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Current Clinical Strategies

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Handbook of Anesthesiology

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1. **Primary ABCD survey** (basic CPR and defibrillation)
   A. Check responsiveness; activate EMS; call for defibrillator.
   B. Airway: assess and manage the airway with noninvasive devices.
   C. Breathing: assess and manage breathing (look, listen, and feel). If the patient is not breathing, give two slow breaths.
   D. Circulation: assess and manage the circulation; if no pulse, start CPR.
   E. Defibrillation: assess and manage rhythm/defibrillation; shock VF/VT up to 3 times (200 J, 300 J, 360 J, or equivalent biphasic) if necessary.
   F.

2. **Secondary ABCD survey** (advanced assessments and treatments)
   A. Airway: place airway device as soon as possible.
   B. Breathing: assess adequacy of airway device placement and performance; secure airway device; confirm effective oxygenation and ventilation.
   C. Circulation: establish IV access; administer drugs appropriate for rhythm and condition.
   D. Differential diagnosis: search for and treat identified reversible causes.

Ventricular Fibrillation and Pulseless Ventricular Tachy?cardia

3. **Primary ABCD**
4. **Assess rhythm after 3 shocks**; continue CPR for persistent or recurrent VF/VT.
5. **Secondary ABCD**
6. **Epinephrine** 1.0 mg IVP, repeat every 3-5 minutes, or vasopressin 40 units IV, single dose, 1 time only.
7. **Resume attempts to defibrillate,** 360 J, within 30-60 seconds.
8. **Consider antiarrhythmics.**
   A. Amiodarone
   B. Lidocaine 1.5 mg/kg IVP, repeat every 3-5 minutes to a total loading dose of 3 mg/kg; then use.
   C. Magnesium sulfate 1-2 grams IV in Torsades de Pointes or suspected hypomagnesemic state or severe refractory VF.
   D. Procainamide 30 mg/min in refractory ventricular fibrillation (maximum total 17 mg/kg).
9. **Defibrillate** 360 J, 30-60 sec after each dose of medication.
10. Consider bicarbonate 1 mEq/kg (if known preexisting bicarbonate responsive acidosis; overdose with tricyclic antidepressant; if intubated and continued long arrest interval; hypoxic lactic acidosis; hypercarbic acidosis).

Asystole

1. Primary ABCD survey.
2. Confirm asystole in two or more leads. If rhythm is unclear and possible ventricular fibrillation, defibrillate

as for VF.
3. Secondary ABCD survey.
4. Consider possible causes: hypoxia, hyperkalemia, hypokalemia, hypothermia, preexisting acidosis, and drug overdose.
5. Consider transcutaneous cardiac pacing (if considered, perform immediately)
6. Epinephrine 1.0 mg IVP, repeat every 3-5 minutes.
7. Atropine 1.0 mg IV, repeat every 3-5 minutes up to total dose of 0.04 mg/kg.
8. If asystole persists, consider withholding or ceasing resuscitative efforts.
   A. Consider quality of resuscitation if atypical clinical features present, or if support for cease-efforts protocols in place

Pulseless Electrical Activity (PEA)

1. Pulseless electrical activity rhythm on monitor, without detectable pulse.
2. Primary ABC survey.
3. Secondary ABC survey.
4. Consider possible causes: pericardial tamponade, tension pneumothorax, hypovolemia, massive pulmonary embolus, hypoxia, hypothermia, drug overdose (such as tricyclics, digitalis, beta-blockers, calcium channel blockers), hyperkalemia, acidosis, massive acute myocardial infarction.
5. Review for most frequent causes: pulmonary embolism (thrombosis), acidosis, tension pneumothorax, cardiac tamponade, hypovolemia, hypoxia, hyperkalemia, hypokalemia, hypothermia, MI, drug overdose.
6. Epinephrine 1 mg IVP, repeat every 3 to 5 minutes.
7. Atropine 1 mg IVP (if rate less then 60 bpm) repeat every 3 to 5 minutes up to a total does of 0.04 mg/kg.

Bradycardia

1. Slow (absolute bradycardia <60 bpm) or relatively slow (rate less than expected relative to underlying conditions or cause)
2. Primary ABC survey.
3. Secondary ABC survey.
4. If unstable (considered unstable if chest pain, shortness of breath, decreased level of consciousness, hypotension, shock, pulmonary congestion, congested heart failure or acute myocardial infarction are present) interventional sequence:
   A. Atropine 0.5-1.0 mg IVP repeated every 3-5 minutes up to 0.04 mg/kg (denervated transplanted hearts will not respond to atropine, go immediately to TCP, catecholamine infusion or both).
   B. Transvenous cardiac pacing (TCP): if patient is symptomatic, do not delay TCP while awaiting IV access or atropine to take effect.
   C. Dopamine 5-20 mcg/kg/min.
   D. Epinephrine 2-10 mcg/min.
5. If stable and not in type II or type III AV heart block, observe.
6. If type II or type III AV heart block, prepare for transvenous pacer (never treat third-degree heart block plus ventricular escape beats with lidocaine).

Tachycardia Overview

1. **Assess and evaluate patient.** Is patient stable or unstable? Are there serious signs and symptoms due to tachycardia?

A. Consider unstable if chest pain, hypotension, CHF, myocardial infarction, ischemia, decreased level of consciousness, shock, dyspnea or pulmonary congestion are present.

2. **If unstable, prepare for immediate cardioversion.**
   A. If ventricular rate is >150 bpm, may give brief trial of medications based on specific arrhythmias. Immediate cardioversion is generally not needed if heart rate is ,150 bpm.
   B. Have available: oxygen saturation monitor, suction device, IV line, intubation equipment.
   C. Premedicate whenever possible (sedative with or without analgesic agent).
   D. **Synchronized cardioversion**
      1. Cardiovert with 100 J, 200 J, 300 J, 360 J. (PSVT and atrial flutter often respond to lower energy levels; start with 50 J).
      2. If delays in synchronization occur and clinical condition is critical, go immediately to unsynchronized shocks.
      3. May need to resynchronize after each cardioversion.
3. **If stable, treat according to arrhythmia.**
   A. Atrial fibrillation/atrial flutter.
   B. Narrow-complex tachycardias.
   C. Stable wide-complex tachycardia: unknown type.
   D. Stable monomorphic VT and/or polymorphic VT.

Tachycardia: Atrial Fibrillation and Atrial Flutter

1. **Evaluation focus: clinical features**
   A. Patient clinically unstable?
   B. Cardiac function impaired?
   C. Wolf-Parkinson-White (WPW) present?
   D. Duration <48 hours or >48 hours?
2. **Treatment focus: clinical evaluation**
   A. Treat unstable patients urgently.
   B. Control heart rate.
   C. Convert the rhythm.
   D. Provide anticoagulation.
3. **Treatment of atrial fibrillation/atrial flutter**
   A. **Rate Control**
      1. If AF >48 hours duration, use agents to convert rhythm with extreme caution in patients not receiving adequate anticoagulation because of possible embolic complications.
      2. Normal cardiac function: use only one of the following agents: calcium channel blockers or beta-blockers.
      3. Impaired heart (EF<40% or CHF): use only one of the following agents: digoxin, diltiazem, or amiodarone.
      4. WPW with preserved heart function: DC cardioversion or use one of the following primary antiarrhythmic agents: amiodarone, flecainide, procainamide, or sotalol.
      5. WPW with impaired heart (EF <40% or CHF): DC cardioversion or amiodarone.
   B. **Convert rhythm**
      1. Normal cardiac function with duration <48 hours: consider cardioversion or any one of the following agents: amiodarone, ibutilide, flecainide, propafenone, or procainamide.
      2. **Normal cardiac function with duration >48 hours**
         A. No cardioversion.
         B. Use antiarrhythmic agents with extreme caution or
         C. Delayed cardioversion with anticoagulation for 3 weeks or
         D. Early cardioversion (IV heparin, TEE to exclude atrial clot, then cardioversion with 24 hours, then anticoagulation for 4 weeks).
         E.

      3. Impaired heart (EF <40% or CHF) duration <48 hours: consider DC cardioversion or amiodarone.

4. Impaired heart (EF <40% or CHF) duration >48 hours: anticoagulation followed by DC cardioversion.
5. WPW with preserved heart function: DC cardioversion or use one of the following primary antiarrhythmic agents: amiodarone, flecainide, procainamide, or sotalol.
6. WPW with impaired heart (EF <40% or CHF): DC cardioversion or amiodarone.

Narrow-Complex Supraventricular Tachycardia

1. **Attempt therapeutic diagnostic maneuver**
   A. Vagal maneuvers (carotid sinus pressure is contraindicated in patients with carotid bruits; avoid ice water immersion in patients with ischemic heart disease).
   B. Adenosine 6 mg rapid IVP (over 1-3 seconds); may repeat with 12 mg rapid IVP in 1-2 minutes for a total of 30 mg.
2. **Junctional tachycardia**
   A. Preserved heart function: no DC cardioversion, amiodarone, beta-blocker, or calcium channel blocker.
   B. Impaired heart (EF <40% or CHF): no DC cardioversion, amiodarone.
3. **Paroxysmal supraventricular tachycardia (PSVT)**
   A. Preserved heart function: (priority order) calcium channel blocker, beta-blocker, digoxin, DC cardioversion, procainamide, amiodarone, sotalol.
   B. Impaired heart (EF <40% or CHF): (priority order) no DC cardioversion, digoxin, amiodarone, diltiazem.
4. **Ectopic or multifocal atrial tachycardia**
   A. Preserved heart function: no DC cardioversion, calcium channel blocker, beta-blocker, amiodarone.

Stable Ventricular Tachycardia

1. Determine if monomorphic or polymorphic. May go directly to cardioversion.
2. **Monomorphic Ventricular Tachycardia**
   A. Normal cardiac function: procainamide or sotalol; may consider amiodarone or lidocaine.
   B. Poor ejection fraction: amiodarone (150 mg IV over 10 minutes) or lidocaine (0.5 to 0.75 mg/kg IVP), followed by synchronized cardioversion.
3. **Polymorphic Ventricular Tachycardia**
   A. Normal baseline QT interval (normal cardiac function): treat ischemia and/or correct electrolytes; consider one of the following medications: beta-blockers, lidocaine, amiodarone, procainamide, or sotalol.
   B. Normal baseline QT interval (poor ejection fraction): amiodarone (150 mg IV over 10 minutes) or lidocaine (0.5 to 0.75 mg/kg IVP), followed by synchronized cardioversion.
   C. Long baseline QT interval (suggests torsades de pointes): correct abnormal electrolytes; consider one of the following medications: magnesium, overdrive pacing, isoproterenol, phenytoin, or lidocaine.

Pediatric Bradycardia

1. Assess and support ABCs as needed, provide oxygen, attach monitor/defibrillator.
2. If unstable bradycardia (poor perfusion, hypotension, respiratory difficulty, altered consciousness):
   A. Perform chest compression if despite oxygenation and ventilation heart rate <60 bpm in infant or child and poor systemic perfusion.
   B. Epinephrine: 0.01 mg/kg IV/IO (0.1 mg/kg ET); may repeat every 3-5 minutes at the same dose; consider alternate medications: epinephrine or dopamine infusions.

C. Atropine: 0.02 mg/kg (minimum dose 0.1 mg); may repeat once; consider first for bradycardia due to suspected increased vagal tone or primary AV block.
D. Consider cardiac pacing.

3. Identify and treat possible causes: hypoxemia, hypothermia, head injury, heart block, heart transplant, toxins/poisons/drugs.

Pediatric Pulseless Electrical Activity

1. **Assess rhythm**
   A. Ventricular fibrillation/pulseless ventricular tachycardia
      1. Attempt defibrillation: up to 3 times if needed; initially 2 J/kg, then 2-4 J/kg, 4 J/kg.
      2. Epinephrine: 0.01 mg/kg IV/IO (0.1 mg/kg ET); may repeat every 3-5 minutes; consider higher doses for second and subsequent doses.
      3. Attempt defibrillation with 4 J/kg within 30-60 seconds after each medication (pattern: CPR-drug-shock).
      4. **Antiarrhythmic**
         A. Amiodarone: 5 mg/kg bolus IV/IO or
         B. Lidocaine: 1 mg/kg bolus IV/IO or
         C. Magnesium: 25 to 50 mg/kg IV/IO for torsades de pointes or hypomagnesemia (maximum 2 gm).
   B. **Pulseless electrical activity or asystole**
      1. Epinephrine: 0.01 mg/kg IV/IO (0.1 mg/kg ET); may repeat every 3-5 minutes; consider higher doses for second and subsequent doses
      2. **During CPR**
         A. Identify and treat causes: hypoxemia, hypovolemia, hypothermia, hyper/hypokalemia, cardiac tamponade, tension pneumothorax, toxins/poisons/drugs, and thromboembolism.
         B. Consider alternate medications: vasopressors, buffers, antiarrhythmics.

Pediatric Tachycardia with Poor Perfusion

1. **Assess patient**, support ABCs, evaluate EKG and QRS duration.
2. **QRS normal for age (approximately <0.08 seconds).**
   A. Probable sinus tachycardia: history compatible, P waves present/normal, HR often varies with activity, variable RR with constant PR, infant HR usually <220 bpm, children HR usually <180 bpm.
   B. Probable supraventricular tachycardia: history incompatible, P waves absent/abnormal, HR not variable with activity, abrupt rate changes, infant HR usually >220 bpm, children HR usually >180 bpm.
   C. Consider vagal maneuvers.
   D. Immediate cardioversion with 0.5 -1.0 J/kg (may increase to 2 J/kg) or
   E. Immediate adenosine (if IV access available): 0.1 mg/kg IV/IO, maximum first dose 6 mg, may double and repeat dose once.
   F. Consider alternative medications
      1. Amiodarone: 5 mg/kg IV over 20-60 minutes or
      2. Procainamide: 15 mg/kg IV over 30-60 minutes.
   G. Identify and treat possible causes: hypoxemia, hypovolemia, hypothermia, hyper/hypokalemia, cardiac tamponade, tension pneumothorax, toxins/poi?sons/drugs, thromboembolism, and pain.
3. **QRS duration wide for age (approximately >0.08 seconds)**
   A. Probable ventricular tachycardia: immediate cardioversion 0.5-1.0 J/kg.
   B. Consider alternative medications
      1. Amiodarone: 5 mg/kg IV over 20-60 minutes or
      2. Procainamide: 15 mg/kg IV over 30-60 minutes or
      3. Lidocaine: 1 mg/kg IV bolus.

Pediatric Tachycardia with Adequate Perfusion

1. **Assess patient,** support ABCs, evaluate EKG and QRS duration.
2. **QRS normal for age (approximately <0.08 seconds).**
   A. Probable sinus tachycardia: history compatible, P waves present/normal, HR often varies with activity, variable RR with constant PR, infant HR usually <220 bpm, children HR usually <180 bpm.
   B. Probable supraventricular tachycardia: history incompatible, P waves absent/abnormal, HR not variable with activity, abrupt rate changes, infant HR usually >220 bpm, children HR usually >180 bpm.
   C. Consider vagal maneuvers.
   D. Establish IV access.
   E. Adenosine: 0.1 mg/kg IV/IO, maximum first dose 6 mg, may double and repeat dose once.
   F. Consult pediatric cardiologist.
   G. Attempt cardioversion: 0.5-1.0 J/kg (may increase to 2 J/kg).
3. **QRS duration wide for age (approximately >0.08 seconds)**
   A. Probable ventricular tachycardia.
   B. Consider: amiodarone: 5 mg/kg IV over 20-60 minutes or procainamide: 15 mg/kg IV over 30-60 minutes or lidocaine: 1 mg/kg IV bolus.
4. Identify and treat possible causes: hypoxemia, hypovolemia, hypothermia, hyper/hypokalemia, cardiac tamponade, tension pneumothorax, toxins/poisons/drugs, thromboembolism, and pain.

Newborn Resuscitation

1. **Normal term neonatal vital signs** (first 12 hours of life)
   A. Heart rate (awake): 100-180 bpm; respiratory rate: 30-60/min; systolic blood pressure: 39-59 mmHg; diastolic blood pressure: 16-36 mmHg.
2. **Ventilation management**
   A. Positive-pressure ventilation should be started for apnea, central cyanosis, and heart rates below 100 beats/min. Ventilate at 40-60 breaths/min (when performed without compression) with an initial pressure of 30-40 cm H_2O to overcome surface tension and open the alveoli. Subsequent breaths should have inspiratory pressures of 15-30 cm H_2O and tidal volumes of 6-8 mL/kg.
   B. Assisted ventilation should be continued until spontaneous respirations are present and the heart rate is greater than 100 beats/min.
3. **Chest compression**
   A. Chest compressions should be started (1) if the heart rate is absent or (2) if the heart rate remains <60 bpm after 30 seconds of adequate assisted ventilation.
   B. The sternum is depressed 0.5-0.75 inch at a rate of 120 beats/min. The compression-ventilation ratio in neonates is 3:1 (intubated) and 5:1 (not intubated).
4. **Vascular Access**
   A. The umbilical vein, the largest and thinnest of the three umbilical vessels, can be cannulated with a 3.5-5.0 F umbilical catheter. The tip of the catheter should be just below skin level and allow free backflow of blood.
5. **Apgar Scores**
   A. The Apgar scoring system enables rapid evaluation of a newborn's condition at specific intervals after birth (1 and 5 minutes of age).
   B. Apgar score at 1 minute correlates best with intrauterine conditions, the 5- and 10-minute Apgar scores correlate best with neonatal outcome.
   C. A normal Apgar score is 8-10. With scores of 5-7 (mildly asphyxiated) supplemental oxygen and stimulation are normally sufficient. Scores of 3-4 (moderately asphyxiated) typically require temporary assisted positive-pressure ventilation with mask and bag. Scores of 0-2 (severely depressed) mandates the immediate initiation of CPR and intubation.

Drugs that may be Given Endotracheally (ALIEN V^2)

1. Atropine, lidocaine, isoproterenol, epinephrine, naloxone, vasopressin, and Valium.
2. Drugs administered via the endotracheal route should be diluted to a volume of 3-5 mL of normal saline or followed by a 3-5 mL normal saline flush, followed by several positive-pressure ventilations.

Score	**0**	**1**	**3**
Heart Rate (BPM)	Absent	<100	>100
Respirations	Absent	Slow, regular	Good, crying
Muscle Tone	Limp	Flexion of extremities	Active motion
Reflex Irritability	No response	Grimace	Cough, sneeze
Color	Blue/pale	Extremities pink	Completely pink

Drug	**Dose**	**Remarks**
Adenosine	0.1 mg/kg	Give rapid IV bolus; max single dose 12 mg
Atropine	0.01-0.02 mg/kg Min dose: 0.1 mg	Max single dose: 0.5 mg in child, 1.0 mg in adolescent
Bretylium	5 mg/kg (may be increased to 10 mg/kg)	Give rapid IV Loading dose
Calcium Chloride 10%	0.2-0.25 mL/kg or 20 mg/kg per dose	Infuse slowly
Dopamine	2-20 mcg/kg/min	Titrate to desired effect
Dobutamine	2-20 mcg/kg/min	Titrate to desired effect
Epinephrine	First dose: IV/IO: 0.01 mg/kg (1:10k) ET: 0.1 mg/kg (1:1000); doses as high as 0.2 mg/kg may be	Epinephrine infusion: 0.05-1.0 mcg/kg/min titrate to desired effect Bolus: 10-20 mcg/kg IV

	effective Subsequent doses: IV/IO/ET: 0.1 mg/kg	
Glucose (D25%)	0.5-1.0 g/kg IV/IO	Max conc of 25% in peripheral vein
Lidocaine	1 mg/kg per dose	Infuse 20-50 mcg/kg/min
Naloxone	0.1 mg/kg up to 5 years or 20 kg	Children over 5 years or 20 kg may be given 2 mg
Prostaglandin E_1	0.05-0.1 mcg/kg/min	Titrate to desired effect
Sodium Bicarbonate	1 mEq/kg per dose or 0.3 x kg x base deficit	Infuse slowly; monitor for apnea, hypotension, hypoglycemia
Valium	0.1-0.25 mg/kg	
Defibrillation	2-4 J/kg	
Cardioversion	0.25-1.0 /kg	

Preoperative Evaluation

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Anesthesia Preoperative Evaluation

1. The goal of the preoperative evaluation is to identify and correct conditions in order to reduce perioperative morbidity and mortality and alleviate patient anxiety.
2. **Anesthesia preoperative interview**
   A. Note the date and time of the interview, the planned procedure, and a description of any extraordinary circumstances regarding the anesthesia.
   B. Current medications and allergies: history of steroids, chemotherapy and herb and dietary supplements (see tables).
   C. Cigarette, alcohol, and illicit drug history, including most recent use.
   D. Anesthetic history, including specific details of any problems.
   E. Prior surgical procedures and hospitalizations.
   F. Family history, especially anesthetic problems. Birth and development history (pediatric cases below).
   G. Obstetrical history: last menstrual period (females).
   H. Medical history; evaluation, current treatment, and degree of control.
   I. Review of systems, including cardiac, pulmonary, neurologic, liver, kidney, gastric reflux, and/or bleeding tendency.
   J.Exercise tolerance.
   K. History of airway problems (difficult intubation or airway disease, symptoms of temporomandibular joint disease, loose teeth, etc).
   L. Physical exam, including airway evaluation, current vital signs, height and body weight, baseline mental status, evaluation of heart and lungs, vascular access.
   M. Overall impression of the complexity of the patient's medical condition, with assignment of ASA Physical Status Class.
   N. Anesthetic plan (general anesthesia, regional, spinal, MAC). The anesthetic plan is based on the patient's medical status, the planned operation, and the patient's wishes.
   O. Documentation that risks and benefits were explained to the patient.
3. **Preoperative laboratory evaluation**
   A. **Hemoglobin:** menstruating females, children less than 1 year old or with suspected sickle cell disease, history of anemia, blood dyscrasia or malignancy, congenital heart disease, chronic disease states, age greater than 50 years, patients likely to experience large blood loss.
   B. **WBC count:** suspected infection or immunosuppression.
   C. **Platelet count:** history of abnormal bleeding or bruising, liver disease, blood dyscrasias, chemotherapy, hypersplenism.
   D. **Coagulation studies:** history of abnormal bleeding, anticoagulant drug therapy, liver disease, malabsorption, poor nutrition.
   E. **Electrolytes, blood glucose, BUN/creatinine:** patients with hypertension, diabetes, heart disease, or disease states with potential for fluid-electrolyte abnormalities. Blood tests performed within 6 months of surgery that show normal results can be used if there has been no intervening clinical event, age greater than 65 years.
   F. **Liver function tests:** patients with liver disease, history of or exposure to hepatitis, history of alcohol or drug abuse, drug therapy with agents that may affect liver function.
   G. **Pregnancy test:** patients in whom pregnancy cannot be reliably ruled out.
   H. **Electrocardiogram:** men over age 40, women over age 45, history or symptoms of cardiac disease, history of diseases associated with cardiac involvement (hypertension, diabetes, morbid obesity, peripheral vascular disease, collagen vascular disease, cocaine abuse). An EKG showing normal results that was performed within 6 months of surgery can be used if there has been no intervening clinical event.

I. **Chest x-ray:** patients with symptoms of pulmonary disease, airway obstruction, cardiac disease, malignancy, history of heavy smoking, age greater than 60 years. A study showing normal results that was performed within one year of surgery can be used if there has been no intervening clinical event.

J. **Urinalysis:** no indication in preanesthetic evaluation; surgeon may request to rule out infection before certain surgical procedures.

K. **Cervical spine flexion/extension x-rays:** patients with rheumatoid arthritis or Down's syndrome. Routine screening in asymptomatic patients is generally not required.

L. **Preoperative pulmonary function tests (PFTs)**

1. There is no evidence to suggest that pulmonary function tests are useful for purposes of risk assessment or modification in patients with cigarette smoking or adequately treated brochospastic disease.
2. **Candidates for preoperative PFTs**
   - A. Patients considered for pneumonectomy.
   - B. Patients with moderate to severe pulmonary disease scheduled for major abdominal or thoracic surgery.
   - C. Patients with dyspnea at rest.
   - D. Patients with chest wall and spinal deformities.
   - E. Morbidity obese patients.
   - F. Patients with airway obstructive lesions.

4. **Pediatric preoperative evaluation:** see section on pediatric anesthesia.

Airway Evaluation

1. **Preoperative evaluation:** assessed by historical interview (ie, history of difficult intubation, sleep apnea) and physical examination and occasionally through of radiographs, PFTs, and direct fiber-optic examination. The physical exam is the most important method of detecting and anticipating difficulties in airway management.

2. **Physical exam**

A. **Mouth**

1. **Opening:** note symmetry and extent of opening (3 finger breadths optimal).
2. **Dentition:** ascertain the presence of loose, cracked, or missing teeth, dental prostheses, and co-existing dental abnormalities.
3. **Macroglossia:** will increase difficultly of intubation.

B. **Neck**

1. **Anterior mandibular space:** evaluated by asking the supine patient to maximally extend the head and measuring the distance between the hyoid bone and the inside of the mentum or between the notch of the thyroid cartilage to the mentum. An inadequate mandibular space is associated with a hyomental distance of <3 cm or a thyromental distance of <6 cm.
2. **Cervical spine mobility (atlantooccipital joint extension):** 35 degrees of extension are normal at the atlantooccipital joint, decreases in extension are associated with increased difficulty of intubation.
3. Evaluate for presence of a healed or patent tracheostomy stoma, prior surgeries or pathology of the head and neck (laryngeal cancer).

3. **Airway classification**

A. **Mallampati classification** (classification of tongue size vs pharynx)

1. **Class 1:** able to visualize the soft palate, fauces, uvula, anterior and posterior tonsillar pillars.
2. **Class 2:** able to visualize the soft palate, fauces, and uvula. The anterior and posterior tonsillar pillars are hidden by the tongue.
3. **Class 3:** only the soft palate and base of uvula are visible.
4. **Class 4:** only the soft palate can be seen (no uvula seen).

B. **Grades of laryngoscopic view**

1. **Grade 1:** full view of the entire glottic opening.
2. **Grade 2:** posterior portion of the glottic opening is visible.
3. **Grade 3:** only tip epiglottis is visible.

4. **Grade 4:** only soft palate is visible.

4. **Predictors of difficult intubation**

A. **Anatomic variations:** micrognathia, prognathism, large tongue, arched palate, short neck, prominent upper incisors, buckteeth, decreased jaw movement, receding mandible or anterior larynx, short stout neck.

B. **Medical conditions associated with difficult intubations**

1. **Arthritis:** patients with arthritis may have a decreased range of neck mobility. Rheumatoid arthritis patients have an increased risk of atlantoaxial subluxation.
2. **Tumors:** may obstruct the airway or cause extrinsic compression and tracheal deviation.
3. **Infections:** of any oral structure may obstruct the airway.
4. **Trauma:** patients are at increased risk for cervical spine injuries, basilar skull fractures, intracranial injuries, and facial bone fractures.
5. **Down's Syndrome:** patients may have macroglossia, a narrowed cricoid cartilage, and a greater frequency of postoperative airway obstruction/croup; risk of subluxation of the atlanto-occipital joint.
6. **Scleroderma:** may result in decreased range of motion of the temporomandibular joint and narrowing of the oral aperture.
7. **Obesity:** massive amount of soft tissue about the head and upper trunk can impair mandibular and cervical mobility, increased incidence of sleep apnea.

ASA Physical Status Classification

1. The ASA (American Society of Anesthesiologists) physical status classification has been shown to generally correlate with the perioperative mortality rate (mortality rates given below).
2. **ASA 1:** a normal healthy patient (0.06-0.08%).
3. **ASA 2:** a patient with a mild systemic disease (mild diabetes, controlled hypertension, obesity [0.27-0.4%]).
4. **ASA 3:** a patient with a severe systemic disease that limits activity (angina, COPD, prior myocardial infarction [1.8-4.3%]).
5. **ASA 4:** a patient with an incapacitating disease that is a constant threat to life (CHF, renal failure [7.8-23%]).
6. **ASA 5:** a moribund patient not expected to survive 24 hours (ruptured aneurysm [9.4-51%]).
7. **ASA 6:** brain-dead patient whose organs are being harvested.
8. **For emergent operations**, add the letter 'E' after the classification.

Preoperative Fasting Guidelines

1. **Recommendations** (applies to all ages)

Ingested Material	Minimum Fasting Period (hrs)
Clear liquids	2
Breast milk	4
Infant formula	6
Non-human milk	6
Light solid foods	6

1. Recommendations apply to healthy patients exclusive of parturients undergoing elective surgery; following these recommendations does not guarantee gastric emptying has occurred.
2. Clear liquids include water, sugar-water, apple juice, non-carbonated soda, pulp-free juices, clear tea, black coffee.
3. Medications can be taken with up to 150 mL of water in the hour preceding induction of anesthesia.

Bacterial Endocarditis Prophylaxis

1. **Antibiotic prophylaxis** is recommended for patients with prosthetic cardiac valves, previous history of endocarditis, most congenital malformations, rheumatic valvular disease, hypertrophic cardiomyopathy, and mitral valve regurgitation.
2. **Prophylactic regimens for dental, oral, respiratory tract, or esophageal procedures**
   A. **Standard regimen**
      1. Adults: amoxicillin 2 g PO 1 hour before procedure.
      2. Children: amoxicillin 50 mg/kg PO 1 hour before procedure.
   B. **Unable to take oral medications**
      1. Adults: ampicillin 2 g IM/IV within 30 min before procedure.
      2. Children: ampicillin 50 mg/kg IM/IV within 30 min before procedure.
   C. **Penicillin allergic**
      1. Adults: clindamycin 600 mg or cephalexin (or cefadroxil) 2 g or azithromycin (or clarithromycin) 500 mg 1 hour before procedure.
      2. Children: clindamycin 20 mg/kg PO or cephalexin (or cefadroxil) 50 mg/kg or azithromycin (or clarithromycin) 15 mg/kg 1 hour before procedure
   D. **Allergic penicillin and unable to take oral medications**
      1. Adults: clindamycin 600 mg IV or cefazolin 1 g within 30 min before procedure.
      2. Children: clindamycin 20 mg/kg IV or cefazolin 25 mg/kg IM/IV within 30 min before procedure.
3. **Prophylactic regimens for genitourinary/gastrointestinal (excluding esophageal) procedures**
   A. **High-risk patients**
      1. Adults: ampicillin 2 g IM/IV plus gentamicin 1.5 mg/kg (not to exceed 120 mg) within 30 min before procedure; 6 hours later ampicillin 1 g IM/IV or amoxicillin 1 g PO.
      2. Children: ampicillin 50 mg/kg IM/IV (not to exceed 2 g) plus gentamicin 1.5 mg/kg within 30 min before procedure; 6 hours later, ampicillin 25 mg/kg IM/IV or amoxicillin 25 mg/kg PO.
   B. **High-risk patients allergic to ampicillin/amoxicillin**
      1. Adults: vancomycin 1 g IV over 1-2 h plus gentamicin 1.5 mg/kg IM/IV (not to exceed 120 mg); complete within 30 min before procedure.
      2. Children: vancomycin 20 mg/kg IV over 1-2 h plus gentamicin 1.5 mg/kg IM/IV; complete within 30 min before procedure.
   C. **Moderate-risk patients**
      1. Adults: amoxicillin 2 g PO 1 hour before procedure, or ampicillin 2 g IM/IV within 30 min before procedure.
      2. Children: amoxicillin 50 mg/kg PO 1 hour before procedure, or ampicillin 50 mg/kg 30 min before procedure.
   D. **Moderate-risk patients allergic to ampicillin/amoxicillin**
      1. Adults: vancomycin 1 g IV over 1-2 hours; complete infusion within 30 min before starting procedure.
      2. Children: vancomycin 20 mg/kg IV over 1-2 hours; complete 30 min before starting procedure.
4. **Miscellaneous notes**
   A. Total dose for children should not exceed the adult dose.
   B. Cephalosporins should not be used in individuals with immediate-type hypersensitivity reaction (urticaria, angioedema, or anaphylaxis) to penicillin.
   C. Patients already taking antibiotics for another reason should be given an agent from a different class for endocarditis prophylaxis.
   D. Patients at risk for endocarditis who undergo open heart surgery should have prophylaxis directed primarily at staphylococci.
   E. Cardiac transplant recipients should probably be considered at moderate risk for endocarditis and receive prophylaxis accordingly.

Premedications

1. The goals of premedications include: anxiety relief, sedation, analgesia, amnesia, antisialagogue effect, increase in gastric fluid pH, decrease in gastric fluid volume, attenuation of sympathetic nervous system reflex responses, decrease in anesthetic requirements, and prophylaxis against allergic reactions.
2. Sedatives and analgesics should be reduced or withheld in the elderly, newborn (<1 year of age), debilitated, and acutely intoxicated, as well as those with upper airway obstruction or trauma, central apnea, neurologic deterioration, or severe pulmonary or valvular heart disease.

Classification	**Drug**	**Adult (mg)**	**Peds (mg/kg)**	**Route**
Barbiturates	Pentobarbital Secobarbital Methohexital Methohexital	50-150	2 2 8 25-30	IM IM IM PR
Non-Barbiturates	Ketamine Ketamine Ketamine Ketamine		0.5-2 2-10 5-6 3-6	IV IM Intranasal PO
Opioids	Fentanyl Fentanyl Sufentanil Morphine Meperidine	25-100 5-15 25-100	0.01-0.02 0.015-0.02 0.0015-0.003 0.05-0.02 1-1.5	IM, IV OFTC Intranasal IM IM
Benzodiazepines	Diazepam Midazolam Midazolam Midazolam Midazolam Midazolam Lorazepam	5-10 1-5 2.5-5 0.2 1-4 15-30 0.125-0.25	0.1-0.3 0.05 0.1-0.2 0.1-0.2 0.4-1.0 0.25-0.35	PO IV IM Intranasal PO SL PR PO, IV PO PO

	Flurazepam Triazolam			
Non-Benzo	Chloral Hydrate		30-50	PO, PR
Antihistamines	Benadryl Phenergan Vistaril	25-75 25-50 50-100	0.5 0.5-1.0	PO, IM IM IM
Alpha-2 Agonist	Clonidine	0.3-0.4	0.004	PO
Antiemetics	Perphenazine Droperidol Ondansetron	5 0.625-1.254	0.05-0.075 0.1	IV IV IV
Anticholinergic	Atropine Scopolamine Glycopyrrolate	0.3-0.6 0.3-0.6 0.2-0.3	0.01-0.02 0.01-0.02 0.01	IM, IV IM, IV IM, IV
H_2 Blockers	Cimetidine Ranitidine Ranitidine	200-400 150-300 50	7.5 1.5	PO, IM, IV PO IV
Antacids	Bicitra	15-60 mL	0.4 mL/kg	PO
Gastric Stim	Metoclopramide	10-20	0.25	PO, IV, IM
OFTC: oral transmucosal fentanyl citrate (lollipop)				

Herb	**Adverse effects**	**Anesthetic considerations**
Echinacea	Unpleasant taste sensation, tachyphylaxis,	May potentiate barbiturate toxicity

	potential hepatotoxicity	
Garlic	Halitosis, prolongation of bleeding time, hypotension	Increased risk of intraoperative hemodynamic instability
Ginger	Prolongation of bleeding time	Increased risk of intraoperative hemodynamic instability
Ginkgo Biloba	Platelet dysfunction	Increased intraoperative/postoperative bleeding tendencies; may decrease effectiveness of IV barbiturates
St John''s Wort	Dry mouth, dizziness, constipation, nausea	Pseudoephedrine, MAOIs, SSRIs should be avoided
Ginseng	Hypertension, insomnia, headache, vomiting, epistaxis, prolonged bleeding time, hypoglycemia	Increased risk of intraoperative hemodynamic instability
Kava Kava	Characteristic ichthyosiform dermopathy	May potentiate effect of barbiturates/benzodiazepines, thereby causing excessive sedation
Feverfew	Aphthous ulcers, gastrointestinal irritability, headache	Increased risk of intraoperative hemodynamic instability
Ephedra (Ma Huang)	Hypertension, tachycardia, cardiomyopathy, cerebrovascular accident, cardiac arrhythmias	May interact with volatile anesthetic agents (eg, halothane) to cause fatal cardiac dysrhythmias; profound intraoperative hypotension, which can be controlled with phenylephrine, not pseudoephedrine

Agent	Effects
Cyclophosphamide	Myelosuppression, hemorrhagic cystitis, water retention, pulmonary fibrosis, plasma cholinesterase inhibition
Nitrogen Mustard	Myelosuppression, local tissue damage
Vincristine	Neurotoxicity, dilutional hyponatremia
Vinblastine	Myelosuppression
Methotrexate	Renal tubular injury
5-Fluorouracil/ARA C	Hemorrhage enteritis, diarrhea, myelosuppression
Adriamycin	Cardiac toxicity; risk factors include total cumulative dose over 550 mg/m^2, concomitant cyclophosphamide therapy, prior history of heart disease, age over 65 years
Bleomycin	Pulmonary toxicity; risk factors include total cumulative dose over 200 mg, concomitant thoracic radiation therapy, age over 65 years.
Mitomycin C	pulmonary toxicity
Cisplatin	renal toxicity, neurotoxicity
Nitrosoureas	myelosuppression, renal pulmonary toxicity
Taxol	hypersensitivity reaction, myelosuppression, cardiac toxicity, peripheral neuropathy
Growth factors	pulmonary edema, pericardial and pleural effusions

Cardiovascular Disease

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Cardiac Disease and Anesthesia

1. **Pertinent cardiac history**
   A. **Cardiac reserve:** limited exercise tolerance in the absence of significant pulmonary disease is the most striking evidence of decreased cardiac reserve. If a patient can climb several flights of stairs without symptoms, cardiac reserve is probably adequate.
   B. **Angina pectoris:** an increase in heart rate is more likely than hypertension to produce signs of myocardial ischemia.
   C. **Previous myocardial infarction:** the incidence of myocardial reinfarction in the perioperative period is related to the time elapsed since the previous myocardial infarction. The incidence of perioperative myocardial reinfarction generally stabilizes at 5-6% after 6 months from the previous myocardial infarction. Mortality after perioperative MI 20-50%. Infarction rate in the absence of a prior MI 0.13%. Most perioperative myocardial reinfarctions occur in the first 48 to 72 hours postoperatively.
   D. **Dysrhythmias:** ventricular dysrhythmia may indicate underlying cardiac disease. Isolated premature ventricular contractions without evidence of underlying cardiac disease are not associated with increased cardiac risk.
   E. **Prior cardiac surgery or PTCA** does not increase perioperative risk.
2. **Contraindications** to elective noncardiac surgery include a myocardial infarction less than 1 month prior to surgery, uncompensated heart failure, and severe aortic or mitral stenosis.
3. **Evaluation of the cardiac patient for noncardiac surgery**
   A. **Risk factors**
      1. **Major risk factors:** unstable coronary syndromes (recent MI, unstable or severe angina, decompensated CHF, significant arrhythmias, severe valvular disease).
      2. **Intermediate risk factors:** mild angina pectoris, prior MI, compensated or prior CHF, diabetes mellitus.
      3. **Minor risk factors:** advanced age, abnormal EKG, arrhythmias, low functional capacity, history of stroke, uncontrolled systemic hypertension.
      4. **Functional capacity:** perioperative cardiac risk is increased in patients unable to meet a 4-MET (climbing a flight of stairs) demand during most normal daily activities.
      5. **Surgery-specific risk:** high risk surgery includes major emergency surgery, aortic or other major vascular surgery, peripheral vascular surgery, long procedures with large fluid shifts or blood loss; intermediate risk surgery includes CEA, head and neck, intraperitoneal, intrathoracic, orthopedic and prostate surgery; low risk surgery includes endoscopic and superficial procedures, cataract and breast surgery.
   B. **Algorithm for preoperative cardiac evaluation**
      1. Step 1: what is the urgency of noncardiac surgery? Certain emergencies do not allow time for preoperative cardiac evaluation.
      2. Step 2: has the patient undergone coronary revascularization in the past 5 years? If so, and if clinical status has remained stable without recurrent symptoms/signs of ischemia, further cardiac testing is generally not necessary.
      3. Step 3: has the patient had a coronary evaluation in the past 2 years? If coronary risk was adequately assessed and the findings were favorable, it is usually not necessary to repeat testing unless the patient has experienced a change or new symptoms of coronary ischemia since the previous evaluation.
      4. Step 4: does the patient have an unstable coronary syndrome or a major risk factors? Elective surgery should be cancelled or delayed until the problem has been identified, evaluated and treated.
      5. Step 5: does the patient have intermediate risk factors? If no, consider functional capacity and level of surgery specific risk to identifying patients most likely to benefit from further noninvasive testing.

6. Step 6: patients with intermediate predictors of clinical risk and moderate or excellent functional capacity can generally undergo intermediate-risk surgery. Further noninvasive testing should be considered for patients with poor functional capacity or moderate functional capacity undergoing high risk surgery.
7. Step 7: patients with minor or no risk factors and moderate or excellent functional capacity are generally safe and don't require further testing. Patients without clinical markers but poor functional capacity who are facing high-risk operations, particularly those with several minor clinical predictors of risk who are to undergo vascular surgery, should be consider for further testing.
8. Step 8: the results of noninvasive testing should be used to determine further preoperative management. In some patients corrective cardiac surgery may be consider before the proposed noncardiac surgery.

C. **Cardiac evaluation studies**

1. **Baseline EKG** is normal in 25-50% of patients with coronary artery disease but no prior myocardial infarction. EKG evidence of ischemia often becomes apparent only during chest pain.
2. **Exercise stress testing:** gives estimate of functional capacity along with the ability to detect EKG changes and hemodynamic response. Highly predictive when ST-segment changes are characteristic of ischemia.
3. **Echocardiography:** evaluates global and regional ventricular function, valvular function, and congenital abnormalities. Detects regional wall motion abnormalities and derives left ventricular ejection fraction.
4. **Dobutamine stress echo:** reliable predictor of adverse cardiac complications.
5. **Technetium-99m:** extremely sensitive and specific for acute MI and for evaluating cardiac function.
6. **Coronary angiography:** gold standard for evaluating cardiac disease. The single most important measurement is the ejection fraction.

4. **Anesthetic management of the cardiac patient for noncardiac surgery**

A. **The overall goal** is to maintain a favorable balance between myocardial oxygen requirements and myocardial oxygen delivery. Maintenance of this balance (by avoiding tachycardia, systemic hypertension, hypoxemia, diastolic hypotension, acidosis) is more important then the specific technique.

B. A common recommendation is to maintain heart rate and systemic blood pressure within 20% of awake values. However, almost 50% of all new ischemic events are not preceded by or associated with significant changes in HR or BP.

C. **Perioperative pain management:** effective pain control leads to a reduction in postoperative catecholamine surges and hypercoagulability.

D. **Intraoperative nitroglycerin:** insufficient data exists to routinely recommend prophylactic IV nitroglycerin in high risk patients.

E. **Premedications** help reduce fear, anxiety and pain, and help prevent sympathetic activation. Continue preoperative cardiac medications up until the time of surgery. Supplemental oxygen should be given to all patients with significant ischemia or who are given sedation.

F. **Monitors** (in addition to standard ASA monitors)

1. **Pulmonary artery catheters:** patients most likely to benefit appear to be those with a recent MI complicated by CHF, those with significant CAD who are undergoing procedures associated with significant hemodynamic stress, and those with systolic or diastolic left ventricular dysfunction, cardiomyopathy, or valvular disease undergoing high-risk operations.
2. **ST-segment monitoring:** use of computerized ST-segment analysis in patients at high risk may improve sensitivity for myocardial ischemia detection. Myocardial ischemia occurs by at least a 1-mm downsloping of the ST segment from baseline. Usually, lead II is monitored for inferior wall ischemia and arrhythmias and V_5 for anterior wall ischemia.
3. **Transesophageal echocardiogram:** ventricular wall motion abnormalities observed by TEE may be the most sensitive indicator of myocardial ischemia but are not practical for routine use and should be reserved for selected high risk patients.

Anesthesia Following Heart Transplantation

1. **Physiology of cardiac transplantation**
   A. The transplanted heart is totally denervated and direct autonomic influences are absent. Resting heart rate, in the absence of vagal influences, is increased (100-120 beats/min).
   B. Ventricular function slightly reduced; cardiac output increases owing to increased venous return ('preload dependent').
   C. Increases in heart rate correspond to catecholamine secretion.
   D. Coronary atherosclerosis accelerated; silent ischemia likely.
   E. Drugs that act indirectly via the autonomic system are ineffective.
   F. Beta-adrenergic receptors remain intact.
2. **Preoperative evaluation**
   A. Evaluation should focus on functional status (activity level) and detecting complications of immunosuppression.
   B. Underlying cardiac disease may be asymptomatic (due to denervation).
   C. Baseline EKG may show both donor and native P waves and a right bundle branch block.
3. **Anesthetic considerations**
   A. Maintain preload.
   B. Sudden vasodilation should be avoided because reflex increases in heart rate are absent. Indirect vasopressors are less effective than direct acting agents because of the absence of catecholamine stores in myocardial neurons.
   C. Atropine and pancuronium will not increases heart rate.

Hypertension

1. **Preoperative evaluation**
   A. History should assess the severity and duration of the hypertension, drug therapy, and the presence of hypertensive complications.
   B. Surgical procedures on patients with sustained preoperative diastolic blood pressures higher than 110 mmHg or with evidence of end-organ damage should be delayed until blood pressure is controlled.
   C. Premedications reduce preoperative anxiety. Cardiac and hypertensive medications should be continued up until the time of surgery.
2. **Anesthetic considerations**
   A. Many patients with hypertension display an accentuated hypotensive response to induction (unmasking of decreased intravascular volume) followed by an exaggerated hypertensive response to intubation.
   B. Techniques used to attenuate the hypertensive response include deepening anesthesia with a volatile agent, giving a bolus of narcotic or lidocaine, pretreatment with a beta blocker or an alpha-2 agonist. In addition, the administration of laryngotracheal lidocaine before placement of the ETT will minimize any pressor response.
   C. Postoperative anticipate excessive increases in systemic blood pressure.

Valvular Heart Disease

1. **Mitral stenosis**
   A. Normal mitral valve are is 4-6 cm^2. Symptoms occur when valve area is reduced to 2.5 cm^2 and become severe with valve area below 1 cm2.
   B. **Complications:** atrial fibrillation (secondary to increased pressure and distention of the LA), pulmonary edema, pulmonary hypertension, right ventricular hypertrophy, and RV failure.
   C. **Anesthetic management**
      1. Give prophylactic antibiotics for prevention of endocarditis.

2. Avoid sinus tachycardia or rapid ventricular response rate during AF.
3. Avoid marked increases in central blood volume associated with over-transfusion and Trendelenburg position.
4. Avoid large decreases in SVR.
5. Avoid hypoxemia or hypovolemia that may exacerbate pulmonary hypertension and evoke RV failure (may consider avoiding N_2O).

2. **Mitral regurgitation**

A. Characterized by left atrial volume overload and decreased left ventricular forward stroke volume. Produces large 'v' waves. Usually due to rheumatic fever and is associated with mitral stenosis; isolated acute MR often reflects papillary muscle dysfunction.

B. **Complications:** LVH, LV failure, LA dilatation, atrial fib, pulmonary edema.

C. **Anesthetic management**

1. Give prophylactic antibiotics for prevention of endocarditis.
2. Avoid sudden decreases in heart rate or increases in SVR (mild decreases in SVR can improve cardiac output as can mild tachycardia).
   A. Avoid hypovolemia.
   B. Monitor the magnitude to the 'v' wave as a reflection of MR flow.
   C. Be aware of the risk of systemic emboli.

3. **Aortic stenosis**

A. **Normal aortic valve area** is 2.5-3.5 cm^2. Severe AS occurs with areas <1 cm^2 and transvalvular pressure gradient greater than 50 mmHg.

B. **Symptoms:** dyspnea, angina, syncope, decreased exercise tolerance, sudden death.

C. **Anesthetic management**

1. Give prophylactic antibiotics for prevention of endocarditis.
2. Maintain normal sinus rhythm. Avoid bradycardia. Always have a cardiac defibrillator available.
3. Avoid sudden decrease in SVR.
4. Optimize intravascular fluid volume. Ensure adequate preload.

4. **Aortic regurgitation**

A. Gradual onset results in marked LVH, increased myocardial oxygen requirements, decrease in aortic diastolic pressure and coronary blood flow. Most often due to infective endocarditis.

B. **Symptoms:** angina pectoris, left ventricular failure.

C. **Anesthetic management**

1. Give prophylactic antibiotics for prevention of endocarditis.
2. Avoid bradycardia, increases in SVR, maintain myocardial contractility, avoid myocardial depressants.

5. **Pulmonary stenosis**

A. Considered severe if pressure gradient is >50 mmHg with a normal CO.

B. **Complications:** right atrial and right ventricular hypertrophy, RV failure, syncope, angina, and sudden death.

C. **Anesthetic management**

1. Give prophylactic antibiotics for prevention of endocarditis.
2. Avoid factors which increase right ventricular O_2 requirements such as tachycardia and increased myocardial contractility.
3. Avoid factors decreasing right ventricular O_2 supply (hypotension).

Disease	**Heart Rate**	**Rhythm**	**Preload**	**After?load**	**Contractility**	**Blood Pressure**
Aortic Stenosis	normal, avoid tachy	sinus is essential	increase or maintain	maintain	maintain	maintain

Aortic Regurg?itation	normal or slight increase	sinus	maintain or increase	reduce	maintain or increase	maintain
Mitral Stenosis	normal. avoid tachy	sinus; AF digitalize	maintain or increase	maintain avoid increase	maintain	avoid hypo-tension
Mitral Regurg?itation	normal or increase	usually AF, digitalize	maintain	reduce	maintain or increase	maintain
Ischemic Heart Dz	slow rate	sinus	maintain	reduce	maintain or decrease	normal at rest
IHSS	normal or slight decrease	sinus, consider pacing	maintain or increase	maintain or slight increase	maintain or decrease	maintain

Pulmonary Disease

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Pulmonary Risk Factors

1. **Risk factors** include preexisting pulmonary disease, thoracic or upper abdominal surgery, smoking, obesity, age greater than 60 years, and prolonged general anesthesia.
2. The two strongest predictors of complications are operative site and a history of dyspnea.
3. **Smoking:** cessation of smoking for 24-48 hours before decreases carboxyhemoglobin levels, shifts the oxyhemoglobin dissociation curve to the right, and increases oxygen available to tissue. Cessation for greater than 8-12 weeks is required to reduce the risk of postoperative pulmonary complications and improve ciliary and small airway function.

Asthma

1. **Preoperative**
   A. Asthma is characterized by airway hyperreactivity, manifest by episodic attacks of dyspnea, cough, and wheezing.
   B. Preoperative evaluation should ascertain the severity and control of the asthma, drug therapy, previous steroid use and history of intubation.
   C. PFTs before and after bronchodilation therapy may be indicated in the patient scheduled for thoracic or abdominal surgery.
   D. Patients should be medically optimized prior to surgery. Patients with active bronchospasm presenting for emergency surgery should undergo a period of intensive treatment if possible.
   E. Bronchodilators should be continued up to the time of surgery. Theophylline levels should be checked preoperatively. Patients receiving steroids should be given supplemental doses (hydrocortisone 100 mg).
2. **Intraoperative management**
   A. The goal during induction and maintenance is to depress airway reflexes so as to avoid bronchoconstriction of hyperreactive airways in response to mechanical stimulation.
   B. Reflex bronchospasm can be blunted prior to intubation by increasing the depth of anesthesia with additional induction agent or volatile agent, or by administering IV or IT lidocaine 1-2 mg/kg.
   C. Intraoperative bronchospasm is usually manifest by wheezing, increasing peak airway pressure, decreased exhaled tidal volumes or a slowly rising wave form on the capnograph. Treatment includes deepening the level of anesthesia, and beta agonists delivered by aerosol or metered dose inhalers.
   D. In ventilated patients maintain PaO_2 and PCO_2 in normal levels, ventilate at slow rates (6-10 bpm), lower TV, long exhalation times.
   E. Patients should be extubated either before airway reflexes return or after the patient is fully awake. Lidocaine may help suppress airway reflexes during emergence.

Chronic Obstructive Pulmonary Disease (COPD)

1. **Preoperative**
   A. Evaluation should determine the severity of the disease and elucidate any reversible components such as infection or bronchospasm.
   B. Routine pulmonary function tests are generally not recommended.
   C. A history of frequent exacerbations, steroid dependence, or need for intubation for respiratory support should prompt particular caution in the evaluation and planning for surgery.
2. **Intraoperative management**

A. Choice of anesthetic technique is less important then the realization that these patients are susceptible to acute respiratory failure in the postoperative period. If general anesthesia is required, airway manipulation should be avoided to decrease bronchospasm.

Endocrinology

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Diabetes Mellitus

1. **Complications:** hyperglycemia, hypoglycemia, ketoacidosis, autonomic neuropathy, coronary artery disease, cerebral vascular resistance, peripheral vascular disease, nephropathy, retinopathy, stiff joint syndrome, and sensory neuropathy.
2. **Evidence of autonomic neuropathy:** impotence, orthostatic hypotension, resting tachycardia, absent variation in heart rate with deep breathing, gastroparesis (vomiting, diarrhea, abdominal distention), asymptomatic hypoglycemia, prolonged QT interval, and sudden death syndrome.
3. **Management of anesthesia**

A. **Preoperative evaluation**

1. **General information:** if possible, diabetic patients should be scheduled for surgery in the morning; correction of hyperglycemia, ketoacidosis, and electrolyte disturbances are important before elective surgery; consider premedicating with metoclopramide (10 mg) and Bicitra.
2. **Patients taking an oral agent**

A. Sulfonylurea agents should be withheld within 12-24 hours of surgery. Other agents can be given until the patient is NPO.

B. Glucose should be checked before and after the procedure.

C.

3. **Insulin-treated type 2 diabetics (NIDDM)**

A. Patients with well controlled NIDDM undergoing a brief procedure may not require any adjustment in insulin regimen. For major surgery consider giving half their normal morning insulin dose and start on a glucose-containing infusion.

B. Blood glucose should be checked before and after surgery.

4. **Insulin-treated type 1 diabetics (IDDM)**

A. Patients must receive exogenous insulin to prevent ketoacidosis.

B. Insulin can be given by sliding scale or continuous infusion.

1. Regular insulin: 50 U/250 cc NS = 0.2 units/cc (flush IV tubing before starting).
2. Insulin rate in U/hr = blood glucose/150 (use 100 as denominator if patient is on steroids, or is markedly obese, or infected). Alternative dosing. 0.1 units/kg/hr.
3.

C. **Morning of surgery:** continue D_5W and insulin infusion (patients undergoing major surgery, consider rechecking plasma glucose and potassium on morning of surgery).

D. **Induction/maintenance:** the choice of drugs or techniques is less important than monitoring/treatment of blood glucose levels.

E. **Intraoperatively:** check blood glucose at the start of surgery and every 1-2 hours intraoperatively. Adjust insulin infusion as needed to keep glucose levels between 110-180 mg/dL.

B. **Other medications/Information**

1. One can estimate the soluble (regular) insulin requirement by taking the daily dose of lente units and multiplying by 1.5. This gives the units of regular insulin required per day.
2. One unit of insulin (IV) will lower the blood sugar by 30-40 mg/dL in a 70 kg person.
3. Ten grams of dextrose will raise the blood sugar by 30-40 mg/dL in an average 70 kg person.
4. Premeds. Zantac (50 mg IVPB) and metoclopramide (10 mg); (consider Bicitra 30 cc).
5. One in four insulin dependent diabetics may have “stiff joint syndrome” and may be difficult to intubate.
6. Diabetics may have gastroparesis as a result of autonomic neuropathy. The drug of choice for a nauseated or vomiting diabetic is metoclopramide 10 mg IV.
7. In obstetrics, the insulin requirements may drop after delivery.

8. Lactate is converted to glucose in the liver. Lactated Ringers may cause a blood glucose elevation.

Drug	**Onset (hrs)**	**Peak (hrs)**	**Duration (hrs)**
Humalog	0.1-0.25	1	3.5-4.5
Regular	0.25-1	1-3	5-7
Semilente	0.5-1	4-6	12-16
Lente, NPH	2-4	8-10	18-24
Ultralente	4-5	8-14	24-36
Chlorpropamide (Diabinese)	24-72		60-90
Glyburide (Micronase)	0.5-1		24-60
Glipizide (Glucotrol)	1-3		12-24
Tolazamide (Tolinase)	4-6		10-18
Acetohexamide (Dymelor)	0.5-1		12-24

Pheochromocytoma

1. **Definition:** catechol-secreting tumors usually located in an adrenal gland. Most pheochromocytomas produce both norepinephrine and epinephrine. Endogenous catecholamine levels should return to normal levels within 1-3 days after successful removal of the tumor. Overall mortality: 0-6%.
2. **Clinical manifestations**
   A. **Triad of symptoms:** tachycardia, headaches, and diaphoresis.
   B. **Signs:** paroxysmal hypertension (hallmark).
   C. **Other manifestations** include flushing, anxiety, tremor, hyperglycemia, polycythemia, cardiomyopathy, intracerebral hemorrhage, decreased intravascular fluid volume, and weight loss.
   D. **Diagnosis** is confirmed by abnormally high levels of catecholamines or catechol metabolites in urine. Assay of 24-hour urinary metanephrine is the most reliable indicator of excess catecholamine secretion.
3. **Preoperative evaluations**
   A. **Prazosin or phenoxybenzamine** may be used to produce preoperative alpha-adrenergic blockade. Ten to 14 days are usually required for adequate alpha-receptor blockade. Beta blockade is instituted after the onset of adequate alpha blockade if dysrhythmias or tachycardia persists.
   B. **Preoperative goals:** blood pressure below 160/95, no ST-T wave changes, restore intravascular fluid volume, and <1 PVC per 5 minutes.
4. **Anesthetic considerations**

A. **Overall goal** is to avoid sympathetic hyperactivity. Critical times are during tracheal intubation, during manipulation of the tumor, and following ligation of the venous drainage of the tumor.

B. **Drugs to avoid**

1. **Histamine releasers:** morphine, curare, atracurium.
2. **Vagolytics and sympathomimetics:** atropine, pancuronium, gallamine, succinylcholine.
3. **Myocardial sensitizers:** halothane.
4. **Indirect catechol stimulators:** droperidol, ephedrine, TCAs, chlorpromazine, glucagon, metoclopramide.

C. **Monitors:** intraarterial catheter in addition to standard monitors.

D. **Combined technique** (general with spinal or epidural) is effective in ablating sympathetic responses while providing good muscle relaxation.

E. **Maintenance** usually with nitrous oxide plus a volatile anesthetic.

Hyperthyroidism

1. **Clinical manifestations** may include: weight loss, heat intolerance, muscle weakness, diarrhea, hyperactive reflexes, nervousness, exophthalmos, sinus tachycardia/atrial fibrillation and fine tremors.
2. **Management of anesthesia**

A. **Preoperative:** all elective surgery should be postponed until the patient is rendered euthyroid with medical treatment. Preoperative assessment should include normal thyroid function tests, and a resting heart rate <85 beats/min. The combination of beta antagonists and potassium iodide is effective in rendering most patients euthyroid in 10 days. Consider esmolol when surgery cannot be delayed.

B. **Intraoperative:** thiopental is the induction agent of choice, since it possesses some antithyroid activity. Drugs that will stimulate the sympathetic nervous system should be avoided (ketamine, pancuronium, indirect-acting adrenergic agonists, etc.). MAC requirements for inhaled agents or anesthetic requirements are not increased with hyperthyroidism. Cardiovascular function and body temperature should be closely monitored. Patients may have exaggerated response to sympathomimetics.

C. **Postoperative:** most serious postoperative problem is thyroid storm, which is characterized by hyperpyrexia, tachycardia, altered consciousness, and hypotension. Most commonly occurs 6-24 hours postoperatively. Treatment includes hydration and cooling, propranolol (0.5 mg increments until heart rate is below 100 beats/min) or esmolol. Consider cortisol (100-200 mg IV), propylthiouracil (250 mg every 6 hours orally) followed by sodium iodide (1 gm IV over 12 hours), and correction of any precipitating cause.

3. **Complications** after total or partial thyroidectomy. Recurrent laryngeal nerve palsy, hematoma formation, hypothyroidism, and hypoparathyroidism.

Hypothyroidism

1. **Clinical manifestations** may include generalized reduction in metabolic activity, lethargy, intolerance to cold, weight gain, constipation, bradycardia, hyponatremia and decreased cardiac function.
2. **Myxedema coma** results from extreme hypothyroidism and is characterized by impaired mentation, hypoventilation, hypothermia, hyponatremia, and CHF. Treatment is with IV thyroid hormones (300-500 mcg of levothyroxine sodium in patients without heart disease).
3. **Management of anesthesia**

A. **Preoperative:** patients with uncorrected severe hypothyroidism or myxedema coma should not undergo elective surgery. Mild to moderate hypothyroidism is not a absolute contraindication to surgery. Patients should be treated with histamine H_2 blockers and metoclopramide because of their slowed gastric emptying times.

B. **Intraoperative:** ketamine is the induction agent of choice because of the exquisite sensitivity of hypothyroid patients to drug-induced myocardial depression. MAC requirements for inhaled agents are

not changed with hypothyroidism. Maintenance usually with nitrous oxide plus supplementation (opioids, ketamine, etc). Monitoring directed toward early recognition of CHF and hypothermia.

C. **Postoperative:** recovery from general anesthesia may be delayed by slowed drug biotransformation, hypothermia, and respiratory depression.

Obesity

1. **Definitions**
   A. **Overweight** is defined as up to 20% above predicted ideal body weight.
   B. **Obesity** is defined as more than 20% above ideal body weight.
   C. **Morbid obesity** is defined as more than twice ideal body weight.
2. **Body mass index**
   A. Clinically the most useful index for defining obesity.
   B. **Body mass index (BMI)** = weight (kg)/height^2 (meters squared).
   C. A BMI greater then 28 kg/m^2 defines obesity; BMI greater than 35 kg/m^2 defines morbid obesity.
3. **Clinical manifestations**
   A. **Cardiovascular:** systemic hypertension, cardiomegaly, congestive heart failure, coronary artery disease and pulmonary hypertension. Cardiac output increases by 0.1 L/min/kg of adipose tissue.
   B. **Pulmonary:** decreased lung volumes and capacities (suggestive of restrictive lung disease), arterial hypoxemia (decreased functional residual volume predisposes the obese patient to a rapid decrease in PaO_2), obesity-hypoventilation syndrome, decreased chest wall compliance.
   C. **Liver:** abnormal liver function tests, fatty liver infiltration.
   D. **Metabolic:** insulin resistance (diabetes mellitus), hypercholesterolemia.
   E. **Gastrointestinal:** hiatal hernia, gastroesophageal reflux.
4. **Obesity-hypoventilation syndrome (Pickwickian syndrome)**
   A. Occurs in 8% of obese patients, commonly in the extremely obese.
   B. Consists of episodes of nasal and oral airflow obstruction during sleep despite continuing respiratory effort. Obstruction is generally due to backward tongue movement and pharyngeal wall collapse (glossoptosis) secondary to interference with the normal coordinated contraction of pharyngeal and hypopharyngeal muscles.
   C. Characterized by hypercapnia, cyanosis-induced polycythemia, right-sided heart failure, and somnolence. The presence of episodic daytime somnolence and hypoventilation in a morbidly obese patient suggests the presence of this syndrome.
5. **Anesthetic considerations**
   A. **Preoperative**
      1. Preoperative evaluation of morbidly obese patients should include a chest x-ray, EKG, arterial blood gas, and pulmonary function tests.
      2. The airway should be carefully examined since these patients are often difficult to intubate as a result of limited mobility of the temporomandibular and atlanto-occipital joints, a narrowed upper airway, and a shortened distance between the mandible and sternal fat pads.
      3. All obese patients are at an increased risk of aspiration and should be considered to have a full-stomach. Pretreatment with H_2 antagonists (both the night before and the morning of surgery), metoclopramide, and sodium citrate should be considered. Avoid sedation.
   B. **Intraoperative**
      1. The risk of rapid decreases in PaO_2 (due to decreased FRC) is significant; therefore, preoxygenation prior to intubation is essential.
      2. Rapid sequence induction/intubation is selected to minimize the risk of pulmonary aspiration. Morbidly obese patients with a difficult airway should be intubated while awake.
      3. Volatile anesthetics may be metabolized more extensively in obese patients. Obese patients are at an increased risk of halothane hepatitis.
      4. Initial drug doses should be based on ideal body weight.
   C. **Postoperative**
      1. Respiratory failure is the major postoperative problem of morbidly obese patients. Other problems include deep vein thrombosis, pulmonary embolism, and wound infections.

2. The semisitting position will optimize the mechanics of breathing (unload the diaphragm) and will minimize the development of arterial hypoxemia.
3. Patients generally should not be extubated until fully awake.

Corticosteroid Therapy Before Surgery

1. Corticosteroid supplementation should be given to patients being treated for chronic hypoadrenocorticism.
2. Consider supplemental corticosteroids in patients being treated with corticosteroids (unrelated of abnormalities in the anterior pituitary or adrenal cortex) or who have been treated for longer than 1 month in the past 6-12 months.
3. **Empiric regimen for perioperative supplementation**
   A. Patients taking less then 10 mg prednisone/day do not require supplementation.
   B. For patients taking greater then 10 mg prednisone/day consider:
      1. For major surgery: give usual daily dose with premedications, cortisol 25 mg IV on induction, then 100 mg IV by infusion over the next 24 hours. Resume daily dose postoperatively.
      2. For minor surgery: give usual daily dose with premedications and cortisol 25 mg IV on induction. Resume daily dose postoperatively.

Liver Disease

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1. **Halothane hepatic dysfunction**
   A. **Two entities**
      1. Mild/transient form related to hypoxia.
      2. Fulminant form possibly secondary to allergic reaction.
   B. Most often occurs in middle-aged obese females with recent exposure to halothane (up to 4 months). Halothane hepatic dysfunction manifest as post-operative fever and elevated liver function tests.
   C. Pediatric patients are less likely to have halothane-related hepatic dysfunction even after repeated exposures at short intervals.
2. **Anesthesia in patients with liver disease**
   A. **Preoperative evaluation and treatment**
      1. **History:** evaluation of type of liver disease, previous or present jaundice, history of gastrointestinal bleeding, previous surgical operations, exposure to drugs/alcohol,
      2. **Physical:** degree of ascites or encephalopathy, hepatosplenomegaly, peripheral edema.
      3. **Lab test:** CBC with platelet count, serum bilirubin, albumin, serum electrolytes, creatinine and BUN, PT/PTT, and liver function tests.
      4. Preop LFTs should be stable or decreasing for elective surgery.
      5. **The coagulation system** function should be evaluated and corrected preoperatively (with vitamin K, FFP, or platelets as needed). Adequate hydration and diuresis (1 mL/kg/hr) should be achieved.
      6. **Premedications:** sedatives should be omitted or the dose decreased.
   B. **Intraoperative management**
      1. Limit volatile anesthetics (by combining with nitrous oxide and opioids) in order to preserve hepatic blood flow and hepatic oxygenation.
      2. Monitor intraoperative blood gases, pH, coagulation, and urine output.
      3. Avoid or use caution in placing esophageal instrumentation.
   C. **Postoperative:** postoperative liver dysfunction is likely to be exaggerated presumably owing to nonspecific effects of anesthetic drugs on hepatic blood flow and subsequent hepatocyte oxygenation.
3. **Alcohol withdrawal syndrome**

A. **Manifestations** (delirium tremens) usually appear 48-72 hours after cessation of drinking. Mortality is about 10%.

B. **Postoperative manifestations:** tremulousness, hallucinations, increased sympathetic nervous system activity (diaphoresis, hyperpyrexia, tachycardia, hypertension), grand mal seizures.

C. **Treatment:** diazepam 5-10 mg IV every 5 minutes until patient becomes sedated) and a beta antagonist (propranolol or esmolol) to suppress sympathetic nervous system hyperactivity.

4. **Intoxicated alcoholic patient**

A. The acutely intoxicated patient requires less

anesthetic, are more prone to gastric regurgitation, and handle stress and blood loss poorly.

Basics of Anesthesiology

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Medical Gas Systems

1. **Oxygen**
   A. Oxygen is stored as a compressed gas at room temperature or refrigerated as a liquid.
   B. The pressure in an oxygen cylinder is directly proportional to the volume of oxygen in the cylinder.
2. **Nitrous oxide**
   A. At room temperature, nitrous oxide is stored as a liquid.
   B. In contrast to oxygen, the cylinder pressure for nitrous oxide does not indicate the amount of gas remaining in the cylinder; 750 psi as long as any liquid nitrous oxide is present (when cylinder pressure begins to fall, only about 400 liters of nitrous oxide remains).
   C. The cylinder must be weight to determine residual volume of nitrous oxide.

	Oxygen	**Nitrous Oxide**	**Carbon Dioxide**	**Air**	**Nitrogen**
Cylinder Color	Green	Blue	Gray	Yellow	Black
Form	Gas	Liquid	Liquid	Gas	Gas
Capacity (L)	625	1590	1590	624	625
Pressure (psi)	2000	750	838	1800	2000

Electrical Safety

1. **Line isolation monitor**
   A. Line isolation monitor measures the potential for current flow from the isolated power supply to ground.
   B. An alarm is activated if an unacceptably high current flow to ground becomes possible (usually 2 mA or 5 mA).
   C. Operating room power supply is isolated from grounds by an isolation transformer. The line source is grounded by the electrical provider while the secondary circuit is intentionally not grounded.
   D.

2. **Electrical shock**
   A. Macroshock
      1. Refers to the application of electrical current through intact skin.
      2. Currents exceeding 100 mA may result in VF.
   B. **Microshock**
      1. Microshock refers to the application of electrical current directly to the heart (ie, guide wires or pacing wires).
      2. Currents exceeding 50 microamps through a ventricular catheter may induce ventricular fibrillation.

3. The national code requires less than 10 microamps maximum permissible leakage through electrodes or catheters that contact the heart. Line isolation monitors do not protect a patient from microshock.
4.

Anesthesia Machine

1. **Safety valves and regulators**
   A. **Outlet check value:** prevents gas cylinders from crossfilling.
   B. **Pressure regulator:** reduces cylinder gas pressure to below 50 psi.
   C. **Fail-safe valve:** closes gas lines if oxygen pressure falls below 25 psi to prevent accidental delivery of a hypoxic mixture.
   D. **Diameter index safety system (DISS):** prevents incorrect gas line attachment to the anesthesia machine.
   E. **Pin index safety system (PISS):** interlink between the anesthesia machine and gas cylinder; prevents incorrect cylinder attachment.
   F. **Second stage oxygen pressure regulator:** oxygen flow is constant until oxygen pressure drops below 12-16 PSI; whereas other gases shut off if oxygen pressure is less than 30 PSI. This ensures that oxygen is last gas flowing.
2. **Flowmeters** on anesthesia machines are classified as constant-pressure, variable orifice flowmeters.
3. **Vaporizers**
   A. **Classification of modern vaporizers**
      1. **Variable bypass:** part of the total gas flow coming into the vaporizer is bypassed into the vaporizing chamber and then returns to join the rest of the gas at the outlet.
      2. **Flow-over:** the gas channeled to the vaporizing chamber flows over the liquid agent and becomes saturated.
      3. **Temperature-compensated:** automatic temperature compensation device helps maintain a constant vaporizer output over a wide range of temperatures.
      4. **Agent specific.**
      5. **Out of circuit:** not in the breathing circuit.
   B. **Vaporizer output** is not influenced by fresh gas flows until very low flow rates (<250 mL/min) or very high flow rates (>15 L/min).
4. **Anesthesia ventilators**
   A. **Power source:** contemporary ventilators have a pneumatic and electrical power source.
   B. **Drive mechanism:** compressed gas is the driving mechanism.
   C. **Cycling mechanism:** time-cycled, and inspiration is triggered by a timing device.
   D. **Bellows classification:** direction of the bellows during expiration determines the classification. Ascending bellows (bellow ascends during expiration) is safer; a ascending bellow will not fill if a disconnect occurs.
   E. Because the pressure relief valve of the ventilator is closed during inspiration, the circuit's fresh gas flows contribute to the tidal volume delivered to the patient. The amount each tidal volume will increase. (fresh gas flow mL/min) x (% inspiratory time) divided by the respiratory rate.
   F. The use of the oxygen flush valve during the inspiratory cycle of a ventilator must be avoided because the pressure-relief valve is closed and the surge of circuit pressure will be transferred to the patient's lungs.

Patient Monitors

1. **Capnogram**
   A. The normal end-tidal to arterial CO_2 gradient (dCO_2) is 2-5 mmHg. This value reflects alveolar dead space (alveoli ventilated but not perfused).

B. **Causes of increased dCO_2.**
1. Decreased pulmonary arterial pressure.
2. Upright posture.
3. Pulmonary embolism. air, fat, thrombus, amniotic fluid.
4. COPD: causes nonvascular air space at the alveolar level.
5. Mechanical obstruction of the pulmonary arteries.
6. Ventilation gas leaving the normal air passages. bronchopleural fistula, tracheal disruption, cuff leak.

C. **Causes of increased end-tidal CO_2**
1. Hypoventilation.
2. Sodium bicarbonate.
3. Laparoscopy (CO_2 inflation).
4. Anesthetic breathing circuit error
   A. Inadequate fresh gas flow.
   B. Rebreathing.
   C. Faulty circle absorber valves.
   D. Exhausted soda lime.
5. Hyperthermia.
6. Improved blood flow to lungs after resuscitation or hypotension.
7. Water in capnograph head

D. **Causes of decreased end-tidal CO_2**
1. Hyperventilation.
2. Inadequate sampling volume.
3. Incorrect placement of sampling catheter.
4. Hypothermia.
5. Incipient pulmonary edema.
6. Air embolism.
7. Decreased blood flow to lungs.
8.

2. **Pulse oximetry**

A. **Oxyhemoglobin** absorbs more infrared light (eg, 660 nm), while deoxyhemoglobin absorbs more red light (eg, 940 nm).

B. The change in light absorption during arterial pulsations is the basis of oximetry determination of oxygen saturation.

C. **Saturation by PaO_2**

100% = 90-100 90% = 60 75% = 40 60% = 30 30% = 20
95% = 70 80% = 50 70% = 35 50% = 27

D. Pulse oximeters that only compare two wavelengths of light will register a falsely high reading in patients with carbon monoxide poisoning because carboxyhemoglobin and oxyhemoglobin absorb light at 660 nm identically.

E. **Methemoglobin** has the same absorption coefficient at both red and infrared wavelengths, resulting in a 1:1 absorption ratio corresponding to a saturation reading of 85%. Thus, methemoglobinemia causes a falsely low saturation reading when SaO_2 is actually greater then 85% and a falsely high reading if SaO_2 is actually less than 85%.

F. **Fetal hemoglobin** and bilirubin do not affect pulse oximeter.

G. **Sources of error**
1. **Dyshemoglobins**
   A. **Carboxyhemoglobin:** because carboxyhemoglobin and oxyhemoglobin absorb light at 660 nm identically, pulse oximeters that only compare two wavelengths of light will register a falsely high reading in patients suffering from carbon monoxide poisoning.
   B. **Methemoglobin:** has the same absorption coefficient at both red and infrared wavelengths, resulting in a 1:1 absorption ratio corresponding to a saturation reading of 85%. Thus, methemoglobinemia causes a falsely low saturation reading when SaO_2 is actually greater then 85% and a falsely high reading if SaO_2 is actually less than 85%.

2. **Intravenous dyes**
   A. **Methylene blue:** an cause large, rapid decrease in saturation without decreases in the actual oxygen saturation.
   B. **Indocyanine green**: causes smaller false decreases in saturation.
   C. **IV fluorescein or indigo carmine** have little effect.
3. **Excessive ambient light:** in cases of reduced pulse amplitude, pulse oximeters may become sensitive to external light sources, such as fluorescent room lights.
4. **Motion artifact.**
5. **Venous pulsations:** pulse oximeter design assumes that the pulsatile component of the light absorbance is due to arterial blood.
6. **Low perfusion.**
7. **Leakage of light** from the light-emitting diode to the photodiode, bypassing the arterial bed.
8. **Penumbra effect:** pulse oximeters whose sensors are malpositioned may display SaO_2 values in the 90-95% range on normoxemic subjects. This so-called "penumbra effect" can cause underestimation at high saturations, overestimation at low saturations, and a strong dependence of the error on instrument and sensor.
9.

3. **Central venous pressure monitoring**
   A. **Indications for central venous pressure monitoring**
      1. Major thoracic procedures involving large fluid shifts or blood losses in patients with good heart function.
      2. Intravascular volume assessment when urine output is unreliable or unavailable (eg, renal failure).
      3. Major trauma.
      4. Frequent blood sampling in patients not requiring an arterial line.
      5. Venous access for vasoactive or caustic drugs.
      6. Chronic drug administration.
      7. Inadequate peripheral intravenous access.
      8. Rapid infusion of intravenous fluids using large cannula.
   B. **Respiratory influences**
      1. **End expiration:** CVP measurements should be made at end expiration because pleural and pericardial pressures approach atmospheric pressure under these conditions.
      2. **Spontaneous ventilation:** during spontaneous breathing, inspiration causes a decrease in intrapleural pressure and juxtacardiac pressure, which is transmitted in part to the right atrium and produces a decrease in CVP.
      3. **Mechanical ventilation:** positive-pressure ventilation causes intrathoracic and juxtacardiac pressure to increase during inspiration, producing a increase in CVP.
      4. **PEEP:** as intrathoracic pressure increases from added PEEP, CVP measurements increases. This may be associated with a reduction in transmural filling pressure, preload, and venous return.
   C. **Central venous pressure abnormalities**
      1. **Atrial fibrillation:** the a wave disappears, and the c wave becomes more prominent since atrial volume is greater at end-diastole. Fibrillation waves may be noticed in the CVP tracing.
      2. **Isorhythmic A-V dissociation or junctional rhythm:** atrial contraction may occur against a closed tricuspid valve, results in cannon 'a' wave.
      3. **Tricuspid regurgitation:** causes "ventricularization" of the CVP trace, with a broad, tall systolic c-v wave that begins early in systole and obliterates the x descent. Unlike a normal v wave, the c-v wave begins immediately after the QRS, leaving only a y descent.
      4. **Tricuspid stenosis:** prominent a wave as the atrium contracts against a stenotic valve; the y descent following the v wave is obstructed.
      5. **Right ventricular ischemia and infarction**
         A. Diagnosis is suggested by arterial hypotension in combination with disproportionate elevation of the CVP as compared to the PCWP. Mean CVP may approach or exceed the mean PCWP.
         B. Elevated right ventricular filling pressure produces prominent a and v waves and steep x and y descents, giving the waveform an M or W configuration.

6. **Pericardial constriction:** central venous pressure is usually markedly elevated, and the trace resembles that seen with right ventricular infarction. prominent a and v waves and steep x and y descents, creating an M pattern. Often the steep y descent in early diastole is short lived, and the CVP in mid-diastole rises to a plateau until the a wave is inscribed at end-diastole (similar to the h wave).

7. **Cardiac tamponade:** venous pressure waveform becomes monophasic with a characteristic obliteration of the diastolic y descent. The y descent is obliterated because early diastolic runoff from atrium to ventricle is impaired by the compressive pericardial fluid collection.

4. **Pulmonary artery catheterization**

A. Indications for a pulmonary artery catheter

1. Poor left ventricular function (EF<0.4, CI<2 L/min/m^2)
2. Assessment of intravascular fluid volume
3. Evaluation of response to fluid or drug administration.
4. Valvular heart disease.
5. Recent myocardial infarction, congested heart failure.
6. Massive trauma.
7. Major vascular surgery.

B. **Contraindications**

1. **Absolute contraindications**
   - A. Tricuspid or pulmonic valvular stenosis.
   - B. Right atrial or right ventricular masses (eg, tumor, clot).
   - C. Tetralogy of Fallot.
   - D.

2. **Relative contraindications**
   - A. Severe dysrhythmias: complete left bundle branch block (because of the risk of complete heart block), Wolff-Parkinson-White syndrome, and Ebstein's malformation (because of possible tachyarrhythmias).
   - B. Coagulopathy.
   - C. Newly inserted pacemaker wires.

C. **Catheter position**

1. Correct position can be confirmed by a chest x-ray. The tip of the catheter should lie in West Zone 3 of the lung (where venous and arterial pressure exceed alveolar pressure 95% of the time).
   - A. Indicators of proper tip placement
   - B. A decline in pressure as the catheter moves from the pulmonary artery into the "wedged" position.
   - C. Ability to aspirate blood from the distal port (eliminating the possibility of overwedging).
   - D. A decline in end-tidal CO_2 concentration with inflation of the balloon (produced by a rise in alveolar dead space).

D. **Complications**

1. **Endobronchial hemorrhage:** the incidence of pulmonary artery catheter-induced endobronchial hemorrhage is 0.06%-0.20%. Risk factors include advanced age, female sex, pulmonary hypertension, mitral stenosis, coagulopathy, distal placement of the catheter, and balloon hyperinflation.
2. **Pulmonary infarction**

E. **Specific conditions**

1. **Cardiac tamponade:** tends to be equalization of all cardiac diastolic pressures, RA=RVD=PAD=PCWP.
2. **Myocardial ischemia:** elevation in PCWP (>15 mmHg) and 'v' waves (>20 mmHg).
3. **RV infarction:** high RA pressures and normal or low PCWP.

F. **Factors affecting PCWP and CO measurements**

1. Thermodilution CO may be inaccurate in patients with tricuspid regurgitation, intracardiac shunts, and atrial fibrillation.
2. PCWP will be greater then LVEDP in mitral stenosis and prolapsing left atrial tumors.
3. PCWP will be less then LVEDP in decreased ventricular compliance and LVEDP > 25 mmHg.

4. With large ‘a’ waves (mitral stenosis, complete heart block, atrial myxoma, early heart failure) use the end-exhalation diastolic PCWP.
5. With large ‘v’ waves (mitral regurgitation, left atrial enlargement, VSD) use the end-exhalation diastolic PCWP.
6. PEEP, by increasing pleural pressure, will artificially elevate the measure PCWP value. To correct for PEEP >10 cmH2O, subtract half the PEEP pressure from the measured PCWP or use the formula: measured PCWP - (PEEP x 0.75)/3.

5. **Mixed Venous Oxygen Saturation (SvO_2)**
   A. **Physiology:** SvO_2 is approximately equal to SaO_2 - (VO_2/CO x Hemoglobin). Normal range: 68-77%.
   B. **Factors:** cardiac output, hemoglobin level, oxygen consumption, and SaO_2.
   C. **Causes of increased SvO_2**
      1. The most common cause is a permanently wedged catheter.
      2. Low VO_2 as seen with cyanide toxicity, carbon monoxide poisoning, increases in methemoglobin, sepsis, and hypothermia.
      3. High cardiac output, as seen with sepsis, burns, left to right shunts, atrioventricular fistulas, inotropic excess, hepatitis, and pancreatitis.
      4. High SaO_2 and high hemoglobin are not common causes.
   D. **Causes of decreased SvO_2**
      1. Decreased Hemoglobin.
      2. Increased VO_2: fever, exercise, agitation, shivering or thyrotoxicosis.
      3. Low SaO_2 with hypoxia, respiratory distress syndrome, or vent changes.
      4. Low cardiac output as seen in MI, CHF, or hypovolemia.
   E. **Pitfalls in continuous venous oximetry**
      1. The most common errors in the continuous measurement of SvO_2 are calibration and catheter malposition. Distal migration of the PA catheter can cause an artifactually high oxygen saturation owing to highly saturated pulmonary capillary blood being analyzed.
      2. The light intensity signal may decrease if there is fibrin or deposition over the fiberoptic bundles, or if the catheter tip is lodged against a vessel wall or bifurcation.
      3. Large fluctuations in the light intensity may indicate an intravascular volume deficit which allows compression or collapse of the pulmonary vasculature (especially during positive pressure ventilation).
      4. Causes of pulse oximetry artifact include, excessive ambient light, motion, methylene blue dye, venous pulsations in a dependent limb, low perfusion, optical shunting.

6. **Chest Radiography**
   A. **Endotracheal tube position:** with the head in the neutral position, the tip of the ET tube should rest at the mid-trachea level, 5 cm above the carina. In adults the T5-T7 vertebral level is a good estimate of carinal position.
   B. **Central venous catheters**
      1. Ideal location the mid-superior vena cava, with the tip directed inferiorly.
      2. The tip of the catheter should not be allowed to migrate into the heart.
      3.

A. **Pulmonary artery catheters**
   1. The tip of the catheter should be below the level of the LA (zone 3) to reduce or eliminate transmission of alveolar pressure to the capillaries.
   2. With an uninflated balloon, the tip of the pulmonary artery catheter should overlie the middle third of the well-centered AP chest x-ray (within 5 cm of the midline). Distal migration is common in the first hours after insertion as the catheter softens and loses slack.
   3.

B. **Intraaortic balloon pump (IABP):** diastolic inflation of the balloon produces a distinct, rounded lucence within the aortic shadow, but in systole the deflated balloon is not visible. Ideal positioning places the catheter tip just distal to the left subclavian artery.

Component	EKG	Cardiac Cycle	Mechanical Event
a Wave	Follows P wave	End diastole	Atrial Contraction
c Wave	Follows the onset of the QRS	Early systole	Isovolemic ventricular contraction, tricuspid motion toward atrium
v Wave	Follows T wave	Late systole	Systolic filling of atrium
h Wave		Mid diastole	Mid-diastolic pressure plateau (occurs with slow heart rates and prolonged diastole)
x Descent		Midsystole	Atrial relaxation, descent of the base, systolic collapse
y Descent		Early diastole	Early ventricular filling, diastolic collapse

Vein	Right Atrium	Right Ventricle	Pulmonary Artery
Internal Jugular Right Left	20 25	30 35	40 50
Subclavian	15	25	40
Antecubital Right Left	40 45	50 55	65 70
Femoral	30	40	55

Condition	**PCWP**	**CO**	**SVR**
Hypovolemic Shock	Low	Low	High
Cardiogenic Shock	High	Low	High
Septic Shock	Low	High	Low

	Normal Range		**Normal Range**
CVP	0-10 mmHg	**CO**	4-8 L/min
Mean RAP	0-10 mmHg	**CI**	2.5-4 L/min/m^2
RVP	15-30/0-10 mmHg	**SV**	60-100 mL
PAP	15-30/5-15 mmHg	**SVI**	35-70 mL/beat/m^2
MPAP	10-20 mmHg	**SVR**	900-1500 dynes/s/cm^5
PCWP	5-15 mmHg	**PVR**	50-150 dynes/s/cm^5
Mean LAP	4-12 mmHg		

Anesthesia Machine Check

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Anesthesia Machine Check List

Check list should be conducted before administering anesthesia.

7. **Emergency** ventilation equipment*
   A. Verify jet ventilator is hooked up and working.
8. **Overview***
   A. **Inspect machine for the following**
      1. Plugged in.
      2. Flowmeters off.
      3. Vaporizers filled and caps tight.
      4. Tanks on machine properly.
      5. No obvious problems.
      6. Breathing circuit attached.
9. **Electrical systems***
   A. Turn on master switch.
   B. Perform battery check.
   C. Turn on all monitors.
10. **High pressure systems***
   A. **Check Oxygen cylinders**
      1. Disconnect wall pipeline.
      2. Open O_2 tank.
      3. Open O_2 flowmeter.
      4. Tank should stay at least 1000 psi.
      5. Close tank.
      6. Low O2 pressure alarm should respond as bobbin falls.
      7. Turn off O_2 flowmeter.
      8. Reconnect pipeline 0O2.
      9.

   B. **Check pipeline pressures** (should read around 50 p.s.i.).
11. **Low pressure systems***
   A. **Test flowmeters**
      1. Adjust flow of gases through their full range, checking for smooth operation.
      2. Check N_2O/O_2 ratio alarm by trying to create a hypoxic mixture (the gas switch should be set for "N_2O/O_2") and verify correct changes in flow.
   B. **Check for low pressure leaks**
      1. **Drager system** (no back-check valve)
         A. Turn O_2 flowmeter to 400 cc/min.
         B. Open vaporizer.
         C. Occlude gas outlet. bobbin should fall.
         D. Open gas outlet. bobbin should rise.
         E. Close vaporizer.
      2. **Ohmeda system** (back-check valve)
         A. Turn of master switch and flowmeters.
         B. Attach suction bulb to common gas outlet.
         C. Squeeze bulb repeatedly until it collapses.
         D. Verify that bulb stays collapsed for 10 seconds.
         E. Repeat with vaporizers turned on.
         F. Turn on switch and turn off vaporizers.
12. **Scavenging system***

A. **Adjust and check scavenging system**

1. Ensure proper connections between scavenging system and both APL (pop?off valve) and the ventilator relief valve.
2. Close scavenge valve, then open 1½ turns.
3. Fully open APL valve and occlude the Y-?piece.
4. With minimum O_2 flow, scavenger reservoir should collapse completely. Verify that PIP valve reads zero (checks negative pressure valve).
5. With O_2 flush activated, allow the scavenger reservoir bag to distend fully and check that PIP valve reads less than 10 cm/H_2O (checks posi?tive pressure pop-off valve).

13. **Breathing system**

A. **Calibrate O_2 monitor***

1. Ensure monitor reads 21% room air.
2. Verify that low O_2 alarm is enabled and functioning.
3. Reinstall sensor in circuit and flush breathing system with 100% oxygen.
4. Verify that monitor now reads greater than 90%.
5.

B. **Check initial status of system**

1. Set selector switch to “bag” mode.
2. Check that breathing circuit is complete, undamaged, and unobstructed.
3. Verify that CO_2 absorbent is adequate.
4. Install any accessory equipment such as humidifier.

C. **Perform leak check of breathing circuit**

1. Set all gas flows to minimum.
2. Close APL valve and occlude Y piece.
3. Pressurize breathing system to 30 cm H_2O with O_2 flush.
4. Ensure that pressure remains fixed for at least 10 seconds.
5. Open APL valve and ensure that pressure decreases.

14. **Manual and automatic ventilation systems**

A. **Test ventilation systems and unidirectional valves**

1. Set appropriate tidal volume (TV), rate, inspiratory flow.
2. Make sure PEEP valve is off.
3. Set to ventilator mode.
4. Place breathing bag on Y-piece.
5. Turn ventilator on and fill bellows with O_2 flush valve.
6. Set O_2 to minimum and other gas flows to “O.”
7. Verify that, during inspiration, bellow delivers appropriate TV and that bellows empties completely on expiration.
8. Set fresh gas flow to about 5 liters per minute.
9. Verify that the ventilator bellows and simulated lungs fill and empty appropri?ately without sustained pressure at end expiration.
10. Check for proper action of unidirectional valves.
11. Turn ventilator off and switch to manual ventilation.
12. Ventilate manually and assure inflation and deflation of artificial lungs and ap?propriate feel of system resistance and compliance.

15. **Monitors**

A. **Check, calibrate, and/or set alarm limits of all monitors**

1. Capnometer and respiratory volume monitor.
2.
3. O_2 analyzer.
4. Pulse oximeter.
5. Set-up and calibrate invasive monitor transducers.
6. Airway pressure high and low alarms.
7. Automatic BP cuff.
8. EKG monitor.
9. Temperature probe available.

10. Transcutaneous O_2.

16. **Final check**
   A. **Check final status of machine**
      1. Vaporizers off.
      2. APL open.
      3. Selector switch should be set to “bag.”
      4. All flowmeters should be zero (O_2 to minimum flow).
      5. Patient suction level adequate.
      6. Breathing system ready to use.
   B. **Additional equipment if needed***
      1. Blood warmers/Level 1.
      2. Bear Hugger.
      3. Warming blanket.
      4. Operating room table works.
      5. Portable oxygen available.

*These steps need not be repeated if same provider uses the machine in successive cases

Pharmacology

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Basic Pharmacology

1. **Stages of general anesthesia**
   A. **Stage 1** (amnesia) begins with induction of anesthesia and ends with the loss of consciousness (loss of eyelid reflex). Pain perception threshold during this stage is not lowered.
   B. **Stage 2** (delirium/excitement) is characterized by uninhibited excitation. Agitation, delirium, irregular respiration and breath holding. Pupils are dilated and eyes are divergent. Responses to noxious stimuli can occur during this stage may include vomiting, laryngospasm, hypertension, tachycardia, and uncontrolled movement.
   C. **Stage 3** (surgical anesthesia) is characterized by central gaze, constricted pupils, and regular respirations. Target depth of anesthesia is sufficient when painful stimulation does not elicit somatic reflexes or deleterious autonomic responses.
   D. **Stage 4** (impending death/overdose) is characterized by onset of apnea, dilated and nonreactive pupils, and hypotension to complete failure of the circulation.
2. **Pharmacokinetics of inhaled anesthetics**
   A. **Anesthetic concentration:** The fraction of a gas in a mixture is equal to the volume of that gas divided by the total volume of the mixture.
   B. **Partial pressure:** The partial pressure of a component gas in a mixture is equal to the fraction it contributes toward total pressure.
   C. **Minimum alveolar concentration (MAC)**
      1. The minimum alveolar concentration of an inhalation agent is the minimum concentration necessary to prevent movement in 50% of patients in response to a surgical skin incision.
      2. The minimum alveolar concentrations required to prevent eye opening on verbal command, to prevent movement and coughing in response to endotracheal intubation, and to prevent adrenergic response to skin incision have been defined. These are called MAC Awake, MAC Endotracheal Intubation, and MAC BAR (for blockade of autonomic response). In general, MAC Awake is 50% MAC, MAC Endotracheal Intubation is 130% MAC, and MAC BAR is 150% MAC. MAC Amnesia, 25% MAC, has defined as the concentration that blocks anterograde memory in 50% of awake patients.
      3. MAC values for different volatile agents are additive. The lower the MAC the more potent the agent.

4. The highest MACs are found in infants at term to 6 months of age. The MAC decreases with both increasing age and prematurity.
5. Factors that increase MAC include hyperthermia, drugs that increase CNS catecholamines, infants, hypernatremia, and chronic ethanol abuse.
6. Factors that decrease MAC include hypothermia (for every Celsius degree drop in body temperature, MAC decreases 2-5%), preoperative medications, IV anesthetics, neonates, elderly, pregnancy, alpha-2 agonists, acute ethanol ingestion, lithium, cardiopulmonary bypass, opioids, and PaO_2 less than 38 mmHg.
7. Factors that have no effect on MAC include duration of anesthesia, gender, thyroid gland dysfunction, hyperkalemia, and hypokalemia.

D. **Alveolar uptake:** The rate of alveolar uptake is determined by the following
1. **Inspired concentration:** A high inspired anesthetic partial pressure (PI) accelerates induction of anesthesia. This effect of the high PI is known as the concentration effect.
2. **Alveolar ventilation:** Increased ventilation increases the rate of alveolar uptake of anesthetic. The net effect is a more rapid rate of rise in the alveolar partial pressure of an inhaled anesthetic and induction of anesthesia.
3. **Anesthetic breathing system:** The rate of rise of the alveolar partial pressure of an inhaled anesthetic is influenced by (1) the volume of the system, (2) solubility of the inhaled anesthetics into the components of the system, and (3) gas inflow from the anesthetic machine.
4. **Uptake of the inhaled anesthetic**
A. **Solubility:** The solubility of inhaled anesthetics is defined as the amount of anesthetic agent required to saturate a unit volume of blood at a given temperature and can be expressed as the blood:gas partition coefficient. The more soluble the agent, the greater the uptake into the pulmonary capillaries. The solubility of the inhalation agent in blood is the most important single factor in determining the speed of induction and recovery in individual patients.
B. **Cardiac output:** A high cardiac output results in more rapid uptake such that the rate of rise in the alveolar partial pressure and the speed of induction are slowed.
C. **Alveolar to venous partial pressure difference**: A large alveolar to venous gradient enhances the uptake of anesthetic by pulmonary blood and tends to slow the rise in the alveolar partial pressure.

E. **Second gas effect:** The ability of the large volume uptake of one gas (first gas) to accelerate the rate of rise of the alveolar partial pressure of a concurrently administered companion gas (second gas) is known as the second gas effect.

F. **Elimination:** Most of the inhaled agents are exhaled unchanged by the lungs. Hyperventilation, a small FRC (function residual capacity), a low solubility, a low cardiac output, or a large mixed venous-alveolar tension gradient increases the rate of decay.

G. **Diffusion hypoxia** results from dilution of alveolar oxygen concentration by the large amount of nitrous oxide leaving the pulmonary capillary blood at the conclusion of nitrous oxide administration. This can be prevented by filling the patient's lungs with oxygen at the conclusion of nitrous oxide administration.

3. **Pharmacokinetics of intravenous anesthetics**

A. **Volume of distribution:** The apparent volume into which a drug has been distributed; dose of drug administered IV divided by the plasma concentration; binding to plasma protein, high degree of ionization, and low lipid solubility limit passage of drugs to tissue and result in small volume of distribution.

B. **Plasma concentration curve.**
1. **Distribution (alpha) phase:** The alpha phase corresponds to the initial distribution of drug from the circulation to tissues.
2. **Elimination (beta) phase:** The second phase is characterized by a gradual decline in the plasma concentration of drug and reflects its elimination from the central vascular compartment by renal and hepatic mechanisms.

C. **Elimination half-time** is the time necessary for the plasma concentration of drug to decline 50 percent during the elimination phase.

D. **Redistribution:** Following systemic absorption of drugs, the highly perfused tissues (brain, heart, kidneys, liver) receive a proportionally larger amount of the total dose; the transfer of drugs to inactive tissue sites (ie, skeletal muscle) is known as redistribution.

E. **Physical characteristics of the drug**

1. Highly lipid-soluble drugs (most intravenous anesthetics) are taken up rapidly by tissues.
2. With water-soluble agents, molecular size is an important determinant of diffusibility across plasma membranes.
3. Degree of ionization: The degree of ionization is determined by the pH of the biophase and the pKa of the drug. Only nonionized (basic) molecules diffuse across the biological membranes.

Local Anesthetics

1. **Mechanism of action of local anesthetics**

A. Local anesthetics prevent increases in neural membrane permeability to sodium ions, slowing the rate of depolarization so that threshold potential is never reached and no action potential is propagated.

B. Local anesthetics gain access to sodium channels only when they are in their activated state. Rapidly firing nerves are more sensitive and, therefore, are blocked first.

2. **Metabolism**

A. **Esters**

1. Ester local anesthetics are predominantly metabolized by pseudocholinesterase (plasma cholinesterase). Cerebrospinal fluid lacks esterase enzymes, so the termination of action of intrathecally injected ester local anesthetics depends upon their absorption into the bloodstream.
2. P-aminobenzoic acid, a metabolite of local anesthetics, has been associated with allergic reactions.

B. **Amides**

1. Metabolized by microsomal enzymes in the liver.
2. Metabolites of prilocaine (o-toluidine derivatives), which accumulate after large doses (greater than 10 mg/kg), convert hemoglobin to methemoglobin. Benzocaine can also cause methemoglobinemia.

3. **Physiochemical factors**

A. **Lipid solubility:** Increased lipid solubility increases potency.

B. **Protein binding:** The greater the protein binding, the longer the duration of action.

C. **pKa:** determines the onset time. The closer the pKa of the local anesthetic is to tissue pH, the greater the fraction of the non-ionized, lipid-soluble form, is available, and the faster the onset.

D. **Ion trapping:** refers to the accumulation of the ionized form of a local anesthetic in acidic environments due to a pH gradient between the ionized and non-ionized forms. This can occur between a mother and an acidotic fetus (ie, fetal distress), resulting in the accumulation of local anesthetic in fetal blood.

E. **Minimum concentration of local anesthetic (Cm)** is the minimum concentration of local anesthetic that will block nerve impulse conduction and is analogous to the minimum alveolar concentration (MAC) of inhalation anesthetics.

4. **Rate of systemic absorption** of local anesthetics (from high to low). Intravenous > tracheal > intercostal > caudal > paracervical > epidural > brachial plexus > sciatic/femoral > subcutaneous.

5. **Adjuvants**

A. Epinephrine may be added to local anesthetics to produce local vasoconstriction, limiting systemic absorption, prolonging duration of effect, and decreasing surgical bleeding.

B. The maximum dose of epinephrine should not exceed 10 mcg/kg in pediatric patients and 200-250 mcg in adults.

C. Adding sodium bicarbonate raises the pH and increases the concentration of nonionized free base. 1 mEq of sodium bicarbonate is added to each 1 mL of lidocaine.

6. **Effects of local anesthetics on organ systems**

A. **Cardiac**

1. Local anesthetics depress myocardial automaticity (spontaneous phase IV depolarization) and reduce the duration of the refractory period.
2. Cardiac dysrhythmia or circulatory collapse is often a presenting sign of local anesthetic overdose during general anesthesia.

3. Intravascular injection of bupivacaine has produced severe cardiotoxic reactions, including hypotension, atrioventricular heart block, and dysrhythmias such as ventricular fibrillation. Pregnancy, hypoxemia, and respiratory acidosis are predisposing risk factors.

B. **Respiratory effects**

1. Lidocaine depresses the hypoxic drive (response to low PaO_2).

2. Apnea can result from phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to local anesthetic agents (eg, postretrobulbar apnea syndrome).

C. **Central Nervous system effects**

1. Early symptoms of overdose include circumoral numbness, tongue paresthesia, and dizziness. Sensory complaints may include tinnitus and burred vision. Excitatory signs (eg, restlessness, agitation, nervousness, paranoia) often precede central nervous system depression (slurred speech, drowsiness, unconsciousness).

2. Tonic-clonic seizures may result from selective blockade of inhibitory pathways. Respiratory arrest often follows seizure activity.

D. **Neurotoxicity**

1. Chloroprocaine has been associated with neurotoxicity. The cause of this neural toxicity may be the low pH of chloroprocaine (pH 3.0).

2. Repeated doses of 5% lidocaine and 0.5% tetracaine have been associated with neurotoxicity (cauda equina syndrome) following infusion through small-bore catheters used in continuous spinal anesthesia. This may be due to pooling of drug around the cauda equina.

3. Transient radicular irritation: manifests as moderate to severe pain in the lower back, buttocks, and posterior thighs that appears within 24 hours after complete recovery from spinal anesthesia (delay reflects the time required for the neural inflammatory reaction to develop). Full recovery generally occurs within 7 days.

E. **Immunologic effects**

1. Allergic or hypersensitivity reactions are very rare with local anesthetics. Esters are more likely to induce an allergic reaction because they are derivatives of p-aminobenzoic acid, a known allergen.

2. Allergic reactions to amides are extremely rare and are probably related to the preservative and not the amide itself. Multidose preparations of amides often contain methylparaben, which has a chemical structure similar to that of p-aminobenzoic acid.

F. **Musculoskeletal effects**. Local anesthetics are myotoxic when injected directly into skeletal muscle.

7. **Eutectic mixture of local anesthetics**

A. **EMLA cream:** a combination of 2.5% lidocaine and prilocaine cream; 45 minutes may be needed before optimal topical anesthesia is produced.

	Plain Solution		**Epinephrine Solution**	
Drug	**Max Dose (mg)**	**Duration (min)**	**Max Dose (mg)**	**Duration (min)**
Procaine Chloroprocaine	400 800	30-60 30-45	600 1000	30-90
Lidocaine Mepivacaine Priloc	300 300 500	30-120 45-90 30-90	500 500 600	120-360 120-360 120-

aine				360
Bupivacaine Etidocaine Ropivacaine	175 300 200	120-240 120-180 120-360	225 400	180-420 180-420

Drug	**Concen ?tration (%)**	**T10 Level (mg)**	**T6 Level (mg)**	**T4 Level (mg)**	**Duration Plain (min)**	**Duration w/epi (min)**
Procaine	10	75	125	200	30-45	60-75
Lidocaine	5.0	25	50-75	75-100	45-60	60-90
Tetracaine*	0.5**	6-10	8-14	12-20	60-90	120-180
Bupivacaine	0.75	6-10	8-14	12-20	90-120	120-150
Ropivacaine	0.5-0.75	6-10	8-14	12-16	90	140

*For hypobaric spinal: tetracaine diluted with sterile water to 0.3% solution
**Preparation concentration of tetracaine is 1%; tetracaine is diluted with 5.0% glucose for hyperbaric solution and normal saline for isobaric solution

Drug	**Usual Conc %**	**Usual Vol (mL)**	**Total Dose (mg)**	**Onset (min)**	**Duration (min)**
Chloroprocain	2-3	15-30	300-900	5-15	30-90

e					
Lidocaine	1-2	15-30	150-500	5-15	60-120
Mepivacaine	1-2	15-30	150-300	5-15	60-180
Prilocaine	1-3	15-30	150-600	5-15	60-180
Bupivacaine	0.25-0.75	25-30	37.5--225	5-15	120-240
Etidocaine	1.0-1.5	15-30	150-300	5-15	120-240
Ropivacaine	0.25-1.0	15-30	75-200	10-20	120-240

Drug	**Plain (mg)**	**Epi (mg)**	**Plain (mg/kg)**	**Epi (mg/kg)**
Amides Bupivacaine Dibucaine Etidocaine Lidocaine Mepivacaine Prilocaine Ropivacaine	175 200	225 400 500 500	2.5 1 4 4.5 4.5 8 3	 7 7
Esters Chloroprocaine Cocaine Procaine Tetracaine	600 500 100	1000 600 200	12 3 12 3	

Neuromuscular Blocking Agents

1. **Depolarizing blockade**

A. Succinylcholine is the only depolarizing muscle relaxant that is made up of two joined acetylcholine molecules. Succinylcholine mimics the action of acetylcholine by depolarizing the postsynaptic membrane at the neuromuscular junction.

B. **Metabolism**

1. Rapid onset of action (30-60 seconds) with a short duration of action (5-10 minutes). Succinylcholine is rapidly metabolized by pseudocholinesterase into succinylmonocholine so that only a fraction (approximately 10%) of the injected does ever reaches the neuromuscular junction.
2. As serum levels fall, succinylcholine molecules diffuse away from the neuromuscular junction.

C. **Adverse side effects of succinylcholine**

1. **Cardiac:** sinus bradycardia, junctional rhythm, sinus arrest. Ganglionic stimulation may increase heart rate and blood pressure in adults. Succinylcholine may produce bradycardia in children after first dose and after second dose in adults.
2. **Hyperkalemia**

A. Normal muscle releases enough potassium during succinylcholine-induced depolarization to raise serum potassium by 0.5 mEq/L.

B. Massive release of intracellular potassium can result from situations where there is a proliferation of extrajunctional receptors. Associated conditions include: patients with thermal injuries, massive trauma, severe intra-abdominal infection, neurologic disorders (spinal cord injury, encephalitis, stroke, Guillain-Barre syndrome, severe Parkinson's disease), ruptured cerebral aneurysm, polyneuropathy, myopathies (eg, Duchenne's dystrophy) and tetanus. This potassium release is not reliably prevented by pretreatment with a nondepolarizer muscle relaxant.

C.

3. **Increased intracranial pressure, increased cerebral blood flow, and increased intraocular pressure.**
4. **Increased intragastric pressure:** the increase in intragastric pressure is offset by an increase in lower esophageal sphincter tone.
5. **Myalgia and myoglobinuria.**
6. **Fasciculations:** can be prevented by pretreatment with a small dose of nondepolarizing relaxant.
7. **Trismus:** patients afflicted with myotonia may develop myoclonus after succinylcholine administration.
8. **Malignant hyperthermia.**
9. **Phase II block** may occur with repeated or continuous infusions and is characterized by tetanic or TOF fade, tachyphylaxis, partial or complete reversal with anticholinesterases.
10. **Prolonged blockade**

A. Decreased plasma cholinesterase in last trimester of pregnancy, liver disease, starvation, carcinomas, hypothyroidism, burn patients, and cardiac failure.

B. Inhibition of plasma cholinesterase.

C. Plasma cholinesterase deficiency.

D. **Pseudocholinesterase abnormalities**

1. **Heterozygous atypical enzyme:** 1 in 50 patients has one normal and one abnormal gene, resulting in a prolonged block (20-30 minutes).
2. **Homozygous atypical enzyme:** 1 in 3000 patients has two abnormal genes, which produce an enzyme with 1/100 the normal affinity for succinylcholine. Blockade may last 6-8 hours or longer.
3. **Dibucaine**, a local anesthetic, inhibits normal pseudocholinesterase activity by 80%, but inhibits the homozygous atypical enzyme by only 20% and the heterozygous enzyme by 40-60%. The percentage of inhibition of pseudocholinesterase activity is termed the dibucaine number. The dibucaine number is proportional to pseudo?cholin?ester?ase function and independent enzyme amount.

E. **Drug interactions with succinylcholine**

1. Cholinesterase inhibitors (echothiophate eye drops and organophosphate pesticides) enhance the action of succinylcholine.

2. Nondepolarizing muscle relaxants antagonize depolarizing phase I blocks. An exception to this interaction is pancuronium, which augments succinylcholine blockade by inhibiting pseudocholin?ester?ase.

3. Other drugs (that potentiate the neuromuscular block) include antibiotics (streptomycins, colistin, polymyxin, tetracycline, lincomycin, clindamycin), antidysrhythmics (quinidine, lidocaine, calcium channel blockers), antihypertensives (trimethaphan), cholinesterase inhibitors, furosemide, inhalational gas, local anesthetics, lithium, and magnesium.

2. **Non-depolarizing neuromuscular agents**

A. Drugs that potentiate nondepolarizing relaxants volatile agents, local anesthetics, calcium channel blockers, aminoglycosides, polymyxins, lincosamines, hexamethonium, trimethaphan, immunosuppressants, high-dose benzodiazepines, dantrolene, and magnesium.

3. **Sensitivity to neuromuscular blockade:** muscles have different sensitivities to muscle relaxants. The most resistant to most sensitive muscles are: vocal cord, diaphragm, orbicularis oculi, abdominal rectus, adductor pollicis, masseter, pharyngeal, extraocular.

4. Common sites of neurostimulation include the ulnar, posterior tibial, peroneal and facial nerves.

	Dose (mg/kg)				**Infusion mcg/kg/min**
Drug	**Intubation**	**N_2O/ Opioid**	**Inhalation**	**Maintenance**	
Succinyl?choline	1.0-1.5			0.04-0.07	10-100
d-Tubocurarine	0.5-0.6	0.3-0.5	0.2-0.3	0.1-0.15	
Metocurine	0.3-0.4	0.15-0.2	0.1-0.15	0.05-0.1	
Pancuro?nium	0.08-0.12	0.05-0.06	0.03	0.01-0.015	
Pipecuron?ium	0.08-0.12	0.04-0.06	0.03	0.005-0.01	
Doxacurium	0.05-0.08	0.02-0.03	0.015-0.02	0.005-0.01	
Atracurium	0.4-0.6	0.3-0.4	0.2-0.3	0.1-0.15	4-12
Cisatracur?ium	0.15-0.2	0.05	0.03-0.04	0.01-0.02	1-2
Vecuronium	0.1-0.2	0.05	0.03-0.04	0.01-0.02	0.8-2.0
Mivacurium	0.15-0.25*	0.1	0.08	0.05-0.1	3-15
Rocuronium	0.6-1.2	0.3-0.4	0.2-0.3	0.1-0.15	8-12
Rapacuronium	1.5-2.5	1.0-1.5	0.6-1.0	0.2-0.5	9-12

*Given in divided doses (0.15 mg/kg followed in 30 seconds by 0.10 mg/kg). For children 2 to 12 years, the recommended dose of is 0.20 mg/kg, administered over 5 to 15 seconds

Drug	ED 95 (mg/kg)	Onset (min)	Duration (min)	Histamine Release	Elimination and Misc
Succinyl?choline	0.25	1	5-10	Rare	Plasma cholinesterase, muscarinic and nicotinic stim
d-Tubo-curarine	0.51	3-5	60-90	+++	70% renal; 20% biliary; autonomic ganglia block
Metocurine	0.28	3-5	60-90	0	80-100% renal; autonomic ganglia blockade
Pancuron?ium	0.07	3-5	60-90	None	70% renal; 15-20% liver; muscarinic block (10-15% HR increase)
Pipecuron?ium	0.07	3-5	60-90	None	90% renal; 10% liver
Doxacurium	0.25-0.4	4-6	60-90	None	35% renal
Atracurium	0.2	3-5	20-35	0	Hofmann elimination and ester hydrolysis, laudanosine
Cisatracurium	0.05	1-2	60	None	Hofmann elimination
Vecuronium	0.05	3-5	20-35	None	10-20% renal; 40-60% biliary; 20% hepatic
Mivacurium	0.08	2-3	12-20	0	plasma cholinesterase
Gallami	2.5	4-5	70-80	None	80-100%

ne					renal; muscarinic block
Rocuronium	0.3	1-2	20-35	None	10-25% renal; 50-70% biliary; 10-20% hepatic
Rapacuronium	0.75-1.0	1 -2	15-20	0	50% hepatic; 25% renal

Anticholinergics

1. Mechanism of action: anticholinergics competitively block binding by acetylcholine and prevent receptor activation acetylcholine.
2. Central anticholinergic syndrome: scopolamine and atropine can enter the central nervous system and produce symptoms of restlessness and confusion that may progress to somnolence and unconsciousness. Other systemic manifestations include dry mouth, tachycardia, atropine flush, atropine fever, and impaired vision.
3. Physostigmine, a tertiary amine anticholinesterase, is lipid-soluble and reverses central anticholinergic toxicity. An initial dose of .01-0.03 mg/kg is recommended and may need to be repeat after 15-30 minutes.
4. Glycopyrrolate does not easily cross the blood-brain barrier, and thus does not cause a central anticholinergic syndrome.

	Atropine	**Scopolamine**	**Glycopyrrolate**
Tachycardia	+++	+	++
Bronchodilation	++	+	++
Sedation	+	+++	0
Antisialagogue	++	+++	++
Amnesia	+	+++	0
O = No effect; + = Minimal effect; ++ = Moderate effect; +++ = Marked effect			

	Edro?phonium	**Neostigmine**	**Pyrido?stigmine**	**Physo?stigmine**

Dose (mg/kg)	0.5-1.0	0.035-0.07 (up to 5 mg)	0.15-0.35	0.01-0.03 (per dose)
Onset (min)	1-3	7-10	10-13	5
Duration (min)	40-70	65-80	80-130	30-300
Renal Excretion (%)	70	50	75	metabolized by plasma esterases
Atropine (mcg/kg)	7-10	15-30	15-20	
Glycopyr?rolate (mcg/kg)	do not use	7	7	
Misc				treatment for anticholinergic overdose

Organ System	**Muscarinic Side Effect**
Cardiovascular	Decreased heart rate, dysrhythmias
Pulmonary	Bronchospasm, increased bronchial secretions
Cerebral	Diffuse excitation (physostigmine only)
Gastrointestinal	Intestinal spasm, increased salivation
Genitourinary	Increased bladder tone
Ophthalmologic	Pupillary constriction

	Midazola	**Diaze**	**Lorazep**

	m (Versed)	**pam** (Valium)	**am** (Ativan)
Relative Potency	3	1	5
Induction	0.05-0.15 mg/kg	0.3-0.5 mg/kg	0.1 mg/kg
Maintenance	0.05 mg/kg prn or 0.25-1.5 mcg/kg/min	0.1 mg/kg prn	0.02 mg/kg prn
Sedation	PO: 0.5-0.75 mg/kg IV: 0.025-0.1 mg/kg IM: 0.07-0.08 mg/kg PR: 0.3-0.35 mg/kg Nasal: 0.25-0.3 mg/kg	2 mg repeated	IV/IM: 0.02-0.08 mg/kg PO: 2-3 mg
Elimination Half-Time (h)	1-4	21-37	10-20

Benzodiazepines

1. **Mechanism of action**
   A. Benzodiazepines selectively attach to alpha subunits to enhance the chloride channel gating function of the inhibitory neurotransmitter GABA.
   B. Benzodiazepine receptors mostly occur on postsynaptic nerve endings in the central nervous system.
2. **Cardiovascular effects**
   A. Minimal cardiovascular effects.
   B. Midazolam reduces blood pressure and SVR more than diazepam.
3. **Respiratory effects:** depression of the ventilatory response to $PaCO_2$.
4. **Cerebral effects**
   A. Reduced cerebral oxygen consumption, cerebral blood flow and ICP.
   B. Prevention and control of grand mal seizures.
   C. Mild muscle relaxation mediated at the spinal cord level.

5. **Miscellaneous effects**
   A. Benzodiazepines reduce MAC by up to 30%. Cimetidine reduces metabolism of diazepam.
   B. Pain during IV/IM injection and thrombophlebitis occurs with diazepam (secondary to its organic solvent propylene glycol).
   C. Erythromycin inhibits midazolam metabolism.
   D. Heparin displaces diazepam from protein-binding sites and increases the free drug concentration.
6. **Reversal**
   A. Benzodiazepines can be reversed by flumazenil (Romazicon). Flumazenil is a competitive inhibitor of GABA.
   B. For reversal of conscious sedation. 0.2 mg IV over 15 seconds. Give additional 0.1 mg IV bolus every 60 seconds to achieve desired effect, to a total of 1 mg. For reversal of overdose. 0.2 mg IV over 30 seconds. If necessary, give 0.3 mg IV 60 seconds later. If no effect, give 0.5 mg boluses every 60 seconds to a total of 3 mg. For reversal of resedation. 0.2 mg IV as required, to a total of 1 mg/hr, or infusion 0.5 mg/hr.
   C. Dose for diagnosis in coma: 0.5 mg IV repeated up to 1.0 mg IV.
   D. Duration of antagonism is brief and may require repeated doses.
   E. Flumazenil may induce seizures, acute withdrawal, nausea, dizziness, agitation, or arrhythmias (particularly in the presence of tricyclic antidepressants).

Opioids

1. **Classification of opioid receptors**
   A. **Mu receptor:** morphine is the prototype exogenous ligand.
      1. **Mu-1:** the main action at this receptor is analgesia, but also responsible for miosis, nausea/vomiting, urinary retention, and pruritus. The endogenous ligands are enkephalins.
      2. **Mu-2:** respiratory depression, euphoria, sedation, bradycardia, ileus and physical dependence are elicited by binding at this receptor.
      3.

   B. **Delta:** modulation of mu receptor, physical dependence. High selective for the endogenous enkephalins, but opioid drugs still bind (leu-enkephalin and beta-endorphin).
   C. **Kappa:** ketocyclazocine and dynorphin are the prototype exogenous and endogenous ligands, respectively. Analgesia, sedation, dysphoria, and psychomimetic effects are produced by this receptor. Binding to the kappa receptor inhibits release of vasopressin and thus promotes diuresis. Pure kappa agonists do not produce respiratory depression.
   D. **Sigma:** N-allylnormetazocine is the prototype exogenous ligand. While this receptor binds many types of compounds, only levorotatory opioid isomers have opioid activity. The sigma receptor binds primarily dextrorotatory compounds. Dysphoria, hypertonia, tachycardia, tachypnea, and mydriasis are the principal effects of this receptor.
2. **Opioid systemic effects**
   A. **Central nervous system effects**
      1. **Analgesia and euphoria.**
      2. **Sedation-hypnosis**
         A. In usual analgetic doses may produce drowsiness, feelings of heaviness, and difficulty concentrating.
         B. Dosages of opioids sufficient to produce apnea and profound analgesia do not always produce hypnosis with resulting risk of intraoperative awareness.
      3. **Respiratory depression.**
         A. Opioids produce a dose-related depression on the ventilatory response to CO_2 by direct effect on respiratory centers resulting in increased arterial carbon dioxide tension, decreased breathing rate, increased tidal volume, decreased minute ventilation and decreased ventilatory response to carbon dioxide
         B. Respiratory rate is not a sensitive indicator of opioid effect.
      4. **Cough suppression.**

5. **Pupillary constriction:** opioids stimulate the Edinger-Westphal nucleus of the oculomotor nerve to produce miosis.
6. **Nausea and vomiting:** opioids directly stimulate the chemoreceptor trigger zone activating the vomiting center proper. The emetic effects are potentiated by stimulation of the vestibular apparatus, making ambulatory patients more prone to vomiting.
7. **Muscle rigidity.**
   A. Large IV doses may produce generalized hypertonus of skeletal muscle, which, in its most severe form, can prevent ventilation.
   B. Benzodiazepine pretreatment may help in preventing rigidity.
8. **CNS toxicity.**
   A. Dysphoria and agitation may occur (higher with meperidine and codeine).
   B. Seizures may be produced by meperidine (normeperidine, major metabolite, is potent convulsant).
   C. ICP may increase if ventilation is not controlled and $PaCO_2$ is allowed to increase.

B. **Cardiovascular effects**
1. **Bradycardia:** opioids produce a specific stimulant effect on the central nuclei on the vagus nerves increasing vagal tone.
2. **Peripheral vasodilation**
   A. Orthostatic hypotension: depress vasomotor center in the medulla.
   B. Venodilation: which may lead to significant pooling of blood.

C. **Histamine release**
1. Histamine release may produce local itching, redness or hives near the site of injection and may cause a decrease in SVR, hypotension, and tachycardia.
2. Morphine and meperidine release histamine, but fentanyl, sufentanil, alfentanil, and remifentanil do not.

D. Smooth muscle effects
1. **Gastrointestinal:** slow gastric emptying, spasm of sphincter of Oddi (less with meperidine), and constipation.
2. **Genitourinary tract:** increases tone of ureter and vesicle sphincter, making voiding difficult (can be reversed with atropine).

E. **Endocrine**: may block stress response to surgery at high doses.
F. **Placenta**: can cross the placenta causing neonatal depression.
G. **Tolerance:** both acute and chronic tolerance may occur.
H. **Physical dependence.**
I. **Drug interactions**: administration of meperidine in a patient taking a monoamine oxidase inhibitor may result in delirium or hyperthermia.

	Meperidine	**Morphine**	**Fentanyl**	**Sufentia**	**Alfentanil**	**Remifentanil**
Comparative Potency	0.1	1	75-125	500-1000	10-25	250
Peak Effect (min)	5-7	20-30	3-5	3-5	1.5-2	1.5-2
Duration	2-3	3-4	0.5-1	0.5-1	0.2-	0.1-0.2

(hr)					0.3	
Half-Life (hr)	3-4	2-4	1.5-6	2.5-3	1-2	0.15-0.3
Clearance (mL/min/kg)	10-17	14.7	11.6	12.7	6.4	40
Vol of Distribution (L/kg)	2.8-4.2	3.2	4.1	2.86	0.86	0.3-0.4
Partition Coefficient (lipid solubility)	38.8	1.4	860	1,778	130	17.9
Protein Binding (%)	60	26-36	84	92	92	80

Indication	**Anesthesia Duration (min)**	**Initial Dose (mcg/kg)**	**Maintenance (Increment or Infusion)**
Incremental Dosing	<30	8-20	3-5 mcg/kg or 0.5-1 mcg/kg/min
	30-60	20-50	5-15 mcg/kg
Continuous Infusion	>45	50-75	0.5-3 mcg/kg/min

Anesthetic Induction	>45	150-300	0.5-3 mcg/kg/min
Blunt Hypertensive Response To Intubation		15-40	

Indication	**Initial Dose**	**Supplemental Dose**	**Continuous Infusion**
Premedication	25-100 mcg		
Sedation (minor procedure)	0.5-2 mcg/kg		
Adjunct to GA	2-50 mcg/kg	25-50	
General Anesthesia	50-150 mcg/kg	25-100	0.5-5 mcg/kg/hr
Postoperative Analgesia	0.5-1.5 mcg/kg		

Indication	**Initial Bolus**	**Bolus**	**Continuous Infusion**
General Anesthesia	1 mcg/kg (over 1 minute)	0.5-1	0.05-2 mcg/kg/min
Sedation	0.5-1 mcg/kg		0.025-2 mcg/kg/min

Indication	**Dose**	**Maintenance**	**Infusion**
GA: Minor Proced	1-2 mcg/kg	10-25 mcg	

ures			
GA: Moderate Procedures	2-8 mcg/kg	10-50	0.5-1.5 mcg/kg/hr
GA: Major Procedures	8-30 mcg/kg	10-50	0.5-1.5 mcg/kg/hr

Drug	**Adult Dosing**	**Pediatric Dosing**
Buprenorphine	0.4 mg IV q 4-6 hr	0.004 mg/kg IV q 6-8 hr
Butorphanol	0.5-2 mg IV q 3-4 hr	Not recommended
Dezocine	2.5-10 mg IV q 2-4 hr	
Nalbuphine	10 mg IV q 3-4 hr	0.1 mg/kg IV q 3-4 hr
Pentazocine	50 mg PO q 4-6 hr	Not recommended

Opioid Antagonist

1. **Naloxone (Narcan)**
   A. **Pure opioid antagonists:** administration results in displacement of opioid agonists from opioid receptors.
   B. 1-4 mcg/kg IV will reverse opioid-induced analgesia and respiratory depression.
   C. **Continuous infusion**, 5 mcg/kg/hr IV, will prevent respiratory depression without altering the analgesia produced by neuraxial opioids.
   D. **Side effects:** sudden antagonism can activate the sympathetic nervous system, resulting in cardiovascular stimulation.

Intravenous Induction Agents

1. **Sodium thiopental (Pentothal)** and other barbiturates
   A. **Preparation:** thiopental is prepared as a 2.5% solution, water-soluble, pH of 10.5, and stable for up to 1-2 weeks if refrigerated.

B. **Mechanism of action:** depresses the reticular activating system, reflecting the ability of barbiturates to decrease the rate of dissociation of the inhibitory neurotransmitter GABA from its receptors.

C. **Pharmacokinetics**

1. **Short duration of action** (5-10 minutes) following IV bolus reflects high lipid solubility and redistribution from the brain to inactive tissues.
2. Protein binding parallels lipid solubility, decreased protein binding increases drug sensitivity.
3. Protein binding of thiopental in neonates is about half that in adults, suggesting a possible increased sensitivity to this drug in neonates.
4. Fat is the only compartment in which thiopental continues to accumulate 30 minutes after injection.

D. **Effects on organ systems**

1. **Cardiovascular:** induction doses cause a decrease in blood pressure (peripheral vasodilation) and tachycardia (a central vagolytic effect).
2. **Respiratory:** barbiturate depression on the medullary ventilatory center decreases the ventilatory response to hypercapnia and hypoxia. Laryngospasm and hiccuping are more common after methohexital than after thiopental.
3. **Cerebral:** barbiturates constrict cerebral vasculature, decreasing cerebral blood flow and intracranial pressure. Barbiturates cause a decline in cerebral oxygen consumption (up to 50% of normal) and slowing of the EEG (an exception is methohexital which activates epileptic foci). This effect may provide some brain protection from transient episodes of focal ischemia (eg, cerebral embolism), but probably not from global ischemia (eg, cardiac arrest).
4. **Renal:** barbiturates decrease renal blood flow and glomerular filtration rate in proportion to the fall in blood pressure.
5. **Hepatic:** hepatic blood flow is decreased.

E. **Adverse effects**

1. Barbiturates are contraindicated in patients with acute intermittent porphyria, variegate porphyria, and hereditary coprophyria.
2. Venous irritation and tissue damage (reflects possible barbiturate crystal formation); intraarterial injection results in severe pain and possible gangrene.
3. Myoclonus and hiccuping.

2. **Etomidate**

A. **Mechanisms of action:** etomidate depresses the reticular activating system and mimics the inhibitory effects of gamma-aminobutyric acid. The disinhibitory effects of etomidate on the parts of the nervous system that control extrapyramidal motor activity contribute to a high incidence of myoclonus.

B. **Pharmacokinetics:** like other barbiturates, redistribution is responsible for decreasing the plasma concentration to awakening levels. Biotransformation is five times greater for etomidate than for thiopental.

C. **Effects on organ systems**

1. **Cardiovascular:** minimal depressant cardiovascular changes.
2. **Respiratory:** less affected with etomidate than thiopental.
3. **Cerebral:** decreases the cerebral metabolic rate, cerebral blood flow, and intracranial pressure (may activate seizure foci).
4. **Endocrine:** induction doses of etomidate transiently inhibit enzymes involved in cortisol and aldosterone synthesis. Long term infusions lead to adrenocortical suppression.

D. **Drug interactions:** fentanyl increases the plasma level and prolongs the elimination half-life of etomidate.

E. **Adverse effects**

1. Myoclonic movements on induction, opioids levels are decreased.
2. High incidence of nausea and vomiting.
3. Venous irritation due to propylene glycol additive.
4. Adrenal suppression.

3. **Propofol**

A. **Mechanisms of action:** propofol increases the inhibitory neuro?trans?mis?sion mediated by gamma-aminobutyric acid.

B. **Pharmacokinetics:** highly lipid solubility. Short duration of action results from a very short initial distribution half-life (2-8 minutes). Elimination occurs primarily through hepatic metabolism to inactive metabolites. Recovery from propofol is more rapid and accompanied by less hangover than other induction agents.

C. **Effects on organ systems**

1. **Cardiovascular:** decrease in arterial blood pressure secondary to a drop in systemic vascular resistance, contractility, and preload. Hypotension is more pronounced than with thiopental. Propofol markedly impairs the normal arterial baroreflex response to hypo?tension.
2. **Respiratory:** propofol causes profound respiratory depression. Propofol induced depression of upper airway reflexes exceeds that of thiopental.
3. **Cerebral:** decreases cerebral blood flow and intracranial pressure. Propofol has antiemetic, antipruritic, and anticonvulsant properties.

D. **Other effects**

1. **Venous irritation:** pain may be reduced by prior administration of opioids or lidocaine.
2. Propofol is an emulsion and should be used with caution if lipid disorder present. Propofol is preservative free.
3. Very low incidence of anaphylaxis.
4. Allergic reactions may reflect patient sensitivity to the solvent, isopro?pylphenol structure of propofol, or sulfite preservative.
5. Occasional myoclonic movement.
6. Subhypnotic doses (10-15 mg) can help treat nausea/vomiting.

4. **Ketamine**

A. **Mechanism of action:** ketamine blocks polysynaptic reflexes in the spinal cord, inhibiting excitatory neurotransmitter effects. Ketamine functionally dissociates the thalamus from the limbic cortex, producing a state of dissociative anesthesia.

B. **Structure:** ketamine is a structural analogue of phencyclidine (PCP).

C. **Pharmacokinetics:** metabolized in the liver to multiple metabolites.

D. **Effects on organ systems**

1. **Cardiovascular:** ketamine increases arterial blood pressure, heart rate, and cardiac output. The direct myocardial depressant effects of ketamine (large doses) are unmasked by sympathetic blockade or patients who are catecholamine depleted.
2. **Respiratory:** ventilation is minimally affected with normal doses of ketamine. Ketamine is a potent bronchodilator.
3. **Cerebral:** ketamine increases cerebral oxygen consumption, cerebral blood flow, and intracranial pressure.

E. **Drug interactions:** nondepolarizing muscle relaxants are potentiated by ketamine. The combination of ketamine and theophylline may predispose patients to seizures.

F. **Adverse effects**

1. **Increased salivation** (can be attenuated by pretreatment with an anticholinergic).
2. **Emergence delirium:** characterized by visual, auditory, proprioceptive and confusional illusions; reduced by benzodiazepine (midazolam) premedication.
3. **Myoclonic movements.**
4. **Increased ICP.**
5. **Eyes:** nystagmus, diplopia, blepharospasm, and increased intraocular pressure.

	Propofol	**Thiopental**	**Etomidate**	**Ketamine**
Induction Dose (mg/kg	1.5-2.5	3-5	0.2-0.6	1-2 (4-8 mg IM)

IV)				
Anesthesia Maintenance	50-300 mcg/kg/min	30-200 mcg/kg/min	10-20 mcg/kg/min	0.5-1 mg/kg IV prn 15-90 mcg/kg/min IV
Sedation	25-100 mcg/kg/min	0.5-1.5 mg/kg	5-8 mcg/kg/min	0.2-0.8 mg/kg IV 2-4 mg/kg IM
Systemic BP	Decreased	Decreased	NC or Dec	Increased
Heart Rate	NC or Dec	Increased	NC or Dec	Increased
SVR	Decreased	Decreased	NC or Dec	Increased
CBF	Decreased	Decreased	Decreased	Increased
ICP	Decreased	Decreased	Decreased	Increased
Respiratory Depression	Yes	Yes	Yes	No
Analgesia	No	No	No	Yes
Emergence Delirium	No	No	No	Yes
Nausea/Vomiting	Decreased	NC	Increased	NC
Adrenocortical Suppression	No	No	Yes	No
NC = no change; Dec = decreased				

Inhaled Anesthetics

1. **Sevoflurane**
   A. **Advantages**
      1. Well tolerated, even at high concentrations, making this the agent of choice for inhalational induction.
      2. Low blood:gas solubility results in sevoflurane providing rapid induction of anesthesia and rapid emergence.
   B. **Disadvantages**
      1. Less potent than similar halogenated agents.
      2. Interacts with CO_2 absorbers. In the presence of soda lime (and more with barium lime) compound A is produced which is toxic to the brain, liver, and kidneys. Thus it is recommended that, in the presence of soda lime, fresh gas flow rates should not be less than 2 L/min, and use of barium lime is contraindicated.
      3. About 5% is metabolized and elevation of serum fluoride levels has led to concerns about the risk of renal toxicity. In theory, sevoflurane should be avoided in the presence of renal failure.
      4. Postoperative agitation may be more common in children then seen with halothane.
2. **Desflurane**
   A. **Advantages**
      1. Rapid onset and offset of effects due to low blood gas solubility. Has the lowest blood gas solubility of the potent inhalational agents.
      2. Stable in the presence of CO_2 absorbers.
      3. Pharmacodynamic effects are similar to those of isoflurane.
   B. **Disadvantages**
      1. Requires a special vaporizer which is electrically heated and thermostatically controlled. Output from the vaporizer is determined by an electronically controlled pressure regulating valve.
      2. Low potency.
      3. Pungency makes it unsuitable for inhalational induction. Irritation of the airways may be of concern in patients with brochospastic disease.
      4. Rapidly increasing the inhaled concentration or exceeding 1.25 MAC can result in significant sympathetic nervous system stimulation with tachycardia and hypertension.
3. **Isoflurane**
   A. **Advantages**
      1. Suitable for virtually all types o surgery.
   B. **Disadvantages**
      1. May have coronary steal effect.
      2. Pungent odor makes unsuitable for inhalational induction.
4. **Enflurane**
   A. **Advantages**
      1. Non-pungent odor (however, rarely used for inhalational induction).
   B. **Disadvantages**
      1. Can cause tonic clonic muscle activity and an epileptiform EEG trace and should not be used in seizure patients.
      2. Increases in cerebral blood flow and ICP more than isoflurane.
      3. Causes some sensitization of the myocardium to catecholamines and tends to decrease arterial blood pressure by decreasing systemic vascular resistance and having a negative inotropic effect.
      4. Causes more respiratory depression than isoflurane or halothane.
      5. 2.4% metabolized, resulting in increased blood fluoride levels. Should not be used for longer than 9.6 MAC hours to avoid fluoride-induced renal toxicity.
      6. May cause hepatic necrosis (vary rare).
5. **Halothane**
   A. **Advantages**
      1. Potent inhalational agent.
      2. Sweet, nonirritating odor suitable for inhalational induction.
   B. **Disadvantages**

1. Requires preservative, 0.01% thymol, the accumulation of which can interfere with vaporizer function.
2. Risk of halothane hepatitis (dysfunction).
3. Sensitizes myocardium to catecholamines more than other agents.
4. Causes vagal stimulation which can result in marked bradycardia.
5. Potent trigger for malignant hyperthermia.

C. **Recommendations**

1. Avoid repeat exposure within 6 months.
2. History of unexplained jaundice or pyrexia after a previous halothane anesthetic is a contraindication to repeat exposure.
3. Use caution with epinephrine. Avoid concentrations >1:100,000.

6. **Nitrous oxide**

A. **Advantages**

1. Powerful analgesic properties.
2. Decreases the MAC and accelerates the uptake of these agents.
3. Appears to be safe in patients with MH susceptibility.

B. **Disadvantages**

1. Decreases myocardial contractility (offset by stimulating effect on the SNS, increasing SVR). Also increases PVR in patients with preexisting pulmonary hypertension.
2. 35 times more soluble than nitrogen in blood, thus causing a rapid increase in the size of air-filled spaces. Also leads to diffusion hypoxia when N_2O is stopped.
3. Supports combustion and can contribute to fires.
4. Increases risk of postoperative nausea and vomiting.
5. May increase intracranial pressure by increasing cerebral blood flow.
6. Inhibits methionine synthetase (prolonged exposure may lead to megaloblastic bone marrow changes).
7. Long-term use can lead to peripheral neuropathy.
8. Possible teratogenic effect.

Agent	**MAC[1]**	**Blood:gas coefficient[2]**	**Vapor pressure[3]**	**Metabolism (%)[4]**
Isoflurane	1.15	1.4	238	0.2
Enflurane	1.68	1.8	172	2-5
Halothane	0.75	2.3	243	15-20
Desflurane	6	0.42	664	<0.1
Sevoflurane	2.05	0.68	157	2%
Nitrous Oxide	105	0.47	38,770	0.0004
1=Minimum alveolar concentration at one atmosphere at which 50% of patients do not move in response to a surgical skin incision.				

2=Blood:gas partition coefficient is inversely related to the rate of induction.
3=Vapor pressure is reported as mmHg at 20EC.
4=Percentage of absorbed anesthetic undergoing metabolism.

	Sevoflurane	Isoflurane/ Desflurane	Halothane	Enflurane	Nitrous Oxide
CO	0	0	9*	99*	0
HR	0	8	0	88*	0
BP	99	99*	9*	99*	0
SV	99	9*	9*	99*	9
Contractility	99	99*	999*	99*	9*
SVR	9	99	0	9	0
PVR	0	0	0	0	8
ICP	8	8	88	88	8
CBF	8	8	88	8	8
Seizures	9	9	9	8	9
Hepatic BF	9	9	99	99	9
RR	8	8	88	88	8
TV	9	9	9	9	9
PaCO2	8	8	8	88	0
*=Dose dependent					

Receptor	Effector Organ	Response to Stimulation
Beta-1	Heart	Increased HR, contractility, conduction velocity
	Fat cells	Lipolysis
Beta-2	Blood vessels,	Dilation

	bronchioles	
	Uterus	Relaxation
	Kidneys	Renin Secretion
	Liver	Glycogenolysis, gluconeogenesis, insulin secretion
Alpha-1	Blood vessels	Constriction
	Pancreas	Inhibit insulin secretion
	Intestine/bladder	Relaxation, sphincter constriction
Alpha-2	Postganglionic	Inhibit NE release
	CNS	Increase potassium conductance
	Platelets	Aggregation
Dopamine-1	Blood vessels	Dilation
Dopamine-2	Postganglionic	Inhibit NE release
Muscarinic	Heart	Decreased HR, contractility, conduction velocity
	Bronchioles	Constriction
	Salivary glands	Secretion stimulation
	Intestine	Contraction, sphincter relaxation, secretion stimulation
	Bladder	Sphincter relaxation
Nicotinic	Neuromuscular junction	Skeletal muscle contraction
	Autonomic	SNS

	ganglia	stimulation

Drug	**Site of Vasodilation**	Advantages	**Side Effects/Problems**
Nitroprusside	Direct dilator (balanced)	Immediate action Easy to titrate No CNS effects	Hypotension Reflex tachycardia Cyanide toxicity Methemoglobin
Nitroglycerin	Direct dilator (venous > arterial	Coronary dilator	Headache Absorbed into IV tubing ETOH vehicle
Hydralazine	Direct dilator arterial >> venous	No CNS effects	Reflex tachycardia, lupus, local thrombophlebitis
Trimethaphan	Ganglionic blocker	Aortic aneurysm Subarachnoid bleed	Anticholinergic effects Decreased cardiac output
Phentolamine	Alpha blocker direct vasodilator	Pheochromocytoma MAO crisis	Reflex tachycardia Tachyphylaxis
Labetalol	Alpha and beta blockers	No overshoot hypotension Maintained CO, HR	Exacerbation of CHF, asthma, AV block, broncho?spasm

Agent	**Dosage Range**	**Onset**	**Duration**
Nitroprusside	0.25-10 mcg/kg/min	30-60 sec	1-5 min
Nitroglycerin	5-200 mcg/min	1 min	3-5 min
Esmolol	0.5 mg/kg over 1 min 50-300	1 min	12-20 min

	mcg/kg/min		
Labetalol	5-20 mg	1-2 min	4-8 hr
Trimethaphan	3-4 mg/min	1-3 min	10-30 min
Phentolamine	2.5-5 mg	1-10 min	20-40 min
Hydralazine	5-40 mg	5-20 min	4-8 hr
Methyldopa	250-1000 mg	2-3 hr	6-12 hr
Nicardipine	5-15 mg/hr	1-5 min	3-6 hr
Fenoldopam	0.1-1.6 mcg/kg/min	4-5 min	<10 min

Drug	**CO**	**PCWP**	**SVR**	**MAP**	**HR**	**CVP**	**PVR**
Norepinephrine	I	I	I	I	NC	I	I
Phenylephrine	D	I	I	I	D	I	I
Epinephrine	I	I	I	I	I	I	I
Dobutamine	II	D	D	I	I	D	D
Dopamine <6 mcg/kg/min	I	I	I	I	I	I	NC
Dopamine >6 mcg/kg/min	I	II	II	II	I	II	I
Digoxin	I	NC	NC	NC	D	NC	NC
Isoproterenol	II	D	D	D	II	D	D
Amrinone	I	D	D	NC	I	D	D
Nitrogly							

cerin 20-40 mcg/min	NC	D	NC	NC	NC	D	NC
Nitroglycerin 50-250 mcg/min	I	D	D	D	I	D	D
Nitroprusside	I	D	D	D	I	D	D
Key: I=Increased; II=Large Increase; D=Decreased; NC=No Change							

Drug	**HR**	**MAP**	**CO**	**PVR**	**Bron?cho-dilation**	**Renal BF**
Phenylephrine	-	+++	-	+++	0	---
Methyldopa	-	--	-	--	0	+
Epinephrine	++	+	++	-/+	++	--
Ephedrine	++	++	++	+	++	--
Norepinephrine	-	+++	-/+	+++	0	---
Dopamine	+/++	+	+++	+	0	+++
Isoproterenol	+++	-	+++	--	+++	-/+
Dobutamine	+	+	+++	-	0	+
HR = heart rate; MAP = mean arterial pressure; CO = cardiac output; PVR = pulmonary vascular resistance; BF = blood flow.						

Drugs and Drips

Acetaminophen (Tylenol)

Actions: inhibits the synthesis of prostaglandins in the CNS and peripherally blocks pain impulse generation; produces antipyresis from inhibition of hypothalamic heat-regulating center (no anti-inflammatory effects).

Indications: treatment of mild-moderate pain and fever.

Dose (children < 12 years): 10-15 mg/kg PO/PR every 4-6 hours as needed (do not exceed 2.6 gm in 24 hours).

Dose (adults): 325-650 mg PO/PR every 4 hours (max 4 gm/24 hrs).

Adverse effects: anemia, blood dyscrasias, nausea, rash, vomiting.

Comments: severe hepatic toxicity on overdose or when combined with alcohol.

Adenosine (Adenocard)

Actions: adenosine slows conduction through the A-V node, interrupting the re-entry pathways through the A-V node.

Indications: PSVT, Wolf-Parkinson-White syndrome.

Dose (adult): 6 mg rapid IV bolus; may be repeated within 1-2 minutes with 12 mg (up to two doses).

Dose (pediatric): 0.1 mg/kg rapid IV; may repeat with 0.2 mg/kg. Max signal dose 12 mg.

Adverse effects: chest pain, facial flushing, hypotension, palpitations, dyspnea, headache.

Comments: Contraindicated in patients with second or third-degree heart block or sick sinus syndrome. Large doses given by infusion may cause hypotension. Not effective in atrial flutter/fibrillation or ventricular tachycardia. Effects antagonized by methylxanthines and potentiated with dipyridamole

Aminocaproic Acid (Amicar)

Actions: inhibits plasminogen activators (fibrinolysis inhibitor), thereby reducing fibrinolysin without inhibiting lysis clot..

Indications: excessive acute bleeding from hyperfibrinolysis, chronic bleeding tendency; antidote for excessive thrombolysis.

Dose: loading dose of 100-150 mg/kg IV over the first 30-60 minutes followed by constant infusion of 33.3 mg/kg/hour for about 8 hours or until bleeding controlled. Most common regimen for adult: 5 gram loading (started prior to skin incision) followed by constant infusion of 1 gm/hour.

Comments: reduced dose by 15-25% in patients with oliguria or ESRD.

Adverse effects: contraindicated in patients with active intravascular clotting, DIC, and bleeding in the kidneys or ureters; hypotension, bradycardia, dysrhythmias, elevated LFT's, thrombosis.

Aminophylline

Actions: inhibits of phosphodiesterase, resulting in bronchodilation with positive inotropic and chronotropic effects.

Loading dose: 5-7 mg/kg IVPB over 15-30 min (6 mg/kg PO).

Maintenance (IV): 1) Children 1-9 years 1 mg/kg/hr
2) Children >9 years 0.8 mg/kg/hr
3) Adult smokers 0.8 mg/kg/hr
4) Adult non-smokers 0.5 mg/kg/hr
5) Adults w/CHF/liver 0.25 mg/kg/hr

Rate determination: cc/hr = dose x body wt (kg) when mixed 1 mg/kg.

Therapeutic level: 10-20 mg/dL.

Adverse effects: nausea, vomiting, anorexia, dizziness, headaches, agitation, tachyarrhythmias, ventricular arrhythmias, palpitations, overdosage hyperreflexia, convulsions, hypotension, tachypnea.

Comments: aminophylline contains about 80% theophylline by weight.

Amiodarone (Cordarone)

Actions: inhibits adrenergic stimulation, decreases A-V conduction and the sinus node function, prolongs the PR, QRS, and QT intervals, and produces alpha- and beta-adrenergic blockade.

Indications: refractory or recurrent ventricular tachycardia or VF, SVT, PSVT, and atrial arrhythmias.

Adult dosage: cardiac arrest: 300 mg IVP; consider repeating 150 mg IVP in 3-5 minutes. Max cumulative dose: 2.2 gm IV/24 hours. Wide-complex tachycardia (stable) administered as followed: (1)

rapid infusion: 150 mg IV over first 10 minutes, may repeat 150 mg every 10 minutes for breakthrough VF/VT; (2) slow infusion: 360 mg IV over 6 hours (1 mg/min); maintenance infusion: 540 mg IV over 18 hours (0.5 mg/min).
Pediatric dose: for refractory VF/VT: 5 mg/kg IV; for perfuming supraventricular and ventricular arrhythmias: loading dose 5 mg/kg IV over 20-60 minutes (repeat to max of 15 mg/kg per day IV).
Adverse effects: may produce vasodilation and hypotension, cause severe sinus bradycardia, ventricular dysrhythmias, AV block, prolong QT interval. May have negative inotropic effects. May cause liver and thyroid function test abnormalities, hepatitis, and cirrhosis. Pulmonary fibrosis may follow long-term use. Increases serum levels of digoxin, oral anticoagulants, diltiazem, quinidine, procainamide, and phenytoin. Use with caution in renal failure.

Amrinone (Inocor)
Actions: phosphodiesterase inhibitor (rapid inotropic agent) causing increase in cardiac output while pulmonary vascular resistance and preload decrease (positive inotropic and vasodilator properties), slightly increases atrioventricular conduction.
Indications. Treatment of low cardiac output states, adjunctive therapy for pulmonary hypertension, intractable heart failure.
Dosage. Loading dose of 0.75 mg/kg is given over 10-15 minutes, followed by a 5-15 mcg/kg/min infusion.
Standard conc. 100 mg in 250 cc NS (do not mix in dextrose solutions).
Comments: Use reduced dose (50-75% of dose) in renal failure. Dose should not exceed 10 mg/kg/24 hours.
Adverse effects: Worsening myocardial ischemia, thrombocytopenia, hypotension, tachyarrhythmias; contraindicated if allergic to bisulfites; hepatic function abnormalities, nausea, vomiting, hypotension, arrhythmias
Comments: Do not dilute in dextrose containing solutions; do not administer furosemide (Lasix) in same IV line.

Aprotinin (Trasylol)
Actions. Inhibitor of several proteases (including trypsin, kallikrein, and plasmin) and inhibits factor XIIa activation of complement.
Indications. Prophylactic use to reduce bleeding and transfusion requirements in high-risk cardiac surgery patients.
Test dose. 1 mL (1.4 mg) administer IV at least 10 minutes before loading dose. After the test dose is given, either the low dose regimen or high dose regimen may be started.
High (standard) dose: 200 mL (280 mg) IV, slowly over 20-30 minutes with patient in supine position; 200 mL (280 mg) into pump prime volume; followed by 50 mL/hr (70 mg/hr) maintenance infusions after loading dose.
Low dose: 100 mL (140 mg) IV, slowly over 20-30 minutes with patient in supine position; 100 mL (140 mg) into pump prime volume; followed by 25 mL/hr (35 mg/hr) maintenance infusions after loading dose.
Adverse effects: allergic reactions and anaphylaxis.
Comments: aprotinin prolongs whole blood clotting time of heparinized blood (prolonged PTT). Patients may require additional heparin even in the presence of adequate anticoagulation by activated clotting time (ACT). All doses of aprotinin should be administered through a central line. No other drugs should be administer in the same line.

Aspirin (Acetylsalicylic Acid)
Actions: irreversibly inhibits platelet cyclo-oxygenase, inhibits the formation of platelet-aggregating substance thromboxane A2 platelet aggregation, acts on hypothalamus heat-regulating center to reduce fever.
Indications: acute ST-elevation infarction, coronary angioplasty, suspected ischemic-type pain.
Adult dose: analgesic and antipyretic: 325-650 mg every 4-6 hours PO or suppository if nauseated or vomiting; myocardial infarction prophylaxis: 160-325 mg/day PO.
Pediatric dose: analgesic and antipyretic: 10-15 mg/kg oral or rectal every 4-6 hours, up to 60-80 mg/kg/24 hours.

Precautions: active peptic ulcer disease (use rectal suppository), history of hypersensitivity or allergy, bleeding disorders, severe hepatic disease, asthma.

Atropine Sulfate

Actions: competitive blockade of acetylcholine at muscarinic receptors; increases cardiac output, dries secretions, antagonizes histamine and serotonin.

Indications: bradycardia; antisialagogue; exercise-induced asthma, antidote for organophosphate pesticide poisoning, mydriasis and cycloplegia.

Dose (antisialagogue): adult 0.2-0.4 mg IV; ped 0.01 mg/kg/dose IV/IM.

Dose (bradycardia): adult 0.5-1.0 mg IV may repeat every 3-5 minutes; pediatric 0.02 mg/kg IV may repeat every 3-5 minutes.

Dose (PEA/asystole): 1 mg IV may repeat every 3-5 minutes.

Minimal dose is 0.1 mg.

Adverse effects: may cause tachydysrhythmias, AV dissociation, premature ventricular contractions, dry mouth, or urinary retention. CNS effects occur at high doses. Increases myocardial oxygen demand.

Comments: not effective in second degree AV block type II; avoid in new third degree block with wide QRS complexes, and hypothermic bradycardia.

Bicarbonate (Sodium Bicarbonate)

Actions: hydrogen ion neutralization.

Indications: metabolic acidosis, gastric hyperacidity, alkalinization agent for urine, treatment of hyperkalemia.

Dose (metabolic acidosis): dosage should be based on the following formula: IV dose in mEq $NaHCO_3$ = [base deficit x weight (kg) x 0.3].

Dose (cardiac arrest): adult: 1 mEq/kg/dose; pediatric: 0.5-1.0 mEq/kg/dose; neonates should receive 4.2% solution.

Adverse effects: may cause metabolic alkalosis, hypercarbia, hyperosmolality. May decrease cardiac output, systemic vascular resistance, and myocardial contractility. Crosses placenta. 8.4% solution is approximately 1.0 mEq/mL. 4.2% solution is approximately 0.5 mEq/mL.

Bretylium (Bretylol)

Actions: initially, release of norepinephrine into circulation, followed by prevention of synaptic release of norepinephrine; suppression of ventricular fibrillation and ventricular arrhythmias; increase in myocardial contractility (direct effect).

Indications: treatment of VF/VF and other serious ventricular arrhythmias.

Dose: 5 mg/kg IV push initially, followed by 5-10 mg/kg every 15-30 min to total of 30-35 mg/kg.

Continuous infusion: 1-2 mg/min (2 gm/250 cc D5W).

Adverse effects: postural and supine hypotension (potentiated by quinidine or procainamide), aggravation of digoxin induced arrhythmias, nausea/vom?iting following rapid injection.

Bumetanide (Bumex)

Actions: loop diuretic with principal effect on the ascending limb of the loop of Henle. Increased excretion of Na, K, Cl, Ca, Mg, phosphate, H_2O.

Indications: edema, hypertension, intracranial hypertension.

Dose: 0.5-1.0 mg IV, repeated to a maximum of 10 mg/day. Continuous infusion of 0.9-1 mg/hour may be more effective then bolus.

Adverse effects: may cause electrolyte imbalance, dehydration, and deafness (rapid infusion ototoxic). Patients allergic to sulfonamides may show hypersensitivity to bumetanide. Effective in renal insufficiency.

Caffeine

Indications: diuretic, relief of post-dural puncture headache.

Dose: adult: 500 mg IV, may repeat at 8 hour intervals whose headache is not relieved; pediatric: 8 mg/kg every 4 hours as needed.

Contraindications: symptomatic cardiac dysrhythmias, peptic ulcer.

Adverse effects: tachycardia, palpitations, headache, insomnia, nervousness, restlessness, gastric irritation, nausea, vomiting, anxiety.

Calcium Chloride

Actions: essential for maintenance of cell membrane integrity, muscular excitation-contraction coupling, glandular stimulation-secretion coupling, and enzyme function.
Indications: hypocalcemia, hyperkalemia, hypomagnesemia, hypotension.
Dose (adult): 500-1000 mg calcium chloride (8-16 mg/kg IV).
Dose (pediatric): 20 mg/kg slow IV.
Adverse effects: ay cause bradycardia or arrhythmia (especially with digitalis), hypertension, increased risk of ventricular fibrillation, and can be irritating to veins.
Comments: Do not mix with sodium bicarbonate; not used routinely in ACLS. 10% CaCl2= 100 mg/mL = 27.2 mg/mL elemental calcium.

Calcium Gluconate

Actions: essential for maintenance of cell membrane integrity, muscular excitation-contraction coupling, glandular stimulation-secretion coupling, and enzyme function.
Indications: hypocalcemia, hyperkalemia, hypomagnesemia, hypotension.
Dose: adult: 15-30 mg/kg IV; pediatric: 60-100 mg/kg slow IV.
Adverse effects: may cause bradycardia or arrhythmia (especially with digitalis), hypertension, increased risk of ventricular fibrillation, and can be irritating to veins.
Comments: do not mix with sodium bicarbonate; not used routinely in ACLS. 10% CaCl2= 100 mg/mL = 9 mg/mL elemental calcium.

Citrate (Bicitra)

Actions: absorbed and metabolized to sodium bicarbonate.
Indications: gastric acid neutralization, premedication.
Dose: 15-60 cc PO
Adverse effects: contraindicated in patients with sodium restriction or severe renal impairment. Do not use with aluminum based antacids.

Dantrolene (Dantrium)

Actions: reduction of calcium release from sarcoplasmic reticulum, prevents or reduces increase in myoplasmic calcium ion concentration.
Indications: malignant hyperthermia, skeletal muscle spasticity, neuroleptic malignant syndrome.
Dose (for MH): 2.5-3 mg/kg IV bolus; if syndrome persists after 30 minutes, repeat dose, up to 10 mg/kg (see MH section).
Adverse effects: muscle weakness, GI upset, drowsiness, sedation, or abnormal liver function; additive effect with neuromuscular blockers.
Comments: mix 20 mg in 60 cc of sterile water. Dissolves slowly.

Desmopressin Acetate (DDAVP)

Actions: increases plasma levels of Factor VIII and von Willebrand factor (vWF) and decreases bleeding times; causes release of tissue plasminogen activator and prostacyclin; antidiuretic activity.
Indications: bleeding uremic patients with platelet dysfunction, cirrhosis, cardiac surgery; improves coagulation in von Willebrand's disease and hemophilia; used as a antidiuretic hormone.
Dose: 0.3 mcg/kg IV over 20 minutes.
Adverse effects: decreased free water clearance from antidiuretic activity, hypotension, thrombosis, decreased serum sodium.

Dexamethasone (Decadron)

Actions: anti-inflammatory and antiallergic effects. Has 25 times the glucocorticoid potency of hydrocortisone.
Indications: cerebral edema from CNS tumors; airway edema.
Dose: load 10 mg IV; maintenance 4 mg IV q6 hours.

Adverse effects: may cause adrenocortical insufficiency (Addison's crisis) with abrupt withdrawal, delayed wound healing, CNS disturbances, osteoporosis, and electrolyte disturbances.

Dextran 40 (Rheomacrodex)

Actions: immediate, short-lived plasma volume expansion; prevents RBC aggregation, decreasing blood viscosity and platelet adhesiveness.

Indications: inhibition of platelet aggregation; improvement of blood flow in low-flow states (eg, vascular surgery); intravascular volume expander.

Dose: adult: load 30-50 mL IV over 30 min, maintenance 15-30 mL/hr; pediatric: <20 mL/kg/24 hr of 10% dextran.

Adverse effects: may cause volume overload, anaphylaxis, bleeding tendency, interference with blood cross matching, or false elevation of blood glucose level. Can cause renal failure.

Digoxin

Actions: positive inotropic effects, negative chronotropic effects, slows conduction velocity through the AV node.

Pharmacokinetics: onset of action is about 30 min following IV.

Indications: CHF, heart rate control in atrial fib/flutter, PSVT.

Dose: supraventricular tachycardia: 10-15 mcg/kg IV in divided doses (0.25-0.5 mg as initial dose and 0.25 mg every 4 hours as subsequent doses until the entire dose is given or heart rate controlled); CHF: 8-12 mcg/kg given in divided doses as above.

Therapeutic level: 0.8-2.0 ng/mL. Maintenance dose affected by body size and renal function.

Adverse effects: symptoms of digoxin toxicity include mental depression, confusion, headaches, drowsiness, anorexia, nausea, vomiting, weakness, visual disturbances, delirium, EKG abnormalities (any arrhythmia) and seizures. Hypokalemia increases risk of digoxin toxicity. Heart block potentiated by beta blockers or calcium channel blockers.

Diltiazem (Cardizem)

Actions: calcium channel antagonist; slows conduction through SA and AV nodes; dilates coronary and peripheral arterioles, and reduces myocardial contractility.

Indications: angina pectoris, temporary control of rapid ventricular rate during atrial fibrillation/flutter; conversion of paroxysmal supraventricular tachycardia to normal sinus rhythm.

Dose: rate control: initial bolus with 0.25 mg/kg (15-20 mg) over 2 minutes, if response is inadequate after 15 minutes, rebolus with 0.35 mg/kg (20-25 mg) over 2 minutes; continuous infusion 5-15 mg/hr (mix 125 mg in 100 cc of D5W) titrate to heart rate.

Adverse effects: hypotension, bradycardia, heart block, impaired contractility, transient increase in LFTs, injection site reaction, flushing, and arrhythmia.

Contraindications: atrial fib/flutter patients with WPW or short PR syndrome, sick sinus syndrome or second- or third-degree AV block except with a pacemaker, wide-QRS tachycardias of uncertain origin, poison/drug-induced tachycardia.

Diphenhydramine (Benadryl)

Actions: H^1 receptor antagonist, anticholinergic, CNS depression.

Indications: allergenic reactions, extrapyramidal reactions, sedation.

Dose: adult: 10-50 mg IV q6-8 hours; pediatric: 5.0 mg/kg/day IV in four divided doses (maximum of 300 mg).

Adverse effects: may cause hypotension, tachycardia, dizziness, urinary retention, seizures.

Dobutamine (Dobutrex)

Action: stimulates beta 1 with minimal effect on beta 2 and alpha receptors, dose dependent increase in cardiac output often with decrease in SVR and PVR, minimal chronotropic effect.

Indications: cardiogenic shock, severe CHF; low cardiac output.

Standard concentration: 250 mg/250 D5W (1 mg/cc).

Dose: 2-20 mcg/kg/min. Titrate to effect.

Adverse effects: tachyarrhythmias (less than dopamine), fluctuations in BP, headache, nausea, can increase ventricular rate in atrial fibrillation.

Comments: Do not mix with sodium bicarbonate.

Dolasetron Mesylate (Anzemet)

Actions: serotonin receptor antagonist.

Indications: postoperative nausea and vomiting.

Dose: 12.5 mg IV; 100 mg PO.

Adverse effects: may cause headaches, dizziness, and hypertension. Use with caution in patients who have or may develop prolongation of cardiac conduction intervals, particularly QTc.

Dopamine (Intropin)

Actions: dopaminergic, alpha and beta adrenergic agonist

Indications: shock, poor perfusion, decreased splanchnic perfusion, low cardiac output, oliguria.

Standard conc.: 400 mg/250 cc D5W = 1600 mcg/cc.

Dosage: range 2-20 mcg/kg/min

2-5 mcg/kg/min: stimulates dopamine receptors, redistribute blood flow to kidneys, inhibits aldosterone.

5-10 mcg/kg/min: beta 1 > alpha receptor stimulation, increased myocardial contractility without marked changes in HR or BP.

10-15 mcg/kg/min: alpha and beta 1 stimulation,

>15 mcg/kg/min: alpha effects predominate.

Adverse effects: tachycardia, arrhythmias, nausea/vomiting; superficial tissue necrosis and sloughing may occur with extravasation, contraindicated in pheochromocytoma.

Comments: do not mix with sodium bicarbonate.

Dopexamine

Actions: synthetic analogue of dopamine, beta-2 and dopamine agonist (little beta-1 or alpha activity); increases CO and heart rate, decreases SVR with little change in blood pressure, increased renal blood flow

Indications: shock, poor perfusion, decreased splanchnic perfusion, low cardiac output, oliguria.

Dose: 1-10 mcg/kg/min titrated to desired effect.

Adverse effects: hypotension (causes vasodilation), tachycardia (atrial).

Droperidol

Actions: dopamine (D_2) receptor antagonist, antiemetic effect, antipsychotic effect, apparent psychic indifference to environment.

Indications: nausea, vomiting, agitation, sedation

Dose (adult) antiemetic: 0.625-2.5 mg prn; sedation: 2.5-10 mg IV prn. **Dose (pediatric) antiemetic**: 0.05-0.06 mg/kg q4-6 hours.

Adverse effects: evokes extrapyramidal reactions in 1%; possible dysphoric reactions; cerebral vasoconstrictor; can decrease blood pressure by alpha blockade and dopaminergic antagonism, used in neuroleptanalgesia, potentiates other CNS depressants.

Ephedrine

Actions: alpha- and beta-adrenergic stimulation, norepinephrine release at sympathetic nerve endings (indirect).

Indications: hypotension.

Dose: 5-50 mg IV prn (adults); 0.1 mg/kg (peds)

Adverse effects: may cause hypertension, dysrhythmias, myocardial ischemia. Avoid giving to patients taking monoamine oxidase inhibitors. Tachyphylaxis with repeated dosing.

Epinephrine (Adrenalin)

Actions: direct alpha and beta adrenergic receptor stimulation, resulting in bronchial smooth muscle relaxation and cardiac stimulation (positive inotrope, chronotrope), dilation of skeletal muscle vasculature (low dose), decreased renal blood flow,

Indications: heart failure, hypotension, cardiac arrest, bronchospasm, anaphylaxis, severe bradycardia.

Standard conc.: 2 mg/250 cc (15 cc/hr = 2 mcg/min).

Dose (cardiac arrest): 1 mg IV every 3-5 minutes during resuscitation; higher doses (up to 0.2 mg/kg) may be used if 1 mg does fails.

Dose (severe brady/hypotension): 2-10 mcg/min infusion; bolus 10-20 mcg.

Dose (bronchospasm): 0.1-0.5 mg SQ/IV every10-15 minutes.

Dose (infusion): start at 2 mcg/min (or 0.05 mcg/kg/min) and titrate to blood pressure and cardiac output; bolus starting at 10-20 mcg; infusion range 0.01- 0.3 mcg/kg/min.

Dose (pediatric-neonates): 0.01-0.3 mg/kg every 3-5 minutes.

Dose (children): 0.01 mg/kg IV every 3-5 minutes; 0.01 mg/kg SQ q15 minutes for bronchospasm.

Adverse effects: may cause hypertension, dysrhythmias, or myocardial ischemia. Dysrhythmias potentiated by halothane. Metabolic effects (increases adipose tissue lipolysis, liver glycogenolysis, inhibits release of insulin). Crosses placenta.

Epinephrine, Racemic (Vaponefrin)

Actions: mucosal vasoconstriction (see epinephrine).

Indications: airway edema, bronchospasm.

Dose: inhaled via nebulizer: 0.5 mL of 2.25% solution in 2.5-3.5 mL of NS q1-4 hr prn.

Adverse effects: see epinephrine.

Ergonovine (Ergotrate)

Actions: constriction of uterine and vascular smooth muscle.

Indications: postpartum uterine atony and bleeding, uterine involution.

Dose: 0.2 mg IV in 5 mL NS given over 1 minute (IV route is used only in emergencies). 0.2 mg IM q2-4 hours for less than 5 doses; then PO: O.2-0.4 mg q6-12 hours for 2 days.

Adverse effects: may cause hypertension from system vasoconstriction, arrhythmias, coronary spasm, cerebrovascular accidents, uterine tetany, or gastrointestinal upset.

Esmolol (Brevibloc)

Actions: selective beta-1 adrenergic blockade. Short half-life (2-9 minutes).

Indications: SVT, myocardial ischemia, hypertension.

Dose (bolus): 5-100 mg IV prn.

Dose (infusion): load 500 mcg/kg bolus over 1 min followed by maintenance starting at 50 mcg/kg/min (1-15 mg/min). To calculate an infusion rate in mL/min, divide mg/min by 10. To calculate an infusion rate in mL/hr, multiply mg/min by 6. Max infusion: 300 mcg/kg/min.

Standard conc.: 10 mg/mL (infusion mix two 2.5 gm ampuls in 500 cc).

Adverse effects: bradycardia, AV conduction delay, hypotension, congestive heart failure, myocardial depression.

Etomidate (Amidate)

Actions: augments the inhibitory tone of GABA in the CNS (produces unconsciousness in approximately 30 seconds).

Indications: IV induction agent for general anesthesia.

Induction dose: 0.2-0.3 mg/kg IV.

Maintenance: 10 mcg/kg/min IV with N 20 and opiate.

Sedation and analgesia: 5-8 mcg/kg/min; used only for short periods of time due to inhibition of corticosteroid synthesis.

Adverse effects: direct cerebral vasoconstrictor, minimal cardiovascular effects, pain on injection, myoclonus may occur in about 1/3 of patients during induction, adrenocortical suppression, nausea/vomiting.

Flumazenil (Romazicon)

Actions: competitive inhibition of GABA.

Indications: reversal of benzodiazepine sedation or overdose.

Dose: first dose 0.2 mg IV over 15 seconds, second dose 0.3 mg IV over 30 seconds, if no adequate response give third dose 0.5 mg IV over 30 seconds, may repeat up to a total of 3 mg.

Adverse effects: seizures, acute withdrawal, nausea, dizziness, agitation, arrhythmias, hypertension.

Drug interactions: do not use in suspected tricyclic drug overdose, seizure-prone patients, unknown drug overdoses.

Furosemide (Lasix)

Actions: increase in excretion of sodium and potassium and water by inhibiting reabsorption in the loop of Henle.

Indications: edema, hypertension, intracranial hypertension, renal failure, hypercalcemia.

Dose (adult): 2-100 mg IV (start with 0.5-1 mg/kg over 1-2 minutes).

Dose (pediatric): 0.2-1 mg/kg

Adverse effects: may cause electrolyte imbalance, dehydration, transient hypotension, deafness, hyperglycemia, or hyperuricemia.

Glucagon

Actions: stimulates adenylate cyclase to produce increased cyclic AMP.

Indications: hypoglycemia, duodenal or choledochal relaxation.

Dose (hypoglycemia): neonates: 0.3 mg/kg/dose, children: 0.025-0.1 mg/kg/dose, adults: 0.5-1.0 mg, may repeat in 20 minutes.

Dose (GI relaxation): 0.25-0.5 mg IV every 20 minutes.

Adverse effects: may cause anaphylaxis, nausea, vomiting, hyperglycemia, or positive inotropic and chronotropic effects; high doses potentiate oral anticoagulants; do not use in presence of insulinoma or pheochromocytoma.

Comments: do not mix in normal saline (use sterile water).

Heparin

Actions: heparin, prepared from bovine lung, facilitates the activation of anti-thrombin III, neutralizes primarily thrombin and factor X.

Indications: anticoagulation.

Loading dose: for thromboembolism: 5000 units IVP; for cardiopulmonary bypass: 300 units/kg.

Maintenance dose: for thromboembolism: start 1000 units/hr and titrate to PTT 1.5 to 2.5 x control; for cardiopulmonary bypass: 100 units/kg/hr IV titrated against activated clotting time.

Standard conc.: 10,000 units/500 cc D_5W (20 units per cc).

Reversal: reverse with protamine sulfate.

Adverse effects: hemorrhage, allergic reactions, thrombocytopenia, altered protein binding, decreased MAP, decreased antithrombin III concentration, altered cell morphology, does not cross placenta.

Hydralazine (Apresoline)

Actions: relaxation of vascular smooth muscle.

Indications: hypertension.

Dose: 2.5-20 mg IV q4 hr or prn.

Adverse effects: hypotension, reflex tachycardia, systemic lupus erythematosus syndrome, or Coombs' test positive hemolytic anemia.

Hydrocortisone (SoluCortef)

Actions: anti-inflammatory, antiallergic, mineralocorticoid effect. Stimulation of gluconeogenesis. Inhibition of peripheral protein synthesis.

Indications: adrenal insufficiency, inflammation and allergy, cerebral edema from CNS tumors, asthma.

Dose: non-life threatening conditions: 50-200 mg IV q2-10 hrs prn. Life threatening conditions: 50 mg/kg IV over several minutes q4-24 hrs.

Adverse effects: may cause adrenocortical insufficiency (Addison's crisis) with abrupt withdrawal, delayed wound healing, CNS disturbances, osteoporosis, or electrolyte disturbances.

Indigo Carmine (Indigotindisulfonate Sodium)

Actions: rapid glomerular filtration causing blue urine.

Indications: evaluation of urine output. Localizing of ureteral orifices.

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

Adverse effects: hypertension from alpha adrenergic stim, lasts 15-30 min.

Insulin

Indications: hyperglycemia.

Actions: facilitation of glucose transport into cells.

Infusion: 50 units regular insulin in 250 cc D_5W or NS (1 U/hr = 5 cc/hr).

Dose: average range is 2-10 units/hour or 0.1 units/kg/hour.

Adverse effects: hypoglycemia, allergic reactions, absorbed by IV tubing.

Isoproterenol (Isuprel)

Actions: synthetic sympathomimetic amine that acts on beta-1 and beta-2 adrenergic receptors; positive chronotrope and inotrope; decreases systemic and pulmonary vascular resistance; increases coronary and renal blood flow.

Indications: bradycardia, shock where increasing HR will increase CO, shock with severe aortic regurgitation, heart failure, pulmonary hypertension, refractory asthma, carotid sinus hypersensitivity, bradycardia in heart transplant patients, refractory torsades de pointes, beta blocker overdose.

Dose: constant infusion 2-20 mcg/min, start at 1-2 mcg/min and titrate to desired heart rate; peds: 0.01 mcg/kg/min.

Standard conc.: 1 mg/250 cc; 15 cc = 1 mcg.

Adverse effects: arrhythmogenic, may increase myocardiac ischemia, hypertension, CNS excitation, nausea/vomiting, pulmonary edema, paradoxical precipitation of Adams-Strokes attacks.

Ketamine

Actions: causes increased HR, CO, cardiac work, and myocardial oxygen requirements; direct stimulation of the CNS leads to increased sympathetic nervous system outflow.

Induction: 0.5-2 mg/kg IV; 6-10 mg/kg IM

Maintenance: 10-100 mcg/kg/min IV

Sedation and analgesia: 0.2-0.8 mg/kg IV; 2-6 mg/kg IM; 3 mg/kg intranasally; 6-10 mg/kg PO.

Adverse effects: ketamine is a direct myocardial depressant, potent cerebral vasodilator, and it should not be used in patients receiving aminophylline (reduces seizure threshold).

Ketorolac (Toradol)

Actions: inhibits prostaglandin synthesis through cyclo-oxygenase inhibition.

Indications: nonopioid, nonsteroidal analgesic for moderate pain.

Single dose (IM): patients <65 years of age: 60 mg; patients >65 years of age, renally impaired and/or less than 50 kg: 30 mg.

Single dose (IV): <65 years of age: one dose of 30 mg; patients >65 years of age, renally impaired and/or less than 50 kg: one dose of 15 mg.

Multiple dosing (IV or IM): <65 years of age: 30 mg every 6 hours. The maximum daily dose should not exceed 120 mg. For patients >65 years of age, renally impaired and patients less than 50 kg should have one-half the above dose. The combined duration of use for parenteral and oral should not exceed 5 days.

Adverse effects: adverse effects are similar to those of other nonsteroidal anti-inflammatory drugs and include peptic ulceration. Contraindicated in patients with active peptic ulcer disease, and renal impairment.

Labetalol (Normodyne, Trandate)

Actions: selective alpha-1-adrenergic blockade with nonselective beta-adrenergic blockade

Indications: hypertension, angina, controlled hypotension.

Dose: 5-10 mg increments at 5 min intervals, may increase dose to 150 mg/dose. Infusion rate: 2-8 mcg/min.

Adverse effects: may cause bradycardia, AV conduction delay, bronchospasm in asthmatics, and postural hypotension.

Lidocaine

Actions: blocks both the initiation and conduction of nerve impulses by decreasing the permeability of the neuronal membrane to sodium ions.
Indications: ventricular dysrhythmias, cough suppression, local anesthesia.
Dose (dysrhythmias): 1-1.5 mg/kg IVP, may repeat up to total of 3mg/kg.
Constant infusion: 1-4 mg/min or 30-50 mcg/kg/min (reduce by half for patients with CHF, hepatic dysfunction, or shock).
Standard concentration: 2 gm/250 cc; (7 cc/hr = 1 mg/min).
Adverse effects: CNS depression, drowsiness, unconsciousness, apprehension, change in vision, vomiting, bradycardia, hypotension, respiratory depression. Avoid in patients with WPW syndrome.
Comments: endotracheal dose 2-2.5 times the IV dose.

Magnesium Sulfate
Actions: central nervous system depressant and anticonvulsant, inhibits release of acetylcholine at the neuromuscular junction, decreases sensitivity of motor end-plate to acetylcholine, decreases muscle excitability, decreases uterine hyperactivity (increasing uterine blood flow).
Indications: pregnancy induced hypertension, hypomagnesemia, torsades de pointes, ventricular arrhythmias due to digitalis toxicity.
Dose (hypomagnesemia): 4-8 gm/100 cc NS or D_5W given over 8 hours.
Dose (pregnancy induced hypertension): initial bolus of 2-4 gm in a 20% solution IV over 5 minutes followed by a continuous infusion of 1-2.5 gm/hr (10-20 gm in 1000 cc of D_5W).
Dose (torsades de pointes): 1-2 g IV; infusion with 0.5-1.0 g/hr IV.
Levels: normal plasma level is 1.5 to 2.2 mEq/L. In the treatment of pregnancy induced hypertension the therapeutic level is 4-5 mEq/l.
Adverse effects: 5-10 mEq/l: loss of deep tendon reflexes and respiratory depression; 10 mEq/l: prolonged PR and QT intervals and widened QRS complexes; 15 mEq/l: SA and AV nodal blocks and respiratory apnea; 25 mEq/l: cardiac arrest.

Mannitol (Osmitrol)
Actions: increase in serum osmolality decreasing brain swelling, osmotic diuresis, and transient expansion of intravascular volume.
Indications: intracranial hypertension, treatment of renal failure, glaucoma, diuresis.
Dose (adult): 0.25-1.0 gm/kg IV as 20% solution over 30-60 minutes (in acute situation, can give bolus of 1.25-25 gm over 5-10 minutes).
Dose (pediatric): 0.2 g/kg test dose, maintenance of 2 g/kg over 30-60 min.
Adverse effects: rapid administration may cause vasodilation and hypotension. May cause pulmonary edema, intracranial hemorrhage, systemic hypertension.

Methohexital (Brevital)
Indications: IV induction agent for general anesthesia.
Induction: 1-1.5 mg/kg IV or 20-30 mg/kg pr (children).
Sedation: 0.2-0.4 mg/kg IV.
Adverse effects: earlier recovery time and less cumulative effect than thiopental, higher incidence of excitatory phenomena (cough, hiccups, involuntary movements), pain on injection, activates epileptic foci (unlike other barbiturates).

Methylene Blue (Urolene Blue)
Actions: low dose promotes conversion of methemoglobin to hemoglobin. High dose promotes conversion of hemoglobin to methemoglobin. Less useful than sodium nitrate and amyl nitrite.
Indications: surgical marker, treatment of methemoglobinemia.
Dose (marker): 100 mg (10 mL of 1% solution) IV.
Dose (methemoglobinemia): 1-2 mg/kg IV. Repeat q1 hr prn.
Adverse effects: may cause RBC destruction (with prolonged use), hypertension, bladder irritation, nausea, diaphoresis. May inhibit nitrate induced coronary artery relaxation. Interferes with pulse oximetry for 1-2 minutes. May cause hemolysis in patients with glucose-6-phosphate-dehydrogenase deficiency.

Methylergonovine (Methergine)

Actions: constriction of uterine and vascular smooth muscle.

Indications: postpartum hemorrhage.

Dose: 0.2 mg IV in 5 mL NS given over 1 minute (IV route is used only in emergencies). 0.2 mg IM q2-4 hours for less than 5 doses; then PO: 0.2-0.4 mg q6-12 hours for 2 days.

Adverse effects: may cause hypertension from system vasoconstriction, arrhythmias, coronary spasm, uterine tetany, or gastrointestinal upset.

Methylprednisolone (Solu-Medrol)

Actions: has 5 times the glucocorticoid potency of hydrocortisone.

Indications: same as hydrocortisone.

Dose (adult): for non-life threatening conditions: 10-250 mg IV q4-24 hr IV over 1 minute. For life threatening conditions: 100-250 mg IV q2-6 hr or 30 mg/kg IV q4-6 hrs.

Dose (pediatric): for life threatening conditions not <0.5 mg/kg/24 hr.

Adverse effects: see hydrocortisone.

Metoprolol (Lopressor)

Actions: beta-1 adrenergic blockade (beta-2 antagonism at high doses).

Indications: hypertension, angina pectoris, dysrhythmia, hypertrophic cardiomyopathy, myocardial infarction, pheochromocytoma.

Dose: 50-100 mg PO q4-24 hr.

Adverse effects: may cause bradycardia, clinically significant bronchoconstriction, dizziness, fatigue, insomnia. Risk of heart block.

Morphine Sulfate

Actions: CNS analgesia, decreases anxiety by sympatholytic effects, venodilator that decreases LV preload, decreases systemic vascular resistance reducing LV afterload.

Indications: pain management, treatment of ischemic pain not relieved by nitroglycerin, acute cardiogenic pulmonary edema.

Dose: pain relief: 2-4 mg IV, repeat every 5-30 minutes to effect.

Precautions: do not administer if hypotensive, use caution in low volume status, may cause respiratory depression.

Milrinone (Primacor)

Actions: phosphodiesterase inhibitor, increases cAMP in the heart, peripheral vasodilator, increases inotropy.

Indications: low output heart failure.

Loading dose: 50 mcg/kg IV over 10 minutes.

Maintenance dose: 0.375-0.75 mcg/kg/min.

Standard concen.: 200 mcg/cc (50 mg/250 cc dextrose or saline).

Adverse effects: increased ventricular ectopy, nonsustained ventricular tachycardia, supraventricular tachycardia; hypotension; headaches.

Comments: the presence of renal impairment may significantly increase the terminal elimination half-life. Do not inject furosemide into IV lines containing milrinone; a precipitate-forming chemical reaction will occur.

Naloxone (Narcan)

Actions: antagonism of narcotic effect.

Indications: reversal of systemic narcotic effects.

Dose (adult): 0.4-2.0 mg IV every 2-3 minutes titrated to effect.

Dose (pediatric): 1-10 mcg/kg IV every 2-3 minutes up to 0.4 mg.

Dose (infusion): 5-10 mcg/kg/hr

Onset/duration: onset 1-2 minutes; duration less than one hour.

Adverse effects: may cause reversal of analgesia, hypertension, arrhythmias, rare pulmonary edema, delirium or withdrawal syndrome. Renarcotization may occur because of short duration of action.

Nicardipine (Cardene)

Actions: dihydropyridine calcium channel blocker.

Indications: short-term treatment of hypertension.

Dose: administer slowly. Initiate therapy at 50 cc/hr (5.0 mg/hr). If desired blood pressure reduction is not achieved at this dose, the infusion rate may be increased by 25 cc/hr every 5 minutes for rapid blood pressure reduction or every 15 minutes for gradual blood pressure reduction up to a maximum of 150 cc/hr. Following achievement of the blood pressure goal, the infusion rate should be decreased to 30 cc/hr.

Contraindications: known hypersensitivity and those with advanced aortic stenosis. Caution when administering in patients with impaired renal or hepatic function or in combination with a beta blocker in CHF patients.

Adverse effects: most commonly hypotension, headache, tachycardia.

Nifedipine (Procardia)

Actions: blockade of slow calcium channels in the heart. Systemic and coronary vasodilation and increase in myocardial perfusion.

Indications: coronary artery spasm, hypertension, myocardial ischemia.

Dose:10-40 mg PO tid; 10-20 mg SL.

Adverse effects: may cause reflex tachycardia, gastrointestinal tract upset, mild negative inotropic effects. Little effect on automaticity and atrial conduction. Sensitive to light.

Nitroglycerin

Actions: vasodilation through nitric oxide-induced relaxation of vascular smooth muscle; greater venous dilation than arterial dilation (decrease of preload > decrease of afterload), coronary artery dilation, decreased systemic vascular resistance, decreased pulmonary vascular resistance.

Indications: myocardial ischemia, hypertension, congestive heart failure, pulmonary hypertension, esophageal spasm.

Dose (bolus): 12.5 to 25 mcg

Dose (infusion): start at 5-10 mcg/min and titrate every 5 minutes until chest pain resolved or hemodynamic state achieved.

Dose (SL): 0.4 mg, repeat x2 at 5 minute intervals as needed.

Dose (spray): 2 metered doses under or onto tongue.

Standard conc. (IV infusion): 50 mg/250 cc; 3 cc/hr = 10 mcg/min.

Adverse effects: reflex tachycardia, hypotension, headache, tolerance and dependence with chronic use. May be absorbed by plastic in IV tubing.

Contraindications: systolic blood pressure <90 mmHg, severe bradycardia, RV infarction, use of Viagra within 24 hours.

Nitroprusside (Sodium Nitroprusside)

Actions: smooth muscle relaxation; arterial dilation greater than venous.

Indications: hypertensive crisis, congestive heart failure, pulmonary hypertension, reduce afterload in acute mitral or aortic valve regurgitation.

Dose (infusion): start at 0.5-1.0 mcg/kg/min and titrate upward every 3-5 minutes to desired effect (up to 10 mcg/kg/min).

Standard concentration: mix 50 mg/250 cc D_5W.

Adverse effects: hypotension, nausea, abdominal cramps, headaches, restlessness, cyanide and thiocyanate toxicity, CO_2 retention, may reverse hypoxic pulmonary vasoconstriction exacerbating intrapulmonary shunting, degraded by light (tubing/container must be covered with aluminum foil); signs of toxicity include tachyphylaxis, metabolic acidosis, and high mixed venous saturation (treatment is with sodium nitrite, sodium thiosulfate, hydroxocobalamin or methylene blue).

Norepinephrine (Levophed)

Actions: alpha 1, alpha 2, beta 1 and beta 2 (min) adrenergic agonist.

Indications: septic shock, cardiogenic shock with hypotension and decreased SVR, refractory hypotension.

Dose (infusion): 0.5-1.0 mcg/min to 30 mcg/min titrated to effect.

Standard conc.: 4 mg/250 cc D_5W; 15 cc/hr = 4 mcg/min.
Adverse effects: hypertension, arrhythmias, myocardial ischemia, increased uterine contractility, constricted microcirculation, ischemic necrosis and sloughing of superficial tissues will result if extravasation occurs (treat with phentolamine 5-10 mg in 10-15 mL saline solution infiltrated into area).

Octreotide (Sandostatin)
Actions: somatostatin analogue that suppresses release of serotonin, gastrin, vasoactive intestinal peptide, insulin, glucagon and secretin.
Indications: relief of flushing, bronchospasm, hypotension from carcinoid tumor.
Dose: 50 mcg IV/SQ prn.
Adverse effects: may cause nausea, decreased gastrointestinal tract motility, transient hyperglycemia.

Ondansetron (Zofran)
Actions: serotonin receptor selective antiemetic.
Indications: prevention of postoperative nausea and/or vomiting.
Adult dose: 4 mg undiluted IV in not less than 30 seconds.
Pediatric dose: 0.05-0.075 mg/kg IV in not less than 30 seconds.
Adverse effects: headache, dizziness, musculoskeletal pain, drowsiness, sedation, shivers, reversible transaminase elevation.

Oxytocin (Pitocin)
Actions: reduced postpartum blood loss by contraction of uterine smooth muscle. Renal, coronary, and cerebral vasodilation.
Indications: postpartum hemorrhage, uterine atony, augment labor.
Dose: uterine atony: 10-40 units in 1000 cc crystalloid; labor induction: 0.0005 -0.002 units/min.
Adverse effects: may cause uterine tetany and rupture, fetal distress. IV bolus can cause hypotension, tachycardia, dysrhythmia.

Phenylephrine (Neo-Synephrine)
Actions: alpha adrenergic agonist (direct) producing vasoconstriction
Indications: hypotension, SVT
Dose: 50-200 mcg bolus or 20-100 mcg/min continuous infusion.
Standard conc.: for bolus 50 mcg/cc; for infusion 40-100 mcg/cc.
Adverse effects: hypertension, reflex bradycardia.

Physostigmine (Antilirium)
Actions: inhibition of cholinesterase, central and peripheral cholinergic effects.
Indications: postoperative delirium, tricyclic antidepressant overdose, reversal of CNS effects of anticholinergic drugs.
Dose: 0.5-2.0 mg IV q15 minutes.
Adverse effects: may cause bradycardia, tremor, convulsions, hallucinations, psychiatric or CNS depression, mild ganglionic blockade, cholinergic crisis.

Procainamide (Pronestyl)
Actions: decreases myocardial excitability and conduction velocity and may depress myocardial contractility, by increasing the electrical stimulation threshold of the ventricle and HIS-Purkinje; has direct cardiac effects.
Indications: atrial and ventricular arrhythmias.
Load: continuous infusion of 20-30 mg/min until (1) arrhythmia suppressed; (2) hypotension ensues; (3) the QRS complex widened by 50%; or (4) a total of 1 gm or 17 mg/kg has been given.
Maintenance: 1-4 mg/min.
Standard concentration: 2 gm/500 cc D_5W: 30 cc/hr = 2 mg/min.
Therapeutic level: 4-10 mcg/mL.
Adverse effects: hypotension, heart block, myocardial depression, ventricular dysrhythmias, lupus, fever, agranulocytosis, GI irritation.
Comments: if cardiac or renal dysfunction present reduce max total dose to 12 mg/kg.

Promethazine (Phenergan)

Actions: antagonist of H1, D2, muscarinic receptors: antiemetic; sedative.

Indications: allergies, anaphylaxis, nausea and vomiting, sedation.

Dose: 12.5-50 mg IV q4-6 hr prn.

Adverse effects: may cause mild hypotension or mild anticholinergic effects. Crosses placenta. May interfere with blood grouping. Intraarterial injection can cause gangrene.

Propofol (Diprivan)

Actions: increases activity of inhibitory GABA synapses; decreases cerebral metabolic rate for oxygen, cerebral blood flow, and ICP; decreases systemic vascular resistance and blood pressure.

Indications: induction/maintenance of anesthesia, sedation, antiemetic.

Induction: 1.5-2.5 mg/kg; reduce w/age >50.

Maintenance: 0.1-0.2 mg/kg/min or 6-12 mg/kg/hr with N_2O and opiate.

Sedation: 25-150 mcg/kg/min.

Dose (antiemetic effect): 10-15 mg IV.

Adverse effects: may cause pain during administration (ain is reduced by prior administration of opioids or lidocaine), hypotension, respiratory depression, allergic reactions.

Propranolol (Inderal)

Actions: nonspecific beta-adrenergic blockade.

Indications: hypertension, dysrhythmias, myocardial ischemia/infarction, thyrotoxicosis, hypertrophic cardiomyopathy, migraine headache.

Dose (Tachyarrhythmias): peds: 0.01-0.1 mg/kg slow IV; adults: 1 mg/dose repeated every 5 minutes to max total of 5 mg1-10 mg IV.

Dose (tetralogy spell): 0.15-0.25 mg/kg/day slow IV

Dose (thyrotoxicosis): 1-3 mg slow IV.

Adverse effects: may cause bradycardia, AV block, hypoglycemia, bronchospasm, CHF, and drowsiness with low doses.

Misc: abrupt withdrawal can precipitate rebound angina.

Prostaglandin E1 (Alprostadil)

Actions: vascular smooth muscle and uterine smooth muscle relaxation.

Indications: pulmonary vasodilator, maintenance of patent ductus arteriosus.

Standard concentration: 500 mcg (1 vial) in 99 cc D_5W (5 mcg/cc).

Dose: start at 0.05 mcg/kg/min (0.01 cc/kg/min); may increase to as high as 0.5 mcg/kg/min.

Adverse effects: hypotension, apnea, flushing, bradycardia.

Comments: rapidly metabolized.

Protamine Sulfate

Actions: antagonist of the anticoagulant effect of heparin.

Indications: reversal of the effects of heparin.

Dose: 1-3 mg per 100 units heparin, maximum 50 mg over 10 minutes, given slowly; 1.3 mg/kg of procaine for each 100 units of heparin present as calculated from the ACT.

Adverse effects: hypotension (rapid injection secondary to histamine release), pulmonary hypertension and allergic reactions (seen in patients receiving procaine containing insulin preparations and in some patients allergic to fish).

Ritodrine (Yutopar)

Actions: beta-2 selective adrenergic agonist that decreases uterine contractility.

Indications: tocolysis (inhibition of preterm labor)

Dose: IV infusion 0.1-0.35 mg/min.

Adverse effects: dose related increases in maternal and fetal heart rate and blood pressure due to beta-1 stimulation. May cause pulmonary edema, insulin resistance, potentiation of dysrhythmias and hypotension by magnesium. Contraindicated in eclampsia, pulmonary hypertension, and hyperthyroidism.

Terbutaline (Brethine)

Actions: beta-1 selective adrenergic agonist.

Indications: bronchospasm and tocolysis.

Dose: bronchospasm (adult): 0.25 mg SQ; repeat in 15 min prn; (ped): 3.5-5.0 mcg/kg SQ. Tocolysis: 10 mcg/kg IV infusion up to 80 mcg/min

Adverse effects: may cause dysrhythmias, pulmonary edema, hypertension, hypokalemia, or CNS excitement.

Vasopressin (Pitressin)

Actions: synthetic analogue of arginine vasopressin; antidiuretic; produces contraction of the smooth muscle.

Indications: diabetes insipidus, GI bleeding, shock-refractory VF.

Dose (GI hemorrhage): start infusion of 0.2-0.4 units/min then titrate.

Dose (cardiac arrest): 40 units IV/IO/ET.

Standard conc. 200 units vasopressin in 250 cc D_5W (0.8 units/mL)

Adverse effects: cardiotoxicity (myocardial ischemia, bradycardia), abdominal cramps, nausea, diarrhea, bowel infarction, water intoxication.

Comments: usually combined with nitroglycerin infusion to reverse cardiotoxic effects while reducing the increase in portal venous resistance.

Verapamil (Calan)

Actions: blockade of calcium channels in heart; prolongation of PR interval with negative inotropy and chronotropy; systemic and coronary vasodilation.

Indications: supraventricular tachycardia, atrial fibrillation or flutter, Wolff-Parkinson-White syndrome.

Dose (adult): 2.5 -10 mg (75-150 mcg/kg) IV over 2 minutes. If no response in 30 minutes, repeat 5-10 mg.

Dose (ped): 0.1-0.3 mg/kg IV, repeat once if no response in 30 minutes.

Adverse effects: may cause severe bradycardia, AV block, excessive hypotension, congestive heart failure. May increase ventricular response to atrial fibrillation or flutter in patients with accessory tracts.

Drip Rates

cc/hr = (mcg/kg/min x 60 x wt(kg))/mcg/cc

mcg/kg/min = [(cc/hr) x (mcg/cc)]/60 x wt(kg)

Drug	**Adult IV Dose**	**Dose Interval**
Ampicillin	1 gm	q4-8 hrs
Cefazolin	0.5-1.0 gm	q4-8 hrs
Cefotetan	1-2 gm	q12 hrs
Clindamycin	600 mg	q6-8 hrs
Erythromycin	0.5-1.0 gm	q6 hrs
Gentamicin	60-120 mg	q8-12 hr (over 20 min)
Metronidazole	500 mg	q6 hrs (over 15 min)
Tobramycin	60-120 mg	q8 hrs

Vancomycin	0.5-1.0 gm	q6-8 hrs (over 30+ min)

Cardiovascular Physiology

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Parameter	**Formula**	**Normal Range**
RA, CVP		0-10 mmHg
LA or LVEDP		4-12 mmHg
RV		15-30/0-10 mmHg
PAS/ PAD		15-30/5-15 mmHg
MPAP	PAD + (PAS-PAD/3)	10-20 mmHg
PCWP		5-15 mmHg
MAP	DBP + (SBP-DBP/3) or	75-110 mmHg
SVR	(MAP-CVP x 79.9)/CO	900-1500 dynes/sec/cm5
PVR	(MPAP-PCWP x 79.9)/CO	50-150 dynes/sec/cm5
$Ca0_2$	(Hgbx1.34)SaO_2+(PaO_2x 0.0031)	16-22 mL O_2/dL blood
CvO_2	(Hgbx1.34)SvO_2+(PaO_2x 0.0031)	12-77 mL O_2/dL blood
C(a-v)O_2	(Hemoglobin x 1.34)(SaO_2 - SvO_2)	3.5-5.5 mL O_2/dL blood
SV	CO x 1000/HR	60-100 mL
CO	SV x HR=VO_2/C(a-v)O_2x10	4-8 liters/min
CI	CO/body surface area	2.5-4.0 L/min/m2
DO_2	CaO_2 x CO x 10	700-1400 mL/min
PAO_2	FIO_2 x (PB-PH_2O)-	

	($PaCO_2$/0.8)	
A-a gradient	[(713)FIO_2-$PaCO_2$ (1.25)]-PaO_2	2-22 mmHg (RA)
Qs/Qt	(CcO_2-CaO_2)/(CcO_2-CvO_2)	0.05 or less
Cr Clearance	(Urine Cr/Serum Cr) x 70	Male=125; Female=105
PaO_2	102-(age/3)	
SvO_2 (mixed)	SaO_2-VO_2/(CO x Hemoglobin)(13.4)	68-77%
VO_2	Hemoglobin x 1.34 x CO x 10 x (SaO_2-SvO_2)	
Fick equation (VO_2)	CO x C(a-v)O_2	225-275 mL/min
Body Mass Index	wt (kg)/ht $(m)^2$	Obese >28;Morbidly >35
V_D/V_T	(PaCO2-P_ECO_2)/$PaCO_2$	Normal: 33%.

Interpretation format	**Normal intervals (each small block=0.04 sec)**		
1. Rate	P-R: 0.12-0.20 msec	Q-T (msec)	HR (bpm)
2. Rhythm	QRS: 0.06-0.10 msec	0.33-0.43	60
3. Intervals		0.31-0.41	70
4. Axis		0.29-0.38	80
5. Hypertrophy		0.28-0.36	90
6. Infarction/ischemia		0.27-0.35	100
7. Ectopy			

Leads with EKG Changes	Injury/Infarct Related Artery	Area of Damage	Associated Complications
V1-V2	LCA:LAD-septal branch	Septum, His bundle, bundle branches	Infranodal block and BBBs
V3-V4	LCA: LAD-diagonal branch	Anterior wall LV	LV dysfunction, CHF, BBBs, complete heart block, PVC
V5-V6 plus 1 and aVL	LCA:circumflex	High lateral wall LV	LV dysfunction, AV nodal block in some
II, III, aVF	RCA: posterior descending branch	Inferior wall LV, posterior wall LV	Hypotension, sensitivity to nitroglycerin and morphine
V4R (II, III, aVF)	RCA: proximal branches	RV, inferior wall LV, posterior wall LV	Hypotension, supranodal and AV-nodal blocks, atrial fib/flutter, PACs, adverse medical reactions

V1-V4 (marked depression)	Either LCA-circumflex or RCA-posterior descending branch	Posterior wall LV	LV dysfunction

Hyperkalemia — Tall peaked T waves, QRS widening, ST elevation, loss of P waves
Hypokalemia — Small T wave/U wave, QRS widening, ST depression
Hypercalcemia — Shortening of QTc
Hypocalcemia — Prolongation of QTc

Location of MI	**Q wave or ST change**	**Reciprocal ST depress**
Anterior	V2-V4 (poor R wave prog)	II, III, AVF
Antero-Septal	V1-V3	
Antero-Lateral	I, aVL, V4-V6	
Lateral	I, AVL, V5-V6	V1, V3
Inferior	II, III, AVF	I, AVL
Posterior	Tall R/T waves V1-V3	V1-V3
Subendocardial (Q wave >0.04 sec	ST depression in ant leads or	

and >25% the height of the R wave)	inferior leads	

LVH Criteria	**RVH Criteria**	**Atrial Hypertrophy**
RV5 or RV6 >26 mm SV1 or SV2 + RV5 or RV6 >35 mm R I +S III >25 mm RaVL >13 mm RaVF >20 mm	R>S in V1 or V3 qR in V1 or V3 RAD Wide QRS	RAH: diphasic P with tall initial component LAH: diphasic P with persistent S in V4-V6 wide terminal component

Left BBB	**Right BBB**
Absence of septal Q waves in V4-V6, I, AVL RR' or M pattern of QRS in I, aVL, V4-V6 2E ST, T wave change I, aVL, V4-V6	RR' or M pattern of QRS in V1-V3 Deep/round S waves in I, aVL,V4-V6 2E ST, T wave change in V1-V3

Atrioventricular Heart Blocks

1. First-degree heart block: PR interval >0.20 sec.
2. Second degree heart block
   A. Mobitz Type I (Wenckebach): PR interval increases until QRS dropped, delta wave.
   B. Mobitz Type II (infra His) PR interval constant until QRS dropped.
3. Third degree heart block: no AV conduction (P has no relation to QRS).

Electrocardiogram Changes

1. Prolonged QT Interval: hypocalcemia, hypokalemia, hypomagnesemia, acute MI, acute myocarditis, procainamide, quinidine, tricyclics.
2. Shortened QT Interval: hypercalcemia, digitalis.
3. LAD: LVH, left ant hemiblock, inferior wall MI.
4. RAD: RVH, left post hemiblock, dextrocardia, pulmonary infarct, RBBB, lateral MI.
5. Pericarditis: diffuse ST elevation concave upward and/or diffuse PR depression and/or diffuse T wave inversion.
6. Digitalis toxicity: ventricular arrhythmias, conduction abnormalities. Quinidine/procainamide: prolonged QT, flattened T wave, QRS widening.
7. Hypothermia: bradycardia, AV junctional, elevated J point, prolong QT.
8. Orthotopic heart transplantation: the patient's original SA node often remains with the original atria, therefore, two P waves can be seen.
9. Head injuries: dysrhythmias and electrocardiographic abnormalities in the T wave, U wave, ST segment, and QT interval are common following head injuries but are not necessarily associated with cardiac injury; they likely represent altered autonomic function.

Electrolyte Abnormalities on EKG

1. Hyperkalemia: tall peaked T waves, QRS widening, ST elevation, and loss of P waves.
2. Hypokalemia: small T wave/U wave, QRS widening, ST depression.
3. Hypercalcemia: shortening of QTc.
4. Hypocalcemia: prolongation of QTc.

EKG Detection of Perioperative Myocardial Ischemia

1. Single lead EKG sensitivity: V5 (75%), V4 (61%), V6 (37%), V3 (33%), II (24%), and all others <14%.
2. Combination leads: leads II and V5 increase sensitivity to 85%, leads V4 and V5 increase sensitivity to 90%, increasing to 96% by combining II, V4, and V5, and to 100% when five leads were used (V2-V5 and II).

Pacemakers

1. **Preoperative pacemaker evaluation**
   A. Determine the indications for the pacemaker.
   B. Determine type of generator, date placed, and the preset rate.
   C. Define pacemaker function.
   D. The patient should be questioned for history of vertigo, syncope, light headedness, or return of any pre-pacemaker symptoms which may reflect dysfunction of the pacemaker.
   E. Labs: serum electrolytes (hypokalemia can increase the negative cell membrane potential increasing threshold for pacemaker to capture).
   F. Chest x-ray: looking for a dislodged electrode or fracture, and the make and model, if available.

First letter: chamb	**Second letter: chambe r**	**Third letter: generat**	**Fourth letter: program**	**Fifth letter: antitachy ?cardia**

er paced	**sensed**	**or response**	**functions**	**functions**
V-Ventricle	V-Ventricle	T-Triggered	P-Program[2]	B-Bursts
A-Atrium	A-Atrium	I-Inhibited	M-Multi-program	N-Normal rate competition[4]
D-Dual chamber	D-Dual chamber	D-Dual chamber	C-Commun?icating	S-Scanning[5]
	O-None (Async)	O-None (Async)	O-None (fixed function)	E-External[6]
		R-Reverse Functions[1]		

1. Pacemaker activated at fast rates only.
2. Rate and/or output only.
3. Telemetry, interrogation (P or M implicit).
4. Paces at normal rate upon sensing tachyarrhythmia (underdrive pacing).
5. Scanning response (such as time extrasystoles).
6. External control (activated by a magnet, radio-frequency, or other means).

7. **Intraoperative management**
   A. The grounding pad for the electrocautery should be placed as far away from pulse generator as possible.
   B. Monitor heart rate during electrocautery with a stethoscope, pulse oximetry, or arterial line.
   C. A magnet placed over the pulse generator will convert a demand (VVI) pacemaker into an asynchronous (VOO) pacemaker.
8. **Indications for temporary pacemaker:** symptomatic sick sinus syndrome, symptomatic hypertensive carotid sinus syndrome with cardio-inhibitory (not just vasodepressor) response, Mobitz type 2 block, acute MI with RBBB or LBBB, comatose trauma patient with bifascicular block, trifascicular block, symptomatic beta blocker overdose.

Respiratory Physiology

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9. **Pulmonary function tests**
   A. **Predicted vital capacity**
      1. Women. (21.78 - [0.101 x age in years]) x height in cm.
      2. Men. (27.63 - [0.112 x age in years]) x height in cm.
   B. **PFTs associated with increased pulmonary morbidity**
      1. FEV_1 < 2 liters.
      2. FEV_1/FVC < 0.5.
      3. Vital capacity < 15 mL/kg.
      4. Maximum breathing capacity < 50% of predicted.
10. **Hemoglobin dissociation curve**
   A. **Factors shifting the curve to the right** (decreasing Hemoglobin affinity for O_2).
      1. Increasing hydrogen ion concentration (decreased pH).
      2. Increased 2,3-DPG concentration.
      3. Increased body temperature.
   B. **P_{50}** (oxygen tension at which hemoglobin is 50% saturated)
      1. Normal adult hemoglobin. 26 mmHg.
      2. Fetal hemoglobin. 19 mmHg.
      3. Parturient maternal hemoglobin. 30 mmHg.
      4. Sickle hemoglobin. 31 mmHg.
      5. Erythrocytes stored for 28 days at 1-6 EC. 17 mmHg.

Condition	**Vital Capacity***	**FEV_1***	**Max Vol Vent***	**Residual Vol***	**DLCO$^+$**	**PaO_2**	**PaCO_2**
Normal	>80	>75	>80	80-120	25-30	80-100	38-42
Restrictive							
Mild	60-80	>75	>80	80-120	-	nl	nl
Moderate	50-60	>75	>80	70-80	--	-	-
Severe	35-50	>75	60-80	60-70	---	-	-
Very	<35	>75	<60	<60	---	--	+
Obstruc?tive							
Mild	>80	60-75	65-80	120-150	nl	-	nl
Moderate	>80	40-60	45-65	150-175	nl	--	-
Severe	-	<40	30-45	>200	-	--	+
Very	-	<40	<30	>200	-	---	++

*Percent predicted; **Percent vital capacity; $^+$

mL/min/mmHg; - = Decrease; + = Increase

Factor	**Abd Surgery**	**Thoracotomy**	**Lob/Pneumonectomy**
FVC	< 70%	< 70%	< 50% or < 2 L
FEV_1	< 70%	< 1 L	< 1 L
FEV_1/FVC	< 50%	< 50%	< 50%
$FEF_{25-75\%}$	< 50%	< 50%	
RV/TLC			> 40%
$PaCO_2$	> 45-50 mmHg	> 45-50 mmHg	
Percent values are percent of predicted			

	Newborn	**1-24 mths**	**7-19 yrs**	**Adult**	**Mixed Venous**	**Venous**
pH	7.37	7.40	7.39	7.37 - 7.44	7.31-7.41	7.31- 7.41
PaO_2	15	90	96	80-100	35-40	30-50
$PaCO_2$	33	34	37	35-45	41-51	40-52
O_2 Sat				>98	60-80	60-85
HCO_3	20	20	22	22-26	22-26	22-28

Parameter	**Neonate**	**Infant**	**5 years**	**Adult**
Resp Rate	40-50	24-30	20	15
TV (mL/kg)	6	6	6	6
Dead space	2 cc/kg			2 cc/kg
FRC (cc/kg)	25	25	35	35
O_2	5-6			2-3

consumption (cc/kg/min)				
Alveolar ventilation	130 cc/kg			60 cc/kg
VA/FRC	4.5:1			1.5:1
Closing volume				Increased
Dead Space				2 cc/kg
Vital Capacity	35 cc/kg			60-70 cc/kg
FRC: functional residual capacity; TV: tidal volume				

Oxygenation and Ventilation

1. **Major causes of hypoxemia**
   A. Low inspired oxygen concentration (decreased FiO_2).
   B. Hypoventilation.
   C. Shunt (normal shunt about 2%): hypoxemia caused by shunt cannot be overcome by increasing the inspired oxygen concentration.
   D. Ventilation perfusion (V/Q) mismatch: common causes of V/Q mismatch are atelectasis, patient positioning, bronchial intubation, one-lung ventilation, bronchospasm, pneumonia, mucus plugging, acute respiratory distress syndrome (ARDS) and airway obstruction.
   E. Diffusion abnormalities.
   F. Cardiac output - oxygen carrying capacity abnormalities (CO/O_2 capacity): as cardiac output or oxygen carrying capacity decrease, oxygen delivery will decrease resulting in hypoxemia.
2. **Causes of hypercarbia**
   A. **Hypoventilation:** common causes include muscle paralysis, inadequate mechanical ventilation, inhalational anesthetics, and narcotics.
   B. **Increased CO_2 production:** including malignant hyperthermia, fever, and thyrotoxicosis.
   C. **Iatrogenic:** common examples include sodium bicarbonate administration and depletion of the CO_2 absorbent.
3. **Methods to improve oxygenation**
   A. Increase FiO_2.
   B. Increase minute ventilation.
   C. Increase cardiac output (and increase oxygen delivery to tissues).
   D. Increase oxygen carrying capacity (hemoglobin).
   E. Optimize V/Q relationships.
   F. Cardiopulmonary bypass.
   G. Decrease oxygen consumption from pain, shivering, or fever.

Arterial Blood Gases

1. **Golden rules of ABGs**
   A. $PaCO_2$ change of 10 corresponds to a pH change of 0.08.
   B. $PaCO_2$ increases HCO_3 concentration. initially by 1; chronically by 3.
   C. $PaCO_2$ decreases HCO_3 concentration. initially by 2; chronically by 5.
   D. pH change of 0.15 corresponds to BE change of 10 mEq/l.
2. **Total body bicarbonate deficit** = base deficit (mEq/L) x patient wt (kg) x 0.4.
3. **Bicarbonate deficit** (HCO_3 deficit) = (total body water) x (24 - HCO_3).

4. **Base excess (BE) or deficit**
   A. BE = HCO_3 + 10(pH - 7.40) - 24.
   B. Base excess or deficit is a calculated value that gives an estimation of "acid load."
   C. Negative values of base excess (ie, deficit) represent metabolic acidosis, and positive values indicated metabolic alkalosis.
5. During apnea, $PaCO_2$ increases 5-6 during the first minute and 3-4 for every minute thereafter.
6. **Henderson-Hasselbach equation**
   A. pH = 6.1 + log[(HCO_3)/(0.03 x $PaCO_2$)]
   B. Modified equation. (H+) = [24 x pCO_2] / HCO_3
   C. pH = pk + log A-/HA
7. **PaO_2 age adjustment:** PaO_2 = 102 - [age in years/3]
8. **Anion gap**
   A. Anion gap = Na - (Cl + HCO_3)
   B. Normal anion gap = 8-16 mEq/l

Increased anion gap (S.L.U.M.P.E.D.) **S**alicylates **L**actate **U**remic toxins **M**ethanol **P**araldehyde **E**thanol/ethylene glycol **D**iabetic ketoacidosis	**Normal anion gap** Renal causes Renal tubular acidosis Carbonic anhydrase inhibitors Lysine or arginine HCl GI bicarbonate loss Diarrhea Pancreatic fistula Ureterosigmoidostomy Addition of HCl Ammonium chloride

Airway Management

?

Tracheal Intubation

1. **Airway innervation**
   A. **Nasal mucosa:** sphenopalatine ganglion a branch of the middle division of cranial nerve V (trigeminal Nerve). The ganglion is located on the lateral wall posterior to the middle turbinate.
   B. **Uvula, tonsils, superior pharynx:** innervated by continued branches from the sphenopalatine ganglion.
   C. **Oral pharynx and supraglottic area:** innervated by branches of CN IX (glossopharyngeal nerve). These branches include lingual, pharyngeal, and tonsillar nerves.
   D. **Trachea:** innervated by the recurrent laryngeal nerve.
   E. **Larynx:** sensory and motor is from the Vagus (CN X)
      1. **Sensory:** above the vocal folds innervated by the internal branch of the superior laryngeal nerve; below the vocal folds innervated by the recurrent laryngeal nerve.
      2. **Motor:** all muscles are supplied by the recurrent laryngeal nerve except for the cricothyroid muscle which is supplied by the external branch of the superior laryngeal nerve.
2. **Common indications for tracheal intubation:** provide patent airway, protection from aspiration, facilitate positive-pressure ventilation, operative position other than supine, operative site near or involving the upper airway, airway maintenance by mask is difficult, disease involving the upper airway, one-lung ventilation, altered level of consciousness, tracheobronchial toilet, severe pulmonary or multisystem injury.
3. **Confirmation of tracheal Intubation**
   A. Direct visualization of the ET tube passing though the vocal cords.
   B. Carbon dioxide in exhaled gases (documentation of end-tidal CO_2 in at least three consecutive breaths).
   C. Bilateral breath sounds.
   D. Absence of air movement during epigastric auscultation.
   E. Condensation (fogging) of water vapor in the tube during exhalation.
   F. Refilling of reservoir bag during exhalation.
   G. Maintenance of arterial oxygenation.
   H. Chest x-ray: the tip of ET tube should be between the carina and thoracic inlet or approximately at the level of the aortic notch or at the level of T5.
4. **Extubation criteria**

	NIF > -20 cm H_2O	Resting min vent < 10 l/min
	RR < 30/min	LOC stable or improving
TV > 5 cc/kg	TV/RR > 10	
VC > 10 cc/kg	Qs/Qt < 20%	
PaO_2 > 65-70 mm (FIO_2 < 40%)	Pmep > +40 cm H_2O	
$PaCO_2$ < 50 mm	Vd/Vt < 0.6	

5. **Complications of endotracheal intubation**
   A. **Complications occurring during intubation:** aspiration, dental damage (chip tooth), laceration of the lips or gums, laryngeal injury, esophageal intubation, endobronchial intubation, activation of the sympathetic nervous system (high BP and HR), bronchospasm.
   B. **Complications occurring after extubation:** aspiration, laryngospasm, transient vocal cord incompetence, glottic or subglottic edema, pharyngitis or tracheitis.
6. **Endotracheal tube recommendations**
   A. **Endotracheal tube size** (mm): for children older then 2 years ETT can be estimated by: Age/4 + 4.
   B. **Length of Insertion (cm) of ETT**
      1. Under 1 year: 6 + Wt(kg).
      2. Over 2 years: 12 + Age/2.

3. Multiply internal diameter (mm) of ETT by 3 to give insertion (cm).
4. Add 2-3 cm for nasal tube.

C. **Pediatrics:** generally use uncuffed tubes in patients under 10 years. When a cuff tube is used maintain endotracheal leak at 15-20 cm H_2O.

Age	**Laryngoscope**	**Endotracheal Tube Size (mm)**	**Distance at Teeth (cm)**	**Suction Catheter (F)**
Neonate <1000 g	Miller 0	2.5	6.5-7.0	5-6
Neonate 1000-2000 g		3.0		
Neonate 2000-3000 g		3.0-3.5		
Term Infant	Miller 0-1 Wis-Hipple 1 Robertshaw 0	3.0-3.5	9-10	6-8
6 months		3.5-4.0	10	8
1 year	Wis-Hipple 1.5 Robertshaw 1	4.0-4.5	11	8
2 years	Miller 2 Flagg 2	4.5-5.0	12	8
4 years		5.0-5.5	14	10
6 years		5.5	15	10

8 years	Miller2-3 Macintosh 2	6.0	16	10
10 years		6.5	17	12
12 years	Macintosh 3	7.0	18	12
Adolescent Adult	Macintosh 3 Miller 3	7.0-8.0	20	12

Endotracheal Intubation Under Anesthesia

1. **Preparation for intubation**

A. **Preoperative evaluation** of the airway will help determine the route (oral or nasal) and method (awake or anesthetized) for tracheal intubation. See preoperative evaluation section for airway exam.

B. **Equipment:** laryngoscope with working light, endotracheal tubes of appropriate sizes, malleable stylet, oxygen supply, functioning suction catheter, functioning IV, and appropriate anesthetic drugs.

C. **Cricoid pressure (Sellick's maneuver):** used to minimize the spillage of gastric contents into the pharynx during the period of time from induction of anesthesia (unconsciousness) to successful placement of a cuffed tracheal tube. An assistant's thumb and index finger exert downward pressure on the cricoid cartilage (approximately 5 kg pressure) so as to displace the cartilaginous cricothyroid ring posteriorly and thus compress the esophagus against the underlying cervical vertebrae.

D. Induction of anesthesia prior to tracheal intubation may include injected and/or inhaled anesthetic drugs.

2. **Orotracheal intubation**

A. **Head position:** place the head in the "sniffing" position if there is no cervical spine injury. The sniffing position is characterized by flexion of the cervical spine and extension of the head at the atlantooccipital joint (achieved by placing pads under the occiput to raise the head 8-10 cm). This position serves to align the oral, pharyngeal, and laryngeal axes such that the passage from the lips to the glottic opening is most nearly a straight line. The height of the OR table should be adjusted to bring the patient's head to the level of the anesthesiologist's xiphoid cartilage.

B. Hold the laryngoscope in the palm of the left hand and introduce the blade into the right side of the patient's mouth. Advance the blade posteriorly and toward the midline, sweeping the tongue to the left. Check that the lower lip is not caught between the lower incisors and the laryngoscope blade. The placement of the blade is dependent on the blade used.

1. **Macintosh (curve) blade:** the tip of the curved blade is advanced into the valleculum (the space between the base of the tongue and the pharyngeal surface of the epiglottis).
2. **Miller (straight) blade:** the tip of the straight blade is passed beneath the laryngeal surface of the epiglottis, epiglottis is then lifted to expose the vocal cords.

C. Regardless of the blade used, lift the laryngoscope upward and forward, in the direction of the long axis of the handle, to bring the larynx into view. Do not use the upper incisors as a fulcrum for leverage because this action may damage the upper incisors and may push the larynx out of sight.

D. The vocal cords should be visualized prior to endotracheal placement. The glottic opening is recognized by its triangular shape and pale white vocal cords. Posteriorly, the vocal cords terminate in the arytenoid cartilages. The tube should be seen to pass between the cords, anterior to the arytenoids. Insert the tube into the pharynx with the right hand from the right side of the mouth; it should pass without resistance through the vocal cords (about 1-2 cm). The endotracheal tube cuff should lie in the upper trachea but beyond the larynx.

E. Once the endotracheal tube is in place, inflate the cuff, confirm endotracheal intubation and secure the endotracheal tube. In order to minimize the pressure transmitted to the tracheal mucosa, the

cuff should be inflated with the least amount of air necessary to create a seal during positive pressure ventilation. For patients intubated outside the operating room, obtain a portable chest x-ray following intubation to confirm tube placement and bilateral lung expansion.

3. **Nasotracheal intubation**

A. A vasoconstrictor should be applied before nasal instrumentation. After anesthesia is induced the mask ventilation is established, the endotracheal tube can be placed.

B. Generously lubricate the nare and endotracheal tube. Soften the endotracheal tube tip by immersing it in hot water. The endotracheal tube should be advanced through the nose directly backward toward the nasopharynx with the Murphy eye orientated anteriorly facing the epiglottis. A loss of resistance marks the entry into the oropharynx.

C. The laryngoscope and Magill forceps can be used to guide the endotracheal tube into the trachea under direct vision (if needed). A fiberoptic bronchoscope can be utilized to direct the tube into the trachea.

4. **Rapid sequence induction/intubation**

A. **Indications:** patients who are at risk for aspiration (eg, history of recent meal, gastroesophageal reflux, pregnancy, trauma) and there is reasonable certainty that intubation should not be difficult.

B. **Method**

1. Nonparticulate antacids, H_2-blockers and metoclopramide may be used preoperatively to decrease the acidity and volume of gastric secretions.

2. Equipment similar to that for any intubation but commonly includes several endotracheal tubes with stylet and cuff-inflation syringe in place, laryngoscope blades, functioning suction, and a patent IV.

3. Preoxygenate with 100% oxygen by mask. Four maximal breaths of 100% oxygen over 30 seconds is as effective as breathing 100% oxygen spontaneously for 3-5 minutes.

4. Premedicate as appropriate (fentanyl, atropine, lidocaine, defasiculating agent).

C. Induction is accomplished with any induction agent. Just before administration of the induction agent, cricoid pressure (Sellick's maneuver) should be applied.

D. Muscle relaxant is usually given to help facilitate intubation. Succinylcholine (1-1.5 mg/kg; use 2.0 mg/kg for infants and children) given immediately after the induction agent. Once the induction agent and muscle relaxant are given, there should be no attempt to ventilate the patient by mask.

E. Intubation should be performed as soon as jaw relaxation has occurred. Cricoid pressure should be maintained until confirmation of tracheal placement of the endotracheal tube has ben confirmed. If the first attempt to intubate fails, cricoid pressure should be maintained continuously during all subsequent maneuvers, while mask ventilation with 100% oxygen is administered.

Conscious Intubation

1. Consideration should be given for patients with suspected or previous history of a difficult intubation, acute processes that may comprise the airway, mandibular fractures or other significant facial deformities, morbid obesity, or cancer involving the larynx.

2. Discuss with the patient the indications, reasons, and the plan.

3. **Preparation:** as with all intubations, appropriate equipment, etc, should be readily available. A plan (and a back-up plan) should be formulated.

4. **Preparing the patient:** consider premedicating with drying agent (ie, glycopyrrolate 0.2 mg IV) 30 minutes before the procedure. If considering a nasal intubation, give 4 drops of 0.25% Norsynephrine to each nare to help minimize bleeding. Other vasoconstrictors include oxymetazoline (Afrin) and cocaine. After standard monitors are placed consider sedation (midazolam, fentanyl, etc.) and titrate to effect.

5. **Topical anesthesia** of the upper airway can be accomplished with various agents (see table) and/or nerve blocks.

6. **Airway nerve blocks**

A. **Sphenopalatine ganglion** (nasal mucosa)

1. Cotton pledgets soaked with anesthetic solution (usually 20% benzocaine or 4% lidocaine) are placed in the nasal cavity at a 30 degree cephalad angulation to follow the middle turbinate back to the mucosa overlying the sphenoid bone.

2. A second set of pledgets is introduced through the nares and passed along the turbinates to the posterior end of the nasal passage.
3. The pledgets should be left in place for at least 2-3 minutes to allow adequate diffusion of local anesthetic.

B. **Lesser and pharyngeal palatine nerves**
1. **Landmarks:** 1 cm medial to the third maxillary molar and 1 cm anterior to the junction of the hard and soft palates (usually 0.5 cm in diameter).
2. Place a cotton pledget soaked with anesthetic solution on this site and wait 1 minute (provides topical anesthesia).
3. Using a 25 g spinal needle create a 90 degree bend 3 cm from the tip. Probe the mucosa with the needle to find the a palatine foramen (usually up to 3), angulate the needle 15 degrees medially and advance 3 cm up the canal. After negative aspiration, inject 1-3 cc of 1-2% lidocaine with epinephrine.

C. **Glossopharyngeal nerve:** insert a 25 g spinal needle into the base of the posterior tonsillar pillar. After negative aspiration, inject 2-3 cc of 1-2% lidocaine with epinephrine. Repeat block on opposite side.

D. **Superior laryngeal nerve**
1. Place the patient supine with the neck extended.
2. Find the thyrohyoid membrane (a soft depression between the hyoid and thyroid bones) and displace the hyoid bone laterally toward the side to be blocked.
3. Insert a 25 g needle off the greater cornu of the hyoid bone, inferiorly, and advance 2-3 mm. As the needle passes through the thyrohyoid membrane, a slight loss of resistance is felt. Inject 2-3 cc of 1-2 % lidocaine with epinephrine. Repeat the block on opposite side.

E. **Translaryngeal (transtracheal) nerve block**
1. Landmarks: cricothyroid membrane (located between the thyroid cartilage superiorly and the cricoid cartilage inferiorly).
2. Insert a 20 g angiocath, bevel up, at the upper edge of cricoid cartilage in the midline. Aspirate for air to confirm placement. Remove the needle, leaving only the angiocatheter. Inject 3-5 cc of 2-4% lidocaine solution at end inspiration. This will usually result in a vigorous cough.

7. **Oral intubations**: after proper preparation of the patient, oral intubation can be accomplished with direct laryngoscopy or indirectly with a rigid stylet fiberoptic laryngoscope (ie, the Bullard blade).

8. **Nasal intubations**

9.

A. After proper preparation, nasal intubation can be accomplished blindly or with the assistance of a direct laryngoscopy and Magill.

B. Blind technique: while listening for breath sounds at the proximal end of the endotracheal tube, advance the tube during inspiration. A cough followed by a deep breath, condensation in the tube from exhaled moisture, and loss of voice suggest tracheal entry.

Fiberoptic-Assisted Tracheal Intubation

1. **Indications:** upper airway obstruction, mediastinal mass, subglottic edema, congenital upper airway abnormalities, immobile cervical vertebrae, verify position of a double-lumen endobronchial tube.
2. **Nasal technique:** after the patient's nares and nasopharynx are anesthetized and vasoconstricted, the tracheal tube is passed through the naris into the posterior nasopharynx. The lubricated bronchoscope is then passed through the tracheal tube until the epiglottis and glottic opening are visualized continuing until the carina is identified. Pass the tube over the scope while the view of the carina is maintained.
3. **Oral technique:** an intubating oral airway (or bite block) is inserted after topicalization of the posterior tongue, soft palate, and lateral oropharyngeal areas. The tracheal tube is inserted about 8-10 cm into the airway and the bronchoscope passed through the tube. The posterior tongue, epiglottis, glottis and carina should be visualized in order. The tube is then passed over the scope while keeping the carina in view.

Medication	**Dose**	**Route**	**Comments**
Cocaine (4-10%)	40-160 mg	Intranasal	Good anesthetic, vasoconstrictor, may cause coronary vasospasm
1% Phenylephrine	1-2 mL	Intranasal	Vasoconstrictor
Cetacaine Spray	2-4 sprays	Oral	Contains benzocaine
2 % Viscous Lidocaine	2-4 mL	Oral	
1% Lidocaine	2-3 mL	Airway blocks	Aspirate before injection
4% Lidocaine	2-3 mL	Transtracheal	Aspirate before injection
Afrin Spray	2-4 sprays	Intranasal	

Transtracheal Ventilation (Cricothyrotomy)

1. **Indications:** can be used as a temporizing measure if mask ventilation and oxygenation become inadequate or is not possible.
2. **Technique:** a catheter (12- or 14-gauge) is connected to a jet-type ventilator, which in turn is connected to an oxygen source capable of delivering gas at a pressure around 50 psi, and inserted into the trachea through the cricothyroid membrane. The gas is delivered intermittently by a hand-held actuator. The duration of ventilation is best assessed by watching the rise and fall of the chest: an I:E ratio of 1:4 seconds is recommended.
3. Oxygenation usually improves rapidly, however, retention of carbon dioxide may limit the duration of the usefulness of the technique.
4. **Complications:** catheter displacement (caused by high pressures created by jet ventilation), pneumomediastinum.

Laryngeal Mask Airway

1. **Indications for LMA**
   A. In place of a face mask or endotracheal tube.
   B. In place of an endotracheal tube, when breathing is being controlled, as long as the inflation pressure is not more than 30 cm H_20.
   C. To aid in the management of the difficult airway (ie, the LMA can be used as a guide for fiberoptic intubation).
2. **Contraindications for LMA**
   A. The LMA does not provide an airtight seal of the airway and, thus, does not protect against gastric regurgitation and pulmonary aspiration.
   B. When controlled ventilation is likely to require a high-inflation pressure of more than 30 cm H_2O.
3. **Insertion of the LMA-Classic**
   A. Propofol (2.5-3.0 mg/kg) is the agent of choice for LMA insertion. Propofol relaxes the jaw and pharyngeal muscles better than thiopental.
   B. The leading edge of the deflated cuff should be wrinkle-free and facing away from the aperture. Lubricate only the back side of the cuff with a water soluble lubricant.
   C. The LMA is held like a pencil and is inserted blindly in the midline with concavity forward while pressing on the anterior shaft with the tip of the index finger toward the hard palate and guiding it toward the pharynx.
   D. When the upper esophageal sphincter is reached, a characteristic resistance is felt. The cuff is then inflated with air (the cuff should be inflated without holding the tube to enable the expanding cuff to find its correct position in the pharynx).
   E. When correctly placed, the black vertical line on the posterior aspect of the tube should always face directly backward, toward the head of the patient.
   F. The LMA should be left in place until the patient can open his mouth on command. During emergence, the patient should not be stimulated (ie, suctioned), and the cuff should not be deflated until the patient can open his mouth on command.
   G. A bite block (or folded gauze) is inserted in the mouth to protect the LMA.
4. **LMA-Fastrach**
   A. **To insert the LMA-Fastrach**
      1. Deflate the cuff of the mask and use a water soluble lubricant on the posterior surface. Rub the lubricant over the anterior hard palate.
      2. Swing the mask into place in a circular movement maintaining contact against the palate and posterior pharynx. Don't use handle as lever.
      3. Inflate the mask, without holding the tube or handle, to a pressure of approximately 60 cm H_2O.
   B. **To insert endotracheal tube and remove the LMA-Fastrach**
      1. Hold the LMA-Fastrach handle while gently inserting the lubricated ET tube into the metal shaft. The use of a standard, curved, PVC ET tube is not recommended.
      2. Advance tube, inflate the ET tube cuff and confirm intubation.
      3. Remove the connector and ease the LMA-Fastrach out by gently swinging the handle caudally. Use the stabilizing rod to keep the ETT in place while removing the LMA-Fastrach until the tube can be grasped at the level of the incisors.
      4. Remove the stabilizing rod and gently unthread the inflation line and pilot balloon of the ET tube. Replace the ET tube connector
5. **LMA as a conduit for tracheal intubation**
   A. The LMA may be used to provide a conduit to facilitate fiberoptic, gum bougie-guided or blind oral tracheal intubation.
   B. Problems include inadequate ET tube length, limitation on ET tube size, and inability to remove the LMA without risking extubation.
6. **Complications of the LMA**
   A. Possibility of regurgitation and pulmonary aspiration.
   B. Oral and pharyngeal mucosa injury during insertion of the LMA.
   C. Laryngospasm and coughing (may occur in lightly anesthetized patient).
   D. Negative pressure pulmonary edema after improper placement in spontaneously breathing patient.

E. The failure to function properly in the presence of local pharyngeal or laryngeal disease.
F. The need for neck extension in the patient with cervical spine disorder.

Size	**Patient**	**Cuff Vol (ml)**	**Largest ETT**
1	Infant up to 6.5 kg	4	3.5
1.5	5-10 kg		4.0
2	Infants/Children up to 20 kg	10	4.5
2.5	Children between 20 - 30 kg	15	5.0
3	Children/small adults over 30 kg	20	6.0*
4	Adults 50-70 kg	30	6.0*
5	Adults 70-100 kg	30	7.0*
6	Adults greater then 100 kg		7.0*

Esophageal Tracheal Combitube

1. **Uses:** emergency airway control in the difficult airway. Available only in one adult size (age >15 years and height >5 feet).
2. **Insertion**
   A. With the head in the neutral position, insert the ETC, with gentle pressure, up to the black marks (teeth should be between black marks).
   B. Inflate the first pilot balloon (blue cuff) with 100 cc. As the cuff is inflated, the combitube will pop out 1 cm.
   C. Inflate the second pilot balloon (white cuff) with 10-15 cc.
3. **Placement**
   A. Ventilate via longer (blue) lumen.
   B. If breath sounds are present, the ETC is in the esophagus; ventilate.
   C. If no breath sounds are heard, change ventilation to shorter lumen #2 (clear) and recheck for breath sounds. If breath sounds are present, the ETC is in the trachea; continue to ventilate.
   D. If no breath sounds or breath sounds faint, attempt to improve seal by adding up to 60 cc to balloon number 1.
   E. If unable to ventilate, deflate both cuffs, pull back 3 cm and reinflate cuffs. Ventilate via blue lumen and check for breath sounds. If still no breath sounds, deflate cuffs, remove ETC and start algorithm over.
4. **Contraindications**

A. Height less than 5 feet (only one size currently available).
B. Intact gag reflex intact (will not tolerate cuff).
C. Presence of esophageal disease (potential for bleeding or rupture).
D. Ingestion of caustic substances (potential for rupture).
E. Upper airway obstruction (foreign body, glottic edema, epiglottis).

5. **Concerns**
A. Potential for nasopharyngeal, oropharyngeal or tracheal mucosal damage or edema (particularly if left in for greater than 2-8 hours).
B. Inability to suction tracheal secretions when in esophageal position.
C. Only one size available; single use makes it expensive.

Bullard Laryngoscope

1. The Bullard laryngoscope, functioning as an indirect fiberoptic laryngoscope, provides direct visualization of the vocal cords. It is available in both adult and pediatric sizes.
2. The advantage of this laryngoscope is that it can be introduced into the oropharynx with minimal mouth opening (oral opening of 0.64 cm required) and the patient can remain in anatomical position.
3. Preloading the intubating stylet involves lubricating the stylet and positioning the endotracheal tube so that the distal end of the stylet projects through the Murphy's eye of the endotracheal tube.
4. The blade is then inserted into the mouth, with the handle in the horizontal plane, and rotated into the vertical plane allowing it to slide around the midline of the tongue and into the posterior pharynx. Gentle traction is applied against the posterior surface of the tongue to obtain visualization of the glottic aperture.
5. With the stylet pointed directly at the glottic opening, the endotracheal tube is advanced under direct vision into the trachea.

Mechanical Ventilation

1. **Types of mechanical ventilators**
A. **Time cycled:** the tidal volume is delivered and inspiration ends after a preset time interval.
B. **Volume cycled:** the tidal volume is delivered and inspiration ends after a preset time interval.
C. **Pressure cycled:** the tidal volume is delivered and inspiration ends when a preset volume is delivered.

2. **Modes of mechanical ventilation**
A. **Intermittent positive-pressure ventilatory modes (IPPV)**.
1. **Controlled mechanical ventilation (CMV):** mechanical breaths are delivered at a preset rate and tidal volume regardless of the pt effort.
2. **Assist-control ventilation (AC):** A preset minute ventilation is delivered regardless of the patient's effort. Ventilator senses patient-initiated spontaneous breath and delivers a preset tidal volume as well.
3. **Intermittent mandatory ventilation (IMV)**: the ventilator provides tidal volume breaths at a preset fixed rate. In between ventilator-delivered breaths, the patient is able to breathe spontaneously at any rate, tidal volume, or pattern.
4. **Synchronized intermittent mandatory ventilation (SIMV):** similar to IMV, ventilatory breaths timed to coincide with spontaneous effort.
5. **Continuous positive airway pressure (CPAP):** a preset level of positive airway pressure is maintained throughout the respiratory cycle. The patient must be spontaneously breathing.
6. **Inspiratory pressure support ventilation (IPS):** a preset pressure is obtained when the patient initiates an inspiratory effort.
B. **Pressure-controlled ventilation**
1. Maximum airway pressure is set on the ventilator, and tidal volume becomes the dependent variable.

2. The duration of inspiration is determined by setting either the inspiratory time or the I:E ratio. Tidal volume is the product of inspiratory flow and inspiratory time.
3. The primary advantage of pressure-controlled ventilation is reduction in peak airway pressure and potential improvement of gas exchange.

C. **High-frequency ventilation**

1. **High-frequency positive pressure ventilation (HFPPV):** similar to conventional ventilation, however, tidal volumes are very small, and cycling frequencies are very fast (60-300).
2. **High-frequency jet ventilation (HFJV):** a small diameter injecting catheter positioned in the central airway pulses gas along the luminal axis under high pressure at a rapid cycling rate.

D. **Pressure-controlled inverse ratio ventilation (PC-IRV**): set by choosing a prolonged inspiratory time such that the time spend during inspiration exceeds expiratory time.

E.

3. **Positive end-expiratory pressure (PEEP)**
4.

A. **Function of PEEP:** increases oxygenation by maximizing the ventilation-perfusion relationship in the lung. PEEP does this by maximizing the FRC (functional residual capacity), keeping lung volumes greater than closing capacity, therefore maintaining airways open and functional.

B. **Adverse effects of PEEP:** decreased cardiac output, hypotension, worsening hypoxia, barotrauma, increased ICP, decreased urine output.

C.

1. **Ventilator Settings**
2.

A. **FIO_2:** normally start with 40% otherwise use 90-100% until first ABG available (1% decrease in FIO_2 = decrease PaO_2 by 7).

B. **PEEP:** initially none; start with 5 cm H_2O and increase in 3-5 cm H_2O increments if PaO_2 less than 60 mmHg with FIO_2 > 50%; over 10 cm H_2O normally requires pulmonary artery catheter.

C. **Rate:** start at 10-14 (for infants start at 25-30).

D. **Tidal volume:** 10-15 ml/kg (infants 8-12 ml/kg).

E. **Mode:** IMV, SIMV, CPAP, A/C, PSV.

Oxygen Therapy

1. **Nasal cannulas:** FIO_2 increases by 3-4%/liter of O_2 given (up to 45%).
2. **Face masks**
3.

A. **Simple mask:** simple mask may deliver oxygen flow rates from 6-15 liters per minute providing FIO_2 of 35-65 percent.

B. **Venturi mask (air entrainment mask):** delivers up to 40% FIO_2.

C. **Partial rebreathing mask:** simple mask with a valveless reservoir bag and exhalation ports. Can deliver up to 80% FIO_2.

D. **Nonrebreathing mask:** simple mask with reservoir bag and unidirectional valve. Can deliver up to 100% FIO_2.

E. **Aerosol face tent:** delivers oxygen form variable oxygen nebulizer over mouth and nose.

Laboratory Values

?

Test	**Normal Value**
Glucose	40-70 mg/dL
Total Protein	20-45 mg/dL
CSF Pressure	50-180 mm H_2O
Leukocytes	Total<4 per mm3
Lymphocytes	60-70%
Monocytes	30-50%
Neutrophils	1-3%

Test	**Normal Value**
Cr Clearance Males Females	 125 mL/min 105 mL/min
Ur Creat	1.0-1.6 g/d
Ur Protein	<0.15 g/d
Ur K	25-100 meg/d
Ur Na	100-260 meg/d

Test	**Normal Value**
Acid Phosphatase	0-5.5 U/L
Albumin	3.5-5.5 g/dL
Alkaline Phosphatase	30-120 U/L
Aminotransferases AST (SGOT) ALT (SGPT)	 0-35 U/L 0-35 U/L
Ammonia	80-110 mcg/dL
Amylase	60-80 U/L
Bilirubin Total Direct	 0.3-1.0 mg/dL 0.1-0.3 mg/dL

Indirect	0.2-0.7 mg/dL
Calcium	8.6-10.5 mg/dL
CO_2	22-30 mEq/L
Chloride	98-106 mEq/L
Total Cholesterol <29 years 30-39 years 40-49 years >50 years	 <200 mg/dL <225 mg/dL <245 mg/dL <265 mg/dL
HDL	30-90 mg/dL
LDL	50-190 mg/dL
CPK	25-145 U/L
Creatinine	0.4-1.5 mg/dL
Ferritin	15-200 ng/mL
Glucose	70-140 mg/dL
Iron	80-180 mcg/dL
Iron-Binding	250-450 mcg/dL
Iron-Sat	20-45
LDH	25-100 U/L
Lipase	49-220 U/L
Magnesium	1.6-2.6 mg/dL
Osmolality	285-295
Phosphorus	2.5-4.5 mg/dL
Protein	5.5-8.0 mEq/L
Sodium	136-145 mEq/L
Triglycerides	<60 mg/dL
Urea Nitrogen	10-20 mg/dL
Uric Acid Males Females	 2.5-8.0 mg/dL 1.5-6.0 mg/dL

	1 month	**6-12 years**	**Adult**
Male			
WBC	5.0-19.5	5.0-13.5	4.5-11.0
RBC	3.0-5.4	4.0-5.2	4.6-6.2
Hemoglobin	14.0-18.0	11.5-15.5	14.0-18.0
Hematocrit	31-55	35-45	42-52
RDW			11.5-

			14.5
Female			
WBC	5.0-19.5	5.0-13.5	4.5-11.0
RBC	3.0-5.4	4.0-5.2	4.2-5.4
Hemoglobin	14.0-18.0	11.5-15.5	12.0-16.0
Hct	31-55	35-45	37-47
RDW			11.5-14.5

Fluid and Electrolyte Management

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1. **Functional fluid compartments**
   A. **Total body water (TBW):** 60% (adult males) and 50% (adult females) of ideal body weight (IBW).
   B. **Intracellular fluid (ICF):** comprises approximately 35% of IBW or 60% of TBW. Principal potassium containing space.
   C. **Extracellular fluid (ECF):** accounts for 25% of IBW or 40% of TBW and is subdivided into interstitial fluid (ISF) and blood volume (BV; about 8% of TBW). Principal sodium containing space.
2. **Guidelines for intraoperative crystalloid fluid replacement**
   A. **Insensible losses:** 2 mL/kg/hr
   B. **Minor trauma/surgery:** 3-4 mL/kg/hr
   C. **Moderate trauma/surgery:** 5-6 mL/kg/hr
   D. **Major trauma/surgery:** 7-8 mL/kg/hr
3. **Maintenance fluid requirements**
   A. **First 10 kg:** 4 mL/kg/hr or 100 mL/kg/day
   B. **Second 10 kg:** 2 mL/kg/hr or 50 mL/kg/day
   C. **>20 kg:** 1 mL/kg/hr or 20 mL/kg/day
4. **Daily electrolyte requirements**
   A. **Na:** 2-3 mEq/kg/24 hours
   B. **K:** 1-2 mEq/kg/24 hours
   C. **Cl:** 2-3 mEq/kg/24 hours
5. **Determinants of perioperative fluid requirements**: basal requirements, preoperative deficits, third-space losses, transcellular fluid losses, effects of anesthetic agents and technique.
6. **Calculated osmolality** = 2 Na + glucose/18 + BUN/2.8 + ethanol/4.6 + isopropanol/6 + methanol/3.2 + ethylene glycol/6.2 (norm 280-295).
7. **Calcium disturbances**
   A. Normal plasma concentration is 8.5-10.5 mg/dL with 50% free ionized 40% protein bound.
   B. Normal free ionized concentration is 4.5-5 mg/dL.
   C. Corrected calcium = measured calcium / [0.6 + (total protein / 8.5)].
   D. For each 1 gm/dL change in albumin there is a corresponding 0.8 mg/dL change in total calcium (free ionized calcium is not affected).
   E. Ionized calcium increases 0.16 mg/dL for each decrease of 0.1 unit in plasma pH.
8. **Glucose**: for each 100 mg/dL glucose above normal there is a corresponding fall in sodium by 1.6 mEq/l.

Fluid	**Glu gm/L**	**Na**	**Cl**	**K**	**Ca**	**HCO_3**	**Kcal/ L**
D_5W	50						170
NS		154	154				
D5 1/4NS	50	38	38				170
LR		130	110	4	3	27	<10

Blood Therapy Management

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1. **Blood loss management**
   A. Estimated blood volume (EBV)
      1. 100-120 mL/kg for premature infant
      2. 90 mL/kg for full-term infant
      3. 80 mL/kg for infants 3 to 12 months
      4. 70 mL/kg thereafter
   B. Replace every 1 mL blood loss with 3 mL crystalloid or 1 cc PRBC
   C. **PRBC**: one unit PRC increases Hct about 3% and Hemoglobin about 1 g/dL
      1. 3 mL/kg PRC increases Hemoglobin about 1 g/dL
      2. 10 mL/kg PRBC increases Hct about 10%
   D. **Max allowable blood loss**=[EBV x (Hct - target Hct)]/ Hct
   E. **Fluid replacement equivalents**
      1. **Crystalloid:** 3 cc/1 cc estimated blood loss [EBL]
      2. **Colloid:** 1 cc/cc EBL
      3. **Whole blood:** 1 cc/cc EBL
      4. Packed red blood cells: ½ cc/cc EBL
2. **Compatibility testing**
   A. **Type specific:** ABO-Rh typing only; 99.80% compatible.
   B. **Type and screen:** ABO-Rh and screen; 99.94% compatible.
   C. **Type and crossmatch:** ABO-Rh, screen, and crossmatch; 99.95% compatible.
   D. **Screening donor blood:** hematocrit is determined, if normal, the blood is typed, screened for antibodies, and tested for hepatitis B, hepatitis C, syphilis, HIV-1, HIV-2, and human T-cell lymphotropic viurses I and II. ALT is also measured as a surrogate marker of nonspecific liver infection.
3. **Blood component therapy**
   A. **Whole blood:** 40% hematocrit; used primarily in hemorrhagic shock.
   B. **Packed red blood cells (PRBC):** volume 250-300 mL with a hematocrit of 70-80%; increases adult Hemoglobin approximately 1 g/dL.
   C. **Platelets**
      1. One platelet concentrate will increases platelet count 5-10x10^9/L; usual dose is 1 platelet concentrate per 10 kg body weight; single-donor platelets obtained by apheresis are equivalent to 6 platelet concentrate; platelets are stored at room temperature; risks of platelet infusions are sensitization reactions due to human leukocyte antigens on cell membranes of platelets and transmission of viral diseases.
      2. A normal platelet count is 150,00-440,000/mm^3. Thrombocytopenia is defined as <150,000/mm^3. Intraoperative bleeding increases with counts of 40,000-70,000/mm^3, and spontaneous bleeding can

occur at counts <20,000/mm^3. During surgery platelet transfusions are probably not required unless count is less then 50,000/mm^3.

D. **Fresh frozen plasma (FFP):** 250 cc/bag; contains all coagulation factors except platelets.

E. **Cryoprecipitate:** 10-20 mL/bag; contains 100 units factor VIII-C, 100 units factor vWF, 60 units factor XIII, and 250 mg fibrinogen; used for factor VIII deficiency and hemophilia A. Fatal hemolytic transfusion reaction: 1:500,000 to 800,000.

F. **Albumisol:** 5% and 25% (heat treated at 60 degrees C for 10 hrs).

4. **Complications of transfusions**

A. **Risk factors** (estimated frequency per unit transfused)

1. Minor allergic reactions (fever, chills, rash): 1-5:100
2. Nonfatal hemolytic transfusion reactions: 1:6,000
3. ABO incompatibility: 1:33,000
4. Anaphylactic shock: 1:500,000
5. Fatal hemolytic transfusion reaction: 1:500,000 to 800,000
6. HIV infection: 1:450,000 to 660,000
7. Hepatitis
   - A. Hepatitis A: 1:1 million
   - B. Hepatitis B: 1:30,000 to 250,000
   - C. Hepatitis C: 1:30,000 to 150,000
8. Bacterial contamination
   - A. Red cells: 1:500,000
   - B. Platelets: 1:12,000
9. Acute lung injury: 1:5,000

B. **Transfusions reactions**

1. **Febrile reactions:** most common nonhemolytic transfusion reaction (0.5-1.0% of transfusions); due to recipient antibodies against donor antigens present on leukocytes and platelets; treat with slow infusion and antipyretics.
2. **Allergic reactions:** occurs in about 3% of transfusions (20% platelet transfusion); caused by immunoglobulin alloantibodies against substances in the donor plasma with activation of mast cells and histamine release; usually presents with abrupt onset of pruritic erythema or urticaria on arms and trunk; minimized with slow infusion and antihistamines.
3.

4. **Anaphylaxis:** occurs in IgA deficient patients who have developed an anti-IgA; immune complex activates mast cells, basophils, etc. resulting in hypotension, dyspnea, laryngeal edema, wheezing and possibly shock; treat like severe allergic reaction.
5. **Acute hemolytic transfusion reaction**: occurs in 1:10,000 with 20-60% mortality; usually due to donor blood ABO incompatibility; complement activation leads to hemolysis and may result in DIC; clinically presents with headache, chills, nausea, vomiting, skin flushing, fever, flank pain, hypotension, dyspnea, bleeding and hemoglobinuria; acute renal failure may occur. Free Hemoglobin in the plasma or urine is presumptive evidence of a hemolytic reaction.
6. **Delayed hemolytic transfusion reaction:** occurs 1:33,000 units PRBCs transfused; usually seen in previously sensitized patients.
7. **Graft vs host disease.**
8. **Transfusion related acute lung injury:** an acute respiratory distress syndrome that occurs within 4 hours after transfusion and is characterized by dyspnea and arterial hypoxemia due to noncardiogenic edema. Treatment is supportive.
9. **Treatment of hemolytic transfusion reactions**
   - A. Stop the transfusion
   - B. Maintain the urine output at a minimum of 75 to 100 mL/hr by the following methods. Generously administer fluids IV and possibly mannitol, 12.5 to 50 grams, given over a 5-15 minute period.
   - C. If IV administered fluids and mannitol are ineffective, than administer furosemide, 20-40 mg IV.

D. Alkalinize the urine since bicarbonate is preferentially excreted in the urine, only 40-70 mEq/70 kg of sodium bicarbonate is usually required to raise the urine pH to 8, whereupon repeat urine pH determinations indicate the need for additional bicarbonate.

E. Assay urine and plasma hemoglobin concentrations. Determine platelet count, PTT, serum fibrinogen level.

F. Return unused blood to blood bank for crossmatch and send blood sample for antibody screen and direct antiglobulin test. Prevent hypotension to ensure adequate renal blood flow.

C. **Metabolic abnormalities**

1. Decreased pH secondary to increased hydrogen ion production.
2. Increase potassium: due to cell lysis; increases with length of storage.
3. Decrease in 2,3 DPG: consumed by RBCs; P_{50} decreases to 18 mmHg after 1 week and 15 mmHg after 3 weeks.
4. Citrate toxicity: citrate metabolism to bicarbonate may contribute to metabolic alkalosis; binding of calcium by citrate could result in hypocalcemia (rare reflecting mobilization of calcium stores in bone and the livers ability to metabolize citrate to bicarbonate.

D. **Microaggregates:** microaggregates consisting of platelets and leukocytes form during storage of whole blood. Micropore filters may decrease help remove these particles.

E. **Hypothermia:** the use of blood warmers (except for platelets) greatly decreases the likelihood of transfusion-related hypothermia.

F. **Coagulopathy disorders**

1. Usually occurs only after massive transfusion (greater than 10 units).
2. **Dilutional thrombocytopenia:** common cause of abnormal bleeding in massive transfusion, responds quickly to platelet transfusions.
3. **Low Factors V and VIII:** factors V and VII are very labile in stored blood and may decrease to levels as low as 15-20% normal, however, this is usually enough for hemostasis.
4. **Disseminated Intravascular Coagulation:** a hypercoagulable state caused by activation of the clotting system leading to deposition of fibrin in microvasculature which causes a secondary activation of fibrinolysis. resulting in consumption of factors and platelets.

5. **Massive transfusions**

A. **Massive transfusion** is defined as the replacement of a patient's total blood volume in less than 24 hours, or as the acute administration of more than half the patient's estimated blood volume per hour.

B. **The use of universal donor blood** (group O, Rh negative blood)

1. Group O, Rh negative blood should be reserved for patients close to exsanguination. If time permits, cross-matched or uncross-matched type specific blood should be administered.
2. Group O, Rh negative blood should not be given as whole blood. The serum contains high anti-A and anti-B titters which may cause hemolysis of recipient red cells.
3. If more than 4 units of group O, Rh negative whole blood is administered, type-specific blood should not be given subsequently since the potentially high anti-A and anti-B titters could cause hemolysis of the donor blood.
4. Patients administered up to 10 units of group O, Rh negative packed red blood cells may be switched to type-specific blood, since there is an insignificant risk of hemolysis from the small volume of plasma administered with packed red blood cells.

6. **Coagulation tests**

A. **Partial thromboplastin time (PTT)**

1. Partial thromboplastin is substituted for platelet phospholipid and eliminates platelet variability.
2. PTT measures the clotting ability of all factors in the intrinsic and common pathways except factor XIII.
3. PTT is abnormal if there are decreased amounts of coagulation factors, patients on heparin, or if there is a circulating anticoagulant present.
4. Normal values are between 25 and 37 seconds.
5.

B. **Activated partial thromboplastin time (aPTT)**

1. An activator is added to the test tube before addition of partial thromboplastin added.

2. Maximal activation of the contact factors (XII and XI) eliminates the lengthy natural contact activation phase and results in more consistent and reproducible results.
3. Normal aPTT is 25-35 seconds.

C. **Prothrombin time (PT)**
   1. Performed by measuring the time needed to form a clot when calcium and a tissue extract are added to plasma.
2. PT evaluates the activity of fibrinogen, prothrombin and factors V, VII and X.
3. Normal PT is 10-12 seconds (depending on control).

D. **International normalized ratio (INR)**
1. Developed to improve the consistency of oral anticoagulant therapy.
2. Converts the PT ratio to a value that would have been obtained using a standard PT method.
3. INR is calculated as $(Pt_{patient}/Pt_{normal})^{ISI}$ (ISI is the international sensitivity index assigned to the test system).

E. **Thromboelastrogram**
1. **Thromboelastrography (TEG)** is a method of testing for global assessment of coagulation. This technique uses a small sample of blood placed in a slowly rotating cuvette at 37EC. A piston is suspended in the cuvette, and as the coagulation proceeds, the tension on the piston is measured. A tracing is generated and several parameters are measured (see below).
2. **Thromboelastrogram parameters**
   A. **r (reaction time):** start of recording until 1 mm deflection (represents initial fibrin formation).
   B. **k (clot formation time):** measured from r until there is a 20 mm deflection in the tracing.
   C. **a (angle):** slope of the increase from r time to k time.
   D. **MA:** maximum amplitude in millimeters, a measure of the maximum clot strength (dependent on fibrinogen, level, platelet numbers, and function).
   E. **A^{60}:** deflection measure at 60 minutes after MA (represents clot lysis and retraction).
   F.

3. **Sonoclot**: the Sonoclot is a test of whole blood that utilizes a warmed cuvette and a suspended piston apparatus, This piston vibrates up and down very rapidly in the blood sample and Sonoclot detects any impedance to this vibration. As a result, the test follows the changes in viscosity over time.
4. **Activated clotting time (ACT)**: the ACT provides a global measurement of hemostatic function and is measured after whole blood is exposed to a specific activator of coagulation. The time for in vitro clot formation after whole blood is exposed to diatomaceous earth (Celite) is defined as the ACT. Normal is 90 to 120 seconds. The linear increase in ACT seen with increasing doses of heparin provides a convenient method to monitor anticoagulant effect of heparin. Although the ACT test is simple, it lacks sensitivity to clotting abnormalities.

7. **Sickle Cell Anemia**

A. Sickle cell anemia is a hemoglobinopathy that results from inheritance of a gene for a structurally abnormal beta globin chain. This results in HbS which has two forms.

B. **Sickle cell trait (HbAS)** is a heterozygous state. Only 1% of the red cells in venous circulation of the heterozygote are sickled. These patients are usually asymptomatic. Vigorous physical activity at high altitude, air travel in unpressurized planes, and anesthesia are potentially hazardous.

C. **Sickle cell disease (HbSS):** homozygous state: 70-98% HbS.

D. **Clinical features**

1. **Signs and symptoms** include anemia (HgB levels 6.5 to 10 gm/dL), obstructive or hemolytic jaundice, joint and bone pain, abdominal and chest pains, lymphadenopathy, chronic leg ulcers, hematuria, epistaxis, priapism, finger clubbing, and skeletal deformities.

2. The disease is characterized by periodic exaggeration of symptoms or sickle cell crisis. There are four main types of crises.

   A. **Vaso-occlusive crises:** caused by sickled cells blocking the microvasculature, characterized by sudden onset of pain frequently with no clear-cut precipitating event.

   B. **Hemolytic crises:** seen in patients with sickle cell disease plus G-PD deficiency, has hematologic features of sudden hemolysis.

C. **Sequestration crises:** sequestration of red blood cells in the liver and spleen causing massive, sudden enlargement, and an acute fall in peripheral hematocrit, this can progress to circulatory collapse.

D. **Aplastic crises:** characterized by transient episodes of bone marrow depression commonly occurring after viral infection.

E.

3. **Anesthetic management**

A. The practice of transfusing these patients to an end point of having 70% hemoglobin A and less than 30% hemoglobin S cells before major surgery remains controversial.

B. To lessen the risk of intraoperative sickling patients should be kept well oxygenated and well hydrated. Avoid acidosis and hypothermia.

8. **Factor VIII deficiency (hemophilia A)**

A. The half life of factor VIII in plasma is 8-12 hours.

B. Treatment consists of lyophilized factor VIII, cryoprecipitate, or desmopressin. Infusion of 1 unit of factor VIII per kg will increase the factor VIII activity level by 2%. Obtain activity levels of 20-40% before surgery.

C. Bleeding episodes related to the level of factor VIII activity (normal 100%).

9. **Factor IX deficiency (hemophilia B; Christmas disease)**

A. The half life of factor IX in plasma is 24 hours.

B. Therapy consists of factor IX concentrates of FFP. For surgical hemostasis, activity levels of 50% to 80% are necessary.

C. Infusion of 1 unit of factor IX per kg of body weight will increase the factor IX activity level by 1%.

Spinal and Epidural Anesthesia

?

General Information

1. **Contraindications to peridural anesthesia**
   A. Absolute contraindications: lack of patient consent, localized infection at injection site, generalized sepsis or bacteremia, allergy to local anesthetics, increased intracranial pressure, coagulopathy.
   B. **Relative contraindications**: localized infection peripheral to regional site, demyelinating CNS disease, chronic back pain or prior lumbar spine surgery, hypovolemia, patients taking platelet inhibiting drugs.
2. **Anatomy**
   A. **Spinal canal:** extends from the foramen magnum to the sacral hiatus.
   B. **Spinal cord:** spinal cord extends the length of the vertebral canal during fetal life, ends at L3 at birth, and moves progressively cephalad to reach the adult position of L1-L2 by 2 years of age.
   C. **Subarachnoid space:** subarachnoid space lies between the pia mater and the arachnoid and extends from S2 to the cerebral ventricles.
   D. **Epidural space** contains nerve roots, fat, lymphatic and blood vessels, and areolar tissue.
   E. **Course of anatomy:** skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, epidural space, and dura.
3. **Physiological changes with spinal and epidural anesthesia**
   A. **Neural blockade**
      1. **Sequence of neural blockade**
         1. Sympathetic block with peripheral vasodilation and skin temperature elevation.
         2. Loss of pain and temperature sensation.
         3. Loss of proprioception.
         4. Loss of touch and pressure sensation.
         5. Motor paralysis.

      2. The above sequence of neural blockade occurs because smaller C fibers are blocked more easily than the larger sensory fiber, which in turn are blocked more easily than motor fibers. As a result, the level of autonomic blockade for a spinal anesthetic extends above the level of the sensory blockade by 2-3 segments, while the motor blockade is 2-3 segments below the sensory blockade. During epidural anesthesia there is not a zone of differential nervous system blockade, and the zone of differential motor blockade averages 4 segments below the sensory level.
      3.
      4. With epidural anesthesia, the local anesthetics act directly on the spinal nerve roots located in the lateral part of the space. To a lesser extent, diffusion of local anesthetic solutions from the epidural space into the subarachnoid space produces spinal cord effects. As a result, the onset of the block is slower than with spinal anesthesia, and the intensity of the sensory and motor block is less.
   B. **Cardiovascular**
      1. **Hypotension:** the degree of hypotension is directly proportional to the degree of sympathetic blockade.
      2. **Blockade above T4** interrupts cardiac sympathetic fibers, leading to bradycardia, decreased cardiac output, and further decrease in BP.
   C. **Respiratory:** with ascending height of the block into the thoracic area, there is a progressive, ascending intercostal muscle paralysis. The diaphragmatic ventilation is mediated by the phrenic nerve, and typically will remain unaffected even during high cervical blockade.
   D. **Visceral effects**
      1. **Bladder:** sacral blockade results in an atonic bladder.
      2. **Intestine:** with sympathectomy, vagal tone dominates and results in a small, contracted gut with active peristalsis.
4. **Complications**

A. **Hypotension:** prehydrating with 500-1000 cc of crystalloid before performing the block will help decrease the incidence of hypotension.
B. **Paresthesia or nerve injury:** during placement of the needle or injection of anesthetic, direct trauma to a spinal nerve or intraneural injection may occur.
C. **Blood tap or vascular injury:** needle may puncture an epidural vein during needle insertion.
D. **Nausea and vomiting:** usually the result of hypotension or unopposed vagal stimulation.
E. **Total spinal:** may see apnea with from direct blockade of C3-C5.
F. **Backache:** overall the incidence of backache following spinal anesthesia is no different from that following general anesthesia.
G. **Postdural puncture headache:** seen 6-48 hours after dural puncture.
H. **Urinary retention:** urinary retention may outlast the blockade.
I. **Infection:** meningitis, arachnoiditis, and epidural abscess.

Spinal Anesthesia

1. **Factors influencing spinal anesthetic**
   A. Dosage.
   B. Drug volume.
   C. Addition of vasoconstrictors to reduce systemic absorption
   D. Baricity of the local anesthetic solution (specific gravity).
   E. Shape of the spinal canal (supine: high point L3-L4; low point T5-T6).
   F. Position of the patient.
   G. Intra-abdominal pressure: as seen with pregnancy, obesity, ascites, or abdominal tumors, increases the blood flow through the epidural venous plexus, reducing the volume of CSF, thus causing the local anesthetic to spread further.
   H. Age (spinal space thought to become smaller with age).

Epidural Anesthesia

1. **Factors influencing epidural anesthesia**
   A. Local anesthetic selected.
   B. Mass of drug injected (dose, volume, and concentration).
   C. Addition of vasoconstrictors to reduce systemic absorption.
   D. Site injection.
   E. Patients over 40 years of age.
   F. Pregnancy (hormonal and/or mechanical factors).
2. **Epidural insertion sites:** cervical interspaces through T4 are best accessed by a median approach, while a paramedian approach for T4-T9 and a median approach for T9-L5.
3. **Complications of epidural anesthesia** (in addition to those listed above)
   A. **Dural puncture:** unintentional dural puncture occurs in 1% of epidural injections performed.
   B. **Catheter complications**
      1. Inability to insert the catheter.
      2. Catheter can be inserted into an epidural vein.
      3. Catheters can break off or become knotted within the epidural space.
      4. Unintentional subarachnoid injection.
      5. Intravascular injection: may result in local anesthetic overdose where large amounts of local anesthetic are used.
      6. Direct spinal cord injury:
      7. Bloody tap: may result from perforation of an epidural vein.
4. **Clinical pharmacology of epidural opioids**
   A. **Hydrophilic opioids** (morphine, hydromorphone)
      1. **Properties:** slow onset, long duration, high CSF solubility, extensive CSF spread.

2. **Advantages:** prolonged single-dose analgesia, thoracic analgesia with lumbar administration, minimal dose compared to IV administration.
3. **Disadvantages:** delayed onset of analgesia, unpredictable duration, higher incidence of side effects, delayed respiratory depression.
4.

B. **Lipophilic opioids** (fentanyl, sufentanil)
1. **Properties:** rapid onset, short duration, low CSF solubility, minimal CSF spread.
2. **Advantages:** rapid analgesia, decreased side effects, ideal for continuous infusion or PCEA.
3. **Disadvantages:** systemic absorption, brief single-dose analgesia, limited thoracic analgesia with lumbar administration

Postdural Puncture Headache

1. **Characteristics of a postdural puncture headache**
A. Postural component (made worse by upright position).
B. Frontal or occipital location.
C. Tinnitus.
D. Diplopia.
E. Young females.
F. Use of a large-gauge needle.
2. **Mechanism:** usually due to a continued leak of CSF through the hole in the dura mater, resulting in low CSF pressure, which causes traction on meningeal vessels and nerves.
3. **Incidence:** the overall incidence is approximately 5-10%.
4. **Treatment of a postdural puncture headache**
A. Oral Analgesics.
B. Bed rest.
C. Hydration (IVF, PO fluids, caffeine containing beverages).
D. Caffeine infusion (500 mg caffeine and sodium benzoate in 1 liter of isotonic crystalloid given over 1-2 hours).
E. Epidural blood patch (placement of 10-20 cc of autologous blood in the epidural space). The success rate is approximately 95%.

Anticoagulant Therapy

1. Do not attempt epidural/spinal if the patient is fully heparinized.
2. **Minidose subcutaneous heparin:** no contraindications to neuraxial block, may consider delaying (about 6 hours) initiation of heparin therapy until after neuraxial block.
3. **Intraoperative coagulation with IV heparin:** delay initiating heparin administration for 1 hour after needle placement, remove the epidural catheter 1 hour before any subsequent IV dose of heparin (assuming 12 dosing) or 2-4 hours after the last dose, consider the use of minimal concentrations of local anesthetic to permit early detection of neurologic changes, blood or difficult neuraxial needle and/or catheter placement does not mandate cancellation of the surgical procedure if it proceeds must perform frequent postoperative monitoring of neurologic status.
4. **Low molecular weight heparin (LMW):** decision to perform a neuraxial block is made on an individual basis, blood or difficult neuraxial needle and/or catheter placement does not mandate cancellation of the surgical procedure if it proceeds must perform frequent postoperative monitoring of neurologic status, consider single dose spinal anesthesia if regional anesthesia is required in patients receiving LMW heparin preoperatively, perform frequent postoperative monitoring of neurologic status.
5. **Oral anticoagulants:** stop anticoagulant and allow normalization of prothrombin time before doing block.
6. **Antiplatelet drugs:** does not interfere with the performance of a block.

7. **Fibrinolytic and thrombolytic drugs:** neuraxial block not recommended with 10 days fo receiving these drugs.

Regional Anesthesia in Pediatrics

1. **Pharmacology**
   A. Protein binding of local anesthetics is decreased in neonates because of decreased of albumin.
   B. Increased volume of distribution may decrease free local anesthetic concentrations.
2. **Spinal anesthesia**
   A. **Hypobaric solutions are most commonly used.**
      1. Bupivacaine 0.75% in 8.25% dextrose, 0.3 mg/kg in infants and children.
      2. Tetracaine 1% with equal volume 10% dextrose, 0.8-1.0 mg/kg in infants and 0.25-0.5 mg/kg in children.
      3. Duration may be prolonged with the addition of epinephrine 10 mcg/kg (up to 0.2 mg).
3. **Complications**
   A. Anesthetic level recedes faster in children than adults.
   B. Hypotension is rare in children under 10 years.
   C. Contraindicated in children with CNS anatomic defects and a history of grade III-IV intraventricular hemorrhage.
4. **Caudal and lumbar epidural anesthesia**
   A. Dural sac in the neonate ends at S3.
   B. Caudal epidural provides excellent postoperative analgesia for circumcision, hypospadias repair, orchiopexy, herniorrhaphy and some orthopedic procedures.
   C. **Caudal catheters**
      1. Infants: 22g catheter placed (40-50 mm) through 20g Tuohy needle.
      2. Children: 20g cath placed (90-100 mm) through 17-18g Tuohy needle.
   D. **Epidural catheters:** older children use 20g cath with 18g Tuohy needle.
   E. **Drugs**
      1. Bupivacaine, 0.125% -0.25% with epinephrine 1.5-2.5 mg/kg (0.5-1 ml/kg) or 0.06 cc local anesthetic/kg/segment (counted from S5).
      2. Ropivacaine 0.25% 2.5 mg/kg (1 ml/kg).
      3. 1% lidocaine, 0.5 cc/kg followed by 0.5% lidocaine, 0.5 cc/kg every hour as needed.
      4. Continuous infusion
         A. Infants and children less than 7 years: load with 0.04 cc/kg/segment of 0.1% bupivacaine (+/- fentanyl 2-3 mcg/cc).
         B. Children older than 7 years load with 0.02 cc/kg/segment of 0.1% bupivacaine (with or without fentanyl 2-3 mcg/cc).
         C. Infusions of 0.1% bupivacaine (with or without fentanyl 2-3 mcg/cc) at 0.1 cc/kg/hr. May be increased up to 0.3 cc/kg/hr.
         D. Fentanyl should not be used in infants under 1 year.
   F. **Ilioinguinal and iliohypogastric nerve blocks**
      1. Effective for orchiopexy and hernia repair.
      2. Bupivacaine 0.25-0.5% with 1/200,000 epi up to a dose of 2 mg/kg is injected just medial to the anterior superior iliac spine in a fan like method. A second injection can be done lateral to the pubic tubercle
   G. **Penile nerve block**
      1. The penile nerve (a branch of the pudendal nerve) is blocked at the base of the penis with a 25-26 g needle with 1-4 mL of bupivacaine 0.25% without epi at the 10:30 and 1:30 positions. Provides 6 hours.

Cutaneous	**Segmental**	**Effects**

Level	Level	
Fifth Digit	C8	All cardioaccelerator fibers (T1-T4) blocked
Inner Aspect of Arm and Forearm	T1-T2	Some degree of cardioaccelerator blockade
Apex of Axilla	T3	
Nipple	T4-T5	Possible cardioaccelerator blockade
Tip of Xiphoid	T7	Splanchnic (T5-L1) may be blocked
Umbilicus	T10	SNS blockade limited to legs
Inguinal Ligament	T12	No SNS blockade
Lateral Foot	S1	

Procedure	**Insertion Site**
Mastectomy	T1
Thoracotomy	T4
Upper Abdominal Surgery	T7-T8
Lower Abdominal	T10
Lower Extremity Above Knee	L1-L2
Lower Extremity Below Knee	L3-L4
Perineal	L4-L5

Problem	**Treatment Options**	**Notes**
Pruritus	Nalbuphine 5-10 mg IV/IM	May be severe

	Diphenhydramine 25-50 mg IV Naloxone 40-80 mcg IV	after intrathecal morphine
Nausea/ Vomiting	Metoclopramide 5-10 mg IV Nalbuphine 5-10 mg IV/IM Naloxone 40-80 mcg IV	
Respiratory Depression	Naloxone 0.1 mg IV prn	Watch for synergism with other sedatives
Urinary Retention	Urinary Catheter	
Blood Pressure Changes	Fluid hydration Ephedrine Phenylephrine	Most likely after meperidine (local anesthetic effects)

Peripheral Nerve Blocks

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General information

1. **Preoperative:** similar to patients receiving general or regional anesthesia, coagulation status should be determined.
2. **Contraindications:** absolute contraindications include lack of patient consent or when nerve blockade would hinder the proposed surgery; relative contraindications include coagulopathy, infection at the skin site, presence of neurologic disease.
3. **Complications** common to all regional nerve blocks: complications to local anesthetics (intravascular injection, overdose, allergic reaction), nerve damage (needle trauma, intraneural injection), and hematomas.
4. **Nerve localization:** paresthesia
   A. Placing a needle in direct contact with a nerve or within the substance of the nerve will stimulate that nerve causing paresthesias.
   B. Injection into a perineural location often results in a brief accentuation of the paresthesia; in contrast, an intraneural injection produces an intense, searing pain that signals the need to immediately terminate the injection.
   C. Correct needle placement can be determined by elicitation of paresthesia, perivascular sheath technique, transarterial placement, and a nerve stimulator.
5. **Nerve block needles**
   A. **Blunt-bevel needle:** designed to minimize trauma upon direct contact with nerves. The angle of the bevel is increased 20-30 degrees, and the sharpness is decreased.
   B. **Insulated needle:** a nonconductor is bonded to the needle except for the last millimeter before the bevel.
   C. **The beaded needle:** a regional needle designed for use with a nerve stimulator.

Peripheral Nerve Blocks

1. **Cervical plexus block**
   A. **Technique:** with the patient's head turned to the opposite side, a line connecting the tip of the mastoid process of the temporal bone and the anterior tubercle of the transverse process of the sixth cervical vertebra (Chassaignac's tubercle, the most prominent of the processes) identifies the approximate plane in which the cervical transverse processes lie. Using a 22 g needle, penetrate the skin over each point, directing the needle in a slightly caudal direction to contact each transverse process. Confirm the position by 'walking' the needle off the tip of the transverse process. Ensure that neither blood nor CSF can be aspirated. Inject 3-5 mL of local anesthetic
   B. **Complications:** blockade of the phrenic nerve, Horner syndrome (ptosis, miosis, enophthalmos, anhydrosis), hoarseness (recurrent laryngeal nerve block), accidental subarachnoid or epidural injection.
2. **Brachial plexus blocks**
   A. **Interscalene block**
      1. **Technique:** the needle is inserted in the interscalene groove at the level of the cricoid cartilage and advanced perpendicular to the skin until a paresthesia is elicited or a transverse spinous process is contacted, at which point 30-40 cc of local anesthetic is injected.
      2. **Indications:** any procedure on the upper extremity, including the shoulder. This technique has a high rate of failure to achieve full block of the ulnar nerve (10-20%) for hand surgery.
      3. **Special contraindications:** contralateral phrenic paresis, severe asthma.
      4. **Side effects:** Horner's syndrome, phrenic paresis.

5. **Complications:** proximity of the vertebral artery makes intraarterial injection possible with rapid progression to grand mal seizure after small amounts are injected. The neural foramina can be reached, and massive epidural, subarachnoid, or subdural injection can occur. Stellate ganglion block results in Horner's sign (myosis, ptosis, anhidrosis). Other complications include recurrent laryngeal nerve block (30-50%) leading to hoarseness, phrenic nerve block, pneumothorax, infection, bleeding, and nerve injury.
6.

B. **Supraclavicular block**
1. **Indications:** procedures on the upper arm, elbow, lower arm and hand.
2. **Special contraindications:** hemorrhagic diathesis, contralateral phrenic paresis.
3. **Side effects:** Horner's syndrome, phrenic paresis.
4. **Complications:** pneumothorax (1-6%) and hemothorax are the most common. Phrenic nerve block and Horner's syndrome may occur.

C. **Axillary block**
1. **Indications:** procedures on the lower arm and hand.
2. **Anatomy:** it should be noted that in the axilla, the musculocutaneous nerve has already left its sheath and lies within the coracobrachialis.
3. **Special contraindications:** lymphangitis (presumed infected axillary nodes).
4. **Complications:** puncture of the axillary artery, intravenous/intra-arterial injection (systemic toxic reaction), postoperative neuropathies (more common when multiple sites of paresthesia are elicited).

3. **Intercostal nerve block**

A. **Technique:** optimally performed with patient prone or sitting, a 22 g needle is inserted perpendicular to the skin in the posterior axillary line over the lower edge of the rib, the needle then is 'walked' off the rib inferiorly until it slips off the rib, after negative aspiration for blood 5 mL of local anesthetic is injected.

B. **Complications:** the principle risks are pneumothorax and accidental intravascular injection of local anesthetic solutions.

4. **Nerve blocks of the lower extremity**

A. **Sciatic nerve block**
1. **Technique:** the patient is placed in the Sim's position (the lateral decubitus position with the leg to be blocked uppermost and flexed at the hip and knee) a line is drawn from the posterior iliac spine and the greater trochanter of the femur, the needle is inserted about 5 cm caudad from the midpoint of this line, and about 25 mL of 1.5% lidocaine or 0.5% bupivacaine or ropivacaine is injected after elicitation of a paresthesia.

B. **Femoral nerve block**
1. **Indications:** surgery of the foot and lower leg.
2. **Technique:** insert short-beveled 22 g block needle in a 30-degree cephalad direction just lateral to the femoral artery and just below the inguinal ligament, fell for 2 'pops' as the needle passes first through the fascia lata and then the fascia iliaca, a nerve stimulator can be used looking for quadricep contractions with a stimulating current of 0.3 mA, inject 15 mL of bupivacaine 0.5%.

C. **3 in 1 block** (femoral, obturator, and lateral cutaneous nerves)
1. Technique: identical to femoral nerve block but a greater volume of local anesthetic used (inject 30 mL)

D. **Ankle block** (requires 5 separate nerve blocks)
1. **Posterior tibial nerve:** insert needle behind the posterior tibial artery and advanced until a paresthesia to the sole of the foot is elicited, inject 5 mL of local anesthetic.
2. **Sural nerve:** inject 5 mL of local anesthetic in the groove between the lateral malleolus and calcaneus.
3. **Saphenous nerve:** inject 5 mL of local anesthetic anterior to the medial malleolus.
4. **Deep peroneal nerve:** inject 5 mL of local anesthetic lateral to the anterior tibial artery at the distal end of the tibia at the level of the skin cease.
5. **Superficial peroneal nerve:** infiltrate a ridge of local anesthetic (10 mL) from the anterior tibia to lateral malleolus.

5. **Intravenous regional neural anesthesia** (Bier Block)

A. **Indications:** forearm and hand surgery of short duration; < 90 minutes.

B. **Technique:** place a small gauge IV as distally as possible, place a double tourniquet around the upper arm, exsanguinate the arm by elevating and wrapping it tightly, with the proximal tourniquet inflated inject 40-50 mL of 0.5% lidocaine (ropivacaine 1.2-1.8 mg/kg can also be used), when the patient feels discomfort from the proximal tourniquet inflate the distal cuff then deflate the proximal cuff.
C. **Complications:** local anesthetic toxicity (the tourniquet should be left inflated for at least 20-30 minutes).

Pediatric Anesthesia

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Pediatric General Information

1. **Vital signs** (see table)
   A. Typical systemic blood pressure in children 1-10 years of age = 90 mmHg + (child's age in years x 2) mmHg.
   B. Lower limits of systemic blood pressure in children 1-10 years of age = 70 mmHg + (child's age in years x 2) mmHg.
2. **Age and approximate weight**
   A. 28 weeks = 1 kg +/- 100 g/wk from 22-30 weeks
   B. <1 year: ½ age (months) plus 4 kg
   C. 1 year to puberty: 2 times age in years plus 10 kg

Age	**RR**	**HR (Awake)**	**HR (Asleep)**	**SBP**	**DBP**
Preterm	60	120-180	100-180	45-60	20-45
Neonate	40-60	100-180	80-160	55-80	20-60
Infant	30-60	100-160	75-160	87-105	53-66
Toddler	24-40	80-110	60-90	90-105	53-66
Preschooler	22-34	70-110	60-90	95-105	55-70
School Age	18-30	65-110	60-90	97-112	57-71
Adolescent	12-16	60-90	50-90	112-128	66-80

Pediatric Developmental Implications

1. **Physiologic differences** (as compared to the adult)
   A. **Cardiac:** cardiac output of neonates and infants is dependent on heart rate, since stroke volume is relatively fixed by a noncompliant and poorly developed left ventricle. The sympathetic nervous

system and baroreceptor reflexes are not fully mature. The hallmark of hypovolemia is hypotension without tachycardia.

B. **Respiratory:** increased respiratory rate; tidal volume and dead space per kg are constant; lower functional residual capacity; lower lung compliance; greater chest wall compliance; small alveoli are associated with low lung compliance; hypoxic and hypercapnic ventilatory drives are not well developed in neonates and infants; neonates are obligate nose breathers.

C. **Temperature:** infants have poor central thermoregulation, thin insulating fat, increased body surface area to mass ratio, and high minute ventilation; heat production in neonates is nonshivering thermogenesis by metabolism of brown fat.

D. **Renal:** normal renal function by 6 months of age; premature neonates may posses decreased creatinine clearance, impaired sodium retention, glucose excretion, and bicarbonate reabsorption.

E. **Metabolic:** hypoglycemia is defined as <30 mg/dL in the neonate, and <40 mg/dL in older children.

F. **Gastrointestinal:** gastric emptying is prolonged, and the lower esophageal sphincter is incompetent, thus the high incidence of gastric reflux.

G. **Central nervous system:** the CNS is the least mature major organ system at birth predisposing the newborn to intraventricular hemorrhages, seizures, respiratory depression, and retinopathy.

2. **Pharmacologic differences** (as compared to the adult)

A. Pediatric drug dosing is based upon a per-kilogram recommendation.

B. **Inhalational anesthetics:** higher alveolar ventilation, relatively low functional residual capacity (ie, a higher ratio of minute ventilation to functional residual capacity) contribute to a rapid rise in alveolar anesthetic concentration. The blood/gas coefficients of isoflurane and halothane are lower in neonates than adults. The minimum alveolar concentration is higher in infants than in neonates or adults. The blood pressure of neonates and infants tends to be more sensitive to volatile anesthetics.

C. **Nonvolatile anesthetics:** an immature blood-brain barrier and decreased ability to metabolize drugs could increase the sensitivity of neonates to effects on IV anesthetics. Dose of thiopental is similar in peds and adults.

D. **Muscle relaxants:** infants require higher doses of succinylcholine per kilogram than do adults because of their larger volume of distribution. Children are more subject to cardiac dysrhythmias, myoglobinemia, hyperkalemia, and MH after succinylcholine than adults. Nondepolarizing agents for infants and children have similar mg/kg requirements.

Pediatric Airway Management

1. **Airway differences** (compared to an adult): large head; tongue larger in relation to the oral cavity; narrow nasal passages; epiglottis is narrow, shorter, U-shaped and protruding; hyoid bone not calcified in the infant; the larynx is higher in the infant neck (C3-4) then in the adult (C5-6) and is angulated and anterior appearing; short trachea and neck; cricoid cartilage (ring) narrowest point of airway in children younger than 8-10 years of age (glottis in adults), obligate nasal breathers (infants less than 6 months)
2. **Pediatric endotracheal tube recommendations** (see airway section)
   A. Uncuffed endotracheal tubes generally used for patients under 10 yrs, however, cuffed tubes have been used safely even in neonates.
   B. Endotracheal (cuffed) tube leak: 15-20 cm H_2O.
   C. Endotracheal tube size (mm): for children older then 2 years ETT can be estimated by: Age/4 + 4.
   D. Length of Insertion (cm) of ETT
      1. Under 1 year: 6 + Wt(kg).
      2. Over 2 years: 12 + Age/2.
      3. Multiply internal diameter (mm) of ETT by 3 to give insertion (cm).
      4. Add 2-3 cm for nasal tube.
3. **Equipment**
   A. **Reservoir bag:** newborn 0.5 L; 1-3 years 1.0 L; 3-5 years 2.0 L; greater than 5 years 3.0 L.
   B. **Arterial catheters:** neonates/infants 24g; less than 5 years old 22g; greater than 5 years old 20 g.
   C. **Central venous catheters** (heparin-coated)

1. Premature 3 Fr, 5 cm, single lumen
2. < 1 year 4 Fr, 8 cm, single lumen
3. 1-2 year 4 Fr, 8 cm, single lumen
4. 3-8 year 4 Fr, 13 cm, single lumen
5. > 8 year 5 Fr, 12 cm, single lumen

Age	**Laryngoscope**	**Endotracheal Tube Size (mm)**	**Distance at Teeth (cm)**	**Suction Catheter (F)**
Neonate <1000 g	Miller 0	2.5	6.5-7.0	5-6
Neonate 1000-2000 g		3.0		
Neonate 2000-3000 g		3.0-3.5		
Term Infant	Miller 0-1 Wis-Hipple 1 Robertshaw 0	3.0-3.5	9-10	6-8
6 months		3.5-4.0	10	8
1 year	Wis-Hipple 1.5 Robertshaw 1	4.0-4.5	11	8
2 years	Miller 2 Flagg 2	4.5-5.0	12	8
4 years		5.0-5.5	14	10
6 years		5.5	15	10
8 years	Miller2-3 Macintosh 2	6.0	16	10
10 years		6.5	17	12
12 years	Macintosh 3	7.0	18	12
Adolescent Adult	Macintosh 3 Miller 3	7.0-8.0	20	12

Age (yrs)	ETT (ID)	BB (Fr)	Univent	DLT (Fr)
0.5-1	3.5-4.0	5		
1-2	4.0-4.5	5		
2-4	4.5-5.0	5		
4-6	5.0-5.5	5		
6-8	5.5-6.0	6	3.5	
8-10	6.0 cuff	6	3.5	26
10-12	6.5 cuff	6	4.5	26-28
12-14	6.5-7.0 cuff	6	4.5	32
14-16	7.0 cuff	7	6.0	35
16-18	7.0-8.0 cuff	7	7.0	35

ETT: endotracheal tube; BB: bronchial blocker; DLT: double lumen tube

Pediatric Preoperative Evaluation (see preoperative section)

1. **History**
   A. Gestational age and weight; events during labor and delivery; neonatal hospitalizations; congenital anomalies; medical, surgical and anesthetic history; allergies; medications.
2. **Physical exam**
   A. Airway exam (see preoperative evaluation section).
   B. Vital signs, height and weight.
   C. General appearance should be noted.
3. **Laboratory testing**
   A. Except as noted below, no routine lab testing should be performed.
      1. Tests should be specific for the patient's coexisting condition and planned surgery.
      2. Infants less then 6 months: preoperative hematocrit.
      3. Preterm infant: glucose, calcium, and coagulation studies.
      4. African-American or mixed race ancestry: sickle cell/anemia screening unless status is known.
      5. In children receiving therapeutic drugs, levels should be checked.

6. Tonsillectomy and adenoidectomy or adenoidectomy: hematocrit (can be done during IV insertion).

4. **Premedications** (see premedication table)
   A. Premedications should be considered for all patients. Parental presence in the operating room during induction is a technique increasing in number with success.
   B. Generally, infants less then 9 months of age require no premedication.
   C. If anticholinergics are consider (generally for children under 1 year of age) they can be administered IV at the time of induction.
5. **Common coexisting disease/illnesses**
   A. **The child with a URI**
      1. A URI within 2-4 weeks of general anesthesia and endotracheal intubation can place the child at an increased risk of perioperative pulmonary complications (wheezing, hypoxemia, atelectasis, laryngospasm).
      2. Factors favoring postponing surgery include purulent nasal discharge, upper airway stridor, croup, lower respiratory symptoms (wheezing), and fever.
      3. Factors favoring performing surgery include clear 'allergic ' reactions, economic hardship on family, few and short 'URI-free' periods, and scheduled surgery may itself decrease frequency of URI's.
   B. **History of prematurity**
      1. Prematurity is defined as birth before 37 weeks gestation or weight less than 2500 grams; premature infants are at increased risk for retinopathy of prematurity and apnea of prematurity.
      2. The former premature infant is at increased risk for the development of postoperative apnea (apnea of prematurity) even after minor surgery and should be monitored overnight if their postconception age (gestational age plus chronological age) is less then 60 weeks.
      3. Risk factors for apnea of prematurity include necrotizing enterocolitis, neurologic problems, anemia, hypothermia, and sepsis.
      4. Regional anesthesia may be associated with a lower incidence.
   C. **Intellectual impairment:** children with severe developmental delay often have several coexisting disease, most often seizure disorders, gastroesophageal reflux, and chronic lung disease.
   D. **Seizure disorders:** anticonvulsant regimen and levels should be documented; anticonvulsants should not be withheld the day of surgery.
   E. **Trisomy 21 (Down's Syndrome):** increased risk of difficult airways, postoperative airway obstruction/croup, sleep apnea and subluxation of the atlanto-occipital joint; routine screening cervical spine radiographs in the asymptomatic child are not indicated.

Anesthesia for Common Pediatric Conditions

1. **Acute airway obstruction** (see table)
   A. **Causes:** laryngotracheobronchitis (croup), epiglottis, and foreign-body aspiration.
   B. **Pathophysiology:** inspiratory stridor is the hallmark of upper airway , supraglottic, and glottic obstruction (croup); wheezing generally indicates intrathoracic airway obstruction (foreign-body aspiration).
   C. **Contributing factors**
      1. **Croup (including postintubation or traumatic croup):** traumatic or repeated intubations, tight fitting ETT, coughing/straining on the ETT, change in patient's position during surgery, intubation greater than one hour, head and neck surgery.
   D. **Treatment**
      1. Total obstruction can occur, adequate preparation for a possible tracheostomy should be made prior to induction of anesthesia.
      2. **Epiglottis:** a slow, gentle, inhalational induction followed by intubation to secure the airway (use ETT 0.5 smaller then usual), only then followed by paralysis, is the preferred approach.
      3. **Acute laryngotracheobronchitis (croup):** aerosolized racemic epinephrine and humidified oxygen; the use of steroids (Decadron 0.3-0.5 mg/kg) is controversial; the need for intubation only

becomes necessary when airway obstruction becomes severe or prolonged enough to lead to respiratory muscle exhaustion and failure.

4. **Foreign-body:** an inhalational induction to induce a state of deep anesthesia, provide IV access, and perform gentle, upper airway endoscopy, removing the foreign object if possible, or securing the airway and bypassing it if not.

2. **Hypertrophic pyloric stenosis**

A. **Manifestations:** persistent vomiting depletes sodium, potassium, chloride, and hydrogen ions, causing hyponatremic, hypokalemic, hypochloremic metabolic alkalosis. Initially, the kidney compensate for the alkalosis by excreting sodium bicarbonate in the urine. Later, as hyponatremia and dehydration worsen, the kidneys must conserve sodium at the expense of hydrogen ion excretion (resulting in paradoxic aciduria). Neonates may be at increased risk for respiratory depression and hypoventilation postoperatively because of persistent metabolic or CSF alkalosis.

B. **Management:** electrolyte abnormalities must be corrected prior to surgery; continuous nasogastric suction; increase risk of aspiration; tracheal intubation may be accomplished awake or after the induction of anesthesia; caudal anesthetic (1.25 mg/kg of 0.25% bupivacaine with epi) is useful for decreasing anesthetic requirements and postoperative pain management.

3. **Inguinal hernia repair**

A. **Manifestations:** in infants often associated with prematurity, a history of RDS, and congenital heart disease; the preterm infant with inguinal hernia is at increased risk for postoperative apnea and pulmonary complications.

B. **Management:** caudal anesthesia (0.25% bupivacaine or ropivacaine 0.75 mg/kg) to decrease intraoperative anesthetic requirements and provide postoperative analgesia.

C. **Complications:** as an intraperitoneal procedure there may be traction on the spermatic cord which can be a stimulus to laryngospasm.

4. **Tonsillectomy and adenoidectomy**

A. **Manifestations:** lymphoid hyperplasia can lead to upper airway obstruction, obligate nasal breathing, and pulmonary hypertension; evidence of airway obstruction, snoring, and apnea should be noted.

B. **Management:** anesthesia induction can be either inhalational or IV and maintenance provided by nearly any means; strict attention to airway patency, hemostasis, and observation until the child is awake and in control of airway and secretions; postoperative vomiting is common.

C. **Postoperative bleeding:** may be evidenced by restlessness, pallor, tachycardia, or hypotension; if reoperation is necessary to control bleeding, intravascular volume must first be restored; rapid-sequence induction with cricoid pressure after gastric suctioning because of the risk of aspiration (blood in the stomach is common).

5. **Myringotomy and insertion of tympanostomy tubes**

A. **Manifestations:** because of the chronic and recurring nature of this illness, it is not uncommon for these patients to have symptoms of an URI on the day of surgery.

B. **Management:** typically very short procedure; inhalational induction common with oxygen, nitrous oxide and volatile agent; intravenous access is usually not necessary.

6. **Ventricular shunts**

A. **Manifestations:** shunts may be required for either internal or external hydrocephalus; all patients should be evaluated for intracranial hypertension (crying, irritability, sudden personality or behavior change, sleepiness, vomiting, and lethargy).

B. **Management:** induction of anesthesia should consider possible elevated ICP (respiration patterns, induction agents); hyperventilation is effective for acute rises in ICP (only hyperventilate suspected of elevated ICP)

	Foreign-Body	**Croup**	**Epiglottis**
Etiology	Aspiration	Viral	Bacterial
Age	6 mos -	6 mos -4	1 yr -

	5 yrs	yrs	adult
Onset	Usually acute	Days (gradual)	Hours
Signs and Symptoms	Cough, voice change, drooling, dysphagia are possible	Low-grade fever, croupy or seal-bark cough, inspiratory stridor, rhinorrhea	Low-pitched inspiratory stridor, pharyngitis, drooling, fever, lethargic to restless, tachypnea, sitting, muffled cough
Obstruction	Supra/subglottic	Subglottic	Supraglottic
Season	None	Winter	None

Anesthesia for Neonatal Emergencies

1. **Congenital diaphragmatic hernia**
   A. **Manifestations:** three types (left or right posterolateral foramen of Bochdalek or anterior foramen of Morgagni) with left most common; a reduction in alveoli and bronchioli (pulmonary hypoplasia) is accompanied by marked elevation in pulmonary vascular resistance; hallmarks include hypoxia, scaphoid abdomen, bowel in the thorax.
   B. **Management:** gastric distention should be minimized by placement of a nasogastric tube and avoidance of high levels of positive-pressure ventilation; sudden fall in lung compliance, blood pressure or oxygenation may signal a contralateral pneumothorax; hyperventilation is recommended to decrease PVR and minimize right-to-left shunting; after hernia reduction, attempts to expand the hypoplastic lung are not recommended.
2. **Tracheoesophageal fistula (TEF)**
   A. **Manifestations:** most common is combination of upper esophagus that ends in a blind pouch and a lower esophagus that connects to the trachea; breathing results in gastric distention; coughing, cyanosis and choking occur with the first feeding; diagnosis made upon failure to pass a catheter into the stomach. Aspiration pneumonia and the coexistence of other congenital anomalies are common
   B. **Management:** awake tracheal intubation after suction of the esophageal pouch (open gastrostomy tube to air if present); correct endotracheal tube position is the tip of the tube distal to the fistula and above the carina; gastrostomy tube is placed after intubation; frequent ETT suction.
3. **Omphalocele and gastroschisis**
   A. **Gastroschisis:** defect in abdominal wall lateral to umbilicus; no hernial sac; no associated congenital anomalies.

B. **Omphalocele:** defect in abdominal wall at the base of the umbilicus; hernial sac present; associated with congenital anomalies (trisomy 21, cardiac anomalies, diaphragmatic hernia, bladder anomalies).

C. **Management:** decompress stomach before induction; intubate awake or asleep; avoid nitrous oxide; insure adequate muscle relaxation; replace third-space fluid loses aggressively; the neonate commonly remains intubated after the procedure and is weaned from the ventilator over the next 1-2 days.

4. **Necrotizing enterocolitis**

A. **Manifestations:** acquired intestinal tract necrosis that appears in the absence of functional or anatomic lesions. Predominantly in premature. Systemic signs include temperature instability, lethargy, respiratory and circulatory instability, oliguria, and bleeding diathesis.

B. **Management:** risk of aspiration; avoid nitrous oxide; the major challenge is maintaining an adequate circulating blood volume and preventing aspiration; third-space fluid replacement may exceed 100-200 mL/kg/hr.

5. **Myelodysplasia**

A. **Manifestations:** failure of neural tube closure can result in abnormalities ranging from spina bifida to myelomeningocele (abnormality involving vertebral bodies, the spinal cord and the brain stem). Ninety percent of myelomeningocele patients have Arnold-Chiari malformation (downward displacement of the brain stem and the cerebellar tonsils through the cervical spinal canal with medullary kinking, blocking normal circulation of the CSF and leads to progressive hydrocephalus.

B. **Management:** patients come to the OR in the prone position; using sterile towels the patient is placed supine for intubation; anesthesia can be induced with inhalational or IV agents; succinylcholine is not contraindicated; anesthetic technique should allow for rapid extubation following surgery; spinal anesthesia (0.5-0.7 mg/kg of hyperbaric tetracaine with epinephrine) can be used in conjunction.

Cardiac Anesthesia

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Pediatric Cardiovascular Physiology

1. **Fetal circulation**
   A. Acidosis, sepsis, hypothermia, hypoxia, and hypercarbia may cause reopening of the fetal shunts and persistence of the fetal circulation.
   B. Diagnosis of persistent pulmonary hypertension of the newborn can be confirmed by measurement of the PaO_2 in blood obtained simultaneously from preductal (right radial) and postductal (umbilical, posterior tibial, dorsalis pedis) arteries. A difference of 20 mmHg verifies the diagnosis.
2. **Closure of the ductus arteriosus**
   A. In the fetus, patency of the ductus arteriosus is maintained by high levels of prostaglandin (PGI_2 and PGE_1).
   B. Functional closure occurs by contraction of the smooth muscle of the ductal wall and usually occurs 10-15 hours after birth. An increase in PO_2 and a decrease in prostaglandins at birth contribute to functional closure.
   C. Permanent anatomic closure of the duct occurs in 4 to 6 weeks.
3. **Closure of the foramen ovale**
   A. Increase in left atrial over right atrial pressure functionally closes the foramen ovale.
   B. Anatomic closure of the foramen ovale occurs between 3 months and 1 year of age, although 20%-30% of adults and 50% of children less than 5 years of age have a probe-patent foramen ovale.
4. **Closure of the ductus venosus**
   A. Decrease in umbilical venous blood flow causes passive closure of the ductus venosus.
   B. The ductus venosus is functionally closed by 1 week of life and anatomically closed by 3 weeks.

Premedication for Adult Cardiac Surgery

1. Traditional premedications have included morphine 0.1-0.15 mg/kg IM, scopolamine 0.3-0.4 mg IM (0.2 mg for patients older than 70 yrs), diazepam 0.15 mg/kg or lorazepam 0.06 mg/kg PO approximately 1-2 hours prior to surgery. With same day admission patients often receive IV fentanyl and a benzodiazepine (ie, versed) to provide anxiolysis and pain relief instead of IM predications.
2. The dose of premedication should be reduced in patients with critical aortic or mitral stenosis, those undergoing cardiac transplantation, patients with CHF, and patients with renal or hepatic dysfunction.
3. Patients on heparin should not receive any IM medications. A common predication for heparinized patients includes diazepam 0.15 mg/kg PO (or lorazepam 0.04 mg/kg) and morphine 1-10 mg IV or a combination of fentanyl and versed IV.
4. Other orders and medications
   A. Current cardiac medications should be continued; diuretics are usually held except in patients with CHF or afternoon cases.
   B. Nasal cannula oxygen (2-4 lpm) should be ordered along with the premedication (oxygen should be given to all sedated patients).

Cardiopulmonary Bypass

1. **Prebypass period**
   A. This period is characterized by variable levels of stimulation. Stimulating periods include sternal splitting and retraction, pericardial incision, and aortic root dissection and cannulation.
   B. **Baseline laboratory data:** ABG, Hct, and ACT, should be obtained.

C. During sternal splitting, the lungs should be deflated.

D. Aortic root dissection and cannulation: this period can be very stimulating and should be treated aggressively with short-acting agents to minimize the risk of aortic tear or dissection (systolic <100 mmHg).

E. **Heparinization**

1. Heparin 300 IU/kg (400 IU/kg if receiving IV heparin): administration is through a centrally placed catheter; aspirate blood both before and after injection.
2. Check an ACT 5 minutes after heparin administration to monitor the degree of anticoagulation. ACT should be greater than 400 seconds prior to initiating cardiopulmonary bypass. If needed, an additional 100-200 IU/kg is administered.

F. **Checklist prior to initiating cardiopulmonary bypass**

1. Ensure adequate heparinization (ACT).
2. Turn nitrous oxide off, place on 100% oxygen.
3. Pulmonary artery catheter should be pulled back 3-5 cm.
4. Turn transesophageal echo off.
5. Check anesthetic depth and muscle paralysis (bolus as needed).
6. Exam the face (color), eyes (pupils), and EEG (if used).
7. Record pre-CBP UOP and fluid administration.

2. **Bypass period**

A. **Checklist/monitoring during cardiopulmonary bypass**

1. Once adequate flows and venous drainage are established, IV fluids, and positive-pressure ventilation are discontinued.
2. The perfusionist should follow the ACT (to ensure adequate heparinization), ABGs (uncorrected), hematocrit, potassium, calcium.
3. Administer additional anesthetic drugs (muscle relaxants, etc).
4. **Watch for the following**

A. **Hypotension**: venous cannula problems (kink, malposition, clamp, air lock), inadequate venous return (bleeding hypovolemia, IVC obstruction), pump problems (poor occlusion, low flows), arterial cannula problem (misdirected, kinked, partially clamped, dissection), vasodilation (anesthetics, hemodilution, idiopathic, allergic), transducer or monitoring malfunction (stopcocks the wrong way).

B. **Hypertension**: pump problems (increased flow), arterial cannula misdirected), vasoconstriction (light anesthesia, response to temperature changes), transducer or monitor malfunction.

C. **Changes in patients' facial appearance**: suffusion (inadequate SVC drainage), unilateral blanching (innominate artery cannulation), obstruction of jugular venous drainage by caval cannula, head position, neck compression.

D. **Low venous return:** aortic dissection, bleeding, pooling of blood, allergic reaction, obstruction of venous tubing, lack of venous return.

B. **Checklist prior to discontinuing cardiopulmonary bypass**

1. Labs: Hct (22-25% ideal), ABGs, potassium, glucose, and calcium.
2. Ventilate lungs with 100% oxygen (consider suctioning first).
3. Core temperature should be normothermic (37EC).
4. Stable rhythm (preferably sinus rhythm) with adequate heart rate (80-100 beats/min); place pacing wires if necessary.
5. Venting of arterial air (verify by TEE if available).
6. All monitors on and recalibrated.
7. If necessary, drug therapy (vasopressors, etc.) should be started.

C. **Discontinuation from cardiopulmonary bypass**

1. Pressure maintenance: transfuse from CPB reservoir to maintain left atrial pressure or PA occlusion pressure. Optimal filling is determined by blood pressure, cardiac output, and direct observation of the heart.

A. Maintenance of hemodynamics

1. Low cardiac output (despite adequate filling pressure and rhythm) may indicate the need for a positive inotrope. Consider dopamine (first- line agent), dobutamine, amrinone and epinephrine.

2. High cardiac output but low BP consider a vasoconstrictor.
3. RV dysfunction: noted by CVP rising out of proportion to left atrial pressure. Treat know causes of elevated PVR (light anesthesia, hypercarbia, hypoxemia, and acidemia). Consider vasodilator therapy (nitroglycerin, nitroprusside) or inotropic support.
4. Hypertension: treated to prevent bleeding at the suture lines and cannulation sites.

2. Look at the heart to evaluate overall function (TEE if available).
3. Return of CPB reservoir blood to patient.

3. **Post cardiopulmonary bypass period**

A. **Hemodynamic stability is the primary goal.**

B. **Protamine:** once hemodynamic stability is achieved and the aortic and vena caval cannula have been removed, protamine can be given.

1. Initially 25-50 mg is given over 5 minutes, and the hemodynamic response is observed.
2. Monitor PA pressures while administering.
3. In general, 1 mg of protamine is administered for each 1 mg of heparin given.
4. After protamine administration, check ACT and compare to baseline. Additional protamine can be given if needed.
5. During transfusion of heparinized pump blood, additional protamine (25-50 mg) should be given.
6. Correct coagulation and other lab adjustments as needed.

C. Prepare the patient for transfer to ICU.

Acid-Base Management During Cardiopulmonary Bypass Surgery

1. **pH-stat:** requires temperature correction for interpretation of blood gases during CPB. Temperature correction can be accomplished by setting the blood gas analyzer to measure the patient's temperature.
2. **Alpha-stat**: requires no temperature correction for interpreting blood gases. The sample is warmed to 37 degrees C and then measured in the blood gas analyzer as any other sample.

Post-Cardiopulmonary Bypass Bleeding

1. **Differential diagnosis:** uncorrected surgical defects, circulating anticoagulants (residual heparin, heparin rebound, protamine anti?coagulation), and platelet defects (thrombocytopenia).
2. **Treatment**

A. **Circulating anticoagulants:** adequate heparinization should be confirmed with ACT, and additional protamine given if needed.

B. **Platelet abnormalities:** given after other coagulation deficiencies have been corrected and no surgically correctable lesion exists.

C. Deficiencies of circulating procoagulants should be corrected by infusing FFP, cryoprecipitate, or fresh donor blood.

3. **Prevention of post-CPB bleeding** (pharmacological factors)

A. **Desmopressin**

1. Synthetic product that increases plasma levels of Factor VIII and Von Willebrand factor and decreases bleeding times.
2. Dosing. 0.3 mcg/kg IV given over 20-30 min.
3. Side effects. Decreased free water clearance from ADH activity, hypotension, thrombosis, decreased serum sodium, hyponatremic seizures.

B. **Aprotinin:** inhibitor of several proteases and factor XIIa activation of complement (see drug section).

C. **Antifibrinolytic agents**

1. **Epsilon aminocaproic acid (Amicar)**

A. Synthetic antifibrinolytic: inhibits proteolytic activity of plasmin and conversion of plasminogen to plasmin by plasminogen activator.

B. Loading dose 100-150 mg/kg IV followed by constant infusion of 10-15 mg/kg/h.

2. **Tranexamic acid**
   A. Similar mechanism as Amicar but is about 10 times more potent.
   B. Loading dose 10 mg/kg IV followed by infusion of 1 mg/kg/hr.
3. **Complications of antifibrinolytics**
   A. Bleeding into kidneys or ureters may thrombose and obstruct the upper urinary tract.
   B. Contraindicated in DIC.
   C. Hypotension may occur with rapid administration.
   D. May be associated with thrombosis and subsequent stroke, myocardial infarction or deep vein thrombosis.

Automatic Implantable Cardioverter Defibrillator (AICD)

1. **Common indications for AICD implantation**
   A. Patients with a history of near-sudden death who have not responded to drug therapy and are not candidates for arrhythmia surgery.
   B. Patients who have had unsuccessful arrhythmia surgery.
   C. Post cardiac arrest patients who have not had an MI and who have no inducible arrhythmia during electrophysiologic testing.
   D. Patients undergoing endocardial resection for recurrent VT.
2. **Contraindications**
   A. Uncontrolled congestive heart failure.
   B. Frequent recurrences of VT that would rapidly deplete the battery.
3. **Intraoperative testing**
   A. The purpose is to establish the defibrillation threshold (ie, the minimum energy required to defibrillate the heart to a stable rhythm). Internal paddles should be readily available in the operative field during the entire procedure, and external patches should also be placed preoperatively.
   B. If all the tests performed with the ECD unit are successful, an AICD is connected to the leads and ventricular fibrillation is again induced to test the newly implanted unit.
4. **Anesthetic considerations**
   A. **Preoperative assessment**
      1. The indication for the AICD should be noted.
      2. Many patients will be taking antidysrhythmic agents at the time of surgery. In theory, the device and defibrillation thresholds should be tested while the patient is on the drug regimen that is planned postoperatively.
      3. Antidysrhythmic agent of concern is amiodarone, which is negative inotropic agent and vasodilator. Amiodarone may cause refractory bradycardia or may precipitate a profound and prolonged hypotensive state postoperatively.
      4.

   B. **Intraoperatively monitoring:** standard monitors and an arterial line are the minimum required monitors. Central venous access may be considered for administration of vasoactive drugs. PA catheter is generally not needed.
   C. **Anesthetic technique:** general anesthesia with nitrous oxide, narcotic, and muscle relaxant anesthetic is most common.
   D. **Other considerations**
      1. Cardioversion is commonly associated with transient hypertension and tachycardia, probably caused by sympathetic outflow.
      2. Multiple intraoperative inductions of VT or VF may cause profound hypotension.
      3. External defibrillator must be available.
      4. The AICD is occasionally inactivated after being placed to avoid the cautery from trigger the AICD to discharge. The AICD is reactivated postoperatively.
5. **Complications**
   A. **Pacemaker interaction**

1. Both temporary and permanent pacemakers may interact with a AICD by interfering with dysrhythmia detection.
2. The AICD can be deactivated when using temporary pacing, especially, A-V sequential pacing.

B. Mechanical. lead fractures, lead insulation breaks, and lead migration.
C. Rate miscounting leading to unnecessary shocks.
D. Infection.

Vascular Anesthesia

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Carotid Artery Surgery (carotid endarterectomy; CEA)

1. **General considerations**
   A. **Indications:** TIAs associated with ipsilateral severe carotid stenosis (>70% occlusion), severe ipsilateral stenosis in a patient with a minor (incomplete) stroke, and 30-70% occlusion in a patient with ipsilateral symptoms (usually an ulcerated plaque), emboli arising from a carotid lesion, large ulcerated plaque.
   B. **Operative mortality is 1-4%** (primarily due to cardiac complications).
   C. **Perioperative morbidity is 4-10%:** stroke is the most common and expected major complication during and after carotid endarterectomy. Hypertension occurs in about 70% of patients undergoing carotid endarterectomy and is associated with an increase in the risk of stroke.
   D. **Complications:** hematoma with tracheal compression, supraglottic edema, cranial nerve injury (cranial nerves VII, IX, X, and XII), myocardial infarction, intraparenchymal hemorrhage, carotid occlusion, intracerebral hemorrhage, embolism.
2. **Preoperative anesthetic evaluation**
   A. Most patients undergoing CEA are elderly and hypertensive, with generalized arteriosclerosis. Preoperative evaluation should include a through cardiac and neurologic evaluation.
   B. **The anesthetic goal** is to maintain adequate cerebral perfusion without stressing the heart. In addition, the patient should be sufficiently responsive immediately after surgery to obey commands and thereby facilitate neurologic evaluation.
3. **Anesthetic management**
   A. **Anesthetic technique** (general or regional anesthesia can be used)
      1. **Regional anesthesia**
         A. Regional anesthesia can be achieved by performing a superficial and deep cervical plexus block, which effectively blocks the C2-C4 nerves. The principal advantage of this technique is that the patient remains awake and can be examined intraoperatively. The need for a temporary shunt can be assessed and any new neurologic deficits diagnosed during surgery.
         B. Disadvantages of regional anesthesia include patient discomfort and loss of cooperation, confusion, panic, or seizures. The awkwardness of these possibilities discourages the majority from using the technique.
      2. **General anesthesia:** commonly thiopental or propofol followed by nitrous oxide plus a volatile and/or opioids for maintenance.
   B. **Monitoring**
      1. Intraarterial blood pressure monitoring is mandatory.
      2. Additional hemodynamic monitoring should be based primarily on the patients underlying cardiac function. Carotid endarterectomy is not usually associated with significant blood loss or fluid shifts.
      3. Cerebral monitoring: electroencephalogram and somatosensory evoked potentials (SSEP) have been used to determine the need for a shunt.
   C. Despite technique mean arterial blood pressure should be maintained at or slightly above the patient's usual range. During carotid occlusion blood pressure should be maintained at or up to 20% higher than the patient's highest recorded resting blood pressure while awake.
   D. Surgical manipulation of the carotid sinus can cause abrupt bradycardia and hypotension. This may be prevented by infiltration of the sinus with local anesthetic. If infiltration has not been performed, then clamp application may cause hypertension and tachycardia since the sinus is now sensing a low pressure.
   E. **Ventilation** should be adjusted to maintain normocapnia. Hypocapnia can produces cerebral vasoconstriction. Hypercapnia can induce intracerebral steal phenomenon.
   F. **Heparin (5000-10,000 units IV)** is usually given prior to occlusion of the carotid artery. Protamine, 50-75 mg, can be given for reversal prior to skin closure.
   G. **Postoperative considerations**

1. Postoperative hypertension may be related to surgical denervation of the ipsilateral carotid baroreceptor. Hypertension can stress and rupture the surgical anastomosis resulting in the development of a wound hematoma, which can rapidly compromise the airway.
2. Transient postoperative hoarseness and ipsilateral deviation of the tongue may occur. They are due to surgical retraction of the recurrent laryngeal and hypoglossal nerves, respectively.

Surgery of the Aorta

1. **Ascending aorta**
   A. Surgery routinely uses median sternotomy and CPB.
   B. Anesthesia is similar to that for cardiac operations involving CPB.
   C. The left radial artery should be used to monitor arterial blood pressure, because clamping of the innominate artery maybe necessary during the procedure.
2. **Aortic arch:** usually performed through a median sternotomy with deep hypothermic circulatory arrest. See section on DHCA.
3. **Descending thoracic aorta**
   A. Generally performed through a left thoracotomy without CPB.
   B. **Monitoring**
      1. Arterial blood pressure should be monitored from the right radial artery, since clamping of the left subclavian may be necessary.
      2. Pulmonary artery catheter is helpful for following cardiac function and intraoperative fluid management.
   C. **Cross clamping of the aorta**
      1. Cross clamping results in a sudden increase in left ventricular afterload which may precipitate acute left ventricular failure or myocardial ischemia in patients with underlying ventricular dysfunction or coronary disease. A nitroprusside infusion is usually required to prevent excessive increases in blood pressure.
      2. **Release hypotension:** following the release of the aortic cross clamp, the abrupt decrease in afterload combined with bleeding and the release of vasodilating acid metabolites from the ischemic lower body can precipitate severe systemic hypotension. Decreasing anesthetic depth, volume loading, and partial or slow release of the cross-clamp may help decrease the severity of hypotension.
   D. **Complications**
      A. **Paraplegia:** the incidence of transient postoperative deficits (11%) and postoperative paraplegia (6%).
      B. The classic deficit is that of an anterior spinal artery syndrome with loss of motor function and pinprick sensation but preservation of vibration and proprioception.
      C. **Artery of Adamkiewicz:** this artery has a variable origin from the aorta, arising between T5 and T8 in 15% , between T9 and T12 in 60%, and between L1 and L2 in 25% of patients.
      D. Measures used to help protect the spinal cord include: use of a temporary heparin coated shunt or partial cardiopulmonary bypass; mild hypothermia; mannitol (related to its ability to lower cerebrospinal pressure by decreasing its production); and drainage of cerebrospinal fluid.
      E. **Renal failure:** infusion of mannitol (0.5 g/kg) prior to cross-clamping may decrease the incidence of renal failure. Low dose dopamine has not been shown to be as effective but may be used as an adjunct for persistently low urine output.
4. **Abdominal aorta**
   A. Either an anterior transperitoneal or an anterolateral retroperitoneal approach is commonly used.
   B. Monitoring includes arterial line, central venous line or pulmonary artery catheter and EKG monitoring with ST segment analysis.
   C. The aorta cross-clamp is usually applied to the supraceliac, suprarenal, or infrarenal aorta. In general, the farther distally the clamp is applied, the less the effect on left ventricular afterload. Heparinization is necessary prior to cross-clamp.
   D. Release of the aortic clamp frequently produces hypotension. The same techniques to prevent release hypotension as discussed above should be used. Cross-clamp placed at the level of the

infrarenal aorta in patients with good ventricular function frequently have minimal hemodynamic changes when the clamp is removed.

E. Fluid requirements are typically increased (up to 10-12 mL/kg/hr) because of the large incision and extensive retroperitoneal surgical dissection. Fluid requirements should be guided by central venous or pulmonary artery pressure monitoring.

F. Renal prophylaxis with mannitol or low dose dopamine should be considered, especially in patients with preexisting renal disease. Clamping of the infrarenal aorta has been shown to significantly decrease renal blood flow, which may contribute to postoperative renal failure.

G. Epidurals are commonly placed both for intraoperative and postoperative use. The combined technique of epidural and general anesthesia decreases the general anesthetic requirement.

Thoracic Anesthesia

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General Considerations

1. The principal goal is to identify patients at risk for both pulmonary and cardiac complications and to start appropriate perioperative therapy.
2. Pulmonary function tests: helpful in identifying patients at increased risk of developing pulmonary complications and in evaluating responses to preoperative pulmonary therapy.
3. Cessation of smoking for at least 4-8 weeks before surgery is associated with decreased incidence of postoperative respiratory complications.
4. Patients undergoing resection of pulmonary tissue are at increased risk of cardiac dysrhythmias because the decrease in available pulmonary vascular bed can cause postoperative right atrial and ventricular enlargement. Consider prophylactic digitalis or beta blockers.

Evaluation of Lung Resectability

1. Initial evaluation includes PFTs, room air arterial blood gas (ABG) and carbon monoxide diffusion capacity (DLCO).
2. If FEV_1 < 50%, FVC <2L, RV/TLC > 50%, maximum breathing capacity (MBC) < 50% pred, or $PaCO_2$ > 40 mmHg, then resection is often contraindicated unless split function PFTs can document that a disproportionate amount of effective ventilation is coming from lung that is not going to be resected.
3. Low FEV_1 or low FVC suggest limited mechanical reserve. An FEV_1 of less than 800 mL is incompatible with life for most adult humans. Mortality is inversely proportional to FEV_1 (patients with high FEV_1 will do well while patients with low FEV_1 can be expected to require post-operative ventilation for a protracted period of time, and may become impossible to liberate from mechanical ventilation).
4. An FVC 3x TV is necessary for an effective cough. Mortality is also inversely proportional to FVC.
5. An RV/TLC of >50% suggests that the patient has near-terminal COPD with airway closing volumes that are approaching TLC. Surgery can be expected to significantly reduce their remaining reserve and may make them impossible to liberate from the ventilator.
6. **Intraoperative management**
   A. General anesthesia in combination with a thoracic epidural is preferred.
   B. Nitrous oxide can be used (limit use during one-lung anesthesia).

One-Lung Anesthesia

1. **Indications for one-lung anesthesia**
   A. Absolute: confined pulmonary infection or bleeding to one side, separate ventilation to each lung, bronchopulmonary fistula, tracheobronchial disruption, large lung cyst, bronchopleural lavage.
   B. Relative: thoracic aortic aneurysm, pneumonectomy, lobectomy, thoracoscopy, sub-segmental resections, esophageal surgery.
2. **Physiology of one-lung anesthesia**
   A. One-lung anesthesia results in a large ventilation-perfusion mismatch, secondary to a large intrapulmonary shunt.
   B. Factors known to inhibit hypoxic pulmonary vasoconstriction include: (1) very high or very low pulmonary artery pressures; (2) hypocapnia; (3) vasodilators; (4) high or low mixed venous oxygen; (5) pulmonary infection; (6) volatile anesthetics.
   C. Factors that decrease blood flow to the ventilated lung: high mean airway pressure; vasoconstrictors; low FIO_2.

D. Carbon dioxide elimination is usually not affected by one-lung anesthesia provide minute ventilation is unchanged.

3. **Double-lumen endotracheal tubes**

A. There are both right and left-sided double-lumen tubes (DLT). Left-sided Robertshaw types are the most common and are designed with a bronchial lumen that has its own cuff and extends distal to the carina. The choice or size of DLT is based on the patients height:

Patient height	Tube size	Depth of insertion
136-164 cm	37 Fr	27 cm
165-179 cm	39 Fr	29 cm
180-194 cm	41 Fr	31 cm

B. Bronchial blockers: involve the placement of a Fogarty embolectomy catheter through a conventional endotracheal tube. The balloon is position in one of the mainstem bronchi, using the fiber-optic bronchoscope for guidance.

C. Univent tube: endotracheal tube containing a bronchial blocker. Once the balloon is inflated, the blocked lung can be vented to the atmosphere and allowed to collapse.

D. Algorithm for checking placement of a double-lumen tube

1. When the bronchial lumen is clamped and the vent opened, breath sounds and chest rise should be minimal on the involved side and normal on the other. There should be no leak at the vent port. The reverse is true when the tracheal lumen is clamped and the vent is opened. It should be noted that auscultation is the least sensitive method to confirm proper placement.
2. Use the fiberoptic bronchoscope to check position.
3. Re-check the position of the tube once you position the patient in the lateral decubitus position.
4. Check the airway pressures during one lung ventilation.

4. **Intraoperative ventilatory management:** higher inspired oxygen concentration usually required, tidal volume 10-12 mg/kg at a rate to maintain the $PaCO_2$ near 35 mmHg; frequent arterial gases should be done to assess oxygenation.

5. **Management of hypoxia during one-lung anesthesia**

A. Confirm tube placement. Increase oxygen to 100%.
B. Change tidal volume (8-15 cc/kg) and ventilatory rate.
C. Periodic inflation of the collapsed lung with 100% oxygen.
D. Continuous insufflation of oxygen into the collapsed lung.
E. Adding 5 cm H_2O of continuous positive airway pressure (CPAP) to the collapsed lung.
F. Adding 5 cm H_2O of positive end expiratory pressure (PEEP) to the ventilated lung.
G. Adding additional CPAP, followed by additional PEEP.
H. Early ligation of the ipsilateral pulmonary artery (in a pneumonectomy).

Obstetrical Anesthesia

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Fundamentals of Obstetrical Anesthesia

1. **Stages of labor**
   A. **First stage:** this stage begins with the onset of regular contractions and ends with full cervical dilation. Pain during the first stage is caused by uterine contractions and cervical dilatation. Pain is carried by the visceral afferent fibers (T10 to L1).
   B. **Second stage:** this stage begins with full cervical dilation and ends with delivery of the infant. Pain at the end of the first stage signals the beginning of fetal descent. Pain in the second stage of labor is due to stretching of the birth canal, vulva, and perineum and is conveyed by the afferent fibers of the posterior roots of the S2 to S4 nerves.
   C. **Third stage:** the period of time from delivery of the infant to delivery of the placenta.
2. **Physiological changes in pregnancy**
   A. **Hematological changes:** increased plasma volume (40-50%), increased total blood volume (25-40%), dilutional anemia (hematocrit 31.9-36.5%).
   B. **Cardiovascular changes:** increased cardiac output (30-50%), decreased systemic vascular resistance (35%), increased heart rate (15-20 bpm).
   C. **Pulmonary changes:** increased minute ventilation (50%), decreased functional residual capacity (20%), airway edema (can make for difficult intubation), decreased $PaCO_2$ and PaO_2 (about 10 mmHg each).
   D. **Gastrointestinal changes:** prolonged gastric emptying, decreased lower esophageal sphincter tone.
   E. **Altered drug responses:** decreased requirements for inhaled anesthetics (MAC), decreased local anesthetic requirements.
3. **Fetal heart monitoring**
   A. **Beat-to-beat variability:** fetal heart rate varies 5-20 bpm with a normal heart rate range of 110-160 bpm; variability is associated with fetal well-being; fetal distress due to arterial hypoxemia, acidosis, or CNS damage is associated with minimal to absent variability of FHR (drug induced loss of variability does not appear deleterious).
   B. **Early decelerations:** slowing of the FHR that begins with the onset of the uterine contraction; caused by head compression (vagal stimulation); not indicative of fetal distress.
   C. **Late decelerations:** characterized by slowing of the FHR that begins 10-30 seconds after the onset of the uterine contraction; reflects hypoxia caused by uteroplacental insufficiency; associated with fetal distress.
   D. **Variable decelerations:** deceleration pattens are variable in magnitude, duration, and time of onset; caused by umbilical cord compression; unless prolonged beyond 30 seconds or associated with fetal bradycardia (<70 bpm) they are usually benign.
4. **Aortocaval compression syndrome**
   A. Caused by impaired venous return when the gravid uterus compresses the inferior vena cava, decreasing venous return to the heart.
   B. **Symptoms:** hypotension, tachycardia, pallor, sweating, nausea, vomiting, and changes in cerebration.
   C. Aortocaval compression is prevented by uterine displacement (lateral position) to increase venous return.
5. **Medications used during labor**
   A. **Vasopressors:** hypotension can result from regional anesthesia, aorto-caval compression, or peripartum hemorrhage. Ephedrine provides both cardiac stimulation and increased uterine blood flow. Ephedrine is the drug of choice for the treatment of maternal hypotension. Phenylephrine, being a pure alpha-adrenergic agent, increases maternal blood pressure at the expense of uteroplacental blood flow.
   B. **Oxytocin (Pitocin)**

1. **Indications:** oxytocin stimulates uterine contractions a, used to induce or augment labor, to control postpartum bleeding and uterine atony.
2. Oxytocin stimulates frequency /force of contractions of uterine smooth muscle and may cause hypotension, dysrhythmias, and tachycardia.

C. **Tocolytics**

1. **Indications:** used to delay or stop premature labor, to slow or arrest labor while initiating other therapeutic measures.
2. **Contraindications:** chorioamnionitis, fetal distress, and preeclampsia or eclampsia (PAH).
3. **Terbutaline and ritodrine**
   A. **Selective beta-2 agonist:** beta-2 stimulation also produces bronchodilation and vasodilation and may result in tachycardia; may cause dysrhythmias, pulmonary edema, hypertension, hypokalemia, or CNS excitement.
   B. **Terbutaline dose:** 10 mcg/min IV infusion; titrate to a maximum dose of 80 mcg/min.
   C. **Ritodrine dose:** IV infusion of 0.1-0.35 mg/min.
4. **Magnesium sulfate** is used most commonly in PAH, but it is also used as a tocolytic (see section on magnesium sulfate).

Anesthesia for Labor and Delivery

1. **Common parenteral pain medications:** meperidine (25-50 mg IV; 50-100 mg IM), morphine (2-5 mg IV), fentanyl (25-50 mcg IV) butorphanol (2mg) and nalbuphine (10 mg) are frequently used to relieve pain and anxiety.
2. **Lumbar epidural blockade**

A. Epidurals are placed after the patient is in active labor (5-6 cm dilated in a primipara, 3-4 cm in a multipara).

B. Place epidural in usual manner after maternal informed consent, hydration, and placement of appropriate monitors.

C. **Test dose:** use 3 mL of lidocaine 1.5% with epi to rule out accidental IV or subarachnoid injection (maybe inconclusive because of heart rate variability in the laboring patient).

D. **Initial epidural block** (options)
   1. Bupivacaine 0.125-0.25%, lidocaine 1%, or chloroprocaine 2% (8-15 mL).
   2. Sufentanil 10-15 mcg or 100-200 mcg fentanyl in 10 mL of saline.
   3. Bupivacaine 0.0625-0.125% + fentanyl 50 mcg or sufentanil 10 mcg.

E. **Subsequent analgesia (options)**
   1. Intermittent: rebolus as needed to maintain maternal comfort.
   2. Continuous infusions options (rate = 8-15 mL/hr)
      A. Bupivacaine 0.04-0.125% + fentanyl 1-2 mcg/mL or sufentanil 0.1-0.3 mcg/mL.
      B. Bupivacaine 0.125% without opiate.
      C.
   3. Patient controlled epidural analgesia (using above mixtures baseline infusion 4-6 mL/hr with controlled bolus of 3-4 mL q20-30 minutes).

F. **Blood pressure** should be monitored every few minutes for 20-30 minutes and every 10-15 minutes thereafter until block wears off.

G. Patients should be maintained in the lateral position and turned side-to-side every hour to avoid a one-sided block.

H. **Sensory level**, adequacy of anesthesia and motor block should be checked regularly. Watch for intravascular or subarachnoid migration.

I. Adjust infusion rate and concentration as needed to control pain.

3. **Intrathecal opioids for labor**

A. Can be used for multiparas in very active labor (6-9 cm) or primiparas who are fully dilated with significant pain. They can also be used for patients in early labor, 2-4 cm dilated, prior to active phase.

B. Fentanyl 10-25 mcg in 1 cc preservative free saline provides about 30-120 minutes of analgesia.

C. Meperidine 10-20 mg provides about 2 hours of analgesia.

D. Sufentanil 10 mcg in 1 cc preservative free saline provides about 60-180 minutes of analgesia.

4. **Spinal anesthesia for labor (saddle block)**
   A. Commonly used if a forceps delivery is required or in the postpartum period, for repair of traumatic lacerations of the vagina or rectum or for removal of retained placenta.
   B. Bupivacaine 1.25-2.5 mg (with sufentanil 10 mcg or fentanyl 25 mcg) or hyperbaric 5% lidocaine 20-40 mg injected intrathecally.
5. **Combined spinal-epidural for labor**
   A. Combined spinal/epidural may be useful for patients presenting in early labor because the spinal can be given to help with early labor pain, while the epidural can be activated after the patient is in active labor.
   B. Spinal: 25 mcg fentanyl or 10 mcg Sufentanil in 1 cc PF saline.
   C. An epidural is initiated as noted above after the pain returns.
6. **Paracervical block:** local anesthetic is injected in the submucosa of the fornix of the vagina lateral to the cervix; only effective during the first stage of labor; high incidence of fetal bradycardia.
7. **Pudendal block:** 10 mL of local anesthetic is placed transvaginally behind each sacrospinous ligament provides complete analgesia for episiotomy and its repair and is sufficient for low forceps deliveries.
8. **General anesthesia:** rarely used and requires intubation.

Anesthesia for Cesarean Section

1. **Anesthetic management**
   A. All patients should have a wedge under the right hip for left uterine displacement (15 degrees) and should receive Bicitra 30 cc PO (metoclopramide 10 mg IV is optional).
   B. Maternal informed consent, hydration (based on clinical setting), and placement of appropriate monitors.
   C. Pre-op labs: hematocrit, hemoglobin, clot to blood bank; patients with PIH check PT/PTT, platelet, and bleeding time prior to block (if used).
   D. If systolic blood pressure falls by 30% or below 90 mmHg, ensure left uterine displacement and increase IV infusion rate. If blood pressure is still not restored, administer 5-15 mg ephedrine IV, repeat prn.
   E. If the block (epidural or spinal) becomes “patchy” prior to delivery of the baby, should be treated with ketamine, 10-20 mg IV, or 40-50% nitrous; after delivery, fentanyl 0.5-1.0 mcg/kg IV and/or versed 0.25-1.0 mg.
   F. If anesthesia remains inadequate with spinal or epidural block, proceed to general anesthesia with endotracheal intubation.
   G. After placenta delivered, oxytocin 20-40 units should be added to IV fluids (if uterine bleeding does not decrease may give Methergine 0.2 mg IM).
2. **Epidural anesthesia**
   A. Place catheter in usual manner and give test dose.
   B. **Local anesthetic options** (15-30 mL total dose in 5 mL increments)
      1. Lidocaine 1.5-2%
      2. Bupivacaine 0.5%
      3. Chloroprocaine 3%
   C. **Additives**
      1. Epinephrine may be added to a maximum conc. of 1:200,000.
      2. Sodium bicarbonate, 1 cc for each 10 cc of local anesthetic, can be added to speed up onset.
   D. **Opioid options**
      1. Fentanyl 50-100 mcg or sufentanil 10-20 mcg.
      2. Duramorph 3-5 mg given after the umbilical cord is clamped, provides 18-24 hours of postoperative pain relief.
3. **Spinal anesthesia**
   A. **Local anesthetic options**
      1. Tetracaine 1% (7-10 mg)

2. Lidocaine 5% (60-75 mg)
3. Bupivacaine 0.75% (8-15 mg)

B. **Additives**
1. Epinephrine 0.2 mg

C. **Opioid options**
1. Fentanyl 10-25 mcg or sufentanil 10 mcg.
2. Duramorph 0.10-0.25 mg provides 18-24 hours of postoperative pain relief.

4. **General anesthesia**

A. Generally reserved for emergency cesarean sections when regional anesthesia is refused or contraindicated, when substantial hemorrhage is anticipated, or when uterine relaxation is required.

B. General anesthesia allows for rapid induction, control of airway, and decreased incidence of hypotension. Aspiration and failed intubation remain a major cause of morbidity and mortality.

C. **Technique**

1. Patients should be premedicated with Bicitra, 30 cc, consider metoclopramide 10 mg, cimetidine, 300 mg, or ranitidine, 50 mg.
2. Position the patient with left uterine displacement. Standard monitors, fetal heart rate monitor.
3. Preoxygenate with 100% oxygen for 3 minutes or 5-6 deep breaths.
4. Rapid-sequence induction with cricoid pressure is performed with thiopental 4-5 mg/kg (ketamine 1 mg/kg for asthmatics and hemodynamically unstable patients) and succinylcholine 1.5 mg/kg.
5. Anesthesia is maintained with a 50% mixture of nitrous and oxygen, combined with a volatile agent (enflurane 0.5-0.75% or isoflurane 0.75%). Use muscle relaxant as necessary. Hyperventilation should be avoided because of adverse effects on uterine blood flow.
6. After the umbilical cord is clamped, a muscle relaxant may be administered (usually one dose of atracurium, 0.5 mg/kg, or vecuronium, 0.05 mg/kg), fentanyl 100-150 mcg, versed 1-2 mg, and nitrous 70%/oxygen 30%, consider discontinuing or using low doses of inhalation agent.
7. Oxytocin (10-40 units/l) is added to the IV infusion after delivery of the placenta to stimulate uterine contraction.
8. Prior to extubation an orogastric tube should be passed to empty the stomach. Extubate when the patient is awake.

Pregnancy-Associated Hypertension (PAH)

1. **Incidence:** 5-15% of all pregnancies and is major cause of obstetric and perinatal morbidity and mortality. The cause is unknown and symptoms usually abates within 48 hours following delivery.
2. PAH is a syndrome manifesting after the 20^{th} week of gestation characterized by hypertension (greater then 140/90 mmHg or a greater then 30/15 mmHg increase from baseline), proteinuria (> 500 mg/day), generalized edema, and complaints of headache.
3. **Severe PAH** is defined as BP > 160/110, pulmonary edema, proteinuria >5 gm/day, oliguria, central nervous system manifestations, hepatic tenderness, or HEELP syndrome.
4. **Eclampsia** occurs when severe PAH progresses to seizures and is associated with a maternal mortality of about 10%.
5. **Predisposing factors:** multiple gestation, major uterine anomalies, chronic hypertension, chronic renal disease, diabetes, polyhydramnios, molar pregnancy, fetal hydrops.
6. **Pathophysiologic alterations**

A. **Hematologic:** decrease in intravascular volume (primarily plasma), disseminated intravascular coagulation characterized initially by reduction in platelets; later by rise in fibrin degradation products, fall in fibrinogen level, increased PT/PTT.

B. **Cerebral:** hyperreflexia, CNS irritability increase, coma, increased intracranial pressure, altered consciousness.

C. **Respiratory:** upper airway and laryngeal edema.

D. **Cardiac:** arteriolar constriction and increase of peripheral resistance leading to increased BP.

E. **Ophthalmic:** retinal arteriolar spasm, blurred vision, retinal edema and possible retina detachment.

F. **Renal:** reduction in renal blood flow and GFR, elevated plasma uric acid (increased levels correlate with severity of disease), deposition of fibrin in glomeruli.

G. **Hepatic:** elevated LFTs, hepatocellular damage or edema secondary to vasospasm, epigastric or right upper quadrant abdominal pain.

7. **General management**

A. **Definitive therapy** includes delivery of fetus and the placenta with symptoms usually resolving within 48 hours.

B. **Antihypertensive drugs:** hydralazine is the agent of choice because it increases both uteroplacental and renal blood flow. Labetalol can also be used. Continuous infusions of nitroprusside can be used in treating hypertensive crisis or acute increases in blood pressure.

C. **Fluid management:** fluids are generally not restricted. Intravascular depletion should be corrected with crystalloids.

D. **Magnesium therapy:** magnesium sulfate is a mild vasodilator and central nervous system depressant. Give 2-4 gram loading dose (slow IV over 5-15 minutes), followed by continuous infusion of 1-3 gm/hour. Therapeutic maternal blood levels of 4-6 mEq/l should be maintained

8. **Anesthetic management**

A. All patients should have a bleeding time, platelet count, coagulation profile, CBC, Mg level, fibrinogen, fibrin split products, electrolytes, uric acid level, and LFTs prior to anesthesia.

B. **Before placing block** prehydrate as guided by clinical exam, urine output, oxygenation, and central venous pressure monitoring (if used).

C. Patients should have blood pressure under control before (DBP<110) starting epidural. Epidural anesthesia is the preferred method of analgesia for vaginal delivery and cesarean section in most patients including those with eclampsia. Spinal can be used. General anesthetics are reserved for fetal distress, coagulopathies or hypovolemia.

D. **Exaggerated edema** of the upper airway structures may require the use of smaller tracheal tubes than anticipated.

E. **Indications for invasive monitoring**

1. Unresponsive or refractory hypertension: increased systemic vascular resistance or increased cardiac output.
2. Pulmonary edema: cardiogenic or left ventricular failure, increased systemic vascular resistance, or noncardiogenic volume overload.
3. Persistent arterial desaturation.
4. Oliguria unresponsive to modest fluid loading: low preload, severe increased systemic vascular resistance with low cardiac output, selective renal artery vasoconstriction.

9. **HELLP syndrome**

A. **HELLP syndrome:** hemolysis, elevated liver enzymes, low platelets.

B. **Incidence:** 4-12% of severe PAH patients.

C. **Reported perinatal mortality:** 7.7-60%; maternal mortality 3.5-24.2%.

D. **Diagnostic criteria:** platelet count less than 100,000/mm^3, hemolysis by peripheral smear and increased bilirubin greater than 1.2 mg/dL, SGOT greater than 70 U/L and LDH greater than 600 U/L.

E. High incidence of maternal complications including abruptio placenta, coagulopathy (DIC, prolonged PT and PTT), acute renal failure, ruptured hepatic hematoma.

Peripartum Hemorrhage

1. **Placenta previa**: abnormal implantation of the placenta in the lower uterine segment; incidence is 0.1-1.0% (higher in subsequent pregnancies); presents with painless vaginal bleeding typically around the 32^{nd} week of gestation; potential for massive blood loss; risk factors include prior uterine scar, prior placenta previa, advanced maternal age, and multiparity.

2. **Abruptio placentae:** premature separation of a normally implanted placenta after 20 weeks of gestation; incidence is 0.2-2.4%; may present with painful vaginal bleeding, hemorrhagic shock, fetal distress, irritable uterus; potential for massive blood loss (blood loss may be concealed), disseminated intravascular coagulation (DIC), acute renal failure; risk factors: hypertension, uterine abnormalities, history of cocaine abuse.

3. **Uterine rupture:** incidence: 0.008-0.1% ; majority are spontaneous without explanation; risk factors: previous uterine surgery, prolonged intrauterine manipulation, rapid spontaneous delivery, excessive oxytocin stimulation; may present with sudden onset of breakthrough pain (although most patients with uterine rupture have no pain) with or without vaginal bleeding, abnormalities in fetal heart rate, irritable uterus; potential for massive blood loss.
4. **Vasa previa:** a condition in which the umbilical card of the fetus passes in front of the presenting part making them vulnerable to trauma during vaginal examination or during artificial rupture of membranes; bleeding here is from the fetal circulation only.
5. **Retained placenta:** incidence is about 1% of all vaginal deliveries and usually requires manual exploration of the uterus; if no epidural or spinal was used analgesia can be provided with IV opioids, nitrous oxide, or small doses of ketamine; if uterine relaxation is required, and bleeding is minimal, nitroglycerin, 50-100 mcg boluses, can be given (occasionally general anesthesia is required for relaxation).
6. **Uterine atony:** occurs in 2-5% of patients; treated with synthetic oxytocins which do not contain vasopressin.
7. **Laceration** of the vagina, cervix or perineum are common.
8. **Uterine inversion** is very rare and is a true obstetrical emergency; general anesthesia is generally required to allow immediate uterine relaxation; these patients can exsanguinate rapidly.

Anesthesia for Nonobstetric Surgery During Pregnancy

1. Approximately 1-2% of pregnant patients require surgery during their pregnancy. Maternal morbidity and mortality is unchanged from that of nonpregnant women, but fetal mortality ranges from 5-35%.
2. **Avoidance of teratogenic drugs:** the critical period of organogenesis is between 15 and 56 days; there is no clear evidence that anesthetics administered during pregnancy are teratogenic.
3. **Avoidance of intrauterine fetal hypoxia and acidosis:** minimized by avoiding maternal hypotension (with left uterine displacement), arterial hypoxemia, and excessive changes in $PaCO_2$.
4. **Prevention of premature labor:** the underlying pathology necessitating the surgery, and not the anesthetic technique, determines the onset of premature labor.
5. **Anesthetic management:** regional anesthesia should be used when possible to minimize fetal exposure; fetal heart rate and uterine activity should be monitored with a doppler and tocodynamometer after the 16^{th} week of gestation; if general anesthesia is chosen it is recommended to use low concentrations of volatile drugs and FIO_2 greater than 50%.

Neuroanesthesia

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Neurophysiology and Neuropharmacology

1. **Cerebrospinal fluid (CSF)**
   A. **Produced at a rate of 0.3 cc/min** primarily by the choroid plexuses of the cerebral (mainly lateral) ventricles. CSF is reabsorbed at a rate of 0.3-0.4 cc/min into the venous system by the villi in the arachnoid membrane.
   B. CSF production is decreased by carbonic anhydrase inhibitors (acetazolamide), corticosteroids, spironolactone, loop diuretics (furosemide), isoflurane, and vasoconstrictors.
   C. **Cerebral spinal fluid volume:** 100-150 mL normal.
2. **Cerebral blood flow**
   A. **Cerebral blood flow rates** averages 50 mL/100 gm/min and represents 15-20% of cardiac output and consumes 20% of the oxygen.
   B. **Cerebral blood flow determinants**
      1. **$PaCO_2$:** for every 1 mmHg change in $PaCO_2$ there is a corresponding change in CBF by 1-2 mL/100 g/min. Cerebral blood flow is directly proportionate to $PaCO_2$ between tensions of 20 and 80 mmHg.
      2. **PaO_2:** no significant increase in CBF until below 50 mmHg.
      3. **Temperature:** cerebral blood flow changes 5-7% per degree Celsius. Hypothermia decreases both $CMRO_2$ and CBF. Cerebral metabolic rate decreases 7% for every 1 degree Celsius reduction in temperature.
      4. **Cerebral perfusion pressure autoregulation:** chronic hypertension shifts the autoregulation curve to the right; autoregulation is impaired in presence of intracranial tumors or volatile anesthetics.
      5. **Hematocrit:** CBF increases with decreasing viscosity (hematocrit). Optimal cerebral oxygen delivery occurs at Hct between 30-34%.
      6. Regionally, CBF and metabolism are tightly coupled. An increase in cortical activity will lead to a corresponding increase in CBF.
      7. Sympathetic tone does not appreciably affect CBF.
3. **Intracranial pressure (ICP)**
   A. **Normal ICP is 5-10 mmHg.**
   B. **Intracranial hypertension** is defined as a sustained increase in ICP above 15 mmHg. When intracranial pressure exceeds 30 mmHg, cerebral blood flow progressively decreases and a vicious cycle is established: ischemia causes brain edema, which in turn increases intracranial pressure, resulting in more ischemia.
   C. Periodic increases in arterial blood pressure with reflex slowing of the heart rate (Cushing response) are often observed and can be correlated with abrupt increases in intracranial pressure lasting 1-15 minutes.
   D. **Cerebral perfusion pressure (CPP)** = MAP - ICP (or CVP).
   E. **Increased intracranial pressure**
      1. **Symptoms:** nausea/vomiting, mental status changes (drowsiness progressing to coma), personality changes, visual changes, neck stiffness, focal deficits, hypertension, bradycardia, absent brain stem reflexes, decerebrate posturing, fixed and dilated pupils, respiratory rhythm changes (irregular rhythm or apnea).
      2. **Signs:** headache, papilledema, posturing, bulging fontanelles in infants, seizures, altered patterns of breathing, cushing's reflex (hypertension and bradycardia).
      3. **Radiologic signs**
         A. X-ray: suture separation, erosion of clinoid process, copper-beaten skull.
         B. **CT/MRI scans:** midline shift, cerebral edema, mass lesions, abnormal ventricular size, obliteration of basal cistern.
         C.

4. **Cushing reflex**
   A. **Cushing reflex:** periodic increases in arterial blood pressure with reflex slowing of the heart is the Cushing response and often observed and correlated with abrupt increases in intracranial pressure (plateau or A waves) lasting 1-15 minutes.
   B. **Cushing triad:** hypertension, bradycardia, respiratory disturbances (late and unreliable sign that usually just proceeds brain herniation).
   C. Continued profound sympathetic nervous system (SNS) discharge during Cushing's reflex may hide a state of hypovolemia. If the Cushing source is taken away by surgical intervention and/or the SNS response is ablated by anesthesia, one may encounter profound and resistant hypotension.
5. **Compensatory mechanisms for increased ICP:** displacement of CSF from the cranial to the spinal compartment, increase in CSF absorption, decrease in CSF production, decrease in total cerebral blood volume.
6. **Treatment of elevated ICP.**
   A. **Reduce cerebral blood volume**
      1. Hyperventilation ($PaCO_2$ 20-25 mmHg). Excessive hyper-ventilation ($PaCO_2$ <20) may cause cerebral ischemia.
      2. Prevent straining or coughing on the endotracheal tube.
      3. Elevation of the head to encourage venous drainage.
   B. **Reduce cerebrospinal fluid volume**
      1. Ventriculostomy or lumbar subarachnoid catheter.
      2. Decrease CSF production with acetazolamide.
      3. Recent studies suggest that administration of hypertonic saline and mannitol reduce the production of CSF and may contribute to the immediate effect of ICP reduction.
      4. Reduce brain volume by decreasing brain water with osmotic diuretics (20% mannitol 0.25 - 1.0 g/kg); mannitol is thought to reduce cerebral swelling by osmotic dehydration, loop diuretics (furosemide 0.5 mg/kg), and steroids (Decadron).
      5. Barbiturates are potent cerebral vasoconstrictors that decrease cerebral blood volume while decreasing cerebral metabolic rate.

4. **Methods of cerebral protection**
   A. **Barbiturates, etomidate, propofol, and isoflurane** may offer protection against focal ischemia and incomplete global ischemia by producing complete electrical silence of the brain and eliminated the metabolic cost of electrical activity; unfortunately, they have no effect on basal energy requirements.
   B. **Hypothermia**
      1. $CMRO_2$ is decreased by 7% for every 1 degree Celsius reduction.
      2. Hypothermia decreases both basal and electrical metabolic requirements throughout the brain; metabolic requirement continue to decrease even after complete electrical silence; most effective method for protecting the brain during focal and global ischemia.
   C. **Calcium channel blockers** (nimodipine and nicardipine) may be beneficial in reducing neurologic injury following hemorrhagic and ischemic strokes.
   D. **Maintenance of optimal cerebral perfusion pressure** is critical (maintaining normal arterial blood pressure, intracranial pressure, oxygen carrying capacity, arterial oxygen tension and hematocrit maintained 30-34%). Hyperglycemia aggravates neurologic injuries and should be avoided.

5. **Pharmacology in neurosurgical patients**
   A. **Inhalational anesthetics**
      1. Volatile agents administered during normocapnia in concentrations higher then 0.6 MAC produce cerebral vasodilation, decreased cerebral vascular resistance, and resulting dose-dependent increases in CBF despite concomitant decreases in $CMRO_2$.
      2. Enflurane increases CSF formation and retard absorption. Halothane impedes CSF absorption but only minimally retards formation.
      3.

   B. Most intravenous agents cause coupled reduction in CBF and $CMRO_2$ in a dose-dependent manner. Ketamine is the only intravenous anesthetic that dilates the cerebral vasculature and increases CBF.

C. All muscle relaxants, except succinylcholine, have no direct effect on CBF and $CMRO_2$. Succinylcholine causes a transient increase in CBF and $CMRO_2$.
D. Opioids in the absence of hypoventilation decrease CBF and possible ICP.

Anesthesia for Craniotomy

1. **Preoperative preparation**
   A. Evidence of increased ICP should be sought (nausea, vomiting, hypertension, bradycardia, personality change, altered LOC, altered patterns of breathing, papilledema, seizures.
   B. Physical examination should include a neurologic assessment documenting mental status and any existing sensory or motor deficits.
   C. CT and MRI scans should be reviewed for evidence of brain edema, a midline shift greater than 0.5 cm, and ventricular size.
2. **Premedication**
   A. Premedication is best avoided when increased ICP is suspected.
   B. Corticosteroids and anticonvulsant therapy should be continued up until the time of surgery.
3. **Monitoring**
   A. In addition to standard ASA monitors, direct intraarterial pressure monitoring and bladder catheterization are indicated for most patients undergoing craniotomy.
   B. Central venous catheter is useful for guiding fluid management, possible treatment of venous air embolism, and to give vasopressors.
4. **Induction and maintenance of anesthesia**
   A. Induction must be accomplished without increasing ICP or compromising CBF. Hypertension, hypotension, hypoxia, hypercarbia, and coughing should be avoided.
   B. Thiopental, propofol, etomidate may be used for IV induction and are unlikely to adversely increase ICP.
   C. Nondepolarizing agents are the muscle relaxants of choice. The hemodynamic response to laryngoscopy can be blunted by pretreatment with lidocaine, labetalol, opioids, and/or esmolol.
   D. Anesthesia is usually maintained with a combination of a opioid, volatile agent, and muscle relaxant. Anesthetic requirements are decreased after craniotomy and dural opening, since the brain parenchyma is devoid of sensation.
5. **Emergence** should occur slow and controlled. Straining, coughing and hypertension should be avoided.

Neurotrauma

1. **Head trauma**
   A. **Glasgow Coma Scale (GCS)** correlates with the severity of injury and outcome. Total score possible = 3-15.
      1. **Best motor response:** 6-obeys commands; 5-localizes pain; 4-withdrawals; 3-flexion: decorticate rigidity; 2-extension: decerebrate rigidity; 1-no motor response.
      2. **Best verbal response:** 5-oriented, conversant; 4-disoriented, conversant; 3-inappropriate words; 2-incomprehensible sounds; 1-no verbalization/response.
      3. **Eye opening:** 4-spontaneous; 3-to verbal stimulation; 2-to pain; 1-no response.
   B. **Cushing triad:** hypertension, bradycardia, respiratory disturbances (late and unreliable sign that usually just proceeds brain herniation).
   C. **Preoperative**
      1. All patients are regarded as having a full stomach and treated as such.
      2. Hypotension in the setting of head trauma is nearly always related to other associated injuries. Correction of hypotension and control of any bleeding take precedence over radiographic studies and definitive neurological treatment because systolic arterial blood pressures of less than 80 mmHg correlate with a poor outcome.

3. Dysrhythmias and electrocardiographic abnormalities in the T wave, U wave, ST segment, and QT interval are common following head injuries but are not necessarily associated with cardiac injury.

D. **Intraoperative**

1. Management is similar to other mass lesions with elevated ICP
2. CPP should be maintained between 70 and 110 mmHg.
3. Dextrose containing solutions may exacerbate ischemic brain damage and should be avoided in the absence of documented hypoglycemia.

2. **Spinal cord injury**

A. Lesions involving phrenic nerve (C3-C5) usually result in apnea requiring intubation and mechanical ventilatory support. Lesions below C5-C6 may cause up to 70% reduction in vital capacity and FEV_1 with impaired ventilation and oxygenation. Lesions involving T1-T4 (cardiac accelerator nerves) may lead to bradycardia, bradydysrhythmias, atrioventricular block and cardiac arrest. T7 or higher is the critical level for significant alveolar ventilation impairment.

B. Spinal shock is seen in high spinal cord injuries lasting from a few hours to several weeks; characterized by loss of sympathetic tone in capacitance and resistance vessels below the level of the lesion; flaccid paralysis; total absence of visceral and somatic sensation below level of injury; paralytic ileus; loss of spinal cord reflexes below level of injury.

C. Autonomic hyperreflexia is associated with lesions above T5, not a problem during acute management (appears following resolution of spinal shock and return of spinal cord reflexes).

D. Methylprednisolone: 30 mg/kg IV loading dose, followed by 5.4 mg/kg/hr for 23 hours may improve the functional recovery if treatment is begun within 8 hrs following injury.

E. Succinylcholine: safe for use during the first 24-48 hours.

Trauma Anesthesia

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Initial Survey and Resuscitation

1. **Five rules of trauma**
   A. The stomach is always full.
   B. The cervical spine is always unstable.
   C. Altered mental status is caused by head injury.
   D. Partial airway obstruction may progress rapidly to complete airway obstruction.
   E. The patient is always hypovolemic.
2. **Airway and breathing**
   A. All patients should have initial stabilization of the cervical spine before any airway manipulation. Assume a cervical spine injury in any patient with multi-system trauma, especially with an altered LOC or a blunt injury above the clavicle. Maintain the cervical spine in a neutral position with inline stabilization when establishing an airway.
   B. The airway should be assessed for patency. All secretions, blood, vomitus, and foreign bodies should be removed. Measures to establish a patent airway should protect the cervical spine. The chin lift or jaw thrust maneuvers are recommended to achieve this task.
   C. All patients should receive supplemental oxygen (face mask, bag-valve mask, endotracheal tube).
   D. Patients who arrive intubated, should have placement confirmed (ie, bilateral breath sounds with good chest rise, direct laryngoscopy, or capnography).
3. **Circulation**
   A. Hypotension following injury must be considered to be hypovolemic in origin until proven otherwise. Volume resuscitation begins immediately with the establishment of intravenous access.
   B. A minimum of two large-caliber intravenous catheters should be established. Blood and fluid warmers should be used.

Trauma Intubations and Anesthetic Management

1. **Indications for airway intervention**
   A. **Airway obstruction.**
      1. Hypoxia and hypercarbia (shock or cardiac arrest).
      2. Controlled hyperventilation (obvious intracranial injury or GCS of < 9).
      3. Protection against pulmonary aspiration (drug overdose).
      4. Airway injury (inhalation injuries).
      5. Sedation for diagnostic procedures (patients who are intoxicated or suffering from possible head injury that are unable to lie still for necessary diagnostic studies).
      6. Prophylactic intubation (patients with impending respiratory failure).
      7. Airway or midface injuries (possible airway compromise)
      8. Large flail segment.
      9.

   B. **Preparation**
      1. All multiple trauma patients should be assumed to have a cervical spine injury and a full stomach. Portable cervical spine x-rays will miss 5% to 15% of injuries. Complete evaluation of the cervical spine may require a CT scan or multiple radiographs and clinical exam. Cervical spine injury is unlikely in alert patients without neck pain or tenderness.

2. Patients who arrive ventilated with an esophageal obturator airway (EOA) should have a more definitive airway placed before the EOA is removed. After the trachea has been intubated, the stomach should be suctioned prior to the removal of the EOA.
3. In alert patients with potential spinal cord injuries, document any movement of extremities before and after intubation.
4. The airway should be examined to detect potentially difficult intubation.
5. Airway equipment (laryngoscope, endotracheal tubes, suction) should be set-up prior to the patients arrival.

C. **Endotracheal trauma intubation**

1. **Preoxygenation**
   A. All patients should be preoxygenated to minimize hypoxia.
   B. Administration of 100% oxygen to an individual with normal spontaneous ventilation for 3 minutes or 4-6 vital capacity breaths will generally result in 95-98% nitrogen washout.
2. **Orotracheal intubation**, facilitated by the use of muscle relaxants and general anesthesia, is the technique of choice for intubating the trachea of trauma patients.
3. **Nasotracheal intubation**
   A. Contraindications to nasotracheal intubation include: apnea; upper airway foreign body, abscess, or tumor; nasal obstruction; central facial fractures; acute epiglottitis (blind technique); basal skull fractures; coagulopathy; and cardiac or other prosthesis.
   B. Not commonly used in trauma patients.
4. **Cricothyroidotomy:** the need for cricothyroidotomy due to severe maxillofacial trauma or an inability to perform oral-tracheal intubation occurs in less than 1% of all trauma patients requiring intubation on admission. It may be used as a primary airway, with injuries to the pharynx for example, or after failure of orotracheal intubation. It may be a full surgical approach or via a percutaneous needle cricothyroidotomy with high flow oxygen.
5. If there is difficulty or delay in intubating the trachea in any trauma patient with respiratory comprise, a tracheotomy or cricothyroidotomy should be performed immediately.

2. **Intraoperative management**

A. Two functioning large bore IVs should be placed before induction. Blood should be available before incision is made, if possible.

B. **Induction**

1. All trauma patients should be assumed to have full stomachs.
2. When general anesthesia is planned, rapid sequence induction with cricoid pressure is the method of choice.
3. Reduced doses of induction agent or no induction agent may be appropriate in severely injured, obtunded patients. Ketamine 0.25-0.5 mg/kg IV is the induction agent of choice in hypovolemia.

C. **Maintenance**

1. Narcotic based anesthetic is recommended for stable patients. For unstable patients, scopolamine/oxygen/pancuronium can be used until hemodynamically stable then small incremental doses. Prophylactic use of scopolamine (0.1-0.2 mg IVP) or midazolam (1-3 mg IVP) may be considered.
2. Avoid using nitrous oxide.
3. The patient should be kept warm (blanket warmer, fluid warmer, and a bear hugger on the upper body or lower body). Hypothermia worsens acid-base disorders, coagulopathies, and myocardial function.

Burns

1. **Preoperative evaluation**

A. First-degree burns are limited to the epithelium, second-degree burns extend into the dermis, and third-degree burns are full thickness.

B. The size of the burn should be estimated as a percentage of the total body surface area (%TBSA).

C. Indications for early intubation include hypoxemia not correctable with oxygen, upper airway edema, or the presence of copious secretions.

2. **Perioperative management**

A. **Cardiovascular system**

1. Burn patients require aggressive fluid resuscitation during the first 24-48 hours.
2. Fluid replacement protocols
   A. Parkland formula: 4.0 cc of Ringer's lactate/kg /%TBSA/24 hours.
   B. Half the calculated fluid deficit is administered during the first 8 hours after the burn injury, and the remainder is given over the next 16 hours. Daily maintenance fluid requirements should be given concurrently.
   C. Early cardiovascular effects include decreased cardiac output, decreased arterial blood pressure, and increased capillary permeability.

B. **Respiratory system**

1. Thermal injury of the face and upper airway are common. Inhalational injury should be suspected in the presence of facial or intraoral burns, singed nasal hairs, a brassy cough, carbonaceous sputum, and wheezing. Before airway edema occurs, endotracheal intubation should be performed.
2. Carbon monoxide poisoning is defined as greater than 20% carboxyhemoglobin in the blood. Tissue hypoxia ensues.
3. Manifestations of carbon monoxide poisoning include irritability, headache, nausea/vomiting, visual disturbances, seizures, coma, or death.
4. Pulse oximetry overestimates the oxyhemoglobin saturation in the presence of carboxyhemoglobin because the absorption spectrum is similar. The classic cherry red color of the skin is a sign of high concentrations of carbon monoxide.

C. **Anesthetic considerations**

1. Succinylcholine is contraindicated 24 hours to 2 years after major burns because it can produce profound hyperkalemia and cardiac arrest.
2. Nondepolarizing muscle relaxants are used when muscle relaxation is required. Burn patients require higher than normal doses of nondepolarizing muscle relaxants.
3. Burn patients may have increased narcotic requirements because of tolerance and increases in the apparent volume of distribution.

Cardiac Tamponade

1. **Manifestations**

A. **Dyspnea, orthopnea, tachycardia:** Beck's triad consists of hypotension, distant heart sounds, distention of jugular veins.

B. **Paradoxical pulse** (>10 mmHg decline in BP during inspiration).

C. The principle hemodynamic feature is a decrease in cardiac output from a reduced stroke volume with an increase in central venous pressure. Equalization of diastolic pressures occur throughout the heart. Impairment of both diastolic filling and atrial emptying abolishes the 'y' descent; the 'x' descent is normal.

D. **EKG:** ST segment changes, electrical alternans.

E. **CXR:** silhouette normal or slightly enlarged.

F. **Transesophageal echo** is the best diagnostic tool.

2. **Anesthetic considerations**

A. **Maintain filling pressures** (to maximize stroke volume): support myocardial contractility with inotropic support if necessary. Avoid bradycardia.

B. Avoid positive pressure ventilation because increased intrathoracic pressure will impede venous return and exacerbate underfilling of the cardiac chambers.

C. **Pre-induction monitors:** standard monitors plus arterial line and central venous line (and pulmonary artery catheter if needed).

D. Hemodynamically unstable patients should be managed with pericardiocentesis (under local anesthesia) prior to induction (the removal of even a small amount of fluid can improve cardiac performance).

E. **Induction:** ketamine is the drug of choice, however, ketamine depresses myocardial contractility and may precipitate hemodynamic deterioration when used in the presence of hypovolemia and maximal sympathetic outflow.

Ophthalmologic Anesthesia

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1. **Physiology of intraocular pressure**
   A. Normal intraocular pressure is maintained between 10 and 22 mmHg.
   B. Intraocular pressure is controlled primarily by regulation of the outflow resistance at the trabecular meshwork. Acute changes in choroidal blood volume can produce rapid increases in intraocular pressure. Hypercapnia can lead to choroidal congestion and increased intraocular pressure. The increases in venous pressure associated with coughing, straining, or vomiting can raise IOP to 30 to 50 mmHg. Similar increases can be seen at intubation. Intraocular pressure can also be increased by extrinsic compression of the globe. The force of the eyelid in a normal blink may cause an increase of 10 mmHg; a forceful lid squeeze can increase IOP to over 50 mmHg A poorly placed anesthesia mask could increase IOP to the point of zero blood flow.
2. **Oculocardiac reflex**
   A. External pressure on the globe or surgical traction (stretch) of extraocular muscles (particularly the medial rectus muscle) can elicit the reflex producing cardiac dysrhythmias ranging from bradycardia and ventricular ectopy to sinus arrest or ventricular fibrillation. Hypercarbia or hypoxemia may increase the incidence and severity of this reflex.
   B. The reflex is trigeminovagal reflex arc. The afferent limb is from orbital contents to the ciliary ganglion to the ophthalmic division of the trigeminal nerve to the sensory nucleus of the trigeminal nerve near the fourth ventricle. The efferent limb is via the vagus nerve. The reflex fatigues with repeated traction on the extraocular muscles.
   C. **Prevention**
      1. Retrobulbar block is not uniformly effective in preventing the reflex (retrobulbar block may elicit the oculocardiac reflex).
      2. Anticholinergic medication can be effective, however caution must be used in the elderly.
      3. Deepen anesthesia.
      4. Factors associated with increased susceptibility to the development of ocularcardiac reflex are anxiety, hypoxia, hypercarbia, and light anesthesia.
   D. **Treatment**
      1. Request the surgeon to stop manipulation.
      2. Assess adequate ventilation, oxygenation, and depth of anesthesia.
      3. If severe or persistent bradycardia, give atropine (7-10 mcg/kg).
      4. In recurrent episodes, infiltration of the rectus muscles with local anesthetics.
3. **Intraocular gas expansion**
   A. A gas bubble may be injected into the posterior chamber during vitreous surgery to flatten a detached retina.
   B. The air bubble is absorbed within 5 days by gradual diffusion.
   C. Sulfur hexafluoride, an inert gas that is less soluble in blood than nitrogen, provides a longer duration (up to 10 days) in comparison with an air bubble.
   D. Nitrous oxide should be discontinued at least 15 minutes prior to the injection of air or sulfur hexafluoride. Nitrous oxide should be avoided until the bubble is absorbed (5 days for air and 10 days for sulfur hexafluoride injection).
4. **Open eye injury**
   A. Considerations include the possibility of recent food ingestion and the need to avoid even small increases in IOP if the injured eye is considered salvageable.
   B. Rapid tracheal intubation facilitated by succinylcholine must be balanced against possible increases in IOP.
5. **Anesthetic drugs**
   A. Most anesthetic drugs either lower or have no effect on intraocular pressure. An exception is ketamine, and possibly etomidate.

B. Ketamine effects are controversial, but is generally felt to moderately increase intraocular pressure. Ketamine increases choroidal blood flow, increases nystagmus, and increases extraocular muscle tone via blepharospasm.

C. Etomidate, which is associated with a high incidence of myoclonus (10-60%), may increase intraocular pressure.

D. Succinylcholine can cause a 5-10 mmHg increase in intraocular pressure for 5-10 minutes. Succinylcholine can potentially increase intraocular pressure by dilating choroidal blood vessels and increases in extraocular muscle tone. Pretreatment with a defasiculating dose of a nondepolarizing muscle relaxant does not reliably eliminate the effect of succinylcholine on intraocular pressure. Nondepolarizing muscle relaxants do not increase intraocular pressure.

6. **Systemic effects of ophthalmic drugs**

A. **Anticholinesterases** (echothiophate, phospholine iodide): systemic absorption leads to inhibition of plasma cholinesterase which may lead to prolongation of the duration of action of succinylcholine. Takes 3 weeks for pseudocholinesterase levels to return to 50% of normal. The metabolism of mivacurium and ester-type local anesthetics may also be affected.

B. **Cholinergics** (pilocarpine, acetylcholine): used to induce miosis; toxicity may manifest in bradycardia or acute bronchospasm.

C. **Anticholinergics** (atropine, scopolamine): used to cause mydriasis; systemic absorption may lead to tachycardia, dry skin, fever, and agitation.

D. **Beta-blockers** (timolol maleate): systemic absorption may cause beta-blockade (bradycardia, bronchospasm, or exacerbation of congestive heart failure). Betaxolol seems to be oculo-specific with minimal side effects.

E. **Carbonic anhydrase inhibitors** (acetazolamide, Diamox): used to decrease aqueous production; induces an alkaline diuresis. Side effects include diuresis and hypokalemic metabolic acidosis.

7. **Retrobulbar blockade**

A. **Technique:** local anesthetic is injected behind the eye into the cone formed by the extraocular muscles. Lidocaine and bupivacaine are the most commonly used local anesthetics. Hyaluronidase, a hydrolyzer of connective tissue polysaccharides, is commonly added to enhance the spread of local anesthetic.

B. **Complications:** retrobulbar hemorrhage, globe perforation, optic nerve atrophy, convulsions, oculocardiac reflex, loss of consciousness, and respiratory arrest.

C. **Post-retrobulbar apnea syndrome:** due to injection of local anesthetic into the optic nerve sheath with spread into the cerebrospinal fluid. Apnea typically occurs within 20 minutes and may last 15-60 minutes. Ventilation must be constantly monitored in patients with retrobulbar blocks.

D. Facial nerve block prevents squinting of the eyelid. Major complications include subcutaneous hemorrhage.

Anesthesia for Select Cases

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Transurethral Resection of the Prostate

1. **Complications**
   A. Intravascular absorption of irrigating fluid: the amount of solution absorbed depends on the hydrostatic pressure of the irrigating fluid, the duration of time sinuses are exposed to irrigating fluid (10 to 30 mL of irrigating fluid is absorbed per minute), and the number and sizes of the venous sinuses opened during resection. Absorption of the irrigating fluid can result in fluid overload, serum hypoosmolality, hyponatremia, hyperglycemia, hyperammonemia, hemolysis.
   B. Autotransfusion secondary to lithotomy position: hypothermia may occur. Bacteremia has an incidence of 10% in patients with sterile urine and an incidence of 50% in patients with infected urine.
   C. Blood loss: related to vascularity of the prostate gland, technique, weight of the prostate resected, length of the operation; blood loss ranges from 2-4 mL/min during resection.
   D. Perforation of bladder or urethra.
   E. Transient blindness: attributed to absorption of glycine and its metabolic byproduct, ammonia, acting as an inhibitory neurotransmitter in the retina.
   F. CNS toxicity: result of oxidative biotransformation of glycine to ammonia.
   G. CNS symptoms: apprehension, irritability, confusion, headache, seizures, transient blindness, and coma, have all been attributed to hyponatremia and hyposmolarity.
2. **Intraoperative management:** regional or general anesthesia can be used; it is important to monitor these patients carefully for signs and symptoms of excessive intravascular absorption of irrigating solution.
3. **Management of TURP (water-intoxication) syndrome**
   A. Obtain serum sodium and arterial blood gas, provide supplemental oxygen, support blood pressure, terminate procedure as soon as possible, consider invasive monitors.
   B. Serum sodium >120 mEq/L: fluid restriction; brisk diuresis with loop diuretics.
   C. Serum sodium <120 mEq/L: loop diuretics; consider hypertonic saline (eg, 3% or 5% saline) infused at a rate which does not exceed 100 mL/hr. Allow sodium to rise by 0.5-2.0 mEq/L/hr; stop hypertonic saline and loop diuretics once sodium is 120-130 mEq/l.

Extracorporeal Shock Wave Lithotripsy

1. **Side effects**
   A. Immersion into the water bath causes peripheral venous compression, resulting in an increase in central blood volume and central venous pressure (about 8-11 mmHg). Some experience hypotension owing to vasodilation from the warm water. In patients with cardiac disease, immersion should be achieved slowly.
   B. During immersion or emersion, cardiac dysrhythmias may occur reflecting changes in right atrial pressure. Shock waves are triggered from the EKG to occur 20 msec after the R wave to minimize the risk of dysrhythmias.
   C. Immersion lithotripsy increases the work of breathing.
2. **Anesthetic Management**
   A. Regional or general anesthesia can be used. Regional anesthesia has the advantage that the patient is awake and cooperative. Regional anesthetic requires a T6 sensory level.
   B. Monitors, epidural catheter insertion site, vascular access sites should be protected with water impermeable dressings.
   C. Maintenance of adequate urine output with IV fluids to help facilitate passage of disintegrated stones. Monitoring of body temperate is useful to detect changes owing to water immersion.

Electroconvulsive Therapy

1. **Side effects**
   A. Increased cerebral blood flow, increased intragastric pressure, apnea.
   B. Cardiovascular response: initial parasympathetic outflow may result in bradycardia, followed by a sympathetic outflow, which produces hypertension, tachycardia and cardiac dysrhythmias, lasting 5-10 minutes.
2. **Anesthetic Management**
   A. Methohexital 0.5-1.0 mg/kg (or thiopental, propofol or etomidate) and succinylcholine 0.25-0.5 mg/kg (or mivacurium or rapacuronium).
   B. Place blood pressure cuff on the opposite arm of the IV and inflate prior to giving of succinylcholine to allow for motor expression of the seizure.
   C. Induced seizures should last longer than 25 seconds and should be terminated if they last longer than 3 minutes.
   D. Administration of an anticholinergic before induction may prevent initial bradycardia; hypertension and tachycardia can be treated with labetalol.

Laparoscopic Surgery

1. **Contraindications** (relative and absolute): increased intracranial pressure, patients with ventriculoperitoneal or peritoneojugular shunts, hypovolemia, CHF, previous abdominal surgery with significant adhesions, morbid obesity, pregnancy, and coagulopathy.
2. **Pulmonary effects:** laparoscopy creates a pneumoperitoneum with pressurized CO_2 (pressures up to 30 cm H_2O). The resulting increase in intra-abdominal pressure displaces the diaphragm cephalad, causing a decrease in lung compliance and an increase in peak inspiratory pressure. Atelectasis, diminished functional residual capacity, ventilation/perfusion mismatch, and pulmonary shunting contribute to a decrease in arterial oxygenation. The high solubility of CO_2 increases systemic absorption which can lead to increased arterial CO_2 levels.
3. **Cardiac effects:** moderate insufflation can increase effective cardiac filling because blood tends to be forced out of the abdomen and into the chest. Higher insufflation pressures (greater than 25 cm H_2O), however, tends to collapse the major abdominal veins which compromises venous return and leads to a drop in preload and cardiac output in some patients. Hypercarbia may stimulate the sympathetic nervous

system and thus increase blood pressure, heart rate, and risk of dysrhythmias.

4. **Management of anesthesia**
   A. **Patient position:** Trendelenburg is often associated with a decrease in FRC, VC, TLV, and pulmonary compliance.
   B. **Anesthetic technique:** general anesthesia with endotracheal intubation.

5. **Complications:** hemorrhage, peritonitis, subcutaneous emphysemas pneumomediastinum, pneumothorax, and venous air embolism. Vagal stimulation during trocar insertion, peritoneal insufflation, or manipulation of viscera can result in bradycardia and sinus arrest.

Liposuction

1. **Potential liposuction perioperative complications**
   A. Pulmonary embolism (thrombus or fat).
   B. Massive fluid dislocation (internal burn).
   C. 60-70% wetting solution absorption.
   D. Organ or vessel perforation with wand.
   E. Lidocaine toxicity.
   F. Hypothermia from several liters of infiltrate.
2. **Anesthetic considerations**
   A. **Fluid balance:** up to 70% of the fluid remains trapped subdermally and is absorbed gradually. Suction extraction of subcutaneous tissue causes a burn-like trauma.
   B. Temperature control.
   C. Lidocaine megadosing: for tumescent infiltration with highly diluted lidocaine and epinephrine doses up to 35 mg/kg are considered safe. Deaths attributed to lidocaine toxicity appear to be caused by terminal asystole subsequent to progressive local anesthetic depression of intra-cardiac conduction and ventricular contractility.

Myasthenia Gravis and Myasthenic Syndrome

1. Myasthenia gravis is characterized by weakness and easy fatigability of skeletal muscle. The weakness is thought to be due to autoimmune destruction or inactivation of postsynaptic acetylcholine receptors at the neuromuscular junction. Muscle strength characteristically improves with rest but deteriorates rapidly with repeated effort.
2. **Osserman classification**
   A. Type I: Involvement of extraocular muscles only.
   B. Type IIa: Mild skeletal muscle weakness, spares muscles of respiration.
   C. Type IIb: More severe skeletal muscle weakness with bulbar involvement.
   D. Type III: Acute onset, rapid deterioration, severe bulbar and skeletal muscle involvement.
   E. Type IV: Late, severe involvement of bulbar and skeletal muscle.
3. **Treatment of myasthenia gravis**
   A. Treatment consists of anticholinesterase drugs, immunosuppressants, glucocorticoids, plasmapheresis, and thymectomy.
   B. Anticholinesterase drugs (usually pyridostigmine) inhibit the breakdown of acetylcholine by tissue cholinesterase, increasing the amount of acetylcholine at the neuromuscular junction.
   C. Cholinergic crisis is characterized by increased weakness and excessive muscarinic effect, including salivation, diarrhea, miosis, and bradycardia.
   D. Edrophonium test: used to differentiate a cholinergic crisis form a myasthenic crisis. Increased weakness after up to 10 mg of intravenous edrophonium is indicative of cholinergic crisis, whereas increasing strength implies myasthenic crisis.
4. **Pre-op predictors for post-op ventilation** (after transsternal thymectomy).
   A. Duration of disease greater than 6 years.
   B. Presence of COPD or other lung disease unrelated to myasthenia.

C. Pyridostigmine dose greater than 750 mg/day.
D. Preoperative FVC less than 2.9 liters.

5. **Anesthetic concerns** muscle relaxants should be avoided. The response to succinylcholine is unpredictable. Patients may manifest a relative resistance, a prolonged effect, or an unusual response (phase II block).
6. **Myasthenic syndrome**, also called **Eaton-Lambert syndrome**, is a paraneoplastic syndrome characterized by proximal muscle weakness, which typically affects the lower extremities. Myasthenic syndrome is usually associated with small-cell carcinoma of the lung. In contrast to myasthenia gravis, the muscle weakness improves with repeated effort and is unaffected by anticholinesterase drugs.
7. Patients with the myasthenic syndrome are very sensitive to both depolarizing and nondepolarizing muscle relaxants.

Anesthesia for Organ Harvest

1. **The donor**
   A. Brain death should be pronounced prior to going to the OR.
   B. **Clinical criteria for brain death**
      1. Cerebral unresponsiveness, irreversible coma.
      2. Brain stem unresponsiveness.
      3. Fixed and dilated pupils, doll's eyes, negative caloric test, absent corneal reflex.
      4. Absent gag and cough reflex, apnea (no respiratory efforts with $PaCO_2$ greater than 60 mmHg).
      5. No posturing (spinal reflexes may be present).
   C. Ancillary tests
      1. Isoelectric electroencephalogram.
      2. Absent CBF by intracranial angiography or nuclear brain scan.
      3. Body temperature less than 95 degrees F.
      4. Absence of drug intoxication or neuromuscular blocking agents.
      5. Corrected metabolic abnormalities.
2. **Donor management**
   A. **Overall goals** are restoration and maintenance of hemodynamic and vascular stability. Hemodynamics should be maintained as follows:
      1. Systolic blood pressure greater than 100 mmHg.
      2. Central venous pressure 10-12 mmHg.
      3. Urine output greater than 100 cc/hour.
      4. P_aO_2 greater than 100 mmHg.
   B. **Physiologic changes associated with brain death**
      1. **Cardiovascular instability** is a common feature, secondary to loss of neurologic control of the myocardium and vascular tree. Fluid resuscitation should be used to keep systolic blood pressure greater than 100 mmHg and mean arterial pressure greater than 70 mmHg.
      2. **Central diabetes insipidus** may occur from hypothalamic failure resulting in extreme salt and water wasting from the kidneys. Massive loss of fluid and electrolytes that may occur. Aqueous Pitressin should be administered in doses of 10 units intravenously every 4 hours to bring urine output down to 150-200 cc per hour.
      3. **Loss of thermoregulatory control:** after brain death, body tempera?ture drifts downward to core temperature.
      4.
      5. **Neurogenic pulmonary edema** may be present.
      6. **Coagulopathy:** the release of tissue fibrinolytic agent from a necrotic brain may initiates coagulopathy.
      7. **Hypoxia:** pulmonary insufficiency secondary to trauma and/or shock should be treated with mechanical ventilation, positive end-expiratory pressure (PEEP), and inspired oxygen fraction sufficient to maintain adequate peripheral oxygen delivery.

8. **Overall hypovolemia** is the most important variable affecting donor organ perfusion. Ringer's lactate should be infused to establish a central venous pressure of 10-12 mmHg. Hematocrit should be maintained about 30%.
9. **Anesthesia** is not needed in the brain dead patient. However, significant hemodynamic responses to surgical stimuli commonly occur in the brain-dead donor during organ harvesting. These responses may reflect some residual lower medullary function (visceral and somatic reflexes). Movement secondary to spinal reflex action should be controlled. Patients are routinely declared dead prior to going to the operating room.

Induced Hypotension

1. **Indications:** to reduce intraoperative blood loss and to produce a relatively bloodless surgical site, to help manage patients who refuse blood transfusions, when reduction in MAP decreases the risk of vessel rupture.
2. **Contraindications:** vascular insufficiency (to brain, heart, or kidney), cardiac instability, uncontrolled hypertension, hypovolemia, polycythemia, allergy to hypotensive agents, increased ICP (controversial), lack of experience or understanding of technique.
3. **Anesthetic considerations**
   A. MAP of 50-60 mmHg in young, healthy patients and MAP of 60-70 mmHg in suitable older patients.
   B. Ventilation should be controlled and aimed at maintaining normocarbia.
   C. Maintain normovolemia, careful volume replacement is essential.
   D. Continuous invasive arterial pressure monitoring is indicated. Consider CVP and PAP.
4. **Anesthetic technique**
   A. Inhalational: has been used as a sole agent; however, not recommended because of the inability to quickly reverse. Commonly used in conjunction with direct vasodilator.
   B. Vasodilator agents: continuous infusions allow easy titration and control of BP, commonly used agents include sodium nitroprusside, nitroglycerin, and trimethaphan.
   C. Beta-adrenergic blockers: decrease MAP by their negative inotropic properties. Labetalol and esmolol are most commonly used. Because of their low hypotensive potency there are commonly used in conjunction with other agents.
5. **Complications:** cerebral ischemia, thrombosis, or edema; acute renal failure; myocardial infarction, congestive heart failure, or arrest; reactive hemorrhage with hematoma formation.

Acute Pain Management

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Acute Postoperative Pain Management

1. **Benefits of epidural analgesia:** superior pain relief, decreased incidence of pulmonary complications, decreased incidence of cardiovascular complications, earlier return of bowel function.
2. **Analgesic delivery systems:** oral (not good for mod/severe pain), IM, IV, PCA, intrathecal (spinal), epidural.
3. **Common postoperative PCA orders:** morphine (30 mg/30 mL prefilled), loading dose 2 mg IV (range 1-4 mg), maintenance dose 1 mg IV, lockout interval 8 minutes (range 6-10 minutes), limit dose to 20 mg over 4 hours, monitor vital signs, assess pain level periodically, record drug administered every 8 hours.
4. **Common postoperative epidural analgesia infusion orders:** select infusion contents, supplemental oxygen, monitor SaO2, observe level of consciousness, record pain score at periodic intervals, naloxone at bedside, morphine for breakthrough pain.
5. **Optimal epidural placement for postop local anesthetic administration**
   A. Thoracotomy: T4-T6.
   B. Upper abdominal/flank: T8.
   C. Lower abdominal: T10-T12.
   D. Lower extremity/pelvic: L2-L4.
6. **Side effects of peridural administered opioids**
   A. **Nausea/vomiting:** opioids in the vomiting center and the chemoreceptor trigger zone in the medulla can cause nausea or vomiting.
   B. **Pruritus:** histamine release may play a small role.
   C. **Respiratory depression**
      1. Patients at risk are the elderly; patients who receive concomitant systemic opiates or sedatives; and patients who have received large doses of spinal opiates.
      2. Early respiratory depression can occur within two hours of spinal opioid administration and is similar to that observed with parenteral administration of an opioid. With hydrophilic agents (ie, morphine), late respiratory depression commonly peaks at 12 or 13 hours after the initial dose but can occur as late as 24 hours.
   D. **Urinary retention.**
   E. **Delayed gastric emptying.**
7. **Management of opioid related side effects**
   A. **Nausea/vomiting**
   B.
      1. Metoclopramide (Reglan): 10-20 mg IV q4 hrs.
      2. Droperidol (Inapsine): 0.625 mg IV q4 hrs; can cause dysphoria, hypotension.
      3.

   C. **Pruritus**
      1. Naloxone (Narcan): 10-40 mcg/hr IV continuous infusion; will not significantly reverse analgesia at recommended doses.
      2. Diphenhydramine (Benadryl): 25-50 mg IV q4 hrs; sedative effect.
   D. **Respiratory depression**
      1. Naloxone (Narcan): 40-100 mcg/bolus titrated q2-3 minutes; larger than necessary dosage may result in significant reversal of analgesia, nausea, vomiting, sweating, and/or circulatory stress.
   E. **Urinary retention**: Foley as needed.

8. **Management fo inadequate analgesia provided by epidural infusion**

   A. **Evaluate proper placement of catheter**

1. Give 5-7 mL of the opioid and local anesthetic solution, if analgesia remains inadequate after 15-30 minutes, give a test dose of local anesthetic (2% lidocaine with epi).
2. If test dose produces a bilateral sensory block catheter location is confirmed and infusion rate was probably insufficient (increase rate).
3. If test dose produces a unilateral block it is likely the catheter is placed laterally, withdrawal catheter 1-2 cm.
4. If test dose produces no response catheter is not in the epidural space. The catheter should be removed and switched to PCA.

Drug	**Bolus Dose (mg)**	**Lockout Interval (min)**	**Continuous Infusion (mg/hr)**
Fentanyl	0.015-0.05	3-10	0.02-0.1
Hydromorphone	0.1-0.5	5-15	0.2-0.5
Meperidine	35,929	5-15	5-40
Methadone	0.5-3.0	10-20	
Morphine	0.5-3.0	5-20	0.5-10
Oxymorphone	0.2-0.8	5-15	0.1-1.0
Sufentanil	0.003-0.015	3-10	0.004-0.03
Pentazocine	5-30	5-15	6-40

Drug	**Bolus Dose**	**Onset (min)**	**Peak (min)**	**Duration (hr)**	**Concen?tration**	**Rate (mL/hr)**
Meperidine	30-100 mg	5-10	12-30	4-6	1 mg/cc	10-20
Morphine	5 mg	20	30-60	12-24	0.1 mg/cc	1-6
Methadone	5 mg	12.5	17	7.2		
Hydromorphone	1 mg	13	23	11.4	0.05 mg/cc	6-8

Fentanyl	100 mcg	4-10	20	2-4	4 mcg/cc	4-12
Diamorphine	5 mg	5	9-15	12.4		
Sufentanil	30-50 mcg	7	25	3-4	5 mcg/cc	10
Alfentanil	15 mcg/kg	15		1-2		

Bupivacaine (%)	**Opioid (concentration)**	**Infusion Rate (mL/hr)**
0.125	Fentanyl 2 mcg/cc	4-8
0.125	Sufentanil 1 mcg/cc	4-8
0.125	Morphine 0.05 mg/cc	4-8
0.0625	Fentanyl 5 mcg/cc	4-10
0.0625	Sufentanil 2 mcg/cc	4-8
0.0625	Morphine 0.1 mg/cc	2-8
	Morphine 0.1 mg/cc	2-8
	Hydromorphone 0.02 mg/cc	1-6

Opioid	Dose	Onset (min)	Duration (hr)
Morphine	0.15-0.6 mg	15-45	8-24
Fentanyl	10-25 mcg	2-5	1-3
Sufentanil	5-15 mcg	2-5	2-4

Drug	**Dose (Adult)**

Butorphanol Tartrate (Stadol)	1-2 mg IV q3-4 hr or 2 mg IM q3-4 hr
Codeine	15-60 mg po q4-6 hr
Hydromorphone HCL (Dilaudid)	2 mg q4-6 hr po or 1-2 mg q4-6 hr SC/IM
Morphine (MS Contin)	30-60 mg q12 hr po
Nalbuphine HCL (Nubain)	10 mg q3-4 hr SC, IM, or IV
Oxycodone HCL	5 mg q6 hr po
Oxymorphone (Numorphan)	1-1.5 mg q4-6 hr SC or IM
Propoxyphene HCL (Darvon)	65 mg q4 hr po (cap)

Generic Name	**Dose**	**Comments**
Acetaminophen	325-650 mg q4-6h PO	Hepatic toxicity follows large doses
Aspirin	325-650 mg q4-6h PO	May cause dyspepsia and GI bleeding, decrease platelet function; less hepatic and renal toxicity
Difflunisal	200-500 mg q8-12h PO	Less irritating to GI tract
Ibuprofen	200-400 mg q8-12h PO	Fewer GI symptoms
Indomethacin	25-50 mg q8h PO	Not recommended in chronic benign pain
Ketorolac	10 mg q6h PO (see drug section for IV dosing)	For acute exacerbations of chronic pain

Naproxen	500 mg initially, then 250 mg q6-8h PO	Slightly more toxic than ibuprofen regarding GI and CNS effects

Chronic Pain Management

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Common Chronic Pain Syndromes

1. **Myofascial pain**
   A. Common cause of somatic pain that is characterized by marked tenderness of discrete points (trigger points) within skeletal muscles and the appearance of tight, ropy bands of skeletal muscle.
   B. The most important aspect of treatment is physical therapy. Infiltration of local anesthetic solution (0.5% lidocaine or 0.25% bupivacaine with cortisol 25 mg) into the trigger point provides analgesia that confirms the diagnosis and permits initiation of physical therapy. Injections can be performed daily.
2. **Lumbosacral radiculopathy** (chronic low back pain)
   A. Pain is usually the result of inflammation of the nerve root or mechanical compression of the dorsal root ganglion.
   B. L5 and S1 nerve roots are most commonly affected. Symptoms consist of low back pain, pain radiating to the lower extremity, motor and sensory loss consistent with the injury of the affected nerve root.
   C. After a full evaluation has been done to rule out the presence of infection or space-occupying lesion, administration of epidural corticosteroids (methylprednisolone 80 mg or triamcinolone 50 mg) into the epidural space as close to the affected nerve root as possible can be performed. If symptoms are improved but still present 1-2 weeks after the initial injection it is acceptable to repeat epidural steroid injection.
3. **Lumbosacral arthropathies**
   A. Degeneration and inflammation of the lumbar facet joints and sacroiliac joints may produce low back pain radiating to the lower extremities that is difficult to distinguish from lumbosacral radiculopathy. Diagnosis is confirmed by prolong relief following injection of local anesthetic into the facet joint.
4. **Intercostal neuralgia**
   A. Characterized by paresthesias and pain in response to touch or movement of the thorax.
   B. Destructive nerve blocks with alcohol or phenol are usually ineffective. Local anesthetic intercostal or paravertebral nerve blocks will provide pain-free intervals, during which time physical therapy can be performed.
5. **Complex regional pain syndrome**
   A. **Type 1 (CRPS type 1: reflex sympathetic dystrophy)**
      1. Refers to disorders that develop as a consequence of trauma affecting the limbs, with or without an obvious peripheral nerve injury. The pain is usually burning in nature and accompanied by diffuse tenderness and pain on light touch. Autonomic nervous system dysfunction is manifested as changes in skin temperature, cyanosis, edema, and hyperhidrosis.
      2. **Treatment:** local anesthetic blockade of the sympathetic chain (cervicothoracic block [stellate ganglion block], lumbar sympathetic block) is useful to confirm the diagnosis. Treatment consist of a series of local anesthetic blocks. Alternate modalities include continuous epidural block, physical therapy, and TENS.
   B. **Type 2 (CRPS type 2: causalgia)**

1. Characterized by burning pain and autonomic nervous system dysfunction associated with major trunk injury. Pain is aggravated by movement or any physical stimulation (light touch). The affected extremity is often warm, dry, and venodilated.
2. **Treatment:** local anesthetic-induced sympathetic blockade may be helpful. Surgical sympathectomy may also be considered.

Postanesthesia Care Unit

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Hemodynamic Complications

1. **Hypotension**
   A. **Causes:** arterial hypoxemia, hypovolemia (most common), decreased myocardial contractility (myocardial ischemia, pulmonary edema), decreased systemic vascular resistance (neuroaxial anesthesia, sepsis), cardiac dysrhythmias, pulmonary embolus, pneumothorax, cardiac tamponade, spurious (large cuff).
   B. **Treatment:** fluid challenge; pharmacologic treatment includes inotropic agents (dopamine, dobutamine, epinephrine) and alpha receptor agonists (phenylephrine, epinephrine). CVP and PA catheter monitoring may be needed to guide therapy.
2. **Hypertension**
   A. **Causes:** enhanced SNS activity (pain, gastric distension, bladder distension), preoperative hypertension, hypervolemia, hypoxemia, spurious (small cuff), increased intracranial pressure, and vasopressors.
   B. **Treatment:** management begins with identification and correction of the initiating cause; various medications can be used to treat hypertension including beta blockers (labetalol 5-10 mg IV, esmolol 10-100 mg IV), calcium channel blockers (nifedipine 5-10 mg SL, verapamil 2.5-5 mg IV), nitroprusside or nitroglycerin; regardless of drug selected it is important to accurately monitor blood pressure.
3. **Cardiac dysrhythmias**
   A. **Causes:** arterial hypoxemia, hypercarbia, hypovolemia, pain, electrolyte and acid-base imbalances, myocardial ischemia, increased ICP, drug toxicity (digitalis), hypothermia, anticholinesterases and malignant hyperthermia.
   B. **Treatment:** supplemental oxygen should be given while the etiology is being investigated; most dysrhythmias do not require treatment.

Respiratory and Airway Complications

1. Respiratory problems are the most frequently encountered complications in the PACU, with the majority related to airway obstruction, hypoventilation, or hypoxemia.
2. **Hypoxemia**
   A. **Causes:** right-to-left intrapulmonary shunt (atelectasis), mismatching of ventilation-to-perfusion (decreased functional residual capacity), decreased cardiac output, alveolar hypoventilation, diffusion hypoxia, upper airway obstruction, bronchospasm, aspiration of gastric contents, pulmonary edema, pneumothorax and pulmonary embolism, obesity, advanced age, and posthyperventilation hypoxia.
   B. **Clinical signs of hypoxia** (restlessness, tachycardia, cardiac irritability hypertension, hypotension) are nonspecific; obtundation, bradycardia, hypotension, and cardiac arrest are late signs.
   C. Increased intrapulmonary shunting relative to closing capacity is the most common cause of hypoxemia following general anesthesia.
   D. **Treatment:** oxygen therapy with or without positive airway pressure. Additional treatment should be directed at the underlying cause.
3. **Hypoventilation**
   A. **Causes:** drug-induced central nervous system depression (residual anesthesia), suboptimal ventilatory muscle mechanics, increased production of carbon dioxide, decreased ventilatory drive, pulmonary, and respiratory muscle insufficiency (preexistent respiratory disease, inadequate reversal of neuromuscular blockade, inadequate analgesia, and bronchospasm).
   B. Hypoventilation in the PACU is most commonly caused by residual depressant effects of anesthetic agents on respiratory drive or persistent neuromuscular blockade.

C. **Treatment:** should be directed at the underlying cause. Marked hypoventilation may require controlled ventilation until contributory factors are identified and corrected.

4. **Upper airway obstruction** (stridor)

A. **Causes:** include incomplete anesthetic recovery, laryngospasm, airway edema, wound hematoma, and vocal cord paralysis. Airway obstruction in unconscious patients is most commonly due to the tongue falling back against the posterior pharynx.

B. **Treatment:** supplemental oxygen while corrective measures are undertaken. Jaw thrust, head-tilt, oral or nasal airways often alleviate the problem.

5. **Laryngospasm and laryngeal edema**

A. Laryngospasm is a forceful involuntary spasm of the laryngeal musculature caused by sensory stimulation of the superior laryngeal nerve. Triggering stimuli include pharyngeal secretions or extubating in stage 2. The large negative intrathoracic pressures generated by the struggling patient in laryngospasm can cause pulmonary edema.

B. **Treatment of laryngospasm:** initial treatment includes 100% oxygen, anterior mandibular displacement, and gentle CPAP (may be applied by face mask). If laryngospasm persists and hypoxia develops, succinylcholine (0.25-1.0 mg/kg; 10-20 mg) should be given in order to paralyze the laryngeal muscles and allow controlled ventilation.

C. **Treatment of glottic edema and subglottic edema:** administer warm, humidified oxygen by mask, inhalation of racemic epinephrine 2.25% (0.5-1 mL in 2 mL NS), repeated every 20 minutes, dexamethasone 0.1-0.5 mg/kg IV maybe considered. Reintubation with a smaller tube may be helpful.

Neurologic Complications

1. **Delayed awakening:** the most frequent cause of a delayed awakening is the persistent effect of anesthesia or sedation. Other causes include recurarization, severe hypothermia, hypoglycemia, and neurologic disorders.

2. **Emergence delirium (agitation):** is characterized by excitement, alternating with lethargy, disorientation, and inappropriate behavior. Potential causes include arterial hypoxemia, hypercapnia, pain, unrecognized gastric dilation, urinary retention, and previous administration of atropine. Treatment includes haloperidol, titrated in 1-2 mg IV increments. Benzodiazepines may be added if agitation is severe. Physostigmine (0.5-2.0 mg IV) may reverse anticholinergic delirium.

Nausea and Vomiting

1. **Risk factors**

A. **Patient risk factors:** short fasting status, anxiety, younger age, female, obesity, gastroparesis, history of postoperative nausea/vomiting or motion sickness.

B. **Surgery-related factors:** gynecological, abdominal, ENT, ophthalmic, and plastic surgery; endocrine effects of surgery; duration of surgery.

C. **Anesthesia-related factors:** premedicants (morphine and other opioids), anesthetics agents (nitrous oxide, inhalational agents, etomidate, methohexital, ketamine), anticholinesterase reversal agents, gastric distention, longer duration of anesthesia, mask ventilation, intraoperative pain medications, regional anesthesia(lower risk).

D. **Postoperative factors:** pain, dizziness, movement after surgery, premature oral intake, opioid administration.

Drug	**Adult Dos**	**Peds Dose**	**Duration**	**Caution Use In**

	e			
Droperidol	0.625-1.25 mg IV/IM	50-75 mcg/kg IV/IM	3-4 hr	Parkinson, hypovolemia
Metoclopramide	10 mg IV/IM (max 20 mg)	0.1 mg/kg (max 5 mg)	1-2 hr	GI obstruction, seizures, Parkinson
Trimethobenz?amide	200 mg IM/PR	<14 kg: 100 mg >14 kg: 100-200 mg	6-8 hr	Benzocaine allergy, Reye's syndrome
Ondansetron	4-8 mg IV	0.1 mg/kg IV	4-6	Prolong cardiac conduction
Dolasetron	12.5 mg IV	0.35 mg/kg	7 hr	Prolong cardiac conduction
Granisetron	1-3 mg IV	10 mcg/kg IV	24 hr	Liver disease
Propofol	10-20 mg IV			
Dexamethasone	8-10 mg IV	0.15-1 mg/kg		
Betamethasone	12 mg IV			
Promethazine	12.5-25 mg IV/IM	0.25-1 mg/kg IV/IM	4 hr	Seizures, hypovolemia, Parkinson

Prochlorperazine	2.5-10 mg IV/IM	0.1-0.15 mg/kg IV/IM	6-12 hr	Seizures, hypovolemia, Parkinson

Pain Control (see section on acute pain management)

1. **Moderate to severe postoperative pain in the PACU**
   A. Meperidine 25-150 mg (0.25-0.5 mg/kg in children).
   B. Morphine 2-4 mg (0.025-0.05 mg/kg in children).
   C. Fentanyl 12.5-50 mcg IV.
2. **Nonsteroidal anti-inflammatory drugs** are an effective complement to opioids. Ketorolac 30 mg IV followed by 15 mg q6-8 hrs.
3. **Patient-controlled and continuous epidural analgesia** should be started in the PACU.

Miscellaneous Complications

1. **Renal dysfunction:** oliguria (urine output less then 0.5 mL/kg/hour) most likely reflects decreased renal blood flow due to hypovolemia or decreased cardiac output.
2. **Bleeding abnormalities:** causes include inadequate surgical hemostasis or coagulopathies.
3. **Shivering** (hypothermia)
   A. Shivering can occur secondary to hypothermia or the effects of anesthetic agents (most often volatile anesthetics).
   B. Shivering should be treated with warming measures (Bair Huggar system). Small doses of meperidine (12.5-25 mg) IV.

Discharge Criteria

1. All patients should be evaluated by an anesthesiologist prior to discharge; patients should have been observed for respiratory depression for at least 30 minutes after the last dose of parenteral narcotic.
   A. Patients receiving regional anesthesia should show signs of resolution of both sensory and motor blockade prior to discharge.
   B. Other minimum discharge criteria include stable vital signs, alert and oriented (or to baseline), able to maintain adequate oxygen saturation, free of nausea/vomiting, absence of bleeding, adequate urine output, adequate pain control, stabilization or resolution of any problems, and movement of extremity following regional anesthesia.

Malignant Hyperthermia

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1. **Definition** malignant hyperthermia is a fulminant skeletal muscle hypermetabolic syndrome occurring in genetically susceptible patients after exposure to an anesthetic triggering agent. Triggering anesthetics include halothane, enflurane, isoflurane, desflurane, sevoflurane, and succinylcholine.
2. **Etiology**: the gene for malignant hyperthermia is the genetic coding site for the calcium release channel of skeletal muscle sarcoplasmic reticulum. The syndrome is caused by a reduction in the reuptake of calcium by the sarcoplasmic reticulum necessary for termination of muscle contraction, resulting in a sustained muscle contraction.
3. **Clinical findings**
   A. **Signs of onset:** tachycardia, tachypnea, hypercarbia (increased end-tidal CO_2 is the most sensitive clinical sign).
   B. **Early signs:** tachycardia, tachypnea, unstable blood pressure, arrhythmias, cyanosis, mottling, sweating, rapid temperature increase, and cola-colored urine.
   C. **Late (6-24 hours) signs:** pyrexia, skeletal muscle swelling, left heart failure, renal failure, DIC, hepatic failure.
   D. **Muscle rigidity** in the presence of neuromuscular blockade. Masseter spasm after giving succinylcholine is associated with malignant hyperthermia.
   E. The presence of a large difference between mixed venous and arterial carbon dioxide tensions confirms the diagnosis of malignant hyperthermia.
   F. **Laboratory:** respiratory and metabolic acidosis, hypoxemia, increased serum levels of potassium, calcium, myoglobin, CPK, and myoglobinuria.
4. **Incidence and mortality**
   A. **Children:** approx 1:12,000 general anesthetics.
   B. **Adults:** approx 1:40,000 general anesthetics when succinylcholine is used; approx 1:220,000 general anesthetics when agents other than succinylcholine are used.
   C. Familial autosomal dominant transmission with variable penetrance.
   D. **Mortality:** 10% overall; up to 70% without dantrolene therapy. Early therapy reduces mortality for less than 5%.
5. **Anesthesia for malignant hyperthermia susceptible patients**
   A. Malignant hyperthermia may be triggered in susceptible patients who have had previous

uneventful responses to triggering agents.

B. Pretreatment with dantrolene is not recommended. If deemed necessary, may give 2.4 mg/kg IV over 10-30 minutes prior to induction.

C. The anesthesia machine should be prepared by flushing the circuit with ten liters per minute of oxygen for 20 minutes. Changing the fresh gas hose will hasten the reduction of the concentration of inhalation agents. Fresh carbon dioxide absorbent and fresh delivery tubing are also recommended.

6. **Malignant hyperthermia treatment protocol**

A. **Stop triggering anesthetic agent immediately,** conclude surgery as soon as possible. Continue with safe agents if surgery cannot be stopped.

B. **Hyperventilate:** 100% oxygen, high flows, use new circuit and soda lime.

C. **Administer dantrolene** 2.5 mg/kg IV; repeat every 5-10 minutes until symptoms are controlled or a total dose of up to 10 mg/kg is given.

D. **Correct metabolic acidosis:** administer sodium bicarbonate, 1-2 mEq/kg IV guided by arterial pH and pCO_2. Follow with ABG.

E. **Hyperkalemia:** correct with bicarbonate or with glucose, 25-50 gm IV, and regular insulin, 10-20 u.

F. **Actively cool patient**

1. Iced IV NS (not LR) 15 mL/kg every 10 minutes times three if needed.
2. Lavage stomach, bladder, rectum, peritoneal and thoracic cavities.
3. Surface cooling with ice and hypothermia blanket.

G. **Maintain urine output** >1-2 mL/kg/hr. If needed, mannitol 0.25 g/kg IV or furosemide 1 mg/kg IV (up to 4 times) and/or hydration.

H. **Labs:** PT, PTT, platelets, urine myoglobin, ABG, K, Ca, lactate, CPK.

I. Consider invasive monitoring: arterial blood pressure and CVP.

J.**Postoperatively:** continue dantrolene 1 mg/kg IV q6 hours x 72 hrs to prevent recurrence. Observe in ICU until stable for 24-48 hrs. Calcium channel blockers should not be given when dantrolene is administered because hyperkalemia and myocardial depression may occur.

Allergic Drug Reactions

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7. **Anaphylaxis**

A. **Anaphylaxis is an allergic reaction** which is mediated by an antigen-antibody reaction (type I hypersensitivity reaction). This reaction is initiated by antigen binding to immunoglobulin E (IgE) antibodies on the surface of mast cells and basophils, causing the release of chemical mediators, including, leukotrienes, histamine, prostaglandins, kinins, and platelet-activating factor.

B. **Clinical manifestations of anaphylaxis**

1. **Cardiovascular:** hypotension, tachycardia, dysrhythmias.
2. **Pulmonary:** bronchospasm, cough, dyspnea, pulmonary edema, laryngeal edema, hypoxemia.
3. **Dermatologic:** urticaria, facial edema, pruritus.

8. **Anaphylactoid reactions**

A. Anaphylactoid reactions resemble anaphylaxis but are not mediated by IgE and do not require prior sensitization to an antigen.

B. Although the mechanisms differ, anaphylactic and anaphylactoid reactions can be clinically indistinguishable and equally life-threatening.

9. **Treatment of anaphylactic and anaphylactoid reactions**

A. **Initial therapy**

1. Discontinue drug administration and all anesthetic agents.
2. Administer 100% oxygen.
3. Intravenous fluids (1-5 liters of LR).
4. Epinephrine (10-100 mcg IV bolus for hypotension; 0.1-0.5 mg IV for cardiovascular collapse).
5.

B. **Secondary treatment**
   1. Benadryl 0.5-1 mg/kg or 50-75 mg IV.
   2. Epinephrine 2-4 mcg/min, norepinephrine 2-4 mcg/min.
   3. Aminophylline 5-6 mg/kg IV over 20 minutes.
   4. 1-2 grams methylprednisolone or 0.25-1 gram hydrocortisone.
   5. Sodium bicarbonate 0.5-1 mEq/kg.
   6. Airway evaluation (prior to extubation).

Venous Air Embolism

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1. **General information**
   A. Air can be entrained into a vein whenever there is an open vein and a negative intravenous pressure relative to atmospheric pressure. This can occur any time the surgical field is above right atrial level (neurosurgical procedures and operations involving the neck, thorax, abdomen, pelvis, open heart, liver and vena cava laceration repairs, total hip replacement).
   B. Incidence is highest during sitting craniotomies (20-40%).
2. **Diagnosis**
   A. **Transesophageal echocardiography (TEE)** is the most sensitive. Sensitivity = 0.015 mL of air/kg/min.
   B. **Doppler:** sensitivity equals 0.02 mL of air/kg/min.
   C. **Decreased PaO_2, TcO_2, and increased ETN_2 (most specific)** normally occur before one sees a sudden decrease in $ETCO_2$ and/or an increase in CVP.
   D. During controlled ventilation of the lungs, sudden attempts by the patient to initiate a spontaneous breath (gasp reflex) may be the first indication of venous air embolism.
   E. **Late signs:** hypotension, tachycardia, cardiac dysrhythmias and cyanosis.
   F. **Consequences depend of the volume and rate of air entry.**
      1. CVP increases 0.4 mL of air/kg/min.
      2. Heart rate increases at 0.42 mL of air/kg/min.
      3. EKG changes occur at 0.6 mL of air/kg/min.
      4. Blood pressure decreases at 0.69 mL of air/kg/min.
      5. A mill wheel murmur is heard at 2.0 mL of air/kg/min.
      6. Embolism of greater than 2.0 mL of air/kg/min is potentially lethal.
3. **Treatment**
   A. Notify surgeon to flood surgical field with saline or pack and apply bone wax to the skull edges until the entry site identified.
   B. Place the patient in the left lateral decubitus position with a slight head-down tilt in an attempt to dislodge a possible air lock.
   C. Nitrous oxide should be discontinued and 100% oxygen given.
   D. The central venous catheter should be aspirated in an attempt to retrieve the entrained air.
   E. Support cardiovascular system with volume, inotropes, and/or vasopressors.
   F. Increase venous pressure with bilateral jugular vein compression.

Latex Allergy

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1. **Risk factors for latex allergy**
   A. Chronic exposure to latex and a history of atopy increases the risk of sensitization. Patients undergoing frequent procedures with latex items (eg, repeated urinary bladder catheterization) are at higher risk.
   B. Patients with neural tube defects (meningomyelocele, spina bifida) and those with congenital abnormalities of the genitourinary tract are at higher risk.
2. **Pathophysiology:** most reactions involve a direct IgE-mediated immune response to polypeptides in natural latex. Some cases of contact dermatitis may be due to a type four hypersensitivity reaction to chemicals introduced in the manufacturing process.
3. **Preoperative evaluation**
   A. **History** in patients at risk, particularly those with co-existing atopy and/or multiple allergies, should include history of balloon or glove intolerance and allergies to medical products used in chronic care (eg, catheters). Elective patients in whom latex allergy is suspected should be referred to an allergist.
   B. **Diagnostic tests:** routine diagnostic testing in the at-risk population is not recommended (only those with a positive history). Available tests:
      1. Skin-prick test: less sensitive than intradermal test but more sensitive than RAST.
      2. Radioallergosorbent test (RAST). An in-vitro test for IgE antibodies in the patient's serum.
      3. Pre-operative medications. Routine preoperative H_1 and H_2 blockers and steroids is no longer recommended.
      4. Scheduling. since latex is an aeroallergen and present in the operating room air for at least an hour after the use of latex gloves, whenever possible your patient should be scheduled as the first case of the day.
4. **Anesthesia equipment**
   A. Common anesthesia equipment that contain latex include gloves, tourniquets, endotracheal tube, ventilator bellows, intravenous injection ports, blood pressure cuffs, and face masks.
   B. Non-latex supplies that are commonly required include glass syringes, drugs in glass in ampules, IV tubing without latex injection ports, neoprene gloves, ambu bag with silicone valves, and neoprene bellows for the Ohmeda ventilator.
   C. The most important precaution is the use of non-latex gloves.
   D. Miscellaneous equipment: sleeve on the fibreoptic bronchoscope, esophageal stethoscope, and cuff on the LMA airway are all non-latex (silicon).
5. **Preoperative preparation**
   A. Check latex allergy cart for supplies. Call pharmacy and order all drugs that may be needed (dispensed in glass syringe).
   B. Notify O.R. nurses. No latex gloves or latex products should come into contact with the patient. Neoprene (non-latex) gloves need to be obtained.
6. **Anesthesia setup and care**
   A. Set up a regular circuit on the anesthesia machine, and use a neoprene reservoir bag. Use plastic masks (adult or pediatric).
   B. Draw up drugs in glass syringes from glass ampules. In an emergency, the rubber stoppers can be removed.
   C. IV infusion setup with two three way stopcocks and no injection ports. (Alternatively tape all injection ports over and do not use).
   D. Use Webril under tourniquet or BP cuff if rubber, angiocaths are safe.
   E. Latex allergy should not alter the choice of anesthetic technique. There are no drugs that are specifically contraindicated.
7. **Diagnosis of latex anaphylaxis**
   A. Anaphylaxis has been reported even in patients pre-treated with H_1, H_2 blockers and steroids and managed in a latex-free environment.
   B. Onset is generally 20 - 60 minutes after exposure to the antigen.
   C. Anaphylaxis presents with the clinical triad of hypotension (most common sign), rash, and bronchospasm.
   D. Serum mast cell tryptase levels are high during an episode and up to 4 hours after. This test will help confirm the diagnosis of anaphylaxis, but it will not identify latex as the antigen.
8. **Treatment of latex anaphylaxis**

A. Treatment of latex anaphylaxis does not differ from the treatment of other forms of anaphylactic reaction.

B. **Primary treatment**

1. Stop administration of latex, administer 100% oxygen.
2. Restore intravascular volume (2-4 L of crystalloid).
3. Epinephrine: start with a dose of 10 mcg, or 0.1 mcg/kg and escalate rapidly to higher doses depending on the response.

C. **Secondary treatment**

1. Corticosteroids (0.25-1 g hydrocortisone or 1-2 g methyl-prednisolone).
2. Diphenhydramine 50-75 mg IV.
3. Aminophylline 5-6 mg/kg over 20 minutes.

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