaine (3 mL)</li></ul>
Maintenance infusion regimens for epidural analgesia	<ul style="list-style-type: none"><li>• Bupivacaine 0.04–0.125% with fentanyl 1–2 mcg/mL at 8–15 mL/h</li><li>• Bupivacaine 0.125% plain at 8–15 mL/h</li></ul>
Epidural bolus	<ul style="list-style-type: none"><li>• Bupivacaine 0.25–0.5% → 5–10 mL</li><li>• Bupivacaine 0.06–0.125% → 8–15 mL</li><li>• Add: fentanyl 1–3 mcg/mL or sufentanil 0.1–0.5 mcg/mL</li></ul>
Maintenance infusion regimen with spinal catheter	Bupivacaine 0.06% at 1–2 mL/h

Common Medications Used in Neuraxial Analgesia & Anesthesia (Continued)	
Scenario	Common Regimens
Spinal for cesarean delivery	Local anesthetic: bupivacaine 8–12 mg or lidocaine 50–70 mg with fentanyl 15 mcg, $\pm$ morphine 150 mcg, $\pm$ epinephrine 100–200 mcg
Epidural for cesarean delivery	3% 2-chloroprocaine or 2% lidocaine with bicarbonate & epinephrine (15–20 mL total)
Epidural morphine for postoperative pain	3–4 mg

Labor Neuraxial Analgesia Management	
Typical Sequence of Events	Remarks
1. Request for analgesia from obstetrician	No evidence to support delay of epidural placement until an arbitrary cervical dilation
2. Perform preanalgesia evaluation, including physical exam, obtain consent	Per ASA practice guidelines for OB anesthesia: a focused H&P may be associated with $\downarrow$ maternal, fetal & neonatal complications
3. Administer antacid prophylaxis	Patients should receive a nonparticulate antacid
4. Wash your hands, use aseptic technique	
5. Place BP cuff and pulse oximeter; take baseline BP; consider fluid bolus	Measure the blood pressure every 2–3 min during procedure
6. Position the patient	Sitting or lateral position are acceptable Sitting may facilitate landmark identification in obese patients
7. If initiating with CSE, administer intrathecal medications	See table above on (Common Medications Used in Neuraxial Analgesia and Anesthesia) for common dosing regimens
8. Place epidural catheter; administer test dose to assess for intravascular/ intrathecal catheter	
9. If initiating using epidural technique, after a negative test dose, bolus local anesthetic through the epidural catheter	
10. Start epidural infusion	Maintenance regimens include intermittent bolus dosing of epidural catheter or continuous infusion of medications $\pm$ PCEA See table above (Common Medications Used in Neuraxial Analgesia and Anesthesia)
11. Monitor BP for 15–20 min after injection	Both ephedrine and phenylephrine are acceptable vasopressors for use in L&D
12. Follow up until delivery	Monitor maternal vital signs, motor blockade & level of analgesia every 2–3 hr; if inadequate analgesia, administer bolus dose of epidural medication

Source: Adapted from *N Eng J Med* 2005;352:655–565 & *Anesthesiology* 2007;106:843–863.

**Regional Anesthesia: Anesthetic Implications of Maternal Physiologic Changes****Technical considerations**

- Lumbar lordosis increased
- CSF return is unaltered
- Reduced sensitivity of “hanging drop” technique

**Hydration**

- Increased fluid requirements to prevent hypotension\*

**Local anesthetic dose requirements†**

- Subarachnoid dose reduced 20% to 33%

\*Relative to that required by nonpregnant women.

†Change in the segmental dose requirement relative to nonpregnant women.

From: Shnider SM, Levinson G. Anesthesia for Cesarean Section. In *Anesthesia for Obstetrics* (3rd ed; Shnider SM, Levinson G eds.) 1993:211–246.

<b>Anesthesia for Cesarean Delivery</b>	
<b>Typical Sequence of Events</b>	<b>Remarks</b>
1. Set up OR	
2. Perform preanesthetic evaluation, including physical examination, obtain consent; assess need for lab work & blood availability	Per ASA guidelines for OB anesthesia, a focused H&P may be associated with ↓ maternal, fetal, & neonatal complications
3. Administer antacid prophylaxis (regardless of the planned mode of anesthesia)	Patients should receive an H2 receptor antagonist or proton pump inhibitor; metoclopramide, and a nonparticulate antacid
4. Prehydrate patient	10–15 mL/kg crystalloid should be administered 0–15 min before initiation of regional blockade
5. ASA standard monitors, position patient for spinal or epidural	Sitting or lateral position both acceptable Sitting may facilitate landmark identification in obese patients
6. Wash your hands, use aseptic technique	
7. Place spinal or epidural	See table (page 25-3) Common Medications Used in Neuraxial Analgesia and Anesthesia
8. If the patient has an indwelling epidural catheter, dose the epidural catheter	Choice of local anesthetic depends on urgency of delivery; 3% CP has a faster onset than 2% lidocaine, therefore may be used for emergency cesarean deliveries
9. Consider epidural morphine after delivery of the infant	Epidural morphine may be used for postoperative analgesia (has an assoc risk of postpartum resp. depression, especially if combined with other systemic analgesics)
10. Place patient in <b>left uterine displacement</b> position (LUD)	Use a wedge or a towel
11. Ascertain sensory level & document	T4–T6 sensory level should be obtained prior to surgical incision
12. Treat hypotension	Ephedrine 5–10 mg or phenylephrine 100 mcg
13. Administer prophylactic antibiotics if appropriate	
14. After the umbilical cord is clamped, administer oxytocin	10–20 U oxytocin in 500 mL LR

General Anesthesia for Emergency Cesarean Delivery	
Typical Sequence of Events	Remarks
1. CS called by obstetrician; maximize placental perfusion & oxygenation en route to OR	<b>Ensure LUD</b> on transport & in OR Administer O <sub>2</sub> on arrival in OR Administer a nonparticulate antacid ASAP
2. Discuss urgency of procedure with obstetricians	Based on urgency, decide if there is time for a regional anesthetic
3. Focused history and physical, including an airway examination	
4. Preoxygenate well, prepare for a rapid sequence induction	
5. Induce general anesthesia after confirmation of surgical team readiness	Thiopental, propofol, etomidate and ketamine all suitable for induction. Succinylcholine or high-dose of NMBD if there is a contraindication to succinylcholine
6. Before delivery, maintain anesthesia with N <sub>2</sub> O 50% or O <sub>2</sub> 100%, 1 MAC halogenated agent	
7. After delivery, increase N <sub>2</sub> O concentration to 66%, decrease halogenated agent to 0.5 MAC	Opioids & benzodiazepines may be administered after delivery of the infant
8. Extubate once patient is awake & meets extubation criteria	

General Anesthesia: Pharmacology During Pregnancy*
<b>Inhalation anesthetics</b>
<ul style="list-style-type: none"> <li>• Minimum alveolar concentration reduced 20% to 40%</li> <li>• Rate of induction increased</li> </ul>
<b>Induction agents</b>
<ul style="list-style-type: none"> <li>• ED<sub>90</sub> of thiopental reduced 35%</li> <li>• Elimination half-life of thiopental prolonged</li> <li>• Elimination half-life of propofol unaltered</li> </ul>
<b>Meperidine</b>
<ul style="list-style-type: none"> <li>• Elimination half-life unaltered</li> </ul>
<b>Succinylcholine</b>
<ul style="list-style-type: none"> <li>• Duration of blockade unaltered (or decreased)</li> <li>• Sensitivity reduced</li> </ul>
<b>Nondepolarizing muscle relaxants</b>
<ul style="list-style-type: none"> <li>• ED<sub>50</sub> of vecuronium reduced</li> <li>• Elimination half-life of vecuronium and pancuronium shortened</li> <li>• Duration of blockade of atracurium unaltered</li> </ul>
<b>Chronotropic agents</b>
<ul style="list-style-type: none"> <li>• Response diminished</li> </ul>
<b>Pressors</b>
<ul style="list-style-type: none"> <li>• Response variable</li> </ul>

\*Changes relative to nonpregnant women.

From: Shnider SM, Levinson G. Anesthesia for Cesarean Section. In *Anesthesia for Obstetrics* (3rd ed; Shnider SM, Levinson G eds.) 1993:211-246.

Anesthesia for Cervical Cerclage Placement	
Anesthesia for cerclage placement similar to anesthesia for cesarean delivery	Common intrathecal regimens include <ul style="list-style-type: none"> <li>• Bupivacaine 0.5–0.75% 12.5 mg ± fentanyl 15–25 mcg</li> <li>• Lidocaine 5% 50–75 mg + fentanyl 15–25 mcg</li> </ul>

<b>Anesthesia for Multiple Gestations</b>	
Exaggerated physiologic & anatomic changes of pregnancy compared with singleton pregnancy	<ul style="list-style-type: none"> <li>• ↓ TLC, ↓ FRC, ↑ maternal metabolic rate → more rapid development of hypoxemia</li> <li>• ↑ Weight gain</li> <li>• ↑ Risk of aspiration (↓ LES tone)</li> <li>• Blood volume ↑'s an additional 500 mL</li> <li>• ↑ C.O.</li> </ul>
↑ Maternal complications	<ul style="list-style-type: none"> <li>• ↑ Risk of preterm labor, PROM</li> <li>• ↑ Risk of preeclampsia</li> <li>• ↑ Perineal trauma</li> <li>• ↑ Risk of uterine atony, antepartum &amp; postpartum hemorrhage</li> </ul>
Anesthetic management for vaginal delivery	<ul style="list-style-type: none"> <li>• Epidural analgesia for labor</li> <li>• Uterine or cervical relaxation may be necessary for delivery of twin B</li> </ul>
Anesthetic management for cesarean delivery	<ul style="list-style-type: none"> <li>• Regional anesthetic preferred technique</li> <li>• If GA, adequate preoxygenation is required</li> <li>• Adequate IV access, T&amp;S</li> </ul>

<b>Anesthetic Implications of Breech Delivery</b>	
Choice of anesthesia depends on mode of delivery	Most breech infants are delivered by cesarean section, usual anesthetic is a spinal blockade For breech vaginal deliveries, epidural analgesia may be initiated
Cord prolapse	If cord prolapse occurs, anesthesia must be immediately initiated (either epidural or GA)
Head entrapment	Fetal head may become entrapped in cervix; IV nitro or GA can be used to facilitate smooth muscle relaxation, followed by high-conc volatile anesthetic

<b>Implications for Vaginal Birth After Cesarean Section (VBAC)</b>
1. 1% risk of uterine rupture—signs include fetal distress, abdominal pain, uterine tenderness, cessation of uterine contractions, palpable fetal parts in abdomen
2. Continuous FHR monitoring, consider intrauterine pressure monitoring
3. IV access
4. Type & screen or cross-match blood available
5. Epidural analgesia <b>not</b> contraindicated; breakthrough pain that is not relieved between uterine contractions may be indicative of uterine rupture

### **Post-Dural Puncture Headache (PDPH)**

Differential dx: Nonspecific headache, migraines, lactation headaches, cortical vein thrombosis, meningitis, subdural hematoma, subarachnoid hemorrhage  
H&P and +/- neuroimaging necessary for correct diagnosis

**Post-Dural Puncture Headache**

Symptoms	Onset & Duration	Treatment
Positional headaches (worse when sitting/standing, improved when supine) Frontal, occipital, or generalized headaches Associated symptoms include diplopia/photophobia, nausea, vomiting, neck stiffness, tinnitus	Onset of headache typically 24–48 h after dural puncture Duration usually 7–14 d	<i>Conservative management</i> includes: 1. ↑ Fluid intake 2. ↑ Caffeine intake. 3. Oral analgesics such as Fioricet (contains acetaminophen, butalbital, caffeine), NSAIDs  <i>Gold standard:</i> Epidural blood patch: 15–30 mL blood sterilely injected into epidural space; relief often immediate, can take 24 hr

**Common Obstetric Drugs**

Drug	Indication	Mechanism	Side Effects
Ephedrine	Hypotension	$\alpha$ - and $\beta$ -agonist (indirect)	HTN, arrhythmias, myocardial ischemia, ↑ uterine blood flow
Phenylephrine	Hypotension	$\alpha$ -agonist (direct)	HTN, reflex bradycardia, inc. uterine vascular resistance
Oxytocin	Stimulate uterine contractions	Activates myometrial oxytocin receptors, increases sodium permeability	Hypotension, tachycardia, flushing, antidiuretic effect
Prostaglandins (15-methyl $\text{PGF}_{2\alpha}$ )	Cervical ripening, uterine atony	Activates uterine smooth muscle contractions	Bronchoconstriction, vasoconstriction, HTN, nausea, vomiting, diarrhea
Ergot alkaloids (methylergonovine)	Uterine atony	Direct activation of uterine smooth muscle	Arterial and venous vasoconstriction, HTN, coronary vasoconstriction, bradycardia
Terbutaline	Inhibit uterine contractions (tocolysis)	$\beta$ -2 receptor agonist	HTN, arrhythmia, myocardial ischemia, pulmonary edema



**Antihypertensive Drugs Used to Prevent or Treat Hypertension During General Anesthesia**

			Special Properties		
Drug	Administration and Dose	Onset and Duration of Action	Effect on Uterine and Placental Blood Flow	Advantages	Disadvantages
Hydralazine (arteriolar vasodilator)	IV bolus: 5–10 mg	Maximum effect requires 20–30 min after IV administration; Duration about 2 hr	Originally thought to improve flow, this has now been questioned	<ol style="list-style-type: none"> <li>1. Easy to administer; no special equipment or monitoring required</li> <li>2. Maintains maternal cardiac output</li> <li>3. Long history of safe use in obstetrics</li> </ol>	<ol style="list-style-type: none"> <li>1. Slow, unreliable onset</li> <li>2. Maternal tachycardia</li> <li>3. Decreased placental blood flow; total distress</li> <li>4. Neonatal thrombocytopenia</li> <li>5. Maternal nausea</li> </ol>
Labetalol ( $\beta$ and $\alpha$ blocker) ( $\beta_2$ agonist)	IV boluses: 10–20 mg up to total of 1–3 mg/kg	IV onset, 1–2 min; Duration 2–3 hr	Improves uterine and placental blood flow	<ol style="list-style-type: none"> <li>1. Easy to administer; no special equipment or monitoring</li> <li>2. Little risk of overshoot</li> <li>3. No associated fetal bradycardia or distress</li> <li>4. Improved placental blood flow</li> <li>5. Few maternal side effects</li> <li>6. Rapid onset</li> <li>7. Becoming widely used by obstetricians and anesthesiologists</li> </ol>	<ol style="list-style-type: none"> <li>1. Large variation in effective dose</li> <li>2. Alone, may not effectively <math>\downarrow</math> BP</li> <li>3. Use with caution in patients with asthma, COPD, or compromised ventricular function</li> </ol>
Nitroglycerin (venodilator)	Constant IV infusion: 5–50 $\mu$ g/min infusion; 25–50 $\mu$ g/500 ml (50–100 $\mu$ g/ml)	Onset less than 2 min; duration only a few minutes	Questionable, depends on state of maternal hydration; has been associated with fetal deterioration	<ol style="list-style-type: none"> <li>1. Rapid onset and dissipation</li> </ol>	<ol style="list-style-type: none"> <li>1. Need IV pump to administer</li> <li>2. Should make up in glass</li> <li>3. May need arterial line</li> <li>4. Great variation in response</li> </ol>

(continued)

**Antihypertensive Drugs Used to Prevent or Treat Hypertension during General Anesthesia (Continued)**

Drug	Administration and Dose	Onset and Duration of Action	Effect on Uterine and Placental Blood Flow	Advantages	Special Properties	Disadvantages
Trimethaphan Ganglionic blocker	IV boluses 1-4 mg IV infusion: 0.3-5 mg/min (1 mg/ml)	IV onset less than 1 min; duration less 5 min	Minimal, if no severe maternal hypotension	1. Reliable, rapid acting 2. Large molecular weight limits total transfer 3. Rapid offset		1. May require arterial line for BP monitoring 2. Interferes with action of pseudo- cholinesterase resulting in prolonged duration of succinylcholine 3. Histamine release (?) 4. May cause mydriasis
Nitroprusside Direct-acting arterial vasodilator	Constant IV Infusion; 0.15-10 µg/kg/min Infusion; 50 mg/ 500 ml 5% dextrose in water (100 µg/ml)	IV onset less than 1 min; duration only a few minutes	Dilates uterine artery in vitro; no ill effects unless severe hypotension present	1. Rapid onset and offset 2. Potent reliable, antihypertensive 3. No ill effects on fetus		1. Unstable solution, protect from light 2. Easy to overshoot, need arterial line 3. Difficult to titrate 4. Increased intracranial pressure 5. Cyanide toxicity; not a problem with short-term use and infusion <3 µg/kg/min ... 6. Tachyphylaxis

From: Shnider SM, Levinson G. Anesthesia for Cesarean Section. In *Anesthesia for Obstetrics* (3rd ed; Shnider SM, Levinson G eds.) 1993:211-246.

## Sedatives and Nonopioid Adjuncts Used for Labor

Class	Drug	Usual Dose	Onset	Duration	Comments
Barbiturates	Pentobarbital (Nembutal)	100–200 mg PO/IM	30–60 min		Possible antianalgesic affect if used alone
	Secobarbital (Seconal)	100 mg PO/IM			Useful only in very early or latent phase labor
Phenothiazines	Promethazine (Phenergan)	25 mg IV/50 mg IM	20 min	4–5 hr	Possible contribution to maternal hypotension, anti-emetic effect, wide use in combination with opioids
	Propiomazine (Largon)	20–40 mg IV/IM	15–30 min IV, 40–60 min IM	1–2 hr IV, 3–4 hr IM	Shorter onset and duration than promethazine, maternal hypotension, respiratory depression greater than with promethazine
Antihistamines	Hydroxyzine (Vistaril)	60 mg IM	50 min	4 hr	Use to prevent nausea and vomiting with opioids, painful on injection, no IV formulation
Benzodiazepines	Diazepam (Valium)	2–5 mg IV/10 mg IM	5 min	1–2 hr/3–4 hr	Use as treatment for eclamptic seizures, an active metabolite, prolonged half-life in neonate, neonatal depression possibly prolonged, neonatal hypotonia and impaired thermogenesis, rare use in labor
Dissociative	Lorazepam (Ativan)	1–2 mg IV/ 2–4 mg IM	20–40 min	6–8 hr	Shorter elimination half-life but longer clinical effect, not routinely used in obstetrics
	Midazolam (Versed)	1–5 mg IV in increments	3–5 min	1–2 hr	Water soluble, good amnesia, short half-life, not used for labor—primarily adjunct after cesarean delivery
	Ketamine (Ketalar)	10–20 mg IV increments, up to 1 mg/kg over 30 minutes	30–60 sec	5 min	Psychoimetic effects with higher doses, not useful for first-stage labor, used just before delivery or as an adjunct to regional anesthesia, higher doses possibly leading to loss of consciousness and increased uterine tone

PO, oral; IM, intramuscular; IV, intravenous.

From: Shnider SM, Levinson G. Anesthesia for Cesarean Section. In *Anesthesia for Obstetrics* (3rd ed; Shnider SM, Levinson G eds.) 1993:211–246.

## Intrapartum Fetal Assessment and Therapy

Fetal Decelerations		
Type of Deceleration	Timing Relative to Contraction	Etiology
Early	Simultaneous to contraction	Vagal reflex to head compression
Late	Onset: 10–30 s after contraction begins End: 10–30 s after contraction ends	Uteroplacental insufficiency, hypoxemia, fetal circulatory decompensation
Variable	Variable in depth, shape, duration	Head or cord compression

## Uteroplacental Blood Flow

$$\text{Uterine blood flow (UBF)} = \frac{\text{uterine arterial pressure (UAP)} - \text{uterine venous pressure (UVP)}}{\text{uterine vascular resistance (UVR)}}$$

## Factors That Decrease UBF

Decrease in UAP • Any factor that causes maternal hypotension will cause ↓ in UAP • UAP is directly proportional to maternal MAP	1. Aortocaval compression 2. Hypovolemia 2° to hemorrhage 3. Sympathectomy
Increase in UVP	1. Vena cava compression 2. Uterine contractions/uterine hypertonus 3. Skeletal muscle hypertonus (Valsalva)
Increase in UVR	1. Catecholamine release 2. Vasopressin release 2° to hypovolemia 3. Vasopressors (ephedrine)

## Placental Transfer of Medications

Inhalational & IV induction agents, local anesthetics → can cross placenta

- However, bupivacaine highly protein-bound & chloroprocaine highly metabolized (fetal conc. of these drugs lower than when lidocaine is used)

Drugs That Do Not Cross the Placenta	
Muscle relaxants	NMBD's cannot cross owing to high molecular weight & ionization
Anticoagulants	Heparin & LMWH cannot cross owing to high molecular weight
Anticholinergics	Glycopyrrolate does not cross easily; atropine does
Insulin	Insulin cannot cross placenta owing to high molecular weight

Assessment of the Newborn: Apgar Score			
Score	0	1	2
Color	Pale or white	Pink body, peripheral acrocyanosis	Pink
HR	Absent	<100	>100
Response to stimulation	None	Grimace	Cough, sneeze
Muscle tone	Flaccid	Some movement	Moving
Respiration	None	Weak, irregular	Crying, regular

## Interpretation of Apgar Score

Normal: 7–10

Moderate impairment: 4–6

Needs resuscitation: 0–3

<p><b>Antepartum Hemorrhage</b> Placenta previa (placenta located over internal os)</p>	<ul style="list-style-type: none"> <li>• Risk factors: Multiparity, age, previous CS, previous previa</li> <li>• Presentation: Painless vaginal bleeding in 2nd or 3rd trimester</li> <li>• Diagnosis: US versus MRI</li> <li>• Anesthetic mgmt: Large-bore IV access, assess volume status, T&amp;C blood</li> <li>• Choice of anesthesia: GA vs regional, consider ↑ risk of accreta if h/o prev CS</li> </ul>
<p>Placental abruption (separation of the placenta from the site of implantation)</p>	<ul style="list-style-type: none"> <li>• Risk factors: HTN, age, tobacco use, cocaine use, trauma, PROM, h/o previous abruption</li> <li>• Presentation: Painful vaginal bleeding, ↑ uterine activity, uterine tenderness</li> <li>• Anesthesia mgmt: Large-bore IV access, assess volume status, assess coagulation status as placental abruption is assoc. with consumptive coagulopathy, T&amp;C blood</li> <li>• Choice of anesthesia: GA vs regional</li> </ul>
<p>Abnormal placentation (abnormally adherent placenta)</p>	<ul style="list-style-type: none"> <li>• Placenta accreta: Abnormally adherent placenta</li> <li>• 3 subtypes:             <ol style="list-style-type: none"> <li>1. Placenta accreta: Adherence to myometrium, not invading muscle</li> <li>2. Placenta increta: Placenta invades uterine muscle</li> <li>3. Placenta percreta: Placenta invades uterine serosa or other pelvic structures</li> </ol> </li> <li>• Risk factors: Previous CS, placenta previa</li> <li>• Diagnosis: Antepartum dx can be made with US/MRI intrapartum dx with difficulty in separating placenta or at laparotomy</li> <li>• Anesthesia mgmt: Large-bore IV access, assess volume status, assess coagulation status, T&amp;C blood</li> <li>• Choice of anesthesia: GA vs regional</li> </ul>
<p>Uterine rupture</p>	<ul style="list-style-type: none"> <li>• Incidence: &lt;1% of pts with previous uterine surgery</li> <li>• Presentation: Vaginal bleeding, hypotension, fetal distress, ± pain</li> <li>• Risk factors: Previous uterine surgery, uterine trauma</li> <li>• Treatment: Emergent CS</li> <li>• Anesthetic mgmt: Large bore IV access, assess volume status, assess coagulation status, T&amp;C blood, GA or extension of epidural anesthesia if pt is hemodynamically stable</li> </ul>
<p>Vasa previa (fetal vessels transverse membranes ahead of fetal presenting part)</p>	<ul style="list-style-type: none"> <li>• Risk factors: Placenta previa, multiple gestation, abnormal placenta, IVF pregnancy</li> <li>• Obstetric management: immediate cesarean delivery</li> <li>• Choice of anesthesia: GA vs regional depending on urgency of CS</li> </ul>

**Postpartum Hemorrhage**

- Incidence: 10% of all deliveries
- Definition: 500 mL EBL in vaginal delivery, 1000 mL EBL for CS
- Anesthetic management:
  - Assess pt volume status, hemoglobin & coagulation status, IV access
  - Send T&S or type & cross-match for blood
  - Administer IV fluid, blood, & vasopressors as indicated

Postpartum Hemorrhage	
Cause	Comments
Uterine atony (risk factors: Multiple gestation, polyhydramnios, chorioamnionitis, precipitous labor, high conc of oxytocin use in labor, high conc of halogenated agents)	<ul style="list-style-type: none"> <li>Consider uterine massage</li> <li>Consider uterotonic agents (oxytocin, cytotec, ergot alkaloids, or prostaglandin)</li> </ul>
Retained placenta	<ul style="list-style-type: none"> <li>Treatment is manual removal of placenta</li> <li>GA for unstable pts, otherwise consider epidural</li> <li>Additional uterine relaxation may be obtained with 50–250 mcg of nitroglycerin or ↑ conc of volatile agent</li> </ul>
Abnormal placentation	See discussion above
Genital trauma (vaginal, vulvar, & cervical lacerations)	Neuraxial analgesia may be extended for repair if pt is hemodynamically stable
Uterine inversion	<ul style="list-style-type: none"> <li>Epidural/spinal anesthesia if pt hemodynamically stable</li> <li>Uterine relaxation may be necessary for replacement of inverted uterus (consider iv nitroglycerin or volatile anesthetics)</li> </ul>

### Hypertensive Disorders of Pregnancy

Differential Diagnosis for Hypertension in Pregnancy				
	Time of Diagnosis	Resolution After Pregnancy	Proteinuria	Comments
Gestational hypertension (gHTN)	<20 weeks' gestation	Yes	No	25% of women with gHTN may develop preeclampsia
Chronic hypertension	<20 weeks' gestation	No	No	
Pregnancy-induced hypertension	>20 weeks' gestation	Yes	No	
Preeclampsia	>20 weeks' gestation	Yes	Yes (>300 mg on 24-h urine sample or a random urine protein of 30 mg/dL)	<ul style="list-style-type: none"> <li>Seizures indicate progression to eclampsia</li> <li>HELLP syndrome (see below)</li> </ul>

### Preeclampsia

- Definition: Hypertension + proteinuria after 20 weeks' gestation
- Pathophysiology: Exact mechanism unknown, may involve imbalance between prostaglandins (thromboxane A & prostacyclin), abnl sensitivity to catecholamines, or antigen-antibody reactions between fetal & maternal tissues
- Treatment: Only definitive cure for preeclampsia is delivery of the infant

Severity of Preeclampsia			
	Blood Pressure Criteria	Proteinuria	Comments
Mild preeclampsia	sBP >140 or 30 mm Hg above normal dBP >90 or 15 mm Hg above normal	<5 g/24 h	Mild preeclampsia may progress to severe preeclampsia; may lead to peripheral edema
Severe preeclampsia	sBP > 160 or dBP > 110	>5 g/24 h	End-organ symptoms may include HA, visual changes, RUQ pain, lab abnormalities (>LFTs, thrombocytopenia), oliguria, or IUGR

<b>Anesthetic Management of Preeclampsia</b>		
<b>Intrapartum management of HTN</b>	Hemodynamic goals include prevention of severe HTN (risk of cerebral hemorrhage) while still maintaining adequate placental perfusion	Hydralazine, labetalol, methyldopa, sodium nitroprusside, or nitroglycerin
<b>Seizure prophylaxis</b>	Mg (4-g bolus, followed by an infusion). Mg ↑ sensitivity to NMBD & ↓ response to vasopressors	Serum Mg conc: 4–6 mEq/L → therapeutic 5–10 mEq/L → QRS widening 10 mEq/L → ↓ deep tendon reflexes 15 mEq/L arrhythmias, respiratory weakness 25 mEq/L cardiac arrest <i>Treatment for Mg toxicity = calcium chloride</i>
<b>Coagulation status</b>	Preeclamptics may become thrombocytopenic due to platelet aggregation & consumption	Check platelet count, check coagulation status if elevated LFTs If pt is not thrombocytopenic or coagulopathic, may use regional anesthesia/analgesia
<b>Monitoring</b>	Consider arterial BP monitoring in pts with severe HTN, or CVP monitoring if volume status unclear or pt is oliguric	

<b>Management of HELLP Syndrome</b>	
Diagnosis	HELLP (hemolysis, elevated LFTs, low platelets) = severe form of preeclampsia; resolution of hemolysis/thrombocytopenia may require 24–72 h
Anesthetic management	Delivery = only treatment for HELLP syndrome—follow management for preeclampsia If parturient is thrombocytopenic, use neuraxial analgesia/anesthesia with caution

### Postpartum Tubal Ligation (PPTL)

- Benefits of immediate PPTL include
  - Ease of access to fallopian tubes (uterus & ovaries are out of pelvis)
  - ↓ Risk of bowel laceration, vascular injury
  - ↓ Duration of hospital stay, ↓ cost (compared to outpatient procedure)

<b>Anesthetic Considerations for Postpartum Tubal Ligation</b>	
NPO status	NPO guidelines apply (see Chapter 1, on Preop Evaluation) Gastric emptying ↓ if opioids have been used
Risk of postpartum hemorrhage (PPH)	Pts at risk for PPH for 24 h Consider uterine bleeding if pt is hemodynamically unstable intraoperatively
Local anesthetic requirements	↓ Requirement; returns to prepregnancy value after 12–36 h postpartum
Regional vs general anesthesia	Regional preferred (spinal or epidural if in situ) Risk of epidural catheter failure ↑ if delivery to surgery interval >8–10 h

<b>Anesthetic Goals for Nonobstetric Surgery in the Pregnant Patient*</b>	
1. Maintain placental perfusion	Avoid maternal hypotension, hypocarbia, hypoxemia, catecholamines
2. Avoid preterm labor	
3. Avoid possible teratogens	Highest risk in 3rd trimester; benzodiazepines, N <sub>2</sub> O, avoid ketamine in 1st trimester (although no evidence that anesthetic drugs cause problems with organogenesis)
4. Antacid prophylaxis	
5. Choice of anesthesia (regional vs GA)	Use regional when possible; if GA, RSI
6. FHR monitoring	Monitor pre- & postop after 16 weeks' gestation

\*Note: elective surgery should be postponed until postpartum if possible

<b>Anesthetic Considerations for Gynecologic Surgery</b>				
<b>Procedure</b>	<b>Indication</b>	<b>Positioning</b>	<b>Choice of Anesthesia</b>	<b>Anesthetic Considerations</b>
1. Hysteroscopy	1. Eval abnormal uterine bleeding 2. Eval cause of infertility	Lithotomy	MAC, GA (LMA or ETT), or neuraxial anesthesia	1. Procedure generally short 2. Consider antiemetic prophylaxis 3. Potential for fluid overload (uterus often distended with a viscous fluid)
2. Dilation and curettage (D&C)	1. Eval of uterine bleeding 2. Incomplete or missed abortion	Lithotomy	Paracervical block, MAC, spinal, or GA (LMA or ETT)	1. Consider antiemetic prophylaxis 2. Rare risk of uterine perforation or hemorrhage
3. Dilation and evacuation (D&E)	1. 2nd-trimester abortion (for fetal or maternal indications)	Lithotomy	Paracervical block, MAC, spinal, GA (LMA or ETT)	1. Consider antiemetic prophylaxis 2. Rare risk of uterine perforation or hemorrhage
4. Conization of the cervix (LEEP procedure)	1. Diagnosis & treatment of cervical dysplasia	Lithotomy	MAC, GA (LMA or ETT), spinal	1. Often short procedures; do not require anesthesia
5. Total abdominal hysterectomy (TAH) ± BSO	1. Uterine or cervical cancer 2. Fibroids unresponsive to hormone therapy	Supine	GA (ETT) ± epidural for postop pain relief, or neuraxial anesthesia (epidural)	1. If hx of chemo, consider cardiac & pulm implications 2. Consider invasive monitoring
6. Vaginal hysterectomy	3. Endometriosis	Lithotomy		3. Potential for blood loss—T&S or cross-match
7. Robotic-assisted laparoscopic hysterectomy		Steep Trendelenburg	GA (ETT)	Same considerations as for TAH & laparoscopic surgery



**Anesthetic Considerations for Gynecologic Surgery (Continued)**

<b>Procedure</b>	<b>Indication</b>	<b>Positioning</b>	<b>Choice of Anesthesia</b>	<b>Anesthetic Considerations</b>
8. Laparoscopic surgery	May be used for many surgeries (e.g., ectopic pregnancy, hysterectomy, postpartum tubal ligation)	Trendelenburg	GA with ETT	<ol style="list-style-type: none"><li>1. Possible resp compromise from CO<sub>2</sub> insufflation (usually 15 mm Hg)</li><li>2. ↑ Min ventilation required if insufflation with CO<sub>2</sub></li><li>3. Consider antiemetic prophylaxis</li></ol>
9. Vulvectomy or other oncologic surgeries	Pelvic malignancy	Supine/lithotomy	GA with ETT ± epidural for postop pain relief	<ol style="list-style-type: none"><li>1. If hx of chemo, consider cardiac &amp; pulm implications</li><li>2. Consider invasive monitoring</li><li>3. Potential for blood loss—T&amp;S or crossmatch</li></ol>

## Anatomy

### Upper Airway

- Infants & small children
  - Larger tongue & shorter mandible relative to oral cavity
  - Larger & narrower epiglottis, angled away from (not parallel to) tracheal axis (*straight blades may improve epiglottic elevation*)
- Infant glottis = cephalad & anterior, creating more acute angle for laryngoscopy
  - Vocal cords have lower anterior attachment
  - ETT can become caught upon anterior commissure of vocal folds
- Nonexpandable cricoid cartilage = narrowest part of infant airway
  - ETT may pass easily through vocal cords but traumatize area beneath (*since this area is smaller than the glottic aperture*)
  - Uncuffed ETT usually used if pt <10 yr of age to prevent subglottic edema/stridor
  - Recommend 20- to 25-cm H<sub>2</sub>O air leak to ensure appropriate fit & limit swelling
- Consider cuffed ETT for major abdominal/thoracic procedures
  - Allows for positive-pressure ventilation
  - Presence of a cuff ↑ external diameter by up to 0.5 mm
  - Stridor more likely when no air leak exists at 30 cm H<sub>2</sub>O
- Neonatal trachea = only 4 cm long; care must be taken to avoid mainstem intubation
- Tracheal diameter = 4–5 mm
  - Airway resistance & laminar flow worsened by any amounts of edema
- Infants—described as **obligate nasal breathers** (2° to immature coordination between resp drive & oropharyngeal sensorimotor input)
  - Since larynx is positioned higher (more posterior) tongue rests against hard & soft palates during quiet breathing
  - Infants develop better control by 4–5 months of age

### Venous Access

- Vascular access often preceded by inhalation induction
  - Limits procedural anxiety & withdrawal reflexes & provides vasodilatation
  - Pt at high risk for aspiration (full stomach) should have awake IV inserted (*parents may be helpful to facilitate this task*)
- Access sites: Include dorsum of hand, antecubital space, prominent scalp veins in infants, saphenous veins adjacent to medial malleoli; intraosseous → consider in presence of severe dehydration/trauma (e.g., burns)
- Umbilical artery catheterization → may allow for quick vascular access in newborn (*placement should be confirmed by x-ray*)
- All IV lines should be carefully examined to make sure there are no air bubbles (*air may cross PFO & result in embolization*)

## Physiology

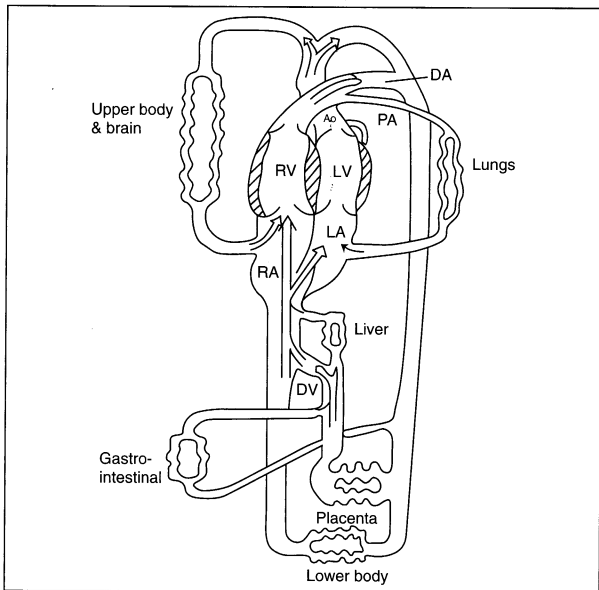
### Transition from Fetal to Neonatal Circulation

- Fetus receives oxygenated blood via umbilical vein
- Series of intracardiac (foramen ovale) & extracardiac (ductus arteriosus & ductus venosus) shunts create parallel fetal circulatory system
  - Allows blood to bypass high resistance of pulmonary vessels
  - Deoxygenated blood is returned to placenta via umbilical arteries
- Transition to neonatal circulation → occurs when umbilical cord is clamped & spontaneous breathing begins
  - Pulm vascular resistance drops, changing blood flow from parallel to series
    - Left-sided intracardiac pressures ↑ to close foramen ovale
    - Highly oxygenated blood & ↓ levels of placental prostaglandins → Stimulate contraction & closure of ductus arteriosus
- Shunts not anatomically closed immediately after birth
  - Some conditions (hypoxia, acidosis, sepsis) → persistent fetal circulation

### Respiratory

- Major conducting airways established by 16th week of gestation
  - Acinus & all distal structures continue development until term
  - Alveoli mature after birth, continue to ↑ in number until 8 yrs of age
- Infant chest wall deforms easily → because of cartilaginous structure
  - Accessory muscles provide limited support (poor anatomic rib configuration)
  - Infantile diaphragm contains 20–25% of fatigue-resistant type I muscle fibers → paradoxical chest wall movement when there is ↑ inspiratory effort

**Figure 26-1 Fetal Circulation.** Reproduced with permission from Davis et al, *Smith's Anesthesia for Infants and Children*, Figure 3-1.



- $\uparrow$  Work of breathing  $\rightarrow$  deterioration into resp failure, esp in premature infant
- FRC similar in both infants & adults per kg
  - Owing to limited elastic recoil, infant closing capacity may near/exceed FRC: Leads to air trapping  $\rightarrow$  when small airways close at end-expiration
  - Cause of age-related changes in  $\text{PaO}_2$
- $\uparrow$  Tracheal compliance in infants; can lead to dynamic tracheal collapse
- Changes in  $\text{PaO}_2$ ,  $\text{PaCO}_2$  & pH control ventilation  $\rightarrow$  act on chemoreceptors
  - Degree of response directly related to gestational & postnatal age
  - Hypoxia stimulates newborn resp effort; high conc of  $\text{O}_2$  may depress it
  - Nonspecific factors (blood glucose, Hct, temp) also affect infant breathing

#### Cardiovascular

- Infant/neonatal myocardium contains less contractile tissue than adult heart
- Neonatal ventricle less compliant during diastole & generates less tension
- Infant ventricle cannot adequately  $\uparrow$  stroke volume when metabolic needs  $\uparrow$
- Cardiac output proportional to changes in heart rate
  - Bradycardia  $\rightarrow$   $\downarrow$  cardiac output; factors contributing to bradycardia (hypoxia, hypercarbia, surgical manipulation) should be corrected
- Consider empiric anticholinergic admin (atropine) to offset laryngoscopy-induced bradycardia

#### Renal

- Kidneys very active in utero & produce copious amounts of urine  
(contribute to maintenance of amniotic fluid volume)
- At birth GFR = 15–20% of adult levels; reaches 50% within 2 weeks & 100% by 1 yr  
(low GFR means infants cannot excrete excessive fluid loads/renal cleared drugs)
- Ability to excrete organic acids poorly developed in neonates  
(causes observed “physiologic acidemia” of newborn)
- Concentrating ability poor & newborns can conc urine only to 600–800 mOsm/kg

## Hepatic

- Gluconeogenesis & protein synthesis begin at 12 weeks gestation (*liver structure near term similar to adults; functional development lags*)
- Preterm & small for gestational age infants usually have diminished glycogen stores
  - Prone to hypoglycemic episodes after delivery
  - Treat hypoglycemia promptly (D10 at 4 mL/kg/hr)
- Albumin levels in preterm infants are often low and affect drug binding & availability
- **Physiologic jaundice:** Due to RBC breakdown & ↑ enterohepatic circ of bilirubin
  - As opposed to pathologic jaundice (encephalopathy from kernicterus)

## Gastrointestinal

- Esophageal tone ↓ in many newborns; reaches adult level ≈ 6 weeks
  - Projectile vomiting after feeding = classic sign of pyloric stenosis
- Meconium (water, pancreatic secretions + intestinal epithelial cells)
  - Usually passed a few hours following delivery
  - Premature infants often have delayed evacuation
    - May also indicate GI dz (meconium ileus/intestinal atresia)

## Hematopoietic

- Neonatal estimated blood volume = 85–90 mL/kg at term, gradually ↓ with age
- HbF: Most prevalent after birth, greater O<sub>2</sub> affinity than HbA (adult)
  - “Physiologic anemia of infancy” due to HbF (replaced with HbA by 3 months)
  - Hg levels rise to 12–13 g/dL by age 2; in adults, they reach 14 for females & 15.5 for males
- Vit K–dependent coag factors ≈ 40% adult levels (2° to immature liver synthesis)
- Prolonged PT normally seen in both preterm & full-term infants

## Neurologic

- Brain growth phases: (1) Neuronal cell division (15–20 wk gestation) & (2) glial cell division (25 wk–2 yr); myelination continues into 3rd yr
- Malnutrition, disruption of blood–brain barrier, & trauma may affect development
- Developmental milestones represent average rate of neurologic maturation
  - Deviations from norm do **not** necessarily indicate significant problems
  - Premature infant’s developmental delay may be considered normal (*depending on degree of prematurity*)

## Temperature Regulation

- Infants lose heat rapidly 2° to: ↑ Surface area:wt ratio, lack of adipose/SQ tissues
  - Infants rely on nonshivering thermogenesis
    - Catecholamine-mediated increase in brown fat metabolic activity
    - → Catecholamines also cause: Pulm & peripheral vasoconstriction, ↑ O<sub>2</sub> use, hypoxia, acidemia
- Effective methods for limiting heat loss include
  - ↑ Ambient room temp, cover infant with thermal insulator, use of heat lamp

## Pharmacology

### Body Fluid Composition

- TBW in infants ≈ 85%, ≈ 60% by 1 yr of age; extracellular water (ECW) dec faster than intracellular water (ICW)
- Fat, muscle, organ wt are age-dependent, affect pharmacodynamics/kinetics
- Infants have greater ECW than adults → volume of distribution for drugs is expanded
  - Drugs with limited tissue uptake may require higher wt-based dosing

### Organ System Maturity

- Enzyme systems involved in biotransformation relatively immature
  - Drugs may have prolonged elimination half-lives

### Protein Binding

- Often only unbound drug is clinically active (many drugs are protein-bound)
- Albumin is major binding protein for acidic drugs (benzos & barbiturates)
- Neonatal albumin quantitatively & qualitatively deficient → dec binding capacity

### Receptors

- Age-related variations in response to drugs may be 2° to receptor sensitivities

## Preoperative Evaluation

### Psychological Assessment

- Use clear, simple language to discuss potential risks (*avoid disclosing info in a child’s presence that may ↑ anxiety*)

- Psychological goals of the preoperative interview
  1. Identify specific causes of anxiety & evaluate potential benefit of preop sedation
  2. Address potential risks pertinent to procedure
  3. Describe reasonable expectations for postop discomfort, side effects
  4. Reassure both parents & patient
- Child-life specialists can facilitate pt education & relieve anxiety
  - Comfort objects may accompany pt into OR
  - Parental presence may be useful; perceived legal risks are largely exaggerated

Age-Based Guidelines for Interacting With Pediatric Patients	
Neonates & infants <9 months	Do not typically fear strangers & separation is usually uncomplicated; preop sedation usually unnecessary & may prolong emergence
Toddlers	Aware of their environment but limited reasoning ability & reality testing Benefit <b>most</b> from preop sedation & parental participation at induction
School-age children	Often do not deal well with loss of control; may admit to fear of waking up during surgery; may be reluctant to ask questions Sedation options should be discussed with parents
Adolescents	Often focus on cosmetic side effects & altered body image after surgery Preop sedation may extend beyond drugs to include meditation or music

Suggested Preoperative Sedation Drugs and Dosing			
Drug	Route	Dose (mg/kg)	Onset Time (min)
Midazolam	IV	0.01–0.03	<5
	Oral	0.5–0.75	15–30
	Nasal	0.1–0.2	15–30
	IM	0.05	5–10
Fentanyl	PR	1–3	10
	IV	0.001–0.005	<5
Ketamine	Oral (“Actiq”)	0.010–0.020	15–20
	IV	1–2 mg/kg	1–2
Methohexital	Oral	5 mg/kg	20–45
	IM	2–3 mg/kg	5–10
Chloral hydrate	PR	20–30 mg/kg	5–10
	PO, PR	30–100 mg/kg	30–60

Nonnarcotic Analgesics for Children			
Drug	Dose	Interval	Route
Ibuprofen	4–10 mg/kg	q6–8h	PO
Naproxen	5–7 mg/kg	q6–8h	PO
Tolmetin	5–7 mg/kg	q6–8h	PO
Choline magnesium salicylate	10–15 mg/kg	q8h	PO
Ketorolac	First dose: 1 mg/kg Repeat dose: 0.5 mg/kg	q6h	IV or IM
Acetaminophen	10–15 mg/kg	q4h	PO
	15–20 mg/kg	q4h	PR

Source: Adapted from Kahan M. Pain management in the critically ill child. In Hamill RJ, Rowlingson JC, eds. *Handbook of Critical Care Pain Management*. New York: McGraw-Hill, 1994:507–521.

Preoperative Evaluation of Pediatric Patients With a Systems Focus	
History	Important Questions and Pertinent Findings
Prenatal care & delivery	Gestational age; Apgar score at birth; duration of intubation & ventilatory support; assoc congenital conditions (BPD, cyanotic heart dz); freq of hospitalizations; review of growth curves (failure to thrive); persistence of apnea/bradycardia

Preoperative Evaluation of Pediatric Patients With a Systems Focus (Continued)	
History	Important Questions and Pertinent Findings
Airway	Dysmorphic features (e.g., Pierre-Robin = assoc with difficult airway); micrognathia, loose teeth; advanced caries
Respiratory	Symptoms of acute/recent URI; asthma; sick contacts; 2nd-hand smoke exposure; presence of wheezing, stridor, nasal flaring, cyanosis; sleep apnea
Cardiac	Murmurs assoc with PFO, PDA, congenital heart dz; freq/duration of cyanotic spells; tachypnea; poor feeding tolerance
Gastrointestinal	Repetitive vomiting; delayed meconium passage; abd distention
Hematologic	Bruising; pallor; family history of sickle cell/thalassemia
Neurologic	Patterns of seizure activity; developmental delay; motor weakness; hypotonia; evidence of ↑ ICP

Estimated Pediatric Vital Sign Parameters				
Age	RR	HR	SBP	DBP
Preterm	55–60	120–180	45–60	20–45
Neonate	40–55	100–160	55–75	20–60
Infant (<6 months)	30–50	80–140	85–105	55–65
1 year	30–35	80–120	90–105	55–65
6 years	20–30	75–110	95–105	50–70
10 years	20–30	80–100	95–110	55–70
16 years	15–20	60–80	110–125	65–80

#### OR Equipment and Setup

- Oral ETT size = (age/4) + 4; depth = (ETT internal diameter × 3)

Suggested ETT Size Selection & Appropriate Insertion Depths			
Age/Weight	Internal Diameter (mm)	Depth (oral, cm)	Depth (nasal, cm)
<1.5 kg	2.5	9.0–10.0	12.0–13.0
1.5–3.5 kg	3.0	9.5–11.0	13.0–14.0
Term	3.5	10.0–11.5	13.5–14.5
3–12 months	4.0	11.0–12.0	14.5–15.0
12–24 months	4.5	12.0–13.5	14.5–16.0

Recommended Laryngoscopic Blade & LMA Sizes			
Age	Blade	Weight	LMA Size
Premature	Miller 0	<5 kg	1
Neonate	Miller 0	5–10 kg	1.5
1–4 years	Miller 1	10–20 kg	2
4–10 years	Miller 2, Mac 2	20–30 kg	2.5
Adolescent	Miller 2, Mac 3	>30 kg	3
Normal/large adults	Miller 2, Mac 3–4	60–90 kg	3–5
Large adults	Miller 2–3, Mac 3–4	>90 kg	5

#### Intravenous Fluids

- Fluid replacement: based on NPO deficit, ongoing maintenance requirement, blood loss, & potential for surgically induced fluid shifts (3rd spacing)
- Lactated Ringer's often appropriate
- Normal saline advised for pts with renal dysfx, mitochondrial myopathym or neuro-surgical procedures
- Dextrose soln for neonates (limited glycogen stores) & diabetes who got hypoglycemic meds
- "Buretrol" or other metered device often used for children <6 months
  - Allows careful control of fluid admin
  - Older children may receive iv fluids through a 60-drop/mL gravity infusion set
  - **Remove all air bubbles** (risk of PFO) from IV tubing & injection ports

## Emergency Drugs

- All emergency drugs should have 1.5-in. 22-gauge needle for emergency IM injection

Suggested Doses of Common Emergency Drugs		
Drug	IV	IM/(SQ)
Atropine	0.01–0.02 mg/kg	0.02 mg/kg
Succinylcholine	1–2 mg/kg	3–4 mg/kg
Ephedrine	0.1–0.2 mg/kg	—
Epinephrine	10 mcg/kg	(10 mcg/kg)

## Techniques

### Induction

Comparison of Pediatric Induction Methods and Commonly Used Drugs		
Technique	Advantages	Disadvantages
Mask induction (sevoflurane)	Brief onset (2–3 min) Avoids awake IV Spontaneous respiration Parental participation Facilitates IV start via vasodilation	Breath-holding/laryngospasm Contraindicated for full stomach/MH Unprotected airway Gases are cold & dry Requires good seal
Intravenous (propofol)	Rapid onset (<30 sec) Minimizes duration of unprotected airway	Anxiety about “shots” Pain upon injection Malfunction Extravasation
Intramuscular (ketamine)	Brief (2–4 min) Can inject at multiple sites Does not require cooperation	Pain upon injection Difficulties with obese children Secretions with ketamine Unprotected airway Nerve injury
Rectal (methohexital)	Rapid onset (1–2 min) Quick clearance	Useful only in young children No prepackaged delivery device Unprotected airway

### Maintenance

- Volatile agents or TIVA-based techniques can be used
  - Drug selection guided by coexistent dz & surgery duration
- “4–2–1 rule” can guide fluid replacement
  - Neonates & infants require additional care to avoid fluid overload (metered devices) & provide glucose supplementation (D5NS)
  - EBV should always be calculated to guide fluid when surgery has high EBL
  - While children tolerate lower Hcts, they also have ↑ metabolic rates & O<sub>2</sub> needs

Pediatric Maintenance Fluid Calculations	
Weight	Rate
<10 kg	4 mL/kg/hr
10–20 kg	40 mL/hr + 2 mL/kg/hr for each kg > 10 kg
>20 kg	60 mL/hr + 1 mL/kg/hr for each kg > 20 kg

Estimated HCT and EBV		
Age	HCT (%)	EBV (mL/kg)
Premature	45–60	90–100
Neonate	45–60	80–90
3–6 months	30–33	70–80
6 months–1 year	32–35	70–80
1–12 years	35–40	70–75
Adult	38–45	60–70

## Clinical Conditions

### Respiratory

#### Apnea of Prematurity

- Newborns <34 wk gestational age → inc risk for perioperative resp complications
  - Immature response to hypoxia & hypercarbia → central apnea
- GA may exacerbate; regional may ↓ incidence of postop spells (does not eliminate); other contributing factors include hypoglycemia, hypothermia, anemia
- Therapies: Positioning (avoid mechanical airway obstruction), resp stimulants (methylxanthine/caffeine 10 mg/kg) in high-risk pts, appropriate monitoring
- Usually premature newborns <60 wk postconception → need continuous cardiorespiratory monitoring for 24 hr postop (*no outpatient procedures*)

#### Meconium Aspiration

#### Prematurity—Perioperative Concerns

- ↑ Risk of hypothermia
- Unable to regulate glucose control
- ↑ Risk of postop apnea (esp. if <50 weeks postconceptional age)
- Retinopathy of prematurity (esp if. <44 week postconceptional age)
- Pulmonary dysfunction

- Presence of thick, meconium stained amniotic fluid during concerning aspiration → may result in profound resp distress & hypoxemia
- Suction nares & oral cavity immediately after delivery
  - Transfer newborn to a radiant warmer & intubate
  - Apply suction & withdraw ETT; repeated until only trace meconium seen
- Positive-pressure ventilation (PPV) should not be used initially
  - Can spread meconium distally into bronchial tree
  - If bradycardia/cyanosis develop → gentle PPV with 100% O<sub>2</sub> pressure

#### Bronchopulmonary Dysplasia (BPD)

- Lung dz of newborn; problematic to accurately define as presentation has varied
- Initially described lung injury from aggressive mech ventilation & high FiO<sub>2</sub>
  - Develop smooth muscle hypertrophy, airway inflammation, pulm HTN
  - Exogenous surfactant, steroids & gentler vent modes → improved survival (*overall dz incidence has not decreased*)
- Babies <30 weeks gestational age →
  - Present with immature lung parenchyma & dysfunctional alveoli
- Pulm dysfx will be persistent to varying degrees (may affect later management)
  - Airway hyperreactivity & resp infections common
  - Supportive care in the OR → gentle ventilation, limit barotrauma, β<sub>2</sub>-agonists
  - Consider need for postop ICU admission

#### Congenital Diaphragmatic Hernia (CDH)

- Diaphragmatic defect → presents at birth with cyanosis, resp distress, scaphoid abd
  - Get herniation of abd contents → result in lung & pulm vessel hypoplasia
  - **Not** simply lung compression & atelectasis
- Surgical correction → postponed several days to optimize pt cardiopulmonary status
  - Severe defects often require more support (ECMO or nitric oxide)
- Anesthetic management
  - Intubation (awake, inhalation or RSI) should minimize gastric distention
  - Maintenance usually volatile + narcotic (avoid N<sub>2</sub>O → risk of pneumothorax)
  - A-line + CVP for blood sampling/fluid resuscitation; temp maintenance important
  - Maintain low pulm vascular resistance → avoid hypoxia & hypercarbia
- Contralateral pneumothorax → sudden cardiovascular collapse & ↓ lung compliance
- Postop: Transfer to NICU intubated & paralyzed

#### Asthma

- Triad of airway inflammation, reversible flow defects, airway hyperreactivity
- Signs & symptoms: Wheezing, dyspnea, chest tightness, coughing
- Preop interview: Freq. of episodes, current meds, hospital admissions, steroid use
- Severe bronchospasm → can restrict airflow so much that wheezing disappears
- Anesthetic management: Supplemental O<sub>2</sub>, bronchodilators, anticholinergics
  - Epinephrine may be required to treat severe episodes of bronchospasm
- Avoid ETT use (may precipitate bronchospasm) for noninvasive procedures



Epiglottitis and Croup		
	Epiglottitis	Croup
Etiology	H. flu (bacterial)	Viral
Age	1-8 yr	6 mo-6 yr
Timing	Fast onset	Gradual onset
X-ray findings	Swollen epiglottis	"Steeple" sign (edematous narrowing of subglottic inlet)
Signs & symptoms	High fever, stridor, drooling	Mild fever, "barking" cough, cyanotic
Anesthetic management	Surgery on standby for emergent surgical airway, usually inhalational induction (sitting position) while pt breathing spontaneously; no awake laryngoscopy (risk of laryngospasm)	Cool mist, racemic epi (nebulized), steroids If needed, perform intubation in OR, consider mask induction
Use smaller-than-normal ETT (due to edema)		

### Foreign Body Aspiration

- Airway manipulation (even minor) → can convert partial into complete obstruction
- Supraglottic foreign body: Careful inhalational induction & gentle upper airway endoscopy to remove foreign body
- Subglottic foreign body: RSI or inhalational induction → followed by rigid bronchoscopy or ETT + flex bronch
- Good communication between surgeon & anesthesiologist essential

### Tetralogy of Fallot

Definition	Pulmonary stenosis, VSD, overriding aorta, RVH
TET spell Management	↑ Ventricular outflow tract obstruction & PVR → inc R-to-L shunt through VSD → cyanosis; Hyperventilation to ↓ PVR, phenylephrine to ↑ SVR (> than PVR) → causes L-R shunt; also can give fluids, propranolol
Anesthetic Management	<b>Goals:</b> ↓ R-to-L shunt (avoid ↑ PVR, ↓ SVR, ↑ heart contractility) <b>Techniques:</b> Hydration, continuation of β-blockade, avoidance of crying <b>Induction:</b> Ketamine (maintain SVR) vs inhalational induction (risk of ↑ PVR due to hypoxia & hypercarbia)
SVR/PVR Management	↓ SVR: Potent volatile agent, histamine-releasing drugs, α-blockers ↑ PVR: Acidosis, hypercarbia, hypoxia, PPV/PEEP, N <sub>2</sub> O

### Upper Respiratory Tract Infections (URIs)

- Children have ≈ 6-8 URIs/yr; most caused by rhinovirus
  - Croup, influenza, strep pharyngitis & allergic rhinitis → may mimic URIs
- URIs ↑ airway reactivity for 4-6 wk following onset of symptoms
  - Potential complications from GA → laryngospasm, bronchospasm, & desat
- Risk factors for resp events: Hx of prematurity, coexistent reactive airway dz, 2nd-hand smoke exposure, ETT, nasal congestion/secretions, airway surgery
- **Not** practical to cancel all children with recent URI; reschedule elective surgery if:
  - Purulent nasal discharge, productive cough, fever > 100°F
  - No change in functional status (appetite, activity), likely to tolerate brief proc
- LMA acceptable technique to avoid unnecessary airway manipulation
  - Consider deep extubations (spontaneously breathing under ≥2 MAC of sevo) to minimize airway irritation during emergence

### Secondary Smoke Exposure

- 2nd-hand smoke leads to inc risk of adverse resp events under GA → laryngo-/bronchospasm, breath-holding, airway obstruction, ↑ oral secretions

### Cardiac

#### Patent Foramen Ovale (PFO)

- Intracardiac shunt → permits fetal circulation in utero (interatrial communication)
  - Usually closes during delivery, soon after infant's 1st breath
  - Pulm vascular resistance falls & L atrial pressures exceed R → closes flap
- Conditions which ↑ R-sided atrial pressures may reopen this conduit → hypoxia
- Paradoxical air embolism: Can occur in pts with PFO → if precautions are not taken

### Atrial & Ventricular Septal Defects (ASD/VSD)

- ASD & VSD → result left-to-right shunts, do not present with systemic hypoxemia unless defects large & volume overload severe

- Small defects usually asymptomatic & hemodynamically stable
  - Over time, shunt flow may → lead to R-heart volume overload & CHF
  - Corrective procedures usually timed according to disease severity
- Anesthetic management
  - Avoid hypoxia & hypercarbia (increased pulm vascular resistance)
  - Conditions which ↑ R-sided heart pressure above L-side may provoke shunt reversal & critical hypoxemia

### Neurologic

**Duchenne muscular dystrophy:** assoc with **malignant hyperthermia**, trigger-free anesthetic techniques should be employed

### Metabolic

#### *Mitochondrial Disease (MD)*

- Diverse group of enzyme complex defects that adversely affect energy metabolism
  - Incidence 1:5000 with variable age of onset & presentation
- Abnl ATP production affects brain, heart, & muscle; can lead to:
  - Seizures, spasticity & developmental delay, hypotonia, cardiomyopathy, arrhythmias, chronic GI dysmotility, delayed growth
- No proven assoc between MD & malignant hyperthermia
  - Pts may be sensitive to propofol, but no clear guidelines regarding its use
  - Be aware of potential for metabolic acidosis
- Normal saline generally recommended for maintenance fluids
  - Lactate admin may cause worsening of symptoms
  - Fluid requirements may be elevated
  - Children may also require glucose supplementation & serial monitoring

### Gastrointestinal

#### *Pyloric Stenosis*

- Obstruction of pyloric lumen usually age 5 wk → persistent, bile-free projectile vomiting
- Condition = medical (not surgical) emergency
  - Infant may be severely dehydrated & have concurrent electrolyte abnl
  - Emesis is H<sup>+</sup> ion rich, causes hypokalemic, hypochloremic metabolic alkalosis
  - Must correct before surgical repair
- ↑ Risk for aspiration
  - Need gastric decompression (NG) immediately before induction
  - Rapid-sequence IV induction with succinylcholine or
  - Awake, oral ETT intubation may require help to physically restrain infant
- Procedure = usually brief, long-acting muscle relaxation unnecessary

#### *Tracheoesophageal Fistula (TEF)*

- Most common presentation (85%) = Type C (proximal esoph atresia w/distal fistula)
- Symptoms: Coughing, excessive drooling, & cyanotic episodes
  - Failure to pass soft-tipped suction catheter into stomach = diagnostic
  - Presence of blind esophageal pouch confirmed by x-ray
- Preop assessment: Focused on resp support, aspiration precautions, & identification of other congenital abnl (echo to rule out endocardial cushion defects)
- Anesthesia management: A-line usually placed
  - Position pt on 30° wedge to avoid passive aspiration of gastric fluid
  - Induction techniques: Should minimize aspiration risk (awake intubation, RSI)
  - Avoid positive-pressure ventilation prior to intubation
    - May cause significant gastric distention, diaphragmatic elevation, & hypoxia
    - Prophylactic gastrostomy may prevent accumulation of gastric air
- Deliberate R-sided mainstem intubation → limit transmission of air across fistula
  - Pass ETT distal to fistula, then withdraw until bilateral breath sounds obtained
- Lung isolation indirectly achieved by surgical compression of nondependent lung
  - May be poorly tolerated (V/Q mismatch); consider intermittent lung reinflation
  - Hypotension may occur → mediastinal structures distortion & ↓ venous return
- Extubate stable pts (with good pain control) to avoid pressure on tracheal suture line
  - If pt remains intubated, only suction with a premeasured catheter that does not extend beyond distal tip of ETT

#### *Gastroschisis & Omphalocele*

- Involve defects of anterior abdominal wall with herniation of visceral components

Comparison of Gastroschisis and Omphalocele		
	Gastroschisis	Omphalocele
<b>Etiology</b>	Omphalomesenteric artery occlusion	Failed gut migration from yolk sac to abdomen
<b>Incidence</b>	1:15,000	1:6000
<b>Presentation</b>	Lateral to umbilicus	Midline
<b>Hernial sac</b>	Absent	Present
<b>Bowel function</b>	Abnormal	Normal
<b>Associated anomalies</b>	Prematurity	Beckwith–Wiedemann syndrome; congenital heart disease; bladder extrophy

- Cover exposed viscera to avoid evaporative heat loss & limit infection
  - Large fluid shifts occur; fluids should be aggressively replaced
  - Serial electrolyte & glucose monitoring important (place A-line/CVP)
- Anesthetic technique
  - Awake intubation or RSI; avoid N<sub>2</sub>O
- Defect closure may → ↑ intra-abdominal pressures which may cause →
  - ↑ Peak airway press, ↓ venous return, hypotension, lower extremity ischemia
- Postop: Usually require mech vent support

**Necrotizing Enterocolitis**

- Etiology multifactorial: Pts usually present with bowel distention & bloody feces
  - Preterm infants <2 weeks gestational age → highest risk
- Intestinal hypoperfusion & ischemia → weakened intestinal wall → may perforate
- Anesthesia management: Place A-line & CVP
  - Resuscitation should include crystalloid & blood products
  - Monitor urine output, avoid N<sub>2</sub>O
  - DIC, thrombocytopenia may occur
- Pts often return for reexploration

Syndromes and Their Anesthetic Implications		
Name	Description	Anesthetic Implications
Adrenogenital syndrome	Inability to synthesize hydrocortisone. Virilization of female.	All need hydrocortisone even if not salt-losing. Check electrolytes.
Alpert's syndrome	Craniofacial abnormalities, syndactyly and potential developmental delay.	Possible hydrocephalus and elevated intracranial pressures. Anticipate potential difficult airway.
Ataxia–telangiectasia	Cerebellar ataxia. Skin and conjunctival telangiectasia. Decreased serum IgA and reticuloendothelial malignancy.	Defective immunity; recurrent chest and sinus infections. Bronchiectasis.
Beckwith syndrome (infantile gigantism)	Birth weight >4000 g. Macroglossia and omphalocele.	Persistent severe neonatal hypoglycemia. Airway problems.
Cherubism	Tumorous lesion of mandibles and maxillae with intraoral masses. May cause respiratory distress.	Intubation may be extremely difficult. May require tracheostomy.
Cretinism (congenital hypothyroidism)	Absent thyroid tissue or defective synthesis thyroxine and goiter.	Airway problems; large tongue, goiter. Respiratory center very sensitive to depression. CO <sub>2</sub> retention common. Hypoglycemia, hyponatremia, hypotension. Low cardiac output. Transfusion poorly tolerated.
Cri-du-chat syndrome	Chromosome 5-P abnormal. Abnormal cry, microcephaly. Micrognathia. Congenital heart disease.	Airway problems; stridor, laryngomalacia. Possibly difficult intubation.

**Syndromes and Their Anesthetic Implications (Continued)**

<b>Name</b>	<b>Description</b>	<b>Anesthetic Implications</b>
Down's syndrome (mongolism)	Microcephaly. Small nasopharynx. Hypotonia. 60% have congenital heart disease. Duodenal atresia in some. Cervical spine abnormalities.	Difficulty airway; large tongue, small mouth. Risk of laryngeal spasm, especially on extubation. Problems of cardiac anomalies.
Duchenne muscular dystrophy	Muscular dystrophy with frequent cardiac muscle involvement. Usually die in 2nd decade. Amount of skeletal muscle involvement and cardiac involvement unrelated.	As for myotonia congenital plus cardiac involvement. Minimal drug dosage. Avoid respiratory depressants, muscle relaxants. Postoperative ventilatory support may be required.
Edward's syndrome (trisomy 18E)	Congenital heart disease in 96%. Micrognathia in 80%. Renal malformations 50–80%. Usually die in infancy.	Possible difficult intubation. Care with renally excreted drugs.
Ehlers–Danlos syndrome	Collagen abnormality with hyperelasticity and fragile tissues. Dissecting aneurysm of aorta. Fragility of other blood vessels. Bleeding diathesis?	CVC—spontaneous rupture of vessels. Angiogram 1% mortality. ECG conduction abnormalities. IV difficult to maintain; hematoma. Poor tissues and clotting defects lead to hemorrhage especially GI tract. Spontaneous pneumothorax.
Familial periodic paralysis	Muscle disease. Hypokalemia, attacks of quadriplegia.	Monitor serum K <sup>+</sup> . Limit use of dextrose. Monitor ECG. Avoid relaxants.
Fanconi syndrome (renal tubular acidosis)	Usually 2° to other disease. Proximal tubular defect. Acidosis, K <sup>+</sup> loss. Dehydration.	Impaired renal function. Treat electrolyte and acid–base abnormalities. Look for 1° disease (galactosemia, cystinosis, etc.)
Homocystinuria	Inborn error of metabolism. Thromboembolic phenomena due to intimal thickening. Ectopia lentis. Osteoporosis. Kyphoscoliosis.	Dextran-80 to reduce viscosity and platelet adhesiveness, increase peripheral perfusion. Angiography may precipitate thrombosis, especially cerebral.
Kartagener's syndrome	Dextrocardia, sinusitis and bronchiectasis. Abnormal immunity.	Chronic respiratory intubation.
Klippel–Feil syndrome	Congenital fusion of two or more cervical vertebrae, leading to neck rigidity.	Difficulty airway and intubation.
Median cleft face syndrome	Varying degrees of cleft face. Frontal lipomas. Dermoids.	Cleft nose, lip, and palate may cause intubation difficulties.
Marfan's syndrome (arachnodactyly)	Connective tissue disorder. Dilated aortic root leads to AI. Aortic, thoracic, or abdominal aneurysm. Pulmonary artery, mitral valve involved. Kyphoscoliosis, pectus excavatum, lung cysts. Joint instability and dislocation.	Care with myocardial depressant drugs. Beware possible dissection of aorta. Lung function poor. Possible pneumothorax. Care in positioning; easily dislocated joints.

(continued)

## Syndromes and Their Anesthetic Implications (Continued)

Name	Description	Anesthetic Implications
Myasthenia congenita	Like adult myasthenia gravis.	Avoid respiratory depressants, muscle relaxants. May require postop IPPV. Problems with anticholinesterase therapy pre- and postop if necessary. Halothane may cause postop shivering and myotonia. Pulmonary complications due to poor cough.
McArdle disease	Glycogen storage disease V.	Muscles affected including cardiac muscle: Care with cardiac depressant drugs.
Pierre Robin syndrome	Cleft palate, micrognathia, glossoptosis. Associated congenital heart disease may occur.	Anticipate difficult airway. Micrognathia & glossoptosis may lead to resp distress, requiring trach or tongue suture to relieve post. oropharyngeal obstruction.
Porphyria	Intermittent porphyria = most common autosomal dominant form. Usual before puberty. Abdominal pain, neurological dysfunction, electrolyte imbalances & psychiatric disturbances characterize acute episodes.	Avoid barbiturates (thiopental, methohexital). Ketamine, etomidate & propofol appear safe. Maintenance of anesthesia with narcotics, volatile agents & nondepolarizing muscle relaxants is recommended. Avoid regional anesthesia in presence of existing neurologic deficit.
Prader-Willi syndrome	Neonate—hypotonia, poor feeding, absent reflexes. Second phase—hyperactive, uncontrollable polyphagia, mental retardation.	Obesity of extreme proportions leading to cardiopulmonary failure.
Scleroderma	Diffuse cutaneous stiffening. Plastic surgery required for contractures and constrictions.	Scarring face and mouth; difficult airway and intubation. Chest restriction; poor compliance. Diffuse pulmonary fibrosis, hypoxia. Veins often invisible and impalpable. Cardiac fibrosis or cor pulmonale. History of steroid therapy.
Stevens-Johnson syndrome	Erythema multiforme, urticarial lesions and erosions of mouth, eyes, and genitalia. Possibly hypersensitivity to exogenous agents (e.g., drugs).	Oral lesions; avoid intubation and esophageal stethoscope. Monitoring difficult because of skin lesions but essential. ECG—fibrillation, myocarditis, pericarditis occur. Temperature control—febrile episodes. Intravenous access—essential but avoid cut-down because of infection. Ketamine probably best anesthetic. Pleural blebs and pneumothorax may occur.
Tay-Sachs disease	Gangliosidosis. Blindness and progressive dementia and degeneration of central nervous system.	No described anesthetic hazard. Progressive neurologic loss leads to respiratory complications. Supportive measures only treatment.

**Syndromes and Their Anesthetic Implications (Continued)**

<b>Name</b>	<b>Description</b>	<b>Anesthetic Implications</b>
Treacher Collins syndrome (mandibulofacial dysostosis)	Micrognathia and aplastic zygomatic arches. Microstomia, choanal atresia. Congenital heart disease may occur.	Possible airway and intubation difficulties. Less severe than Pierre Robin deformity.
von-Hippel-Lindau syndrome	Retinal or CNS hemangioblastoma. (Posterior fossa or spinal cord.) Associated with pheochromocytoma & renal, pancreatic, or hepatic cysts.	Problems due to associated pheochromocytoma, renal and hepatic pathology.
Von Recklinghausen disease (neurofibromatosis)	Café-au-lait spots. Tumors in all parts of CNS. Peripheral tumors associated with nerve trunks. Increased incidence pheochromocytoma. Honeycomb cystic lung changes. Renal artery dysplasia and hypertension.	Screen for pheochromocytoma (urinary VMA). Should be investigated for lung function. Tumors may occur in the larynx and right ventricular outflow tract. Care with renally excreted drugs if kidneys involved.
Wilson's disease (hepatolenticular degeneration)	Decreased ceruloplasmin causes abnormal copper deposits, especially in liver and CNS motor nuclei. Renal tubular acidosis.	Hepatic failure 2° to fibrosis. IV induction (propofol, ketamine, etomidate) acceptable. Apnea uncommon with succinylcholine administration, despite pseudocholinesterase deficiency. Consider reduced dosing of renally-excreted drugs.
Wolff-Parkinson-White syndrome	ECG abnormality—short PR, prolonged QRS with phasic variation in 40%. Associated with many cardiac defects. Anomalous conduction path between atria and ventricles. Delta wave may be present on ECG.	Scopolamine preferred to atropine as drying agent. Tachycardia due to atropine or apprehension may change ECG and suggest infarction, with ST-segment depression. Paroxysmal SVT on induction of anesthesia or during cardiac surgery has been reported. Should be treated with digitalis, propranolol, pacemaker if necessary. Neostigmine may accentuate W-P-W pattern.

Source: Adapted from Pajewski TN. *Anesthesiology Pocket Guide*. Philadelphia: Lippincott-Raven, 1997.

# AMBULATORY ANESTHESIA

URSULA GALWAY

AMBULATORY 27-1

## Patient/Procedure Selection

- ASA status: ASA 1 & 2 preferred
  - ASA 3 & 4 only if disease is compensated/stable
- Avoid pts: Unevaluated medical problems/acute illness
  - Disease that will require hospitalization after surgery
- Procedures: Must have low postop complication rates
  - Immediate postop surgical care manageable at home
  - Avoid high anticipated blood loss/major invasive cases
- Preop eval: Consider preadmission testing/telephone interview
  - Remind NPO guidelines, lab testing as needed

## Postop Nausea & Vomiting (PONV)

- Incidence: 20–30% of general surgical pts, 70–80% of high-risk pts
- Impact: Accounts for 0.1–0.2% of unanticipated hospital admissions
  - ↑ PACU length of stay & costs, ↓ pt satisfaction

Risk Factors for PONV		
Patient Factors	Anesthetic Factors	Surgical Factors
Female	Volatile anesthetics	Surgical Procedure
Nonsmokers	Nitrous oxide	(ophthalmologic, gynecologic, breast, abdominal, laparoscopy)
History of PONV or motion sickness	(controversial) Opioids	Surgical duration: Each additional 30 min raises PONV risk by 60%

Source: Adapted from Gan et al. SAMBA guidelines for the management of PONV. *Anesth Analg* 2007;105:1615–1628.

- Reduction of risk (*Anesth Analg* 2007;105:1615–1628)
  - Regional techniques (general anesthesia has 11-fold ↑ risk of PONV)
  - Propofol (19% ↓ PONV risk compared to inhalational agents)
  - Avoid nitrous oxide (12% ↓ PONV risk), inhaled anesthetics, neostigmine
  - Minimize opioids
  - Ensure adequate hydration

Simplified Risk Score for PONV		Prediction and Prophylaxis of PONV		
Risk Factors	Points	No. of Points	% Risk of PONV	Prophylaxis
Female	1	0	10% (low risk)	
History of PONV/ motion sickness	1	1	21% (low risk)	No prophylaxis
Nonsmoker	1	2	39% (mod risk)	Monotherapy
Perioperative opioids	1	3	61% (high risk)	Multimodal therapy/consider TIVA
		4	79% (high risk)	

Source: Adapted from Apfel et al. A simplified risk score for predicting PONV: conclusions from cross-validations between two centers. *Anesthesiology* 1999;91:693–700.

Antiemetic Drugs				
Class	Drug	IV Dose	Timing	Side Effects
5HT <sub>3</sub> Antagonists	Ondansetron	4 mg	End of surgery	Headaches
	Granisetron	0.35–1.5 mg		
	Dolasetron	12.5 mg		
	Tropisetron	2 mg		
Steroid	Dexamethasone	4–6 mg	At induction	
Butyrophenones	Droperidol	0.625–1.25 mg	End of surgery	QT prolongation, drowsiness, extrapyramidal effects
	Halopridol	0.5–2 mg		
Phenothiazines	Promethazine	6.25–25 mg	End of surgery	Drowsiness, agitation, extrapyramidal effects
	Prochlorperazine			

(continued)

**Antiemetic Drugs (Continued)**

Class	Drug	IV Dose	Timing	Side Effects
Antihistamine	Dimenhydrinate	1 mg/kg		Sedation
Other	Propofol	20 mg	In PACU	Resp depression
	Scopolamine	Trans patch	4 hr before end of surgery	Sedation, confusion dry mouth
	Ephedrine	0.5 mg/kg IM	End of surgery	
	Metoclopramide	10–20 mg	1 hr before end of surgery	Extrapyramidal effects

- PONV rescue strategy
  - If initial agent is ineffective → give drug from a different class
  - Repeat droperidol and 5-HT3 antagonists q6h
  - Repeat administration of dexamethasone not recommended
- Discharge criteria
  - Evaluation can be based on formal scoring system (also see Aldrete score in Chapter 13) or RN/MD assessment
  - Oral intake: Not required prior to discharge
  - Voiding: Usually required prior to discharge if pt received neuraxial anesthesia, gynecologic, hernial, anorectal, or genital surgery
  - Spinal/epidural anesthesia
    - Pt must have return of sensation and no motor block
  - Upper extremity nerve blocks
    - Discharged can occur before full return of motor/sensory function
    - Patient must be given instructions to protect numb limb from injury

**Modified Postanesthetic Discharge Scoring System (Score ≥9 for discharge)**

<b>VITAL SIGNS</b>	
Within 20% of preop value	2
20–40% of preop value	1
40% of preop value	0
<b>AMBULATION</b>	
Steady gait/no dizziness	2
With assistance	1
None/dizziness	0
<b>NAUSEA/VOMITING</b>	
Minimal	2
Moderate	1
Severe	0
<b>PAIN</b>	
Minimal	2
Moderate	1
Severe	0
<b>SURGICAL BLEEDING</b>	
Minimal	2
Moderate	1
Severe	0

Source: Adapted From Twersky RS. *Ambulatory Anesthesia Handbook*, 1st ed. St. Louis: Mosby-Year Book, 1995.



Multimodal Pain Management Strategy

**Step 3: Severe Postop Pain**

Step 1 and 2 strategies

**AND**

Local anesthetic peripheral neural blockade  
(with or without catheter)

**AND**

Use of sustained-release opioid analgesics

**Step 2: Moderate Postop Pain**

Step 1 strategy

**AND**

Intermittent doses of opioid analgesics

**Step 1: Mild Postop Pain**

Nonopioid analgesic

Acetaminophen, NSAIDs, COX-2 selective inhibitors

**AND**

Local anesthetic infiltration

Source: Adapted from Crews JC. Multimodal pain management strategies for office-based and ambulatory procedures. *JAMA* 2002;282:629-632.

**Summary of Options for Postop Pain Management**

Route	Therapy Class	Medication/Dose
INTRAVENOUS/ INTRAMUSCULAR	OPIOIDS	Fentanyl 25-100 mcg/kg q30-60 min Hydromorphone 0.2-2 mg q4-6h Meperidine 25-50 mg q3-4h Morphine 1-10 mg q2-6h
	NSAIDs MIXED AGONISTS/ ANTAGONISTS	Ketorolac 30 mg Butorphanol 20 mcg/kg Nalbuphine 0.25 mcg/kg
ORAL	NSAIDs	Ibuprofen 400-800 mg q4-6h Ketorolac 10-20 mg q4-6h Naproxen 500 mg q6-8h
	COX-2 INHIBITORS	Celecoxib 200-400 mg q12h
	OPIOID/ NONOPIOID COMBINATIONS	Acetaminophen/propoxyphene napsylate (Darvocet) q4-6h Acetaminophen/oxycodone (Percocet) q4-6h Acetaminophen/codeine (Tylenol with codeine) q4-6h Acetaminophen/hydrocodone (Vicodin) q4-6h
	OPIOIDS	Hydrocodone 5-10 mg q4-6h Morphine 10-30 mg q3-4h Oxycodone 5 mg q3-6h
TRANSDERMAL	OTHERS OPIOIDS	Acetaminophen 325-1000 mg q4-6h Fentanyl patch 25-100 mcg/h q72h
INTRANASAL	OPIOIDS	Fentanyl Meperidine Butorphanol
LOCAL ANESTHETICS		Neuraxial anesthesia Regional nerve block Local infiltration by surgeon Continuous subcutaneous catheter
NON PHARMOLOGICAL METHODS		Heat/cold therapy, massage, TENS relaxation, hypnosis, acupuncture, biofeedback

# ANESTHESIA FOR AESTHETIC SURGERY & SURGERY OUTSIDE OF THE OPERATING ROOM

RUCHIR GUPTA • PADMA SURAMPUDI

OUTSIDE OR 28-1

## Anesthesia for Aesthetic Surgery

### Liposuction

- Most commonly performed plastic surgery procedure
- Performed by inserting hollow rods into skin & suctioning subcutaneous fat
- Tumescent liposuction most common form of liposuction
- Wet technique: Inject less fluid than amount of fat to be removed

### Anesthetic Techniques

- Pain can be controlled with the infused lidocaine + IV/oral opioids
- Low-volume procedures (wet technique) → consider deep sedation/GA/regional
- Large-volume procedures (tumescent liposuction) → consider local or sedation
- Limit IV fluids if large volume of solution is being infused

### Complications

- Lidocaine toxicity (can occur postprocedure 2° to slow systemic absorption)
- Volume overload/CHF
- Hypovolemia; bleeding; pulmonary embolism; hypothermia

## Practice Advisory on Liposuction

### Anesthetic Infiltration Solutions

1. For small-volume liposuction, wetting solutions with local anesthetics may provide sufficient pain relief without additional anesthesia; pts or providers, however, may prefer sedation/general anesthesia even with small-volume liposuction
2. Avoid bupivacaine (Marcaine) as additive to infiltrate soln (severe side effects, slow elimination, & lack of toxicity reversal)
3. Lidocaine given in large volumes may cause systemic toxicity. Preventive measures:
  - Limit lidocaine dose to 35 mg/kg (this level may not be safe in pts with low protein levels/conditions where lidocaine by-products accumulate)
  - Calculate dose for total body weight & ↓ lidocaine conc if necessary
  - Utilize superwet rather than tumescent technique
  - Avoid lidocaine when utilizing general/regional anesthesia
4. Avoid epinephrine in pts with pheochromocytoma, hyperthyroidism, severe HTN, cardiac dz, or peripheral vas dz
5. Consider staged infiltration of various sites to ↓ effects of excess epinephrine

Source: Adapted from Iverson RE. *Plast Reconstr Surg* 2004;113(5):1478-1490.

### Blepharoplasty

- Technique: Usually local anesthesia + MAC
- After sedation, local anesthesia is injected into eyelid skin, rendering area numb
- Avoid bucking & coughing during emergence (may ↑ risk of bleeding)
- Consider propofol/remifentanyl infusion for wake-up

### Breast Augmentation

- Technique: Usually GA (separation of pectoralis muscle may require paralysis); MAC with local anesthesia may be used; consider antiemetic prophylaxis

### Breast Reduction

- Technique: Usually GA; paravertebral block/epidural may be used

### Rhytidectomy (Facelift)

- Technique: Usually GA or MAC
- Beware of airway fire when performing MAC + supplemental O<sub>2</sub> (proximity of Bovie & O<sub>2</sub>)
- Avoid bucking and coughing during emergence (may increase risk of bleeding)

## Anesthesia Outside the Operating Room

### General Considerations/Safety

- Thorough preoperative eval of every pt is essential
- All patients receiving any form of anesthesia **must** have ASA monitors during anesthesia care

- Transport equipment should be available (bag-valve-mask with O<sub>2</sub> tank)
- Emergency drugs should be available & IV access assessed
- Postop care & standards are same as for OR based anesthesia
- Pay attention to possible allergies to contrast dye

### ASA Guidelines for Non-OR Anesthetizing Locations

1. Reliable O<sub>2</sub> source, including backup supply
2. Adequate & reliable suction
3. Adequate & reliable scavenging system if anesthetic gases are to be used
4. Self-inflating resuscitator bag capable of delivering 0.9 FiO<sub>2</sub>; adequate drugs, supplies, & equipment for the planned activity
5. Adequate monitoring equipment to adhere to ASA Monitoring Standards
6. Sufficient electrical outlets
7. Sufficient site for equipment
8. Immediate availability of emergency cart with defibrillator & emergency drugs
9. Reliable 2-way communication
10. Observation of all applicable building & safety codes and facility standards

Source: Adapted from ASA Standards for Basic Intraoperative Monitoring.

### CT/MRI/Interventional Neuroradiology

CT: General Considerations

- Pt should wear lead with thyroid shield at all times while in CT scanner

CT: Monitors

- Regular OR anesthesia monitors may be used
  - Standard ASA monitors required if any anesthesia given

CT: Anesthetic Considerations

- Anesthetic options range from mild sedation to general anesthesia
- Patient factors to consider: Pt cooperation, claustrophobia, comorbidities, age, mental status, length of scan
- Ensure adequate length of IV lines, anesthesia circuit, monitoring wires

Special Procedures in the CT Suite

*Stereotactic Brain biopsy*

- Metal frame placed to perform procedure (usu. with local + benzodiazepine)
- Technique: MAC, titrate sedation carefully to avoid airway compromise if GA necessary, awake fiberoptic intubation may be safest technique

*Percutaneous Vertebroplasty*

- Indication: Reverse vertebral collapse in osteoporotic patients
- Technique: Usually MAC (or GA if pt in excessive pain)
- Patient is in prone position → consider pelvic/chest support to avoid impinging on abdomen and interfering with ventilation

MRI: General Considerations

- Indications for anesthesia care: Children, mentally challenged, claustrophobic pts, pts with resp difficulty, hemodynamically unstable pts, chronic pain pts
- Distinct features of anesthesia in MRI:
  - Powerful magnet
    - Remove ferromagnetic equipment: Stethoscopes, credit cards, USB drives, pens, keys, IDs, beepers, cell phones
    - Metals safe: Beryllium, nickel, stainless steel, tantalum, & titanium
  - Difficulty accessing airway
    - Carefully titrate sedatives & have monitors facing clinician at all times

MRI: Monitors

- Nonferrous monitoring equipment needed
- Nonmagnetic laryngoscopes for emergencies
- Ensure adequate length of IV lines, anesthesia circuit, monitoring wires

Interventional Neuroradiology

*General Considerations*

- Standard ASA monitors; if arterial line necessary, can be radial or through femoral sheath
  - Femoral sheath a-lines → only MAP is useful
- Technique: GA if motionless state required; sedation if rapid neurologic testing essential or for most diagnostic scans

*Deliberate Hypertension*

- May be necessary to help radiology catheters flow to desired location
- Usually 20–40% above baseline; phenylephrine infusion may be useful

*Deliberate Hypotension*

- May be required in carotid endarterectomy/arterio-venous malformation procedures
- Various approaches may be used (↑ anesthesia, labetalol, vasodilators—nitroprusside/nitroglycerin/hydralazine)

*Embolization of Arteriovenous Malformation (AVM)*

- Polyvinyl alcohol (PVA) injected into feeding vessels of AVM
- Approach: MAC (can continuously monitor neuro status) or GA
- Systemic heparinization may be required
- Complications: Hemorrhage 2° to anticoagulation (can reverse with protamine) hemorrhage 2° to thrombus (can ↑ BP by 20–40 mm Hg); ↑ ICP (treat with hyperventilation, head ↑, mannitol, furosemide)

*Cerebral Aneurysms*

- Uses balloons, coils, or liquid polymer solution to endovascularly treat the aneurysm
- Usually performed under general anesthesia, a-line should be placed
- Important to have OR available in case of rupture & urgent need for surgical repair

*Central Intraarterial Thrombolysis*

- Treatment of stroke if <6 hr from onset of symptoms
- Usually performed under MAC (neurologic assessment is desirable)

*Endoscopy & ERCP (Endoscopic Retrograde Cholangiopancreatography)**General Considerations*

- Most upper and lower endoscopies are performed without an anesthesiologist
- Lateral position for lower endoscopy; lateral/supine for upper endoscopy
- Important to have access to airway at all times

*Technique*

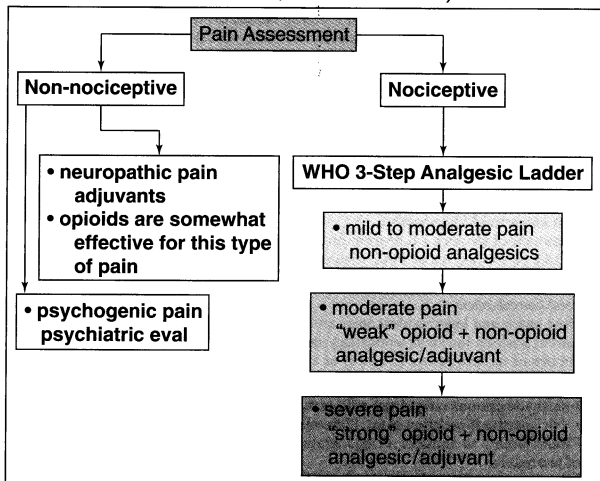
- Anesthetic options range from mild sedation to general anesthesia
  - For anesthesia sedation cases: Midazolam/fentanyl/propofol combination often used
- Patient factors to consider: Pt cooperation, comorbidities, age, mental status, length of procedure
- **Upper endoscopy:** Consider pharyngeal topical anesthesia (lidocaine, benzocaine) prior to endoscope insertion
- Postop pain: Relatively low, usually from air used for inflation
- **ERCP:** May be performed in supine, lateral or prone position; pt can have significant pain during bile duct dilatation

# MANAGEMENT OF CHRONIC PAIN

NALINI VADIVELU • CHRISTIAN WHITNEY

**Chronic pain** = persistence of pain >3 months, without any biological value

**Figure 29-1** Algorithm for pharmacologic chronic pain management. (Adapted from The Texas Cancer Pain Initiative, Texas Cancer Council.)



Nociceptive pain = tissue pain (somatic or visceral); non-nociceptive pain = neuropathic pain

## Low Back Pain (LBP)

- Incidence: 15% of population; most common cause of job-related disability cost = \$100 billion/yr; risks related to physical aspects of work

Differential Diagnosis of Low Back Pain	
Mechanical Causes	Nonmechanical Causes
Sprain Spondylosis of annulus, facet, or disc Compression fx, spinal stenosis Alignment disorders (scoliosis, kyphosis, spondylolisthesis)	Infection Cancer
Causes of Referred Pain	Inflammatory Spondyloarthropathic Causes
Gastrointestinal disease (pancreatitis, cholecystitis) Renal disease (kidney stones, pyelonephritis) Pelvic disease (prostatitis, endometriosis)	Ankylosing spondylitis Inflammatory bowel disease Reiter's syndrome Psoriatic spondylitis Paget's disease Osteochondrosis

## Workup of Low Back Pain

- AP & lateral lumbar spine x-rays  
(diagnose lumbosacral radiculopathy/arthropathies/herniated discs)
- Neurologic exam
  - Peripheral motor function, sensory system, deep tendon reflexes
- Physical exam/range of motion
  - Straight leg raise test (supine or sitting position)
    - Elevation of leg to <60° is abnormal
    - Radiation of pain below knee (not just to hamstrings) = positive
- Rectal, abd, & pelvic exams necessary to rule out pathology in prostate, bladder, abd, & pelvis

**Disc Herniation—Neurologic Exam**

- In most cases numbness corresponds to level of disc involved, with motor weakness & reflex loss
- 95% of all disc herniations occur at L5 & S1

<b>Cervical and Lumbar Disk Herniation Patterns</b>					
<b>Disc</b>	<b>Root</b>	<b>Pain/ Paresthesias</b>	<b>Sensory Loss</b>	<b>Motor loss</b>	<b>Reflex loss</b>
C4–C5	C5	Neck, shoulder upper arm	Shoulder	Deltoid, biceps, infraspinatus	Biceps
C5–C6	C6	Neck, shoulder, lat. arm, radial forearm, thumb & index finger	Lat. arm, radial forearm, thumb & index finger	Biceps, brachioradialis	Biceps, brachioradialis, supinator
C6–C7	C7	Neck, lat. arm, ring & index fingers	Radial forearm, index & middle fingers	Triceps, extensor carpi ulnaris	Triceps, supinator
C7–T1	C8	Ulnar forearm and hand	Ulnar half of ring finger, little finger	Intrinsic hand muscles, wrist extensors, flexor dig profundus	Finger flexion
L3–L4	L4	Anterior thigh, inner shin	Anteromedial thigh and shin, inner foot	Quadriceps	Patella
L4–L5	L5	Lat. thigh and calf, dorsum of foot, great toe	Lat. calf and great toe	Extensor hallucis longus, ± foot dorsiflexion, invers. & evers.	None
L5–S1	S1	Back of thigh, lateral posterior calf, lat. foot	Posterolat. calf, lat. and sole of foot, smaller toes	Gastrocnemius ± foot eversion	Achilles

Note: lumbar disk protrusion tends to compress the root corresponding to the level of the vertebra below it.

**Spinal Stenosis—Neurologic Exam**

- Pain along lateral aspect of leg while walking (pseudoclaudication)
- Straight leg raising test usually negative
- Sacroiliitis presents with pain over involved joints

**Treatment of Low Back Pain**

*Pharmacologic Treatments*

- NSAIDs, acetaminophen, tramadol, antidepressants, muscle relaxants, anticonvulsants, opioids
- Encourage pts to be active: Physical therapy; stretching exercises, massage, ice, heat, electrical muscle stimulation, work-hardening programs, exercise rehabilitation
- Consider treatment of associated depression/anxiety

*Interventional Procedures*

- Diagnostic nerve & facet blocks
- Selective joint injections
- Spinal endoscopy
- Nerve ablations & selective rhizotomies
- Intradiscal distraction therapies
- Epidural steroid injections
  - Commonly done to treat radicular low back pain
  - Common agents: Betamethasone (Celestone) 12–18 mg  
Methylprednisolone (Depomedrol) 80–120 mg  
Triamcinolone (Kenalog) 75 mg
  - 1–2% lidocaine or normal saline can be added to injectate
  - Up to 3–4 injections may be performed if clinically indicated in 12-mo period (at least 2-wk interval between injections)

**Osteoarthritis (Joint Disease)**

*Pathophysiology*

- Predominately affects long bone joints, also affects other synovial joints
- Most common form of joint dz, incidence ↑ with age

- Major cause of work-related disability in men aged >50 yr
- Microfractures, bursitis, osteophytes, ↑ capsular pressure → pain

#### Diagnosis

- Detailed history & physical
- Radiologic evidence of loss of joint space & sclerosis

#### Treatment

- Nonpharmacologic therapies
  - Appropriate footwear, range-of-motion exercises, muscle strengthening, physical & occupational therapy
- Pharmacologic therapies
  - Acetaminophen, opioids, Tramadol, topical capsaicin, NSAIDs

### Sickle Cell Disease

#### Pathophysiology

- Hemoglobin synthesis disorder, most common in African Americans
- Pain = most common symptom & can be present throughout life
- Infection in kids, with stroke & trauma in adults → most common causes of death
- Dehydration, fatigue, cold weather → can precipitate pain

#### Diagnosis

- Detailed history & presence of pain
- Hemoglobin electrophoresis, genetic testing

#### Treatment

- Analgesics, nonopioids, & opioids

### Angina Pectoris & Myocardial Infarction

#### Pathophysiology

- Pain associated with release of lactate,  $K^+$ , prostaglandins, bradykinin, & adenosine from damaged tissue (in presence of ischemia)
- Substances sensitize heart sensory nociceptors → cause pain

#### Diagnosis

- Physical signs: Chest pain tightening, radiating shoulder pain, presence of nausea, cold & clammy hands
- Tests: ECG, myocardial enzyme testing, echo, & stress testing

#### Treatment

- Nonpharmacologic therapy
  - Exercise, weight reduction, HTN control, smoking cessation
- Pharmacotherapy
  - Nitrates,  $\beta$ -blockers,  $Ca^{2+}$  blockers, blood thinners, CABG
  - Spinal cord stimulation if above measures fail

### PERIPHERAL NEUROPATHIC PAIN

- Causes include diabetes, cancer, herpes zoster, infections, trauma, autoimmune dz
- Injury to peripheral nerves often assoc with autonomic nerve disorders

#### Diabetic Neuropathy

##### Pathophysiology

- Associated with sensory, motor & autonomic symptoms
- Neuropathic pain syndrome → involves abnl nerve activity & destruction
- Painful diabetic neuropathy occurs in 20–24% of U.S. diabetic pts

##### Symptoms

- *Sensory*: Range from painful sensations (burning, pricking) to nonpainful symptoms (numbness, tingling)
- *Motor*: Distal impaired fine coordination & proximal weakness (difficulty climbing stairs)
- *Autonomic*: Lack of sweating, diarrhea, urinary retention, impotence

##### Treatment

- Preventive & symptomatic measures
- Common drugs: Tricyclics (amitriptyline), anticonvulsants (pregabalin & 5-hydroxytryptiline), norepinephrine uptake inhibitors (duloxetine)

#### Postherpetic Neuralgia (PHN)

- Pain > 3 months after healing of acute rash of herpes zoster
- Acute treatment (herpes zoster)—antiviral drugs
- PHN—treat with TCAs, gabapentinoids, 5% lidocaine patches, opioids

### Complex Regional Pain Syndrome (CRPS)

- Painful condition can develop after distal limb injury
- Features include: Pain, autonomic dysfx, dystrophic changes
  - Pain after *noxious event* → CRPS I (RSD)
  - Pain after *nerve injury* → CRPS II (causalgia)

#### Treatment

- Meds: Gabapentinoids, antidepressants, opioids,  $\alpha$ -adrenergic agonists, mexiletine, GABA agonists, topical clonidine, & transdermal lidocaine
- Physical therapy (stress loading, range of motion, isometric strengthening, & functional rehab)
- Procedures: Sympathetic blocks, dorsal column stimulators, implantable intrathecal pumps for neuraxial analgesia

Definition of Pain Terms	
Allodynia	Painful response to nonpainful stimulus
Dysesthesia	Unpleasant abnormal sensation (spontaneous or evoked)
Hyperalgesia	↑ Sensitivity to stimulation
Hyperpathia	Painful syndrome with abnormally painful response to stimulus
Hypoalgesia	↓ Pain in response to normally painful stimulus
Hypoesthesia	↓ Sensitivity to stimulation
Paresthesia	Abnormal sensation (spontaneous or evoked)

### Occipital Neuralgia

- Definition: Radiating pain spasm syndrome in suboccipital area
- Symptoms: Headache radiating to back, sides, & front of head, pain & eye pressure
- Etiology: Nerve entrapment (from trauma, nerve lesions, localized inflammation, infection)
- Incidence: More common in women
- Treatment: Oral medications, local injections, massage, heat, physical therapy

### Headache

#### Primary Headache Syndromes

- Tension: Associated with muscle contraction in neck or lower head
- Migraine: See below
- Cluster: Periodic, paroxysmal, brief, sharp, orbital headache that may awaken from sleep ± lacrimation, rhinorrhea, conjunctival injection, or unilateral Horner's synd.

#### Secondary Causes of Headaches

- Vascular: Stroke, intracerebral hemorrhage, SAH, subdural hematoma, AVM, unruptured aneurysm, arterial hypertension, venous thrombosis
- Infection: Meningitis, encephalitis, abscess
- Brain tumor
- CSF disorder: ↑ (hydrocephalus) or ↓ (s/p LP)
- Trigeminal neuralgia
- Extracranial: Sinusitis, TMJ syndrome, temporal arteritis

#### Clinical Evaluation (JAMA 2006;296:1274)

- History: Quality, severity, location, duration, time of onset, precipitants/relieving factors
- Associated symptoms (visual  $\Delta$ s, nausea, vomiting, photophobia)
- Focal neurologic symptoms
- Head or neck trauma, constitutional symptoms
- Medications, substance abuse
- General and neurologic examination
  - *Warning signs that should prompt neuroimaging:*
    - Worst ever, worsening over days, wakes from sleep
    - Vomiting, aggravated by exertion or Valsalva
    - Fever, abnl neurologic exam, aura, cluster-type headache



**Headache (Continued)****Migraine****Epidemiology**

- Affects 15% of women and 6% of men; onset usually by 30 y/o.

**Clinical Manifestations** (*Lancet* 2004;363:381; *JAMA* 2006;296:1274)

- Unilateral or bilateral, retro-orbital, throbbing or pulsatile headache; lasts 4–72 h
- Often accompanied by nausea, vomiting, photophobia
- “**POUNding**”: Pulsatile; duration 4–72 h; Unilateral; Nausea & vomiting; Disabling  
Likelihood ratio (LR) 3.5 if 3 criteria are met, LR 24 if  $\geq 4$  criteria are met
- Classic (18%) = visual aura (scotomata with jagged or colored edge) precede headache
- Common (64%) = headache without aura
- Complicated = accompanied by stereotypical neurologic deficit that may last hrs
- Precipitants: Stress, hunger, foods (cheese, chocolate) and food additives (MSG), fatigue, alcohol, menstruation, exercise

**Treatment** (*NEJM* 2002;346:257)

- Eliminate precipitants
- Prophylaxis: TCA,  $\beta$ -blockers, CCB, valproic acid, topiramate (*JAMA* 2004;291:965)
- Abortive therapy:  
ASA, acetaminophen, caffeine, high-dose NSAIDs  
Metoclopramide IV, prochlorperazine IM or IV  
5-HT<sub>1</sub> agonists (“triptans”); contraindic. in pts w/ complicated migraine, CAD, prior stroke  
Combination of triptan + NSAID more efficacious than either single agent alone (*JAMA* 2007;297:1443)  
Ergotamine, dihydroergotamine; use with caution in pts with CAD

**Myofascial Pain Syndrome (MPS)**

- Common cause of musculoskeletal pain
- Condition where characteristic myofascial trigger points are tender
  - Pressing on points refers pain to other areas
  - Trigger points usually at muscle junction with fascia or within a muscle
- Exam: Trigger points appear as taut bands of muscle tissue on palpation
- Treatment: Massage, vapocoolant spray, muscle stretch, trigger-point injection, improved nutrition, exercise, biofeedback, low-dose TCAs

**Trigger-Point Injection—Local Anesthetic or Steroids**

- Raise small skin wheal with 25-ga needle & local anesthetic
- Inject 1–3 mL local (0.25–0.5% bupi or 2% lido) with 22 ga needle into trigger point (after negative aspiration)
- For corticosteroid injection, use larger needle (18–21 ga)
  - Triamcinolone 40 mg/mL
  - Methylprednisolone 40 mg/mL
  - Dexamethasone 4 mg/mL
- If 3 or more trigger point injections at same site fail (spaced 2 wk apart)  
Consider injection of botulinum toxin A (Botox)

**Fibromyalgia**

- Rheumatologic pain disorder
- Comprises widespread musculoskeletal pain, sleep disorders, fatigue, depression, autonomic dysfx
- Women more affected than men, etiology unknown
- Diagnosis: Must rule out conditions causing similar symptoms  
(*rheumatoid arthritis & lupus*)  
11 or more of 18 points located from neck to knees must be painful to touch

**Treatment**

- Psychological & social support
- Meds: Pregabalin 75 mg PO bid initial dose → up to max of 450 mg PO bid antidepressants, opioids

**Cancer Pain**

- Somatic pain, neuropathic pain, cancer related bone pain & visceral pain
- Multiple etiologies: Plexopathies, bone/brain metastasis, procedure related pain (post-mastectomy pain syndrome, phantom pain, radiation-induced neuritis, polyneuropathy from chemotherapy)

Treatment

- Treatment should be symptomatic & combined with oncologic treatments
- Follow the 3-step WHO analgesic ladder (see below)

### 3-Step WHO Analgesic Ladder

#### Step 3: Severe Postop Pain

Step 1 and 2 strategies

**AND**

Local anesthetic peripheral neural blockade  
(with or without catheter)

**AND**

Use of sustained-release opioid analgesics

#### Step 2: Moderate Postop Pain

Step 1 strategy

**AND**

Intermittent doses of opioid analgesics

#### Step 1: Mild Postop Pain

Nonopioid analgesic

Acetaminophen, NSAIDs, COX-2 selective inhibitors

**AND**

Local anesthetic infiltration

Source: Adapted from Crews JC. Multimodal pain management strategies for office-based & ambulatory procedures. *JAMA* 2002;282:629-632.

### TREATMENT OF CHRONIC PAIN

- Chronic pain best treated with multidisciplinary approach
  - Pain is not an isolated symptom
  - Complementary nonpharmacologic approaches include:
    - Exercise programs, treatment of depression, progressive muscular relaxation, acupuncture, biofeedback, guided imagery, & hypnosis
- **Oral/IV medications**
  - Opioids, NSAIDs, antidepressants, anticonvulsants,  $\alpha$ -2 agonists, gabapentinoids, corticosteroids
- **Epidural steroid injections (ESIs)**
  - For treatment of LBP & spinal stenosis
  - Intralaminar, transforaminal or caudal
  - Intra-articular steroid injections (triamcinolone, betamethasone, and methylprednisolone)
- **Transcutaneous electric nerve stimulation (TENS)**
  - Low-voltage electric stimulation applied to skin
- **Peripheral nerve blocks**
  - For diagnosis or treatment
  - Occipital/trigeminal/facial/lateral femoral cutaneous nerve blocks
  - Brachial plexus block, intercostal block, paravertebral block
  - Ilioinguinal/genitofemoral nerve blocks, lumbar sympathetic block
- **Stellate ganglion block**
  - *Definition:* Cervical thoracic sympathetic ganglion block of inferior cervical & 1st thoracic ganglion
  - *Indications:* CRPS (I & II), phantom pain, refractory angina, vascular insufficiency (Raynaud's syndrome), frostbite, scleroderma
  - *Side effects:* Horner's syndrome, warm extremity

#### Stellate Ganglion Block—Technique

- Point of entry: Between trachea & carotid sheath at level of cricoid cartilage & Chassignac's tubercle (C6)  
*(stellate ganglion lies at C7, but enter at C6 to prevent damage to pleura)*
- After contacting C6, withdraw needle 1-2 mm *(gets needle out of longus colli muscle)*
- Ensure neg. aspiration, then give **1st test dose** (0.5 mL of 0.25% bupi or 2% lido)
- **2nd test dose** should be 3 mL of local with 1:200,000 *(to rule out IV placement)*
- Inject 12-15 mL 0.25% bupi in 3-mL aliquots with intermittent aspiration
- Place pt into sitting position *(facilitates spread onto stellate ganglion)* for 10 min
- Pt should be observed for 1 hr after the block

**Stellate Ganglion Block—Complications**

- Infection
- Vascular injury (*carotid artery/internal jugular vein*)
- Neural injury (*vagus, brachial plexus*)
- Pneumothorax
- Hemothorax
- Intravascular injection (*vertebral artery*)
- Epidural/intrathecal block
- Brachial plexus anesthesia
- Recurrent laryngeal n. block (*with resultant hoarseness*)
- Phrenic n. paralysis (*with elevated hemidiaphragm*)
- Chylothorax

**Provocative Discography**

- Performed to determine if back pain is discogenic (arising from disc)
- Involves injection of contrast media into disc in question (pain generator)
  - Test = positive when concordant pain is produced
- Treatment of discogenic pain
  - Initial therapy = dynamic lumbar stabilization exercises
  - If pain persists → percutaneous intradiscal RF ablation or spinal fusion

**Spinal cord stimulation (SCS)**

- Indications: CRPS, periph vascul dz, failed back surgery syndrome, & angina (*occasionally phantom pain, postherpetic neuralgia, spinal cord injury*)
- Mechanism: Rectangular pulses delivered to epidural space via implanted electrodes (connected to pulse generator)
- Efficacy: 50% pain relief in about 50% of people

**Intrathecal pumps:** Deliver agents into intrathecal space

opioids (morphine), local anesthetics, clonidine, baclofen, ziconotide

**Intraspinal tunneled catheter** e.g., provides neuraxial analgesia

(intrathecal/epidural) frequently done for the treatment of advanced cancer pain

**Neurolytic procedures**

- Performed to relieve visceral pain in cancer pts with short life expectancy
- Blocks include: Celiac plexus block, superior hypogastric plexus blocks & neurolysis of ganglion of impar (sympathetic ganglion in sacrococcygeal junction)
- Techniques: Injections of alcohol or phenol; cryotherapy; RF lesioning

**Technique of Celiac Plexus Block**

- Performed at L1; pt usually in prone position
- 22-ga Quincke spinal or Chiba needle placed 5–7 cm lateral to midline → insert to depth of 7–9 cm
- Inject contrast dye to ensure correct placement
- Inject 15–20 mL of 0.5% bupiv, or 10–15 mL of 2% lido, or 10–15 mL of 0.5% ropiv
- Inject neurolytic agent (10–15 mL of 50% alcohol or 7–12% phenol)

**Cryotherapy**

- **Definition:** Extreme cold to freeze & destroy nerves & tissue
- **Mechanism:** Cell injury by necrosis (resulting from freezing & thawing)
- **Technique:** Liquid nitrogen most commonly used (argon & N<sub>2</sub>O possible) (*desired temp usu. –50 to –60°C*); usually one or two 5- to 30-s freeze-thaw cycles performed q4–6wk

**Radiofrequency (RF) neurolysis**

- Destroys nerve conduction for 3–18 mo
- Indications: Denervate facet joints, splanchnic nerve (for abd pain), cranial nerve ganglion (trigeminal ganglion)
- Only pulsed RF is recommended for peripheral nerves
  - Usually done at 42–45°C for 120–180 s
  - Forms a lesion if neural temp exceeds 45°C

**Facet Blocks**

- Pain relief for neck & back pain (pain elicited by neck/back extension)
- Blocks medial branch of posterior ramus of spinal nerve
  - For severe & prolonged pain, consider denervation of facet joints by RF
- High temp 80°C applied for 90 s at each level

## Nonliving Donors

### Cadaveric Organ Grafts

- Donors usually brain-dead, without evidence of untreatable infection or extracranial malignancy
- Brain death criteria (also see Chapter 33, Ethical Issues in Anesthesia)
  - Comatose without spontaneous movement or response to painful stimuli
    - Rule out causes of reversible cerebral dysfunction
  - Lack of brainstem activity
    - Assess brainstem for lack of brainstem reflexes
      - *Pupillary response to light, corneal reflex, oculocephalic reflex (doll's eye), oculovestibular reflex (cold caloric testing), gag & cough reflex, facial motor response*
- Apnea test
  1. Preoxygenated pt with 100% O<sub>2</sub> for 10 min & confirm normal PCO<sub>2</sub>
  2. Turn off ventilator & administer O<sub>2</sub> via T-piece
  3. After 7–10 min, PaCO<sub>2</sub> >60 mm Hg & no resp effort confirms lack of brainstem control (positive apnea test)
- Other means by which to test brainstem activity: Transcranial Doppler, EEG, AEPs

### Non-Heart Beating Organ Donors

- Organs removed only after cardiac arrest ensues (longer warm ischemia time)
- Legal & ethical issues complicate widespread acceptance
- Less favorable outcomes (↑ rate of major biliary complications)

### Organ Preservation Techniques

- Recommended time limits for storage before reperfusion
  - *Kidney—1 to 2 d; Heart—6 hr; Liver—18 hr*

### Intraoperative Management of Procurement

- Pathophysiologic changes in brain death
  - Hypotension, ↓ cardiac output, myocardial dysfx, & ↓ SVR
  - ↓ oxygenation from neurogenic pulm edema, diabetes insipidus
  - Electrolyte disturbances: ↑ Na<sup>+</sup>, ↓ K<sup>+</sup>
  - Hyperglycemia, coagulopathy, hypothermia
- General anesthetic goals
  - SBP >100 (MAP 70–110 mm Hg)
  - PO<sub>2</sub> >100 mm Hg
  - Urine output 1–1.5 mL/kg/hr
  - Hemoglobin >10.0 g/dL
  - CVP 5–10 mm Hg
  - FIO<sub>2</sub> <40% (as tolerated) for lung procurement
- Anesthesia
  - General with positive-pressure lung ventilation
  - Long-acting nondepolarizing muscle relaxants
  - Volatile anesthetics & narcotics to control hemodynamics
    - Surgical stimulation may cause hemodynamic responses (i.e., ↑ BP) via spinal cord pathways
  - Brain death criteria pts have no pain perception (analgesia is **not** required)
  - Pressor of choice = dopamine, others as needed
  - 50–200 mL of blood required for pretransplant testing
- Specific requirements depend on which organs are harvested
  - Pancreas harvest: May need to irrigate oro/nasogastric tube with Betadine solution to maintain sterility
  - Lung & heart harvest: Pull back CVP & PA catheter if cross clamping needed
  - Liver harvest: Phentolamine or alprostadil usually given just before or during cross clamping (↓ SVR & allows for even distribution of preservation solution)
  - Heparin bolus (20,000 to 30,000 U) upon request by surgeon

## Living Kidney Donor

### Donor Criteria and Evaluation

- Donor kidney fx tested (determine Cr, Cr clearance, urine protein excretion)

### Low Morbidity & Mortality

- Potential complications include pneumothorax & subcutaneous emphysema

**Anesthetic Considerations**

- GA with ETT, 1–2 large-bore IVs
- Epidural catheter usually not placed
- Positioning: R or L lateral (with table flexed & kidney rest elevated)
- Generous fluid requirements (must place Foley); goal urine output 10–20 mL/kg/hr  
*mannitol or furosemide may be used to maintain urine output*
- Heparin given before renal vessels are clamped (3000–5000 U IV)
- Protamine may be given after kidney is dissected free & blood supply tied off

**Surgical Procedure**

- Laparoscopic living donor nephrectomy more popular than open approach  
*hand-assisted approach through additional incision also commonly utilized*
- Potential complications: Pneumothorax & subcutaneous emphysema

**Living Liver Donor****Donor Criteria**

- Imaging (CT/MRI) & lab eval (LFTs/coags) to assess liver fx & anatomy
- No consensus on who can donate: Factors associated with poor performance →  
↑ donor age, graft steatosis, ↑ graft ischemia times, ↑ ICU days, ↑ inotrope requirement

**Pediatric Recipients**

- Usually need only L lateral or L hepatic lobe (not the entire liver)

**Adult Living Donors**

- May need only L lateral, L hepatic, or R hepatic lobe  
(leaves donor with 1/3 the original liver mass)

**Morbidity & Mortality**

- Complication rates vary from 0–67%, crude morbidity rate = 37%
- As of 2003, there were 3 U.S. deaths from living liver donation, 8 worldwide

**Surgical Procedure**

- R-sided subcostal incision vs chevron incision  
→ Liver mobilized & vascular structures dissected free  
→ Liver transected  
→ Bile duct & vascular structures oversewn, hemostasis achieved, incision closed

**Anesthetic Considerations**

- GA with ETT; 2 large-bore IVs; A-line; ± CVP
- PRBCs should be available (although blood loss usually <1 L)
- Consider preop thoracic epidural  
(some do not place—fear autoanticoagulation after hepatic resection)
- Oro/nasogastric tube for gastric decompression (improves exposure)
- Liver manipulation may cause hypotension from ↓ venous return

**Living Lung Donor**

Recipient usually receives 1 lung lobe from 2 different living donors (1 LLL & 1 RLL)

**Morbidity & Mortality**

- Mortality low, morbidity high (61% in 1 study)  
*complications: Reexploration, pleural effusion, hemorrhage, phrenic n. injury, pericarditis, pneumonia, ileus*

**Anesthetic Considerations**

- GA with ETT; large-bore IV access; A-line
- Positioning: Lateral decubitus
- ± Thoracic epidural catheter
- Heparin given just before lobar artery ligation

**Surgical Procedure**

- Thoracotomy incision
- Usually take left lower lobe (LLL) or right lower lobe (RLL)

<b>Contraindications to Solid Organ Transplantation</b>	
<b>Absolute Contraindications</b>	<b>Relative Contraindications</b>
<ul style="list-style-type: none"> <li>• Active uncontrolled infection</li> <li>• Severe cardiopulmonary/medical condition (pt unfit for surgery)</li> <li>• Inability to tolerate immunosuppression (AIDS)</li> <li>• Continued drug/alcohol abuse</li> <li>• Brain death</li> <li>• Extrahepatic malignancy</li> <li>• Inability to comply with medical regimen</li> <li>• Lack of psychosocial support</li> </ul>	<ul style="list-style-type: none"> <li>• Noncompliance</li> <li>• History of drug abuse</li> <li>• Advanced age</li> <li>• Psychological instability</li> <li>• HIV infection</li> </ul>

## Anesthesia for Kidney Transplantation

### Indications

- Polycystic kidney disease
- Diabetes mellitus–related kidney failure
- Hypertensive kidney disease
- Glomerular disease
- Tubulointerstitial disease
- Other familial or congenital diseases

### Preoperative Evaluation

- Check electrolytes the morning of surgery (delay surgery if  $K^+ > 6.0$  mEq/L)
- Should have dialysis within 24 hr of surgery
- Typical comorbidities
  - CAD = major cause of death in ESRD pts before & after transplant
  - Electrolyte abnl, HTN, DM, delayed gastric emptying, acidosis, anemia
  - CHF (from vol overload & compensatory concentric cardiomyopathy)
  - Coagulopathies (qualitative platelet defect in uremic pts), pericarditis

### Intraoperative Management

- Standard monitors (avoid placing BP cuff on fistula arm)
- Consider A-line (if indicated by comorbidities)
- Consider central line—CVP monitoring, ability to give thymoglobulin *may be difficult to place (prior dialysis lines)*

### Induction & Maintenance

- Usually GA (RSI if gastroparesis suspected—i.e., long-standing diabetes)
- Spinal & epidural not typically implemented (platelet dysfx in uremic pts)
- Avoid enflurane & sevoflurane (inorganic fluoride byproduct may accumulate)
- Paralytics
  - Consider avoiding succinylcholine (if elev.  $K^+ 0.5$  mEq upon induction)
  - Vecuronium & pancuronium may have prolonged effects
  - Atracurium & cisatracurium not affected by ESRD (Hoffman degradation and nonenzymatic ester hydrolysis)
- Narcotics
  - Morphine, meperidine, oxycodone metabolites can accumulate & prolong duration
  - Fentanyl, sufentanil, alfentanil, remifentanil may be safer alternatives

### Surgical Procedure

- 8–10 cm arced incision from pubic symphysis to anterior superior iliac spine
- Graft anastomoses usually made to external iliac vein & artery  
*External iliac artery & vein clamped for anastomoses*
- Graft warm ischemia time is usually about 15–30 min
- Bladder filled via Foley catheter (to facilitate ureteral anastomosis to bladder)
- Native kidney only removed if pt has intractable HTN or chronic infection

### Specific Intraoperative Considerations

- Hypotension may ensue with unclamping of iliac vessels & graft reperfusion  
Avoid  $\alpha$ -adrenergic agents that cause graft vessel vasoconstriction (phenylephrine)  
Low-dose dopamine (3–5 mcg/kg/min) may be a better option
- Heparin may be requested before clamping of iliac vessels
- $\uparrow$  Preload (CVP of 12–15 & MAP  $>60$ ) before unclamping/reperfusion by administering 0.9 NS (3–5 L may be needed) or colloid
- Mannitol may act as free radical scavenger & help diurese kidney after reperfusion (furosemide also used); goal urine output  $>0.5$  mL/kg/hr
- Ca-blocker admin before vessel anastomosis may prevent reperfusion injury
- Consider bicarbonate infusion for significant metabolic acidosis (pH  $<7.2$ )

### Immunosuppressive Agents

- Typical combination: Corticosteroids, cyclosporine (or tacrolimus), & azathioprine (or mycophenolate mofetil)
- Can delay cyclosporine & tacrolimus a few days & use antithymocyte globulin instead

### Postoperative Management

- Pt usually extubated
- Goal urine output  $>0.5$  mL/kg/hr

## Anesthesia for Liver Transplantation (Also see Chapter 18, General Surgery)

### General

- 1-yr survival following transplant 80–90%; 5-yr survival: 60–80%
- Organ allocation: Based on MELD (model of end-stage liver dz) or PELD (pediatric) score

### Preoperative Evaluation

- Underlying diagnoses of recipients  
Hepatitis C (21%), EtOH (16%), cryptogenic cirrhosis (10%), primary biliary cirrhosis (9%), primary sclerosing cholangitis (8%), fulminant (6%), autoimmune (5%), hep B (5%), EtOH + hep C (4%), cancer (4%)
- Extrahepatic manifestations of liver disease  
Correctible problems include coagulopathy (platelet & FFP admin) pleural effusions (thoracentesis)

### Extrahepatic Manifestations of Liver Disease

- |          |   |
|----------|---|
| • Pulm:  | Portopulmonary HTN, hepatopulmonary syndrome, pleural effusions   |
| • CV:    | Hyperdynamic circulation (↑ cardiac output & ↓ SVR)   |
| • GI:    | Portal HTN, esophageal varices, ascites   |
| • CNS:   | Encephalopathy, ↑ ICP (with fulminant hepatic failure)  |
| • Heme:  | Thrombocytopenia (↓ thrombopoietin & hypersplenism), ↓ clotting factors (↓ decreased synthetic fx, DIC, fibrinolysis) |
| • Renal: | Oliguria, renal insufficiency, hepatorenal syndrome   |

### Intraoperative Management

- Venous access
  - Large-bore peripheral access (RICC line or 8.5-Fr peripheral IV)
  - 8.5- or 9-Fr central venous catheter
  - May need additional access if PA catheter is in lumen of 8.5- or 9-Fr central catheter
- Standard monitors; A-line preinduction; CVP; consider PA catheter & TEE
- Equipment
  - Stat lab must be close by & available
  - Rapid infuser systems (Level I, Belmont, etc.) set up & available
  - Blood products available (usually 10 U FFP, 10 U PRBCs, & platelets)
  - Venovenous bypass machine (with perfusionist) available
  - Cell saver

### Induction and Maintenance

- Usually RSI (for "full stomach" precautions) or awake intubation
- Pts often coagulopathic (use care when placing lines, ETT, NG tube)
- inhalational agents, narcotics, & muscle relaxants during maintenance
- Avoid ketamine—can ↑ seizure activity
- Consider correcting coagulopathies early (rather than later)
- Maintain normothermia

### Postoperative Management

- Peripheral nerve injuries commonly due to positioning
- Following skin closure, patient brought to ICU (usually intubated)

### Phases of a Liver Transplant Operation

#### The Preanhepatic (Dissection) Phase

- Primary purpose: Dissection of porta hepatis & mobilization of native liver
- Hypotension: Surgical bleeding, ascites/effusion drainage, clamping/pressure on abd veins
- Bleeding risks: Portal HTN, coagulopathy & prior abdominal surgery
- Metabolic alterations: ↑ K<sup>+</sup>, metabolic acidosis, ↓ Ca<sup>2+</sup> (from citrate toxicity)
- Coagulopathy: Underlying factor deficiencies, thrombocytopenia & dilutional coagulopathies
- Hypothermia (must be corrected, or will worsen coagulopathy)
- Ensure adequate urine output & euvolemia

#### The Anhepatic Phase

- Begin = clamping hepatic artery & portal vein; end = reperfusion of donor liver
- Vessels perfusing liver are clamped & old liver removed
- Warm ischemic time: Begins when donor liver removed from ice, end when reperfused; should limit warm ischemia time to between 30 & 60 min
- Portal vein, inferior vena cava (IVC) & hepatic artery usu. clamped during this phase
- Venovenous bypass occasionally required for pts who do not tolerate cross clamping
  - Blood diverted from portal vein & IVC into SVC, usu. via axillary vein
  - Advantages: Avoids renal/splanchnic engorgement, maintains preload, ↑ renal perfusion; may ↓ blood loss, ↓ transfusion requirements
  - Disadvantages: ↑ Risk of air embolus/DVT, seroma, nerve injury, wound lymphocele

### Phases of a Liver Transplant Operation (Continued)

- Piggyback technique may also obviate need for IVC clamping
  - Native hepatic veins fashioned into a cuff (serves as a receptacle for suprahepatic IVC of the donor liver); cava to cuff anastomosis performed & native IVC need not be clamped
- Preparation for reperfusion:  $\uparrow K^+$  & acidosis prevalent in absence of functioning liver
- Aggressively treat with furosemide, albuterol,  $\uparrow$  ventilation, insulin/D50 and/or bicarb

### The Neohepatic Phase

- Begins with allograft reperfusion; end with completion of biliary anastomosis
- Allograft flushed of air, debris & residual preservative solution
- Unclamping of donor liver  $\rightarrow$  embolic debris,  $\uparrow K^+$ , metabolic acidosis,  $\downarrow Ca^{2+}$ , hypothermia, hypotension, hypovolemia, release of cytokines & other destabilizing agents
- Postreperfusion syndrome (PRS)  $\rightarrow$   $\downarrow$  MAP of 30% from baseline, lasts  $\geq 1$  min, within 5 min of reperfusion
  - Can see arrhythmias,  $\downarrow$  SVR,  $\downarrow$  cardiac output, vasodilation,  $\uparrow$  L ventricular filling pressure, R ventricular dysfx
  - Contributing factors:  $\uparrow K^+$ ,  $\downarrow Ca^{2+}$ , acidosis, & blood loss
  - Air embolus & thromboembolus may be seen via echo or inferred via PA-line
  - Treatment: Minimize  $\uparrow K^+$ ,  $\downarrow Ca^{2+}$ , acidosis; usually resolves within 30 min
- Postreperfusion coagulopathy  $\rightarrow$  may also follow graft reperfusion
  - Due to (1) release of heparin or (2) tissue plasminogen activator (tPA)  $\rightarrow$  1 $^\circ$  fibrinolysis
  - Heparin effect reversible with heparinase on thromboelastography (TEG)
  - Cryoprecipitate, FFP & antifibrinolytics (epsilon aminocaproic acid, tranexamic acid) may treat 1 $^\circ$  fibrinolysis
  - Protamine will treat heparin effect
  - Refractory coagulopathy may indicate graft failure
- Indicators of good graft function: Resolution of coagulopathy & metabolic acidosis, return of normoglycemia & bile production; renal dose dopamine may offer some renal protection

### Anesthesia for Lung Transplantation

#### Indications

- COPD, idiopathic pulmonary fibrosis, cystic fibrosis (CF),  $\alpha_1$ -antitrypsin deficiency, PPH (primary pulmonary HTN)
- Less frequently: Sarcoidosis, retransplantation, Eisenmenger's syndrome

#### Indications for Heart-Lung Transplantation (HLT)

- Pts with lung transplant indication & significant left ventricular dz
- Most commonly PPH, CF, & Eisenmenger's syndrome

#### Single-lung Transplantation (SLT) vs Bilateral Sequential Lung Transplantation (BSLT)

- BSLT = 1 lung transplanted followed by a repeat procedure on contralateral side

#### Preoperative Evaluation

- Lab values: ABO compatibility of donor & recipient
- Functional data (including PFTs) & indicated cardiac testing
- Pts may have difficult lying flat (poor pulm function)

#### Intraoperative Considerations

- Standard monitors + A-line, central line, PA catheter; consider TEE (assess RV fx)
- 2 large-bore IVs;  $\pm$  epidural catheter

#### Induction and Maintenance

- Lung isolation: Double-lumen tube, univent tube, or ETT + bronchial blocker
- Avoid  $N_2O$  (presence of bullous emphysematous dz, pulm HTN, intraop hypoxemia)
- Fluid management usually conservative (helps with postop management)

#### Surgical Procedure for Single Lung Transplantation

- Posterolateral thoracotomy position (need for rapid access to cannulation sites for emergent cardiopulmonary bypass may affect positioning)
- Incision usually anterior thoracotomy with partial sternotomy
- Sequence of surgical events
  - $\rightarrow$  Structures for lung to be resected are dissected free
  - $\rightarrow$  Pneumonectomy completed
  - $\rightarrow$  Atrial/pulm vein anastomosis made, followed by bronchus & pulm artery
  - $\rightarrow$  Circulation then restored to graft & ventilation begun
  - $\rightarrow$  Process repeated for other side during bilateral sequential lung transplantation

#### Specific Anesthetic Considerations

- Lung recipients susceptible to pulm HTN & R ventricular dysfx during 1-lung vent
- Hypoxemia common in 1-lung ventilation; consider using:



- $\text{FiO}_2$  of 1.0
- PEEP of 10 as tolerated to dependent lung
- CPAP to nondependent lung
- **Nitric oxide (NO)**
  - Advantages:
    - ↓ Pulm vascular resistance & improves oxygenation
    - NO preferentially reaches ventilated areas, causing ↑ blood flow, improvements in  $\dot{V}/\dot{Q}$  mismatch & improved oxygenation
    - ↓ Inflammatory response to surgery or trauma
    - Impedes microbial growth
    - Activates guanylate cyclase in platelets to attenuate platelet aggregation & adhesion
  - Disadvantages:
    - Methemoglobinemia, NO metabolite-related lung injury, ↓ sensitivity of exhaled  $\text{N}_2\text{O}$  monitoring
    - Rapid discontinuation of NO in pulm vasculature prevents systemic vasoconstriction & results in systemic hypotension
- Cardiopulmonary bypass (CPB) indications
  - Adequate oxygenation cannot be maintained despite ventilatory/pharmacologic interventions & PA clamping by surgeons
  - Inability to ventilate
  - Development of RV dysfx
- May see hypotension with restoration of graft blood flow after anastomosis
- At end of procedure, eval of pt for tube exchange to single lumen is performed, although high PEEP requirements & oropharyngeal edema may preclude it

## Anesthesia for Heart Transplantation

### General Information

- 1-year survival = 86% in 1990s
- Poor survival due to paucity of donor organs, devices (e.g., left ventricular assist devices—LVAD) used to provide a bridge to transplant

### Most Common Indications

- New York Heart Association class III or IV heart failure (despite optimal therapy)
- Ischemic and idiopathic dilated cardiomyopathies
- Viral, infiltrative, postpartum, congenital heart dz—related failure
- Occasionally for refractory angina, unmanageable arrhythmia, diastolic failure

Possible Contraindications to Heart Transplantation	
Severe ↑ pulm vascular resistance (>6 Wood units) (orthotopic procedure)	Age >55 years
Continued illicit drug/tobacco use, noncompliance	Uncontrolled malignancy
Significant irreversible renal, hepatic, or pulm dysfunction	Obesity
Coexisting systemic illness with a poor prognosis	Previous malignancy
Diabetes mellitus with end-organ damage	Osteoporosis
Active infective process (hepatitis B & C)	Active peptic ulcer disease
Amyloidosis (cardiac dz may recur)	

Source: Adapted from Miller. *Miller's Anesthesia*, 6th ed. Philadelphia: Elsevier,

### Perioperative Assessment

- Donor heart function worsens with ischemic time >6 hr
- Pt usually not NPO (owing to short notice of graft availability)
- Pt may receive extensive levels of cardiovascular support
  - Meds—warfarin, vasopressor support, ACE inhibitor, dobutamine
  - Devices—LVAD, pacemaker/AICD
- Immunosuppressive meds & antibiotics
- Ensure blood products available

### Intraoperative Management

- Large-bore IV access, standard monitors, preinduction A-line, CVP & PA catheter, TEE
- Induction and maintenance
  - Consider high-dose narcotic induction
  - Neuromuscular blockade with nondepolarizing agent
  - May need inotropic support upon induction
  - Standard heparin dosing for pre-CPB anticoagulation
  - See (Chapter 16, Anesthesia for Cardiac Surgery) for detailed notes on CPB
- Separation from CPB

- Transplanted heart denervated (will not mount tachy-/bradycardic responses)
- Only direct-acting sympathomimetics work for inotropic/chronotropic effects
  - Isoproterenol, epinephrine, milrinone, dobutamine
- LV function is generally adequate, however, RV dysfunction often seen
- Strategies to lower PVR
  - High  $\text{FiO}_2$ ; avoid hypercapnia/hypothermia
  - Optimize airway pressures & tidal volumes
  - Use nitrates,  $\text{PGE}_1$ , prostacyclin, & inhaled NO as indicated
  - Use CVP/TEE to guide fluid management
  - Consider use of RV assist device

#### Surgical Procedure

- Incision median sternotomy
- Recipient heart excised (except for L atrial tissue with pulmonary veins)
  - Biatrial approach—excises both atria (mandating bicaval anastomosis)
  - Classic approach—atria transected at grooves
- Aortic cannulation high, near the arch

#### Specific Anesthetic Considerations

- Anticipate previous cardiac surgery (redo sternotomy)
  - Structures may be adhered to sternum & ruptured upon entry
  - Presence of LVAD/RVAD
- Pts with hemodynamic instability may need extracorporeal membrane oxygenation (ECMO) prior to induction
- No specific anesthetic strategies for posttransplant anesthesia delivery
  - May see a delayed response to catecholamines
  - Anticipate a denervated heart with absence of vagal tone

## General Considerations

- Aging assoc with ↑ perioperative mortality & morbidity
  - Perioperative cardiovascular events
  - Postoperative infections
  - Perioperative airway and pulmonary problems
  - Postoperative CNS dysfunction

## Systemic Changes

### Cardiovascular System

- ↓ Arterial elasticity causes: ↑ Afterload, ↑ SBP, LVH, ↓ baroreceptor reflex
- ↑ Incidence of cardiac arrhythmias, CAD, CHF, aortic stenosis, atherosclerosis, HTN, diastolic dysfunction, diabetes

### Pulmonary System

- ↓ Compliance causes:  $\dot{V}/\dot{Q}$  mismatch, ↑ residual volume, ↓ alveolar surface, ↑ closing capacity, ↑ chest wall rigidity, ↓ PaO<sub>2</sub>
- ↑ Incidence of diminished response to hypoxia & hypercapnia, ↓ muscle strength, ↓ cough, ↑ incidence of pneumonia, COPD, lung cancer

### Renal System

- RBF, ↓ nephron mass, little change in serum creatinine due to ↓ muscle mass, ↓ Na concentrating/diluting ability → dehydration, fluid overload
- Increased incidence of: Perioperative ARF

### Nervous System

- ↓ Nervous system mass and neuronal density, ↓ neurotransmitters and neuroreceptors
- ↓ Dosage requirements of local and general anesthetics, ↑ incidence of postop delirium, ↑ recovery time from general anesthesia
- Local epidural anesthetic results in ↑ cephalad spread

## Pharmacologic Considerations

- Plasma albumin → main plasma binding protein for drugs
- ↑ Central compartment & ↑ serum drug concentration after bolus administration
- Drug elimination may be prolonged with impaired renal function

## Avoiding Overdose in Elderly Pts

- Administer reduced concentration of inhaled agent depending on age
  - Should ↓ agent % because MAC and MAC-awake decrease with advancing age:
    - MAC for 70–85-yr-old = 65–85% MAC for 40-yr-old
  - Lighter level (e.g., BIS 40–60), shorter-duration anesthetics now considered safer than deeper (BIS 20–40), longer-duration ones
- Correct IV dose for
  - Advancing age: Should reduce IV drug dose, e.g.,
    - Fentanyl: Dose for 80-yr-old = 50% dose for 20-yr-old
    - Propofol: Induction dose for 75-yr-old = 0.8–1.2 mg/kg
  - Obesity (increasing in elderly)
    - Induction agents, opioids—base on lean body weight
    - Neuromuscular blocking agents—base on lean body weight plus 1/3 (total body weight – lean body weight)
  - Allow for slower onset time—i.e., allow sufficient time for initial drug bolus to reach peak affect before giving additional drug

### Time (min) to Peak Effect After Bolus Dose

Drug	Young Adult	Elderly Adult
Succinylcholine (1 mg/kg)	1.2	1.6
Cisatracurium (0.1 mg/kg)	3.0	4.0
Rocuronium (1.0 mg/kg)	1.0	1.3

- Take into account longer duration of action in elderly
- Neuromuscular block agents
  - Duration prolonged by age if steroid-based, mild hypothermia, increased intensity of neuromuscular blockade (as required for intra-abd surgery), diabetes mellitus, dosing based on total body weight (see above), residual potent inhaled agent (high muscle solubility), respiratory acidosis (see below)

- Use short- or intermediate-acting rather than long-acting
- Consider cisatracurium (unaffected by age)
- Reversal: Recommended in the elderly
- Avoid respiratory acidosis: Keep end-tidal  $\text{CO}_2 \leq$  normal during reversal (do not allow  $\text{CO}_2$  retention as stimulus of spontaneous ventilation prior to/during reversal)
- Meperidine: Avoid except for 10–20 mg IV to treat shivering
  - Accumulates with repeat dosing
  - Active, toxic (seizures) metabolite
  - Anticholinergic activity—can cause tachycardia, agitation
  - Acute serotonergic syndrome with MAO inhibitors
- Other opioids ( $\uparrow$  brain sensitivity with increasing age)
  - Elderly require lower doses for pain relief
  - Morphine, sufentanil, alfentanil, and fentanyl may be 2 $\times$  as potent
  - Owing to  $\downarrow$  clearance and volume of central compartment  $\rightarrow$  adjust infusion rates
- Premedications
  - Reduce dose of benzodiazepines
  - Metoclopramide may  $\uparrow$  risk of extrapyramidal effects

## EKG Lead Placement & Utility

- 12 leads
  - Limb leads: I, II, III, aVR, aVL, aVF
    - II, III, aVF—inferior
    - I, aVL—lateral
  - Precordial chest leads: V1, V2, V3, V4, V5, V6
    - V1, V2—septal
    - V3, V4—anterior
    - V5, V6—lateral
- Coronary arteries
  - LAD: V1, V6, anterior, septal, & inferior
  - Circumflex: I, aVL, V5, V6, lateral & posterior (inferolateral)
  - RCA: II, III, aVF, inferior & posterior (inferolateral)
- Intraoperative monitoring
  - II = best for detecting arrhythmias
  - V = best for detecting ischemia

### Steps for ECG Interpretation

- Rhythm
  - Check for P before every QRS
  - Check PR intervals to assess for AV block
  - Check QRS intervals to assess for bundle branch blocks

### Rate

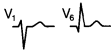
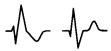
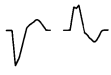
Count large boxes between R waves on ECG (each large box = 0.2 s)

- 1 large box = 300 bpm
- 2 boxes = 150 bpm
- 3 boxes = 100 bpm
- 4 boxes = 75 bpm
- 5 boxes = 60 bpm
- 6 boxes = 50 bpm
- Examine axis; ST segments; T, Q, and U waves; QRS width & progression, hypertrophy (see below)

**Axis** = direction of vector which represents heart's overall wave of depolarization

Interpretation of Axis in Vertical Plane		
Lead I	Lead AVF	Axis
Positive	Positive	Normal
Positive	Negative	Left axis deviation
Negative	Positive	Right axis deviation
Negative	Negative	Extreme right axis deviation

- Atrioventricular Conduction System
  - **1st-degree AV block**—PR interval increased  $>0.2$  s
  - **2nd-degree AV block**
    - **Mobitz type I** (Wenkebach)—AV delay (PR interval) increases with each beat, until QRS is dropped after P wave
      - Treatment—only if symptomatic: Atropine, isoproterenol, permanent pace
    - **Mobitz type II**—sudden unpredictable dropped QRS not associated with progressive PR interval prolongation
      - *Caution: May progress to 3rd degree heart block*
      - Treatment—permanent pacemaker
  - **3rd degree AV block** (complete heart block)
    - No relationship between P wave & QRS—"AV dissociation"
    - Treatment—permanent pacemaker
  - **Bundle-branch block**

Bundle Branch Blocks		
Normal		Initial depol. is left-to-right across septum (R in V <sub>1</sub> & Q in V <sub>6</sub> ; absent in LBBB) followed by LV & RV free wall, with LV dominating (nb, RV depol. later and visible in RBBB).
RBBB		<ol style="list-style-type: none"> <li>1. QRS ≥ 120 msec (100–119 = incomplete)</li> <li>2. RSR' in R precordial leads (V<sub>1</sub>, V<sub>2</sub>)</li> <li>3. Wide S wave in I, V<sub>5</sub>, and V<sub>6</sub></li> <li>4. ± ST↓ or TWI in R precordial leads</li> </ol>
LBBB		<ol style="list-style-type: none"> <li>1. QRS ≥ 120 msec (100–119 = incomplete)</li> <li>2. Broad, slurred, monophasic R in I &amp; V<sub>6</sub> (± S if cardiomeg)</li> <li>3. Absence of Q in I, V<sub>5</sub>, and V<sub>6</sub></li> <li>4. Displacement of ST &amp; Tw opposite major QRS deflection</li> <li>5. ± PRWP, LAD, Q's in inferior leads</li> </ol>

Bifascicular block: RBBB + LAHB/LPHB; Trifascicular: 1° AVB + RBBB + LAHB/LPHB

- **Right bundle-branch block (RBBB)**
  - Examine QRS in V<sub>1</sub> & V<sub>2</sub>
  - Right ventricular depolarization delayed
  - LBBB makes it difficult to determine infarction on ECG
- **Left bundle-branch block (LBBB)**
  - Examine QRS in V<sub>5</sub> or V<sub>6</sub>
  - Left ventricular depolarization delayed
  - Difficult to determine infarction on ECG
- **Atrial flutter**
  - Regular atrial activity; 180–350 bpm; ventricular rate 150 bpm (2:1 AV block)
  - ECG: "F waves," "sawtooth" pattern, flutter waves
  - Treatment
    - Unstable → immediate electrical cardioversion
    - Burst pacing (temporary or permanent pacemaker)
    - Medical therapy (β-blockers, Ca<sup>2+</sup>-channel blockers)
    - Radiofrequency catheter ablation (RFA)
- **Atrial fibrillation**
  - Irregular atrial activity at 350–600 bpm, ventricular rate 160
  - ECG: Wavy baseline, absent P waves
  - Treatment
    - Unstable → immediate electrical cardioversion
    - Chemical cardioversion (Class IA, IV, III antiarrhythmics)
    - Antiarrhythmic drugs
    - Anticoagulation
    - Rate control: β- or Ca<sup>2+</sup>-channel blockers, digoxin
    - Maze procedure
- **Paroxysmal SVT**
  - Ventricular rate 140–250 bpm
  - ECG: Narrow complex, P waves hidden in QRS complexes  
(QRS may be slightly widened, not more than 0.14 s)
  - Treatment: Vagal maneuvers, β- or Ca<sup>2+</sup>-channel blockers, radiofrequency ablation
- AV Reentrant Tachycardia
  - **Wolf-Parkinson-White**
    - PR interval shortened, delta wave, wide QRS
    - Treatment: β- or Ca<sup>2+</sup>-channel blockers, radiofrequency ablation
- Ventricular Arrhythmias
  - **Premature ventricular beats**
    - Widened QRS
    - **Couplet**—two in a row; **Bigeminy**—every other beat is PVC
  - **Ventricular tachycardia**—3 or more PVCs in row, 100–200 bpm
    - Nonsustained VT (NSVT)—persists for <30 s
    - Sustained VT—persists for ≥30 s
    - Treatment
      - Symptomatic: Electrical cardioversion followed by antiarrhythmic drugs; follow ACLS protocol
      - Asymptomatic NSVT: β-blockers, implantable cardioverter-defibrillator (ICD) in pts at high risk
      - Unstable: Defibrillation as if ventricular fibrillation

- **Torsades de pointes**
  - Polymorphic VT with varying amplitudes of QRS twisting about the baseline
  - Treatment: Magnesium 1–2 g IV followed by infusion
- **Ventricular fibrillation**
  - Chaotic irregular appearance without discrete QRS waveforms
  - Treatment: See ACLS protocol; ICD if arrhythmia not associated with acute MI

### Hypertrophy

- **Right atrial hypertrophy**
  - Large, biphasic P wave with tall initial component
- **Left atrial hypertrophy**
  - Large, biphasic P wave with wide terminal component
- **Ventricular hypertrophy**
  - **Right ventricular hypertrophy**
    - R wave  $>S$  in V1 (R wave becomes progressively smaller from V1 to V6)
    - S wave persists in V5 & V6
    - Right axis deviation with slightly widened QRS
    - Rightward rotation in horizontal plane
  - **Left ventricular hypertrophy**
    - S wave in V1 + R wave in V5  $>35$  mm
    - Left axis deviation with slightly widened QRS
    - Leftward rotation in horizontal plane
    - Inverted T wave that slants downward gradually but upward quickly

### Electrolyte Imbalances

- Hypokalemia
  - Flattened T wave
  - U waves
- Hyperkalemia
  - Peaked T waves
  - Wide or flat P wave
  - Wide QRS
- Hyper-/hypocalcemia
  - Hypercalcemia—shortened QT
  - Hypocalcemia—prolonged QT

### Drug Effects

- Digitalis toxicity
  - Inverted or flattened T waves
  - Shortened QT interval

### Pulmonary Embolism

- Right axis deviation
- Acute RBBB
- Inverted T waves in V1 to V4 from right ventricular overload
- Wide S in I large Q; and inverted T in III

### Pericarditis

- Diffuse ST-segment elevation (looks similar to acute MI, usually more universal in nature)
- May see subsequent inverted T waves (similar to acute MI)

### Hypothermia

- J wave or Osborne wave

## Informed Consent

- Should be read & signed by patient prior to administering sedatives
- Principle of patient autonomy (patient may accept/refuse treatment)
- Contains description of procedure, potential risks and benefits
- Incapacitated pts (under influence of meds, altered consciousness, incompetent, disabled) → next of kin/health-care proxy/court-appointed guardian should provide consent
- Telephone consent acceptable, preferably cosigned by a witness
- Consent may be waived in an emergency situation
- Resuscitation efforts generally do not require informed consent because they are considered emergency interventions and consent is implied
- For non-English speakers, use an official interpreter (not family or care team member) whenever possible
- In addition to standard language of the consent form, document additional potential complications & procedures as discussed with patient

- **Advance directive** → instructions given by an individual specifying what should be done for his or her health should he or she no longer be able to make decisions
- **Living will** → addresses specific directives regarding treatment course to be taken by caregivers (may forbid certain interventions—e.g., intubation, CPR) if pt unable to give informed consent
- **Health-care power of attorney** → appoints an individual (a proxy) to make health-care decisions should pt become incapacitated
- **Mental competency** → Legal term; pt's ability to make rational informed decisions
  - Adults are presumed to be competent
  - **Only a court** can declare a person incompetent
  - **Physician opinion** of incompetency = **opinion only**

## Brain Death

- Definition = permanent absence of brain & brainstem function
- Must rule out confounding factors  
(*drug/toxins, hypothermia <32 degrees, metabolic derangements, Guillain-Barré syndrome, locked-in syndrome*)

## Brain Death Criteria—Adults & Children

Coma	Absence of gag reflex
Absence of motor responses	Absence of coughing in response to tracheal suctioning
Absence of pupillary responses to light	Absence of sucking and rooting reflexes
Absence of corneal reflexes	Absence of respiratory effort at PaCO <sub>2</sub> of 60 mm Hg or 20 mm Hg > pts nl value
Absence of caloric (vestibulo-ocular) reflexes	

## Required Interval Between Two Evaluations for Brain Death Determination

Term to 2 mo	48 hr
2 mo to 1 yr old	24 hr
>18 yr old	2 evals not required

## Required Confirmatory\* Testing

Term to 2 mo	2 confirmatory tests
2 mo to 1 yr old	1 confirmatory test
>1 yr old	Optional

\*Confirmatory tests: Cerebral angio, EEG, transcranial Doppler ultrasound, cerebral scintigraphy

## End-of-Life Issues

- **DNR/DNI is not automatically suspended during surgery**
- In case of DNR/DNI, must clearly document that status & communicate with medical & nursing staff to avoid providing unwanted treatment
- Specific measures not to be performed should be clearly documented by physician (e.g., intubation, chest compressions, defibrillation, invasive line placement, vasopressors)



- In cases of medical futility: Physician has duty to counsel medical decision maker (next of kin, legal guardian) & explain possibility of DNR/DNI status & potential for withdrawal of life-sustaining measures
- Medical decision maker should receive info about pt's prognosis prior to making end-of-life decisions for pt

### **Pediatric/Minor (<18 yr) Patients**

- Physicians must obtain informed consent from a parent or surrogate before a child can undergo any medical intervention
- Consent for pregnancy termination procedure dependent on state laws
- Pediatric patients' wishes should be included in decision-making process when appropriate

### **Jehovah's Witnesses (JW)**

- JW pts usually will not accept blood or blood products (even under lifesaving circumstances)
- Obtain informed consent, discuss options, & document preoperative discussion with pt regarding products pt will/will not accept
- Special legal considerations may apply to minors, incompetent individuals, emergency procedures
- Physicians may opt out of providing care for a JW patient
- JW may agree to some blood conservation (special cell-saver) techniques
- Generally prohibited
  - Allogenic transfusion of whole blood, red cells, white cells, platelets, plasma
  - Autologous (preoperative donated) blood/blood products
- May be acceptable (discuss with JW)
  - Cell-saver scavenging, cardiopulmonary bypass, dialysis, plasmapheresis
    - If blood does not come out of a continuous circuit with pt
  - Epidural blood patch
  - Blood plasma fractions
    - Albumin, globulins, clotting factors—factors VIII & IX
    - Erythropoietin
    - PolyHeme (blood substitute solution—chemically modified human Hgb)
    - Hemopure (blood substitute solution—chemically stabilized bovine Hgb)

# EMERGENCY ALGORITHMS

TRACY PALUMBO DOVICH

The protocols described below are based on the recommendations given in the *AHA 2005 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*

## ADVANCED CARDIAC LIFE SUPPORT (ACLS)

### ABCD Survey

#### Airway/Breathing

- Maintain patent airway, give supplemental oxygen
- Place advanced airway
  - Administer continuous CPR rather than in cycles
  - 5 cycles of CPR = 2 min
- Adult, child, and infant
  - Continuous ventilation @ 8–10/min
  - Continuous compressions @ 100/min
- Newborn (with or without airway)
  - Continuous ventilation @ 30/min
  - Continuous compressions @ 90/min

#### Circulation

- Check for a pulse

Adult	Carotid
Child	Carotid/femoral
Infant	Brachial

- If a pulse is present, continue rescue breathing

Rescue Breathing	Breaths/Min
Adult	10–12
Child	12–20
Infant	12–20

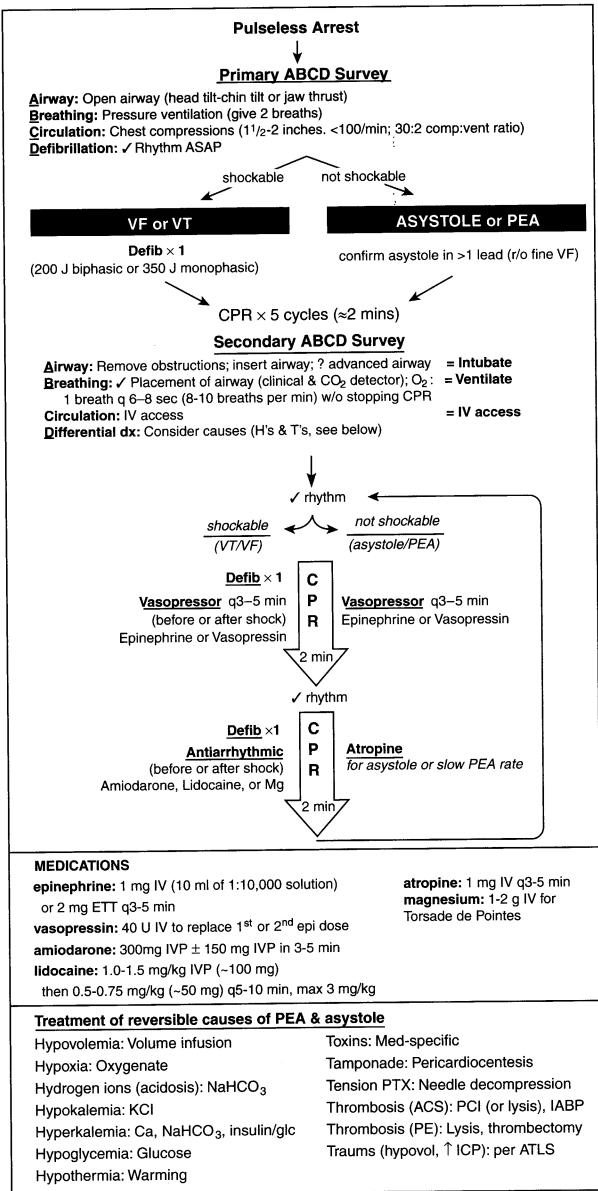
- Reassess pulse every 2 min
- If a pulse is not present within 10 s or pt shows signs of poor perfusion, begin chest compressions
- Chest compressions
  - Minimize interruptions between compressions
  - CPR should be resumed immediately after defibrillation
  - Complete chest recoil between compressions
  - If 2 rescuers are present, roles should be switched every 2 min to prevent fatigue
  - Continue cycles of CPR until a defibrillator or additional help arrives

	Adult (>8yr old)	Child (1 yr old–puberty)	Infant (<1 yr old)
<b>Chest Compressions Location</b>	Center of sternum	Lower ½ of sternum	Lower ½ of sternum
<b>Depth</b>	1½–2 in.	⅓–½ depth of chest	⅓ depth of chest
<b>Technique</b>	Heels of both hands	Heels of both hands	2 fingers (1 rescuer) thumbs encircling hands (2 rescuers)
<b>Rate (per min)</b>	100	100	100
<b>Compression/ventilation ratio</b>	1 or 2 rescuers 30:2	1 rescuer 30:2 2 rescuers 15:2	1 rescuer 30:2 2 rescuers 15:2

- Rhythm checks should not be longer than 10 s
- Should be done after 5 cycles of CPR have been completed (2 min)
- Pulse checks should be done only if an organized rhythm is restored
- Drug administration and definitive airway placement should minimally interrupt compressions

- **Intravascular access should be obtained**
  - Intravenous (preferred)
    - Peripheral (does not interfere with CPR)
    - Central (faster onset of medications, may interfere with CPR)
  - Intraosseous (IO) access
    - May be safely used if difficult IV access
  - Endotracheal route
    - Not desirable
    - Last resort if IV or IO cannot be obtained
    - Dose: 2–2.5 × standard IV dose diluted in 5 to 10 mL NS
    - Drugs that can be administered through ETT
      - Lidocaine, Atropine, Narcan (not recommended), Epinephrine, Vasopressin
- **Defibrillation**
  - Prompt defibrillation is critical when a patient displays a shockable rhythm
  - Pediatric patients (aged 1–8 yr or puberty)
    - Use pediatric dose-attenuating system
    - First shock: 2 J/kg, subsequent shocks: 4 J/kg
  - No recommendation for or against use of AEDs for infants
- **Differential Diagnosis**
  - Diagnose and treat throughout resuscitation

Figure 34-1 ACLS protocols.



**MEDICATIONS**

<p><b>epinephrine:</b> 1 mg IV (10 ml of 1:10,000 solution) or 2 mg ETT q3-5 min</p> <p><b>vasopressin:</b> 40 U IV to replace 1<sup>st</sup> or 2<sup>nd</sup> epi dose</p> <p><b>amiodarone:</b> 300mg IVP ± 150 mg IVP in 3-5 min</p> <p><b>lidocaine:</b> 1.0-1.5 mg/kg IVP (~100 mg) then 0.5-0.75 mg/kg (~50 mg) q5-10 min, max 3 mg/kg</p>	<p><b>atropine:</b> 1 mg IV q3-5 min</p> <p><b>magnesium:</b> 1-2 g IV for Torsade de Pointes</p>
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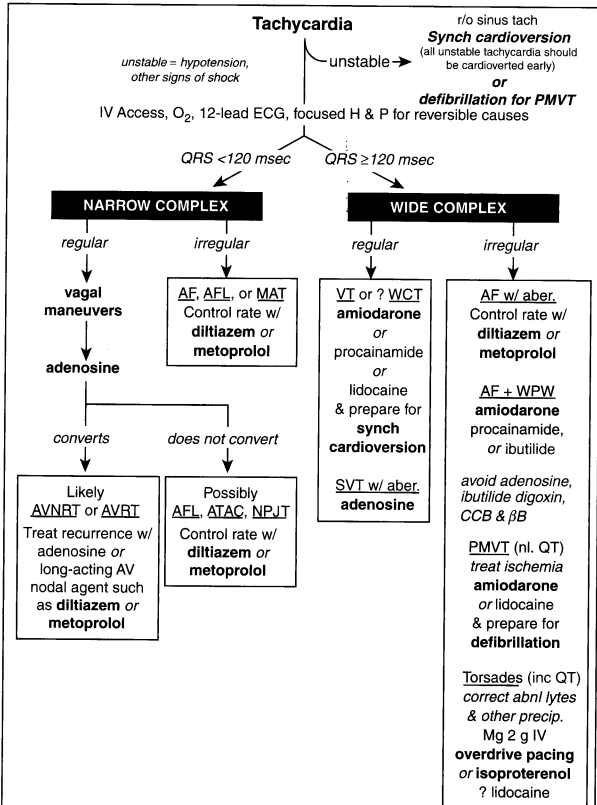
**Treatment of reversible causes of PEA & asystole**

<p>Hypovolemia: Volume infusion</p> <p>Hypoxia: Oxygenate</p> <p>Hydrogen ions (acidosis): NaHCO<sub>3</sub></p> <p>Hypokalemia: KCl</p> <p>Hyperkalemia: Ca, NaHCO<sub>3</sub>, insulin/glc</p> <p>Hypoglycemia: Glucose</p> <p>Hypothermia: Warming</p>	<p>Toxins: Med-specific</p> <p>Tamponade: Pericardiocentesis</p> <p>Tension PTX: Needle decompression</p> <p>Thrombosis (ACS): PCI (or lysis), IABP</p> <p>Thrombosis (PE): Lysis, thrombectomy</p> <p>Traums (hypovol, ↑ ICP): per ATLS</p>
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ALGORITHMS 34-3

(Adapted from ACLS 2005 Guidelines, Circ 2005;112 (Suppl 1): IV-58)

Figure 34-2 ACLS protocols.

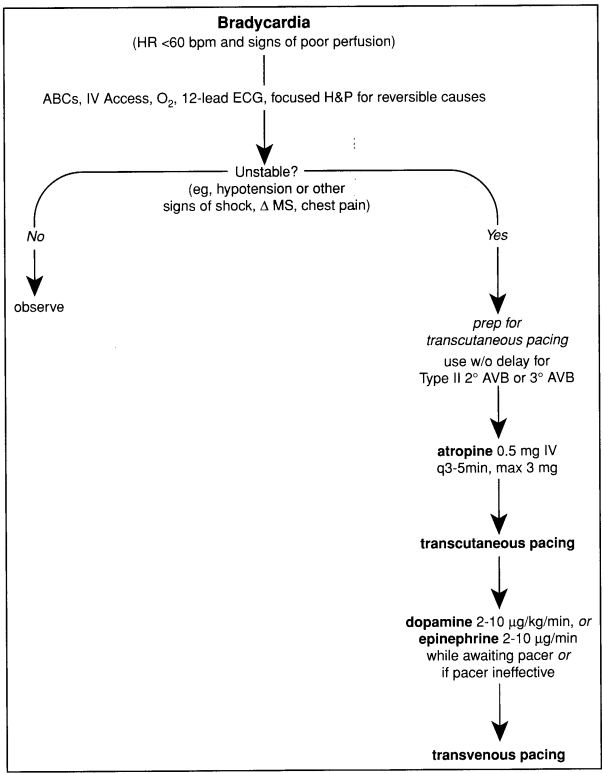


ALGORITHMS 34-4

<b>CARDIOVERSION</b>	<b>MEDICATIONS</b>
<p><u>Ancillary equipment</u></p> <ul style="list-style-type: none"> <li>Pulse oximeter</li> <li>Suction</li> <li>IV access</li> <li>Intubation equipment</li> </ul> <p><u>Premedicate</u></p> <ul style="list-style-type: none"> <li>midazolam 1-5 mg</li> <li>fentanyl 100-300 µg</li> <li>titrate to effect</li> </ul> <p><u>Synchronized cardioversion</u></p> <p><b>100, 200, 300, 360 J</b> or biphasic equivalent</p>	<p><b>adenosine:</b> 6 mg <i>rapid</i> IVP then 20 cc NS bolus, 12 mg IVP q2 min × 2 if needed</p> <p><b>amiodarone:</b> 150 mg IV over 10 min</p> <p><b>diltiazem:</b> 15-20 mg IV over 2 min, 20-25 mg 15 min later if needed, 5-15 mg/h</p> <p><b>ibutilide:</b> 1 mg over 10 min, repeat × 1 if needed</p> <p><b>lidocaine:</b> 1-1.5 mg/kg IVP, repeat in 5-10 min</p> <p><b>metoprolol</b> 5 mg IV q5 min × 3</p> <p><b>procainamide</b> 17 mg/kg at 50 mg/min (<i>avoid if EF low</i>)</p> <p><b>verapamil</b> 2.5-5 mg IV over 2 min, 5-10 mg 15-30 min later if needed</p>

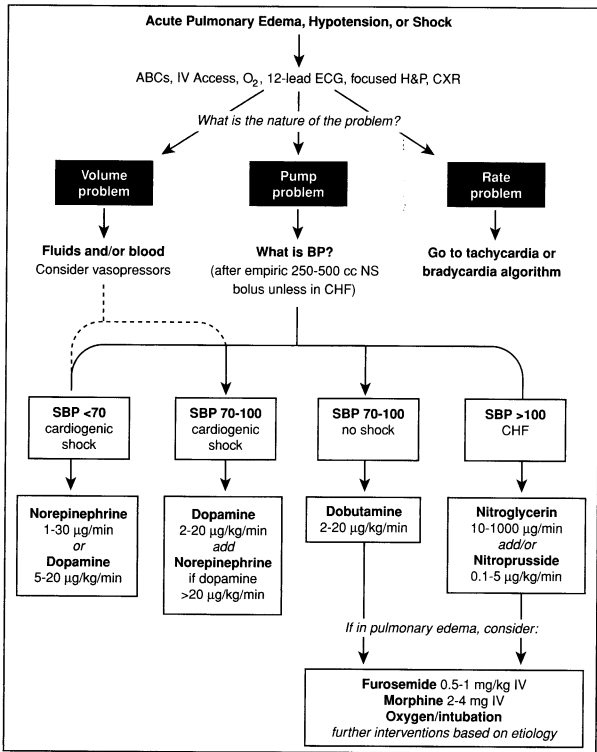
(Adapted from ACLS 2005 Guidelines, Cinc 2005;112 (Suppl 1) IV-67)

Figure 34-3 ACLS protocols.



(Adapted from ACLS Guidelines, Cinc 2005;112(Suppl I) IV-67)

Figure 34-4 ACLS protocols.



ALGORITHMS 34-6

(Adapted from ACLS 2005 Guidelines)

Adult Drug Dosages			
Drug	Indication	IV Dose	ETT Dose
Adenosine	SVT	6 mg, repeat dose with 12 mg	
Amiodarone	SVT, VT/VF, AF/flutter Pulseless VF/VT	150 mg over 10 min, then 1 mg/min 300 mg, repeat with 150 mg	
Atropine	Asystole	1 mg q3–5min, max 0.04 mg/kg <b>note: &lt;0.5 mg can lead to paradoxical bradycardia</b>	1 mg/10 mL dilution, 2–2.5× IV dose
	Bradycardia	0.5–1.0 mg q5min, max 0.04 mg/kg	
Calcium	Hypocalcemia, hyperkalemia, hypermagnesemia	Chloride: 5–10 mg/kg Gluconate: 12–30 mg/kg	
Diltiazem	AF with RVR, reentrant tachycardia	0.25 mg/kg bolus, may repeat bolus 0.35 mg/kg; 5–15 mg/hr infusion	
Dobutamine	Systolic heart failure	2–20 mcg/kg/min	
Dopamine	Oliguria Hypotension, CHF	1–5 mcg/kg/min 5–20 mcg/kg/min	
Epinephrine	Pulseless VT/VF, asystole	1 mg q3–5min	2–2.5 mg
	Hypotension	0.1–1 mcg/kg/min	
	Bronchospasm, anaphylaxis	0.1–0.25 mg	
Glucose/ Dextrose	Hypoglycemia	25–50 g	
Lidocaine	Refractory VT, PVCs	1–1.5 mg/kg IV, repeat 0.5–0.75 mg/kg q5–10 min, max 3 mg/kg; 15–50 mcg/kg/min infusion	2–2.5 × IV dose
Magnesium	Hypomagnesemia, Torsades de pointes	1–2 g	
Naloxone	Narcotic overdose	0.4–2 mg, titrated q2–3 min in 0.2 mg increments	Least desirable route; supported only by anecdotal evidence
Procainamide	Atrial and ventricular arrhythmias	Load: 20 mg/min until toxicity or up to 17 mg/kg; maintenance: 1–4 mg/min	
Sodium bicarbonate	Cardiac arrest	1 mEq/kg (after established ventilation), as per ABG	
	Metabolic acidosis	Base deficit × wt (kg) × 0.2	
Vasopressin	Pulseless VT/VF	40 U × 1 dose	2–2.5× IV dose
Verapamil	SVT, AF/flutter, WPW	2.5–5 mg over 2 min, repeat 5–10 mg; max total 20 mg	



## Pediatric Advanced Life Support (PALS)

Pediatric resuscitation is recommended for children from age 1 yr to start of puberty.

### Recognize the Need of CPR

- Sudden cardiac arrest
  - Uncommon in the pediatric population
- Majority of the events are asphyxia, usually not a primary cardiac cause
  - Usually present as asystole or bradycardia
  - VF and PEA are less common
    - Likely to be the rhythm in a sudden witnessed collapse
- With an unwitnessed arrest, perform BLS immediately and then obtain a AED (CPR first)
- With a witnessed arrest, defibrillate as soon as possible, then CPR (defibrillate first)
- Regarding the infant (<1 yr old) population, there are no data to support or refute the use of defibrillation

Ventricular Fibrillation/Ventricular Tachycardia (pulseless)

#### • Pediatric pulseless arrest algorithm

- Designed to minimize interruptions of compressions
- Rhythm checks are performed after 5 cycles (or 2 min) after shock
  - After a definitive airway is placed, CPR is continuous
- Diagnose and treat underlying causes throughout resuscitation

#### • PEDIATRIC PULSELESS ARREST ALGORITHM SEQUENCE (Figure 34-5)

- Perform BLS while obtaining a defibrillator
- If a shockable rhythm is seen, defibrillate
  - 2 J/kg on the first attempt, 4 J/kg on subsequent attempts
  - Paddles size: Adult size for children > 10 kg  
Infant size for children < 10 kg
  - Largest paddles that can fit on the chest with a 3 cm distance between the paddles are recommended
- After 1 shock, immediately resume compressions
  - CPR before subsequent shocks is associated with a higher rate of success of defibrillating the rhythm
- After 5 cycles of CPR, check the rhythm
- If a shockable rhythm is present, defibrillate (4 J/kg) and resume compressions
- Check the rhythm after 5 cycles and give epinephrine during compressions while charging the defibrillator
  - Drug timing is less important than continuous compressions
  - Standard-dose epinephrine (0.01 mg/kg) should be given every 3–5 min
- After defibrillation, resume CPR for 5 cycles
- Check the rhythm and defibrillate if shockable (4 J/kg), then resume CPR and administer amiodarone or lidocaine
  - Amiodarone 5 mg/kg IV
  - Lidocaine 1 mg/kg IV
- Treat torsades de pointes with magnesium
  - Magnesium 25–50 mg/kg IV
- If the patient develops an organized rhythm, check for a pulse
  - If a pulse is present, supportive care should begin
  - When no pulse is palpable, continue CPR
- If an organized rhythm is achieved but shockable rhythm recurs, give amiodarone during chest compressions before defibrillation

Asystole and PEA

- Similar in causes and treatment
- Grouped together in Pediatric Pulseless Arrest Algorithm
- Pediatric pulseless arrest algorithm (Figure 34-5)
  - After rhythm is determined, begin CPR
  - Administer epinephrine every 3–5 min, minimizing interruptions of compressions
  - Diagnose and treat underlying factors

Bradycardia With Cardiorespiratory Compromise

- Give supportive care including supplemental oxygen and adequate ventilation
- Evaluate heart rate and perfusion
  - If heart rate is below 60 bpm and poor perfusion is still evident after ventilation is supported, start compressions
  - If still persistent, consider drug therapy (epinephrine, isoproterenol, or atropine) or transcutaneous pacing
  - If stabilized, give supportive care and observe
  - If pulse is absent, follow recommendations for pulseless arrest

### Tachycardia With Cardiorespiratory Compromise

- Give supportive care including supplemental O<sub>2</sub>
- Evaluate rhythm and QRS complex
  - **Narrow complex tachycardia**
    - Sinus
      - Diagnose and treat cause
    - Supraventricular tachycardia (SVT)
      - Vagal stimulation
      - Adenosine
      - Synchronized cardioversion (0.5–1 J/kg, repeat 2 J/kg)
      - Amiodarone or procainamide
      - Consider cardiology consultation
  - **Wide complex tachycardia**
    - Ventricular tachycardia (VT)
      - Synchronized cardioversion (same dose as above)
      - Amiodarone or procainamide
      - Consider cardiology consultation

### Vascular Access/Drug Administration

- Intravenous
  - Preferred route
- Intraosseous
  - Safe and effective if intravenous can not be achieved
  - Onset of action is similar to IV route
  - Recommended in cardiac arrest when IV access is not yet established
- Endotracheal
  - Can be used if other routes are not accessible
  - Drugs that can be administered through ETT
    - Lidocaine
    - Atropine
    - Epinephrine
    - Narcan supported only by anecdotal evidence
  - Recommended dose = 2.5× standard dose in 5 mL normal saline followed by 5 ventilations
  - Optimal doses unknown
    - ETT size for children ages 1–8 yr = (age in yr + 4)/4

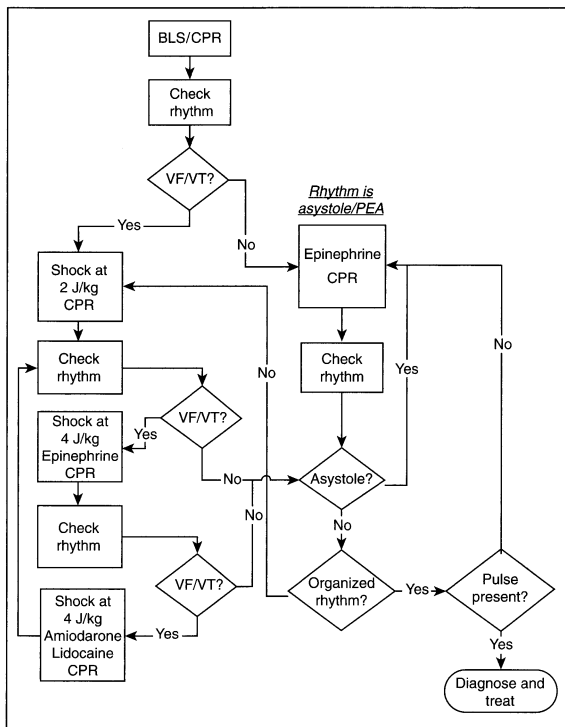
Pediatric Drug Dosages			
Drug	Indications	IV dose	ETT dose
Adenosine	SVT	<50 kg: 0.05–0.1 mg/kg, repeat dose by increasing 0.05–0.1 mg/kg q1–2min, max 0.3 mg/kg >50 kg: Adult dose	
Amiodarone	Pulseless VT/VF	5 mg/kg; up to 15 mg/kg or 300 mg	
Atropine	Bradycardia	0.02 mg/kg q5min, max total 1 mg child, 2 mg adolescent <b>Note: &lt;0.1 mg can lead to paradoxical bradycardia</b>	0.03 mg/kg ETT
Calcium chloride	Hypocalcemia, hyperkalemia, hypermagnesium	20 mg/kg	
Dobutamine	Systolic heart failure	2.5–15 mcg/kg/min	
Dopamine	Hypotension	1–20 mcg/kg/min	
Epinephrine	Cardiac arrest	0.01 mg/kg (0.1 mL/kg 1:10,000) q3–5min, max 1 mg	0.1 mg/kg (0.1 mL/kg 1:1000) in 1–2 mL diluent, max 10 mg
	Hypotension	0.1–1 mcg/kg/min	
	Anaphylaxis	0.01 mg/kg q20min	
Glucose/Dextrose	Hypoglycemia	0.25 g/kg, 6–9 mg/kg/min	

(continued)

Pediatric Drug Dosages (Continued)			
Drug	Indications	IV dose	ETT dose
Lidocaine	Refractory VT,	1 mg/kg, max 100 mg	2-3 mg/kg
	PVCs	20-50 mcg/kg/min	
Magnesium	Hypomagnesemia, Torsades de pointes	25-50 mg/kg over 10-20 min, max 2 g	
Naloxone	Narcotic overdose	0.005-0.01 mg/kg q3-5min	Least desirable route; supported only by anecdotal evidence
Procainamide	Atrial and ventricular arrhythmias	Load: 3-6 mg/kg, max 100 mg; Maintenance: 20-80 mcg/kg/min	
Sodium bicarbonate	Cardiac arrest	1 mEq/kg (after established ventilation)	
	Metabolic acidosis	Base deficit $\times$ wt (kg) $\times$ 0.3	
Verapamil	SVT	<1 yr old: 0.1-0.2 mg/kg over 2 min q30min 1-15 yr old: 0.1-0.3 mg/kg, max 5 mg, repeat 15 min, max 10 mg	

### Pediatric Pulseless Arrest Algorithm

Figure 34-5 Pediatric Pulseless Arrest Algorithm, based on 2005 AHA Guidelines for CPR & ECC.



# COMMON MEDICAL PHRASES IN SPANISH

SALOMON MAYA

## Initial Evaluation/Preop

<b>What is your name?</b>	<i>Cómo te llamas?</i>
<b>Do you know where you are?</b>	<i>Sabes adonde estas?</i>
<b>What type of surgery are you having today?</b>	<i>Que tipo de cirugía vas a tener hoy?</i>
<b>Where does it hurt?</b>	<i>Dónde le duele?</i>
<b>How old are you?</b>	<i>Cuántos años tiene?</i>
<b>How much do you weigh in pounds?</b>	<i>Cuánto pesas en libras?</i>
<b>Are you pregnant?</b>	<i>Estas embarazada?</i>
<b>Are you allergic to anything?</b>	<i>Eres alérgico a alguna medicina?</i>
<b>What reaction do you experience?</b>	<i>Que tipo de reacción te da?</i>
<b>Are you taking any medication?</b>	<i>Estas tomando algún medicamento?</i>
<b>Have you been hospitalized before?</b>	<i>Has estado hospitalizado alguna vez?</i>
<b>Have you had surgery in the past?</b>	<i>Has tenido alguna cirugía en el pasado?</i>
<b>Do you drink alcohol? How much?</b>	<i>Tomas alcohol? Cuánto?</i>
<b>Do you smoke?</b>	<i>Usted fuma?</i>
<b>How many cigarettes per day?</b>	<i>Cuántos cigarrillos fumas por día?</i>
<b>Do you use any illicit drugs?</b>	<i>Usted usa algunas drogas ilegales?</i>
<b>Do you have any problems with your heart?</b>	<i>Tienes algún problema con tu corazón?</i>
<b>Do you get chest pains? Heart attacks?</b>	<i>Usted le da dolores del corazón? Has tenido algún ataque del corazón?</i>
<b>Do you exercise?</b>	<i>Haces ejercicio?</i>
<b>Can you walk two flights of stairs?</b>	<i>Puedes caminar dos pisos de escaleras?</i>
<b>Do you experience an irregular heartbeat?</b>	<i>Tu corazón se siente rápido o irregular?</i>
<b>Do you have problems with your lungs? Asthma?</b>	<i>Tienes algún problema con tus pulmones? Asma?</i>
<b>Do you get short of breath when you walk?</b>	<i>Te falta el aire cuando caminas?</i>
<b>Do you have digestive problems?</b>	<i>Tienes algún problema con tu estómago/intestinos?</i>
<b>Do you have acid reflux?</b>	<i>Tienes acidez?</i>
<b>Do you take medicine for your reflux?</b>	<i>Tomas alguna medicina por tu acidez?</i>
<b>Do you wake up with metallic taste?</b>	<i>Te levantas en la mañana con un sabor metálico en la boca?</i>
<b>Have you been vomiting? When?</b>	<i>Has vomitado? Cuando?</i>
<b>Do you have any problems with your liver?</b>	<i>Tienes algún problema con tu hígado?</i>
<b>Do you have any problems with your kidneys?</b>	<i>Tienes algún problema con tus riñones?</i>
<b>Do you have diabetes?</b>	<i>Tienes diabetes?</i>
<b>Do you have problems with easy bleeding?</b>	<i>Tienes problemas con sangrando fácil?</i>
<b>Are you taking any blood thinners?</b>	<i>Estas tomando alguna medicina para la sangre?</i>
<b>Do you have any problems with your brain?</b>	<i>Tienes algún problema con tu cerebro?</i>
<b>Do you have any numbness or tingling?</b>	<i>Tienes entumecimientos o sientes hormigueos?</i>

<b>Has anyone told you it was difficult to place a breathing tube in your mouth?</b>	<i>Alguien te dijo que fue difícil poner un tubo de respiración en tu boca?</i>
<b>Ever had any problems with anesthesia?</b>	<i>Has tenido algún problema con anestesia en el pasado?</i>
<b>Have you had a recent cough or sore throat?</b>	<i>Has tenido algún tos o dolor de garganta reciente?</i>
<b>When did you last eat or drink?</b>	<i>Cuando fue la última vez que comiste o tomaste algo?</i>
<b>Do you have any muscular pains in your body?</b>	<i>Tienes algún dolor musculares en el cuerpo?</i>
<b>Is your movement limited in any way?</b>	<i>Esta usted limitado a moverse de alguna manera?</i>

### Physical Exam

<b>I am going to check your eyes.</b>	<i>Voy a revisar tus ojos.</i>
<b>Look straight at the light.</b>	<i>Mira directo a la luz.</i>
<b>Open your mouth. Do you have full range of motion of your neck?</b>	<i>Abre la boca. Tienes algún problema con el rango de movimiento de tu cuello?</i>
<b>Open your mouth as wide as possible.</b>	<i>Abre la boca lo más posible?</i>
<b>Stick out your tongue.</b>	<i>Saque la lengua.</i>
<b>I am going to check your teeth.</b>	<i>Voy a revisar tus dientes.</i>
<b>Do you wear dentures?</b>	<i>Usa usted dentadura postiza?</i>
<b>Now I am going to examine your chest.</b>	<i>Ahora, voy a revisar tu pecho.</i>
<b>I am going to listen to your heart.</b>	<i>Voy a escuchar tu corazón.</i>
<b>I am going to listen to your lungs.</b>	<i>Voy a escuchar tus pulmones.</i>
<b>Breathe through your mouth.</b>	<i>Respira con la boca abierta.</i>
<b>Does it hurt to breathe?</b>	<i>Te duele al respirar?</i>
<b>I am now going to place your IV.</b>	<i>Ahora voy a poner su suero.</i>
<b>Take a deep breath.</b>	<i>Respira hondo.</i>
<b>Now I am going to examine your abdomen.</b>	<i>Ahora voy a revisar su abdomen.</i>

### Regional Anesthesia

<b>Now we're going to place an epidural/spinal/block.</b>	<i>Ahora vamos a poner su epidural/spinal/anestesia regional.</i>
<b>Please sit up straight.</b>	<i>Por favor, siéntese derecho.</i>
<b>Please slouch.</b>	<i>Por favor encogerse.</i>
<b>Relax your shoulders.</b>	<i>Encogerse los hombros.</i>
<b>Chin to your chest.</b>	<i>Barbilla a tu pecho.</i>
<b>Push your back out toward me.</b>	<i>Empuje tu espalda hacia mí.</i>
<b>Numbing medication going in.</b>	<i>La medicina está entrando.</i>
<b>You will feel pressure.</b>	<i>Vas a sentir presión.</i>
<b>Any pain on the right or the left?</b>	<i>Tienes algún dolor en la derecha o izquierda?</i>
<b>Do you have ringing in your ears?</b>	<i>Le pitan los oídos?</i>
<b>I'm going to do a test. Are you numb here?</b>	<i>Vamos hacer una prueba. ¿Estas entumecido/paralizado aquí?</i>
<b>Your legs are getting numb?</b>	<i>Tus piernas están entumecido/dormido?</i>
<b>Does your arm/leg feel numb?</b>	<i>El brazo/pierna te siente entumecido/dormido?</i>
<b>Feel weak?</b>	<i>Sin fuerza?</i>
<b>Any metallic tastes in your mouth?</b>	<i>Tienes algún sabor metálico en la boca?</i>

**Induction**

<b>Can you move onto the operating room table?</b>	<i>Te puedes mover a la cama de operacion?</i>
<b>Don't worry.</b>	<i>No se preocupe.</i>
<b>Take a deep breath.</b>	<i>Respire profundo.</i>
<b>It's just oxygen.</b>	<i>Es puro oxigeno.</i>
<b>You might feel some burning in your IV.</b>	<i>Puedes sentir un poco de quemadura en el suero.</i>
<b>You might start to feel a little sleepy.</b>	<i>Puedes empezar de sentir un poco de sueño.</i>
<b>You're going to feel some pressure on your neck.</b>	<i>Vas a sentir un poco de presion en en cuello.</i>
<b>Everything is OK.</b>	<i>Todo esta bien.</i>
<b>You are going to go to sleep now.</b>	<i>Te vas a dormir ahora.</i>

**Emergence**

<b>Open your eyes.</b>	<i>Abre los ojos.</i>
<b>Take a deep breath.</b>	<i>Respire profundo.</i>
<b>Do you have (phlegm/ sputum)?</b>	<i>Tienes flema/esputo?</i>
<b>Do you have to vomit?</b>	<i>Tienes que vomitar?</i>
<b>Turn your head to the right.</b>	<i>Mueve la cabeza a la derecha.</i>
<b>Can you lift your head up?</b>	<i>Puedes levantar tu cabeza?</i>
<b>Do your arms feel weak?</b>	<i>Los brazos te siente debil?</i>
<b>Squeeze my left hand.</b>	<i>Apriete mi mano izquierda.</i>
<b>Wiggle your toes.</b>	<i>Mueve los dedos del pie.</i>
<b>Do you have pain?</b>	<i>Tienes dolor?</i>
<b>Can you hear me?</b>	<i>Me puedes oir?</i>
<b>Can you talk?</b>	<i>Puedes hablar?</i>
<b>Can you breathe?</b>	<i>Puedes respirar?</i>
<b>Please don't move.</b>	<i>Por favor no se mueve.</i>
<b>Try to calm down.</b>	<i>Trate de calmarse.</i>
<b>I am going to give you some oxygen.</b>	<i>Te voy a dar un poquito de oxigeno.</i>
<b>This is just an oxygen mask.</b>	<i>Esto es una mascara de oxigeno.</i>
<b>We will put you on a stretcher.</b>	<i>Lo vamos a poner en una camilla.</i>
<b>You will be fine.</b>	<i>Vas estar bien.</i>
<b>Smile.</b>	<i>Sonrie.</i>
<b>Squeeze my right hand.</b>	<i>Apriete mi mano derecha.</i>

**Simple Commands**

<b>Move!</b>	<i>Muevase!</i>
<b>Breathe!</b>	<i>Respire!</i>
<b>Speak!</b>	<i>Hable!</i>
<b>Open!</b>	<i>Abra!</i>
<b>Turn!</b>	<i>Voltee!</i>
<b>Be still!</b>	<i>Quieto!</i>
<b>Bend!</b>	<i>Doble!</i>
<b>Listen!</b>	<i>Oiga!</i>

# NORMAL LABORATORY VALUES

MARK A. HOEFT

## Hematology

Complete blood count (CBC)	
White blood cells (WBC)	4.5–11.0 × 10 <sup>3</sup> /mm <sup>3</sup>
Red blood cells (RBC)	
Adult males	4.50–5.90 × 10 <sup>6</sup> /mm <sup>3</sup>
Adult females	4.00–5.20 × 10 <sup>6</sup> /mm <sup>3</sup>
Hemoglobin (HGB)	
Adult males	13.5–17.5 g/dL
Adult females	12.0–16.0 g/dL
Hematocrit (HCT)	
Adult males	41.0%–53.0%
Adult females	36.0%–46.0%
Mean corpuscular volume (MCV)	
Male (adult)	78–98 m <sup>3</sup>
Female (adult)	78–102 m <sup>3</sup>
Mean corpuscular hemoglobin (MCH)	26.0–34.0 pg/cell
Mean corpuscular hemoglobin concentration (MCHC)	31.0–37.0 g/dL
Platelet count	150–350 × 10 <sup>3</sup> /mm <sup>3</sup>
Differential count, peripheral	
Neutrophils	40–70%
Bands	0–10%
Lymphocytes	22–44%
Monocytes	4–11%
Eosinophils	0–8%
Basophils	0–3%
Erythrocyte sedimentation rate (ESR)	
Females	1–25 mm/hr
Males	0–17 mm/hr
Red cell distribution width	11.5–14.5%
Reticulocyte count	0.5–2.5% red cells
Sodium, urine	Diet Dependent
Urinalysis	
pH	5.0–9.0
Specific gravity	1.001–1.035
Chemical screens	Negative

## Coagulation

Activated clotting time (ACT)	70–180 sec
Bleeding time	2–9.5 min
D-Dimer	<500 ng/mL
Fibrinogen	150–400 mg/dL
Fibrinogen degradation products	<2.5 g/mL
INR	0.8–1.2
Partial thromboplastin time, activated (aPTT)	22.1–35.1 sec
Prothrombin time (PT)	10.3–13.2 sec
Thrombin time	16–24 sec

## Chemistry

Alanine (ALT, SGPT)	
Female	7–30 U/L
Male	10–55 U/L
Albumin	3.3–5.0 g/dL
Alkaline phosphatase	
Female	30–100 U/L
Male	45–115 U/L
Amylase	3–100 U/L
Anion gap	3–15 mmol/L
Aspartate (AST, SGOT)	
Female	9–32 U/L
Male	10–40 U/L
Bilirubin	
Total	0.0–1.0 mg/dL
Direct	0.0–0.4 mg/dL

Blood urea nitrogen (BUN)	8–25 mg/dL
Calcium, ionized	1.14–1.30 mmol/L
Calcium, serum	8.5–10.5 mg/dL
Carbon dioxide	23–31.9 mEq/L
Chloride	100–108 mEq/L
Creatinine, serum	0.6–1.5 mg/dL
Fractional excreted sodium (FENa)	<1%
Gamma glutamyltransferase (GGT)	
Female	5–36 U/L
Male	8–61 U/L
Glomerular filtration rate, estimate (eGFR) by age	
20–29	116 mL/min/1.73 m <sup>2</sup>
30–39	107 mL/min/1.73 m <sup>2</sup>
40–49	99 mL/min/1.73 m <sup>2</sup>
50–59	93 mL/min/1.73 m <sup>2</sup>
60–69	85 mL/min/1.73 m <sup>2</sup>
70+	75 mL/min/1.73 m <sup>2</sup>
Glucose, blood (fasting)	
Normal	70–110 mg/dL
Diabetes mellitus	>125 mg/dL
Glucose, 2 hr postprandial	<120 mg/dL
Iron, serum	
Females	30–160 mcg/dL
Males	45–160 mcg/dL
Iron binding capacity (TIBC)	228–428 mcg/dL
Lactic acid, plasma	0.5–2.2 mmol/L
Lactate dehydrogenase (LDH)	110–210 U/L
Lipase	1.3–6.0 U/dL
Magnesium	1.4–2.0 mg/dL
Methemoglobin	0.4–1.5% of total hemoglobin
Osmolality, serum	280–296 mOsm/kg
Osmolality, urine	Diet-dependent
Phosphorous	2.6–4.5 mg/dL
Potassium	3.4–4.8 mEq/L
Protein, total	6.0–8.3 g/dL
Sodium	135–145 mEq/L
Thyroid stimulating hormone (TSH)	0.5–5.0 uU/mL
Thyroxine, free (T4)	0.9–1.8 ng/mL
Thyroxine, total (T4)	4.5–10.9 mcg/dL
Triiodothyronine, total (T3)	60–181 ng/dL
Uric acid, serum	
Male	3.6–8.5 mg/dL
Female	2.3–6.6 mg/dL

### Cardiac Enzymes

Creatine phosphokinase (CPK, CK)	
Females	40–150 U/L
Males	60–400 U/L
Creatine kinase, isoenzyme	
CK-MB	<7.0 ng/mL
Relative index	≤3.5%
Troponin T	<0.10 ng/mL

### Blood Gas

Arterial blood gas	
pH	7.35–7.45
HCO <sub>3</sub> <sup>-</sup>	23–31.9 mmol/L
PaCO <sub>2</sub>	35–45 mm Hg
PaO <sub>2</sub> (room air)	80–100 mm Hg
Base excess	-2 to +2 mEq/L
Venous blood gas tensions	
pH	7.32–7.42
PmVO <sub>2</sub>	35–50 mm Hg
PmVCO <sub>2</sub>	38–50 mm Hg



Oxygen percent saturation (sea level)

Arterial

95–98%

Venous

60–85%

**Antibiotics**

Gentamicin (therapeutic)

Peak

4–8 mcg/mL

Trough

<2.1 mcg/mL

Vancomycin (therapeutic)

Peak

15–35 mcg/mL

Trough

<10.1 mcg/mL

Reference: *Laboratory Handbook* 2007. Department of Pathology, Massachusetts General Hospital. Accessed March 18, 2008. <<http://mghlabtest.partners.org>>

# APPENDIX A: FORMULAE AND QUICK REFERENCE

## CARDIOLOGY

Hemodynamic Parameters	Normal Value
Mean arterial pressure (MAP) = $\frac{SBP + (DBP \times 2)}{3}$	70–100 mmHg
Heart rate (HR)	60–100 bpm
Right atrial pressure (RA)	≤6 mmHg
Right ventricular (RV)	Systolic 15–30 mmHg Diastolic 1–8 mmHg
Pulmonary artery (PA)	Systolic 15–30 mmHg Mean 9–18 mmHg Diastolic 6–12 mmHg
Pulmonary capillary wedge pressure (PCWP)	≤12 mmHg
Cardiac output (CO)	4–8 L/min
Cardiac index (CI) = $\frac{CO}{BSA}$	2.6–4.2 L/min/m <sup>2</sup>
Stroke volume (SV) = $\frac{CO}{HR}$	60–120 ml/contraction
Stroke volume index (SVI) = $\frac{CI}{HR}$	40–50 ml/contraction/m <sup>2</sup>
Systemic vascular resistance (SVR) = $\frac{MAP - \text{mean RA}}{CO} \times 80$	800–1200 dynes × sec/cm <sup>5</sup>
Pulmonary vascular resistance (PVR) = $\frac{\text{mean PA} - \text{mean PCWP}}{CO} \times 80$	120–250 dynes × sec/cm <sup>5</sup>

("Rule of 6s" for PA catheter-measured pressures: RA ≤6, RV ≤30/6, PA ≤30/12, WP ≤12)  
(1 mmHg = 1.36 cm water or blood)

### Fick Cardiac Output

Oxygen consumption (L/min) = CO (L/min) × arteriovenous (AV) oxygen difference

CO = oxygen consumption / AV oxygen difference

Oxygen consumption must be measured (can estimate w/ 125 ml/min/m<sup>2</sup>, but inaccurate)

AV oxygen difference = Hb (g/dl) × 10 (dl/L) × 1.36 (ml O<sub>2</sub>/g of Hb) × (S<sub>a</sub>O<sub>2</sub> - S<sub>v</sub>O<sub>2</sub>)

S<sub>a</sub>O<sub>2</sub> is measured in any arterial sample (usually 93–98%)

S<sub>v</sub>O<sub>2</sub> (mixed venous O<sub>2</sub>) is measured in RA, RV, or PA (assuming no shunt) (normal ~75%)

$$\therefore \text{Cardiac output (L/min)} = \frac{\text{Oxygen consumption}}{\text{Hb (g/dl)} \times 13.6 \times (S_aO_2 - S_vO_2)}$$

### Shunts

$$Q_p = \frac{\text{Oxygen consumption}}{\text{Pulm. vein } O_2 \text{ sat} - \text{Pulm. artery } O_2 \text{ sat}} \quad (\text{if no R} \rightarrow \text{L shunt, PV } O_2 \text{ sat} \approx S_aO_2)$$

$$Q_s = \frac{\text{Oxygen consumption}}{S_aO_2 - \text{mixed venous } O_2 \text{ sat}} \quad (\text{MVO}_2 \text{ drawn proximal to potential L} \rightarrow \text{R shunt})$$

$$\frac{Q_p}{Q_s} = \frac{S_aO_2 - \text{MV } O_2 \text{ sat}}{\text{PV } O_2 \text{ sat} - \text{PA } O_2 \text{ sat}} \approx \frac{S_aO_2 - \text{MV } O_2 \text{ sat}}{S_aO_2 - \text{PA } O_2 \text{ sat}} \quad (\text{if only L} \rightarrow \text{R and no R} \rightarrow \text{L shunt})$$

## Valve area

$$\text{Gorlin equation: Valve area} = \frac{\text{CO}/(\text{DEP or SEP}) \times \text{HR}}{44.3 \times \text{constant} \times \sqrt{\Delta P}} \quad (\text{constant} = 1 \text{ for AS, } 0.85 \text{ for MS})$$

$$\text{Hakki equation: Valve area} \approx \frac{\text{CO}}{\sqrt{\Delta P}}$$

## Coronary Artery Anatomy

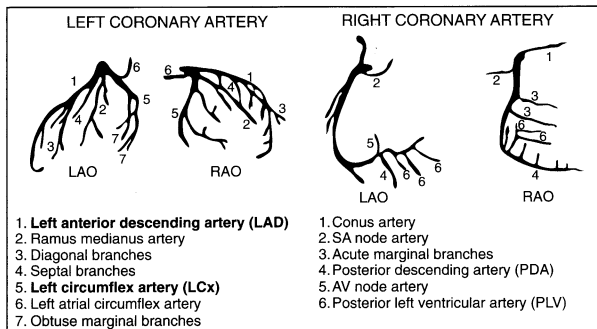


Figure App.-1 Coronary arteries.

(From Grossman WG. *Cardiac Catheterization and Angiography*, 4th ed. Philadelphia: Lea & Febiger, 1991, with permission.)

## PULMONARY

**Dead space** = lung units that are ventilated but not perfused

**Intrapulmonary shunt** = lung units that are perfused but not ventilated

$$\text{Alveolar gas equation: } P_A O_2 = [F_I O_2 \times (760 - 47)] - \frac{P_a CO_2}{R} \quad (\text{where } R = 0.8)$$

$$P_A O_2 = 150 - \frac{P_a CO_2}{0.8} \quad (\text{on room air})$$

**A-a gradient** =  $P_A O_2 - P_a O_2$  [normal A-a gradient  $\approx 4 + (\text{age}/4)$ ]

**Minute ventilation** ( $\dot{V}_E$ ) = tidal volume ( $V_T$ )  $\times$  respiratory rate (RR) (normal 4-6 L/min)

**Tidal volume** ( $V_T$ ) = alveolar space ( $V_A$ ) + dead space ( $V_D$ )

$$\text{Fraction of tidal volume that is dead space} \left( \frac{V_D}{V_T} \right) = \frac{P_a CO_2 - P_{\text{expired}} CO_2}{P_a CO_2}$$

$$P_a CO_2 = k \times \frac{\text{CO}_2 \text{ Production}}{\text{alveolar ventilation}} = k \times \frac{\dot{V}_{CO_2}}{\text{RR} \times V_T \times \left( 1 - \frac{V_D}{V_T} \right)}$$

## NEPHROLOGY

**Anion gap (AG)** =  $\text{Na} - (\text{Cl} + \text{HCO}_3)$  (normal =  $[\text{alb}] \times 2.5$ ; typically  $12 \pm 2$  mEq)

**Delta-delta ( $\Delta\Delta$ )** =  $[\Delta \text{AG (i.e., calc. AG - expected)}] / \Delta \text{HCO}_3$  (i.e.,  $24 - \text{measured HCO}_3$ )

**Urine anion gap (UAG)** =  $(\text{U}_{\text{Na}} + \text{U}_{\text{K}}) - \text{U}_{\text{Cl}}$

**Calculated osmoles** =  $(2 \times \text{Na}) + \left(\frac{\text{glc}}{18}\right) + \left(\frac{\text{BUN}}{2.8}\right) + \left(\frac{\text{EtOH}}{4.6}\right)$

**Osmolal gap (OG)** = measured osmoles - calculated osmoles (normal < 10)

**Estimated creatinine clearance** =  $\frac{[140 - \text{age}(\text{yrs})] \times \text{wt}(\text{kg})}{\text{serum Cr}(\text{mg/dl}) \times 72}$  ( $\times 0.85$  in women)

**Fractional excretion of Na ( $\text{FE}_{\text{Na}}$ , %)** =  $\frac{\left[ \frac{\text{U}_{\text{Na}}(\text{mEq/L})}{\text{P}_{\text{Na}}(\text{mEq/L})} \times 100\% \right]}{\left[ \frac{\text{U}_{\text{Cr}}(\text{mg/ml})}{\text{P}_{\text{Cr}}(\text{mg/dl})} \times 100(\text{ml/dl}) \right]} = \frac{\text{U}_{\text{Na}}}{\text{P}_{\text{Na}}} \times \frac{\text{P}_{\text{Cr}}}{\text{U}_{\text{Cr}}}$

### Corrected Na in hyperglycemia

estimate in all Pts: corrected Na = measured Na +  $\left[ 2.4 \times \frac{(\text{measured glc} - 100)}{100} \right]$

however,  $\Delta$  in Na depends on glc (*Am J Med* 1999;106:399)

$\Delta$  is 1.6 mEq per each 100 mg/dl  $\uparrow$  in glc ranging from 100-440

$\Delta$  is 4 mEq per each 100 mg/dl  $\uparrow$  in glc beyond 440

**Total body water (TBW)** =  $0.60 \times \text{IBW}$  ( $\times 0.85$  if female and  $\times 0.85$  if elderly)

**Free H<sub>2</sub>O deficit** =  $\text{TBW} \times \left( \frac{[\text{Na}]_{\text{serum}} - 140}{140} \right) \approx \left( \frac{[\text{Na}]_{\text{serum}} - 140}{3} \right)$  (in 70 kg Pt)

**Trans-tubular potassium gradient (TTKG)** =  $\frac{\text{U}_{\text{K}}}{\text{P}_{\text{K}}} \times \frac{\text{P}_{\text{Osm}}}{\text{U}_{\text{Osm}}}$

## HEMATOLOGY

Heparin for Thromboembolism	
80 U/kg bolus 18 U/kg/h	
PTT	Adjustment
<40	Bolus 5000 U, $\uparrow$ rate 300 U/h
40-49	Bolus 3000 U, $\uparrow$ rate 200 U/h
50-59	$\uparrow$ Rate 100 U/h
60-85	no $\Delta$
86-95	$\downarrow$ Rate 100 U/h
96-120	Hold 30 min, $\downarrow$ rate 150 U/h
>120	Hold 60 min, $\downarrow$ rate 200 U/h

(Circ 2001;103:2994)

Heparin for ACS	
<b>STEMI w/ fibrinolysis</b>	
60 U/kg bolus (max 4000 U) 12 U/kg/h (max 1000 U/h)	
<b>UA/NSTEMI</b>	
60-75 U/kg bolus (max 5000 U) 12-15 U/kg/h (max 1000 U/h)	
PTT	Adjustment
<40	Bolus 3000 U, $\uparrow$ rate 100 U/h
40-49	$\uparrow$ Rate 50 U/h
50-70	no $\Delta$
71-85	$\downarrow$ Rate 50 U/h
86-100	Hold 30 min, $\downarrow$ rate 100 U/h
101-150	Hold 60 min, $\downarrow$ rate 150 U/h
>150	Hold 60 min, $\downarrow$ rate 300 U/h

(ACC/AHA 2004 Guideline for STEMI)

✓ PTT q6h after every change (half-life of heparin is ~90 min)

✓ PTT qd or bid once PTT is therapeutic

✓ CBC qd (to ensure Hct and plt counts are stable)

**OTHER**

**Ideal body weight (IBW)** = [50 kg (men) or 45.5 kg (women)] + 2.3 kg/inch over 5 feet

$$\text{Body surface area (BSA, m}^2\text{)} = \sqrt{\frac{\text{height (cm)} \times \text{weight (kg)}}{3600}}$$

		Disease	
		present	absent
Test	⊕	a (true ⊕)	b (false ⊕)
	⊖	c (false ⊖)	d (true ⊖)

$$\text{Prevalence} = \frac{\text{all diseased}}{\text{all patients}} = \frac{a + b}{a + b + c + d}$$

$$\text{Sensitivity} = \frac{\text{true positives}}{\text{all diseased}} = \frac{a}{a + c} \quad \text{Specificity} = \frac{\text{true negatives}}{\text{all healthy}} = \frac{d}{b + d}$$

$$\oplus \text{ Predictive value} = \frac{\text{true positives}}{\text{all positives}} = \frac{a}{a + b}$$

$$\ominus \text{ Predictive value} = \frac{\text{true negatives}}{\text{all negatives}} = \frac{d}{c + d}$$

$$\text{Accuracy} = \frac{\text{true positives} + \text{true negatives}}{\text{all patients}} = \frac{a + d}{a + b + c + d}$$

$$\oplus \text{ Likelihood ratio} = \frac{\text{true positive rate}}{\text{false positive rate}} = \frac{Se}{1 - Sp}$$

$$\ominus \text{ Likelihood ratio} = \frac{\text{false negative rate}}{\text{true negative rate}} = \frac{1 - Se}{Sp}$$

$$\text{Odds} = \frac{\text{probability}}{1 - \text{probability}} \quad \text{Probability} = \frac{\text{odds}}{\text{odds} + 1}$$

**Posttest odds** = pretest odds × LR

# APPENDIX B: ANESTHESIA MACHINE CHECKOUT RECOMMENDATIONS

This checkout, or a reasonable equivalent, should be conducted before administration of anesthesia.

These recommendations are valid only for an anesthesia system that conforms to current and relevant standards and includes an ascending bellows ventilator and at least the following monitors: capnograph, pulse oximeter, oxygen analyzer, respiratory volume monitor (spirometer), and breathing system pressure monitor with high- and low-pressure alarms.

- Verify backup ventilation equipment is available and functioning\*
- High-pressure system
  - Check oxygen cylinder supply\*
  - Check central pipeline supplies\*
- Low-pressure system
  - Check initial status of low-pressure system\*
  - Perform leak check of machine low-pressure system\*
  - Turn on machine master switch and all other necessary electrical equipment\*
  - Test flowmeters\*
- Adjust and check scavenging system\*
- Breathing system
  - Calibrate O<sub>2</sub> monitor\*
  - Check initial status of breathing system
  - Perform leak check of breathing system
- Test manual and automatic ventilation systems and unidirectional valves
- Check, calibrate, and/or set alarm limits of all monitors
- Check final status of the machine
  - Vaporizers off\*
  - APL valve open
  - Selector switch to “Bag”
  - All flowmeters to zero
  - Patient suction level adequate
  - Breathing system ready to use

\*Note: If an anesthesia provider uses the same machine in successive cases, the steps denoted by an asterisk need not be repeated or may be abbreviated after the initial checkout.

Adapted From *Clinical Anesthesia Procedures of the Massachusetts General Hospital*, 7th ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

# APPENDIX C: SETTING UP THE OR FOR AN ANESTHESIA CASE

Adapted from "The CA-1 Survival Guide, 2008" Brigham and Women's Hospital

## MNEMONIC: "SOAP M"

### S: Suction

Make sure suction is on, plugged in tightly to the canister, on at full blast

### O: Oxygen

Check that pipeline supply is between 50–55 psi and connected to the wall

Check that the cylinder in the back of the machine has adequate oxygen

(> 1000 psi) in case of emergency

Calibrate O<sub>2</sub> sensor

Make sure an Ambu Bag is available

### A: Airway

Check all laryngoscopes and handles

Choose appropriate ETT and check cuff for leaks. Have an ETT stylette available

Make sure an LMA is available

Oral airway, bite block, and tape

Stethoscope

### P: Pharmacy

Draw up main drugs needed for the case (including a sedative, induction agent, paralytics)

Make sure emergency drugs (epinephrine, atropine, extra succinylcholine) are available

Make sure vaporizers are adequately filled

Make sure appropriate antibiotics are available

Set up appropriate drips and have a functioning drug infusion pump

### M: Machine/Monitors

Checking the machine (see Anesthesia Machine checkout recommendations, Appendix B)

Checking the monitors

Check that the BP cuff is present and of appropriate size

Check that you have a pulse oximeter, EKG cables and electrodes

Check that the nerve stimulator is present and functioning

Fluid warmer and patient warmer devices

Arm straps

A-line, CVP, PA and EEG monitors as indicated

# APPENDIX D: MANAGEMENT OF MALIGNANT HYPERTHERMIA

## EMERGENCY THERAPY FOR MALIGNANT HYPERTHERMIA

MH Hotline 1-800-644-9737

Outside the US: 1-315-464-7079

### Diagnosis vs Associated Problems

#### Signs of MH:

- Increasing  $\text{ETCO}_2$
- Trunk or total body rigidity
- Masseter spasm or trismus
- Tachycardia/tachypnea
- Mixed respiratory and metabolic acidosis
- Increased temperature (may be late sign)
- Myoglobinuria

#### Sudden/Unexpected Cardiac Arrest in Young Patients:

- Presume hyperkalemia and initiate treatment (see #6)
- Measure CK, myoglobin, ABGs, until normalized
- Consider dantrolene
- Usually secondary to occult myopathy (e.g., muscular dystrophy)
- Resuscitation may be difficult and prolonged

#### Trismus or Masseter Spasm with Succinylcholine

- Early sign of MH in many patients
- If limb muscle rigidity, begin treatment with dantrolene.
- For emergent procedures, continue with non-triggering agents, evaluate and monitor the patient, and consider dantrolene treatment.
- Follow CK and urine myoglobin for 36 hours
- Check CK immediately and at 6 hour intervals until returning to normal. Observe for dark or cola-colored urine. If present, liberalize fluid intake and test for myoglobin.
- Observe in PACU or ICU for at least 12 hours.

#### Acute Phase Treatment

##### 1. GET HELP. GET DANTROLENE – Notify Surgeon

- Discontinue volatile agents and succinylcholine.
- Hyperventilate with 100% oxygen at flows of 10 L/min. or more.
- Halt the procedure as soon as possible; if emergent, continue with non-triggering anesthetic technique.
- Don't waste time changing the circle system and  $\text{CO}_2$  absorbant.

##### 2. Dantrolene 2.5 mg/kg IV rapidly, through large-bore IV if possible

To convert kg to lb, for amount of dantrolene, give patients 1 mg/lb (2.5 mg/kg approximates 1 mg/lb).

- Dissolve the 20 mg in each vial with at least 60 ml sterile, preservative-free water for injection. Prewarming (not to exceed  $39^\circ\text{C}$ ) the sterile water may expedite solubilization of dantrolene. However, to date, there is no evidence that such warming improves clinical outcome.
- Repeat until signs of MH are reversed.
- Sometimes more than 10 mg/kg (up to 30 mg/kg) is necessary.
- Each 20 mg bottle has 3 gm mannitol for isotonicity. The pH of the solution is 9.

##### 3. Bicarbonate for metabolic acidosis

- 1–2 mEq/kg if blood gas values are not yet available.

##### 4. Cool the patient with core temperature $>39^\circ\text{C}$ . Lavage open body cavities, stomach, bladder, or rectum. Apply ice to surface. Infuse cold saline intravenously. Stop cooling if temp. $<38^\circ\text{C}$ and falling to prevent drift $<36^\circ\text{C}$ .

##### 5. Dysrhythmias usually respond to treatment of acidosis and hyperkalemia.

- Use standard drug therapy **except calcium channel blockers, which may cause hyperkalemia or cardiac arrest in the presence of dantrolene.**

##### 6. Hyperkalemia—Treat with hyperventilation, bicarbonate, glucose/insulin, calcium.

- Bicarbonate: 1–2 mEq/kg IV.
- Insulin: For **pediatric**, 0.1 units insulin/kg and 1 ml/kg 50% glucose or for **adult**, 10 units regular insulin IV and 50 ml 50% glucose.



- Calcium chloride 10 mg/kg or calcium gluconate 10–50 mg/kg for life-threatening hyperkalemia.
  - Check glucose levels hourly.
- 7. Follow**  $\text{ETCO}_2$ , electrolytes, blood gases, CK, core temperature, urine output and color, coagulation studies. If CK and/or  $\text{K}^+$  rise more than transiently or urine output falls to less than 0.5 ml/kg/hr, induce diuresis to  $>1$  ml/kg/hr and give bicarbonate to alkalinize urine to prevent myoglobinuria-induced renal failure. (See D below.)
- Venous blood gas (e.g., femoral vein) values may document hypermetabolism better than arterial values.
  - Central venous or PA monitoring as needed and record minute ventilation.
  - Place Foley catheter and monitor urine output.

### Post Acute Phase

- A.** Observe the patient in an ICU for at least 24 hours, due to the risk of recrudescence.
- B.** Dantrolene 1 mg/kg q4–6h or 0.25 mg/kg/hr by infusion for at least 24 hours. Further doses may be indicated.
- C.** Follow vitals and labs as above (see #7 above)
- Frequent ABG as per clinical signs
  - CK every 8–12 hours; less often as the values trend downward
- D.** Follow urine myoglobin and institute therapy to prevent myoglobin precipitation in renal tubules and the subsequent development of Acute Renal Failure. CK levels above 10,000 IU/L is a presumptive sign of rhabdomyolysis and myoglobinuria. Follow standard intensive care therapy for acute rhabdomyolysis and myoglobinuria (urine output  $>2$  ml/kg/hr by hydration and diuretics along with alkalinization of urine with Na-bicarbonate infusion with careful attention to both urine and serum pH values).
- E.** Counsel the patient and family regarding MH and further precautions; refer them to MHAUS. Fill out and send in the Adverse Metabolic Reaction to Anesthesia (AMRA) form ([www.mhreg.org](http://www.mhreg.org)) and send a letter to the patient and her/his physician. Refer patient to the nearest Biopsy Center for follow-up.

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Anesthesiology



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