FIRST AID FOR THE®

ANESTHESIOLOGY BOARDS

AN INSIDER'S GUIDE

Summaries of high-yield topics prepare you for in-service and board exams and recertification

Mnemonics, tables, and images **reinforce essential concepts**

Written by residents and reviewed by faculty

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INTRODUCTION

Guide to the ABA Part 1 Examination

INTRODUCTION

The ABA examination can be a great source of stress for anesthesiology residents and anesthesiologists. It is required for both primary certification and recertification for practicing anesthesiologists. According to the ABA a board certified anesthesiologist is "a physician who provides medical management and consultation during the perioperative period, in pain medicine and in critical care medicine. At the time of application and at the time of initial certification, a diplomate of the Board must possess knowledge, judgment, adaptability, clinical skills, technical facility, and personal characteristics sufficient to carry out the entire scope of anesthesiology practice without accommodation or with reasonable accommodation." The examination not only provides a means for the ABA to evaluate if a candidate has attained a certain level of proficiency to practice anesthesiology but also for patients to be comfortable that a candidate has achieved a level of clinical knowledge and competency required to provide good clinical care.

This chapter is meant to provide basic information about the ABA exam and provide some useful tips to help conquer this integral step in providing for board certification. Detailed information is also available on the ABA website (www.theaba.org) and the booklet of information published on the website.

Check the ABA website for detailed and updated information: www.theaba.org

ABA–THE BASICS

Certification Requirements

From the ABA website, the following are the requirements for a candidate to be eligible to take the board certification exam. At the time of certification by the ABA, the candidate must:

A. Hold an unexpired license to practice medicine or osteopathic medicine in at least one state or jurisdiction of the United States or province of Canada that is permanent, unconditional and unrestricted. Further, every United States and Canadian medical license the applicant holds must be free of restrictions.
Candidates for initial certification and ABA diplomates have the affirmative obligation to advise the ABA of any and all restrictions placed on any of their medical licenses and to provide the ABA with complete information concerning such restrictions within 60 days after their imposition or notice, **whichever first occurs.** Such information shall include, but not be limited to, the identity of the state medical board imposing the restriction as well as the restriction's duration, basis, and specific terms and conditions. Candidates and diplomates discovered **not** to have made disclosure may be subject to sanctions on their candidate or diplomate status.

B. Have fulfilled all the requirements of the continuum of education in anesthesiology.

C. Have on file with the ABA a Certificate of Clinical Competence with an overall satisfactory rating covering the final six-month period of clinical anesthesia training in each anesthesiology residency program.

D. Have satisfied all examination requirements of the Board.

E. Have a professional standing satisfactory to the ABA.

F. Be capable of performing independently the entire scope of anesthesiology practice without accommodation or with reasonable accommodation.

Exam Registration

*The **standard deadline** for a complete application is **December 15.** Check the ABA website for late deadline each year.*

Registration for the exam must be done using the ABA Electronic Application System, via the ABA website at www.theABA.org. Registration is available once the application is accepted by the ABA.

The **ABA Part 1 examination** is administered once each year. Test dates are August 3–4, 2009, and August 2–3, 2010. The **standard deadline** for the ABA to receive a completed application and the application fee is **December 15** of the year immediately preceding the year in which the Part 1 examination is to be administered.

For examination in 2009, the **late deadline** by which the ABA must receive a completed application, the application fee and the late fee is January 15, 2009.

For examination in 2010, the late deadline by which the ABA must receive a completed application, the application fee and the late fee is December 31, 2009.

Application fee of $750 is due at the time of the application and a separate examination fee of $500 is due when the candidate accepts the examination opportunity. A late fee of $1050 will be charged if the application is received after the deadline has passed. The late deadline by which the ABA must receive the application and late fee changes each year so please refer to the ABA website.

The ABA notifies candidates of the exact date, time, and location of their examination and the rules for its conduct at least two months before the date of examination. Candidates who inform the Board that they are canceling their examination appointment are charged a cancellation fee of $200 and may forfeit the examination opportunity. Notice of cancellation must be in writing and must include a check in the amount of the cancellation fee.

Once an examination opportunity is accepted by the candidate, scheduling and accommodations must be made with a Pearson testing center via the website.

Exam Structure

The ABA certification exam is currently a **one-day** computer-based test administered at numerous test centers around the country. The exam is 250 questions divided into two 125-question blocks during the day. You are allowed 2 hours and 20 minutes for each section with an optional 20-minute break in the middle of the two sections. During the time allotted for each block, you can answer test questions in any order as well as review responses and change answers. Examinees cannot go back and change answers from the previous block.

Exam Content

*The ABA certification exam is 250 questions broken into two 125-question blocks. **K-type questions are no longer on the exam.***

The ABA Part 1 (Written) Examination has three types of item formats: A-type, R-type, and G sets. K-type questions will no longer be on the examination. Examples of all item formats are found on the ABA website at www.theaba.org/Home/anesthesiology_initial_certification#entrance under Part 1 Examination Item Formats.

Most questions are relevant to the practice of general anesthesiology and the clinical science behind it. The questions consist of five major sections, including basic sciences, clinical sciences, organ-based basic and clinical sciences, clinical subspecialties, and special problems in anesthesiology. A detailed content outline can be found on the ABA website at www.theaba.org/pdf/ITEContentOutline2007.pdf.

Exam Score Reporting

Once you have finished the exam, a printed sheet will be given to you confirming that you have completed the exam and stating that you will be receiving your scores in 8–10 weeks. This sheet will be given to you by the examination center.

RECERTIFICATION EXAM/MAINTENANCE OF CERTIFICATION IN ANESTHESIOLOGY (MOCA)

The MOCA (Maintenance of Certification in Anesthesiology) program will now replace the Recertification in Anesthesiology program. Check ABA website for details.

The American Board of Anesthesiology is replacing its Recertification in Anesthesiology program with the **Maintenance of Certification in Anesthesiology (MOCA®) program**. Currently, either program is available to ABA diplomates who were certified prior to January 1, 2000. The ABA issued diplomates certified on or after January 1, 2000, a certificate that is valid for 10 years. Diplomates with a time-limited certificate must satisfactorily complete the MOCA before their time-limited certificate expires to maintain diplomate status in the specialty. The Recertification in Anesthesiology program is not available to these diplomates. The Recertification in Anesthesiology program will close in 2009. The MOCA program is available to all ABA diplomates, whether or not their initial certification is time-limited. MOCA is a 10-year program, and diplomates with a time-limited certificate must complete the program before their current certification expires in order to maintain their diplomate status. The exam for MOCA requirement needs to be taken in years 7–10 and may be taken up to twice a year.

The content of the exam is based on clinical practice and up to 25% of questions may (currently) be omitted. **After 2009, all questions must be answered.** Majority of the knowledge for the exam is based on clinical knowled and not textbook knowledge. The ABA MOCA/Recertification Examinatio comprised of 200 questions, of which approximately one-half of the questi are in General Anesthesia and approximately one-twelfth of the questions in each of the following areas: Pediatric Anesthesia, Cardiothoracic Anesthes Neuroanesthesia, Critical Care Medicine, Obstetrical/Gynecologic Anesthes and Pain Medicine.

i m p or tn t

Detailed information regarding deadlines, application process and fees can be found on the ABA website at www.theaba.org/Home/anesthesiology_maintenance.

TEST PREPARATION ADVICE

The ABA test tends to focus on many detailed concepts of anesthetic management involving pharmacology and physiology. Assuming that you have done well during your residency and maintained some level of self-study, First Aid can be a good tool to guide you. However, you should use other sources since First Aid is only one review tool; other resources such as textbooks, articles, other review books and especially question books are imperative in attaining a good score. Score your practice test questions, and look closely at the items you missed. By making sure you understand the reasons the answers you selected were incorrect, you'll be in a better position to interpret the questions correctly the next time. In addition, there are a number of high-quality board review courses offered around the country. Board review courses are very expensive but can help those who need some focus and discipline

Ideally, you should start your preparation early in your **first year of residency,** especially if you are planning on starting a demanding job or fellowship right after residency. Cramming in the period between the end of residency and the exam is **not advisable.** Also, realize that most of the scenarios are ones that you have seen during your three years of residency or been exposed to during morning lectures, grand rounds, and individual teaching sessions.

TEST-TAKING SKILLS TO SURVIVE

At this point in your life, you have taken enough standardized exams to know how to survive these kinds of exams. However, these are just some survival tips for the big day:

- Eat a good breakfast the morning of the exam—nutrition is an important thing to get you through the day.
- Don't worry about what to bring—the test center will provide you with a writing board, markers, earphones, and earplugs. Just wear something comfortable.
- Go through the tutorial—it does not take out of your time to take the exam and will prevent you from wasting time "looking for things" on the screen.
  - For longer questions, read the question stem and scan the options, and **then** go back and read the case. You may get your answer without having to read through the whole case.
  - Answer each question—there's no penalty for guessing, so you should **never** leave a question blank.
  - Good pacing is key. You need to leave adequate time to get to all the questions. If you don't know the answer within a short period, make an educated guess and move on.
- Identify key words—they may help you focus on what they are trying to ask you. Don't panic with "impossible" questions. They may be **experimental questions** that won't count. Again, take your best guess and move on.

*Identify **key words** to focus on what the question is asking.*

TESTING AND LICENSING AGENCIES

The American Board of Anesthesiology
4101 Lake Boone Trail, Suite 510
Raleigh, NC 27607-7506
919-881-2570
Fax: 919-881-2575
www.theaba.org/

Pearson VUE Professional Testing Center
www.vue.com

Federation of State Medical Boards (FSMB)
P. O. Box 619850
Dallas, TX 75261-9850
817-868-4000
Fax: 817-868-4099
www.fsmb.org

Educational Commission for Foreign Medical Graduates (ECFMG)
3624 Market Street
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Fax: 215-386-9196
www.ecfmg.org

SECTION I

The Fundamentals of Anesthetic Management

- Anesthetic Pharmacology
- Physiology and Anesthesia
- Preoperative Evaluation
- Anesthesia Machines
- Monitoring and Equipment
- Airway Management
- Regional Anesthesia
- Postoperative Recovery

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CHAPTER 1

Anesthetic Pharmacology

INHALATIONAL ANESTHETICS

Fast induction (↑ rate of rise of alveolar concentration) caused by:

↓ volatile solubility

↓ cardiac output

↓ alveolar venous partial pressure gradient

↑ alveolar ventilation

↑ Fi

Slow induction (↓ rate of rise of alveolar concentration) caused by:

↑ volatile solubility

↑ cardiac output

↑ alveolar venous partial pressure gradient

↓ alveolar ventilation

- **Pharmacokinetics:** What the body does to a drug (uptake, plasma/tissue concentration, and elimination).
- **Pharmacodynamics:** What the drug does to the body.
- **Fi** (inspired concentration of anesthetic) is determined by three factors: (1) fresh gas flow rate; (2) volume of the breathing system; and (3) absorption of the gas by the machine and circuit. In order to deliver the actual concentration of inhalational anesthetics:
  - **Fresh gas flow** must be high enough to minimize rebreathing.
  - **Volume of the anesthetic breathing system** (countering maximum alveolar concentration) should be minimal.
  - **Circuit absorption** of inhalational anesthetics should be minimal.
- **Fa** (alveolar concentration) is determined by three factors: (1) uptake; (2) alveolar ventilation; and (3) concentration and second gas effect.
- **Uptake** has three components:
  - **Blood solubility:** Unlike other drugs, the clinical effects of inhalational anesthetics are not determined by its blood concentration, but rather by its partial pressure in the brain; an ↑ solubility reduces the partial pressure of the anesthetic gas and slows induction.
  - **Alveolar blood flow:** ↑ blood flow to the alveolus causes ↑ uptake, which slows induction by removing more anesthetic from the gaseous phase, resulting in a ↓ alveolar concentration.
  - **Alveolar-venous partial pressure difference:** The partial pressure of venous blood indicates uptake of gas by tissues other than the clinical effect site (i.e., the brain); a larger difference in partial pressure between alveolar gas and the venous blood signifies ↑ tissue uptake, resulting in ↓ alveolar concentration.
- ↑ **alveolar ventilation** replaces anesthetic that is taken up into the bloodstream:
  - A ventilation-perfusion (V/Q) mismatch (shunt or dead space) lowers the arterial partial pressure of the anesthetic secondary to mixing of blood from areas of ↑ ventilation.
  - This effect is more pronounced with less soluble agents such as nitrous oxide.
- Concentration effect and second gas effect:
  - **Concentration effect** has two components: *Concentrating effect:* A reduction in lung volume caused by anesthetic uptake will concentrate the remaining alveolar gas. *Augmented inflow effect:* In replacing the volume of anesthetic taken up by the tissue, a higher alveolar ventilation rate results in a higher alveolar ventilation.
  - **Second gas effect**: When nitrous oxide is mixed with a companion gas such as oxygen or volatile agents, the ↑ vapor pressure and insoluble nature of nitrous oxide will enable it to be absorbed from the alveolus first, resulting in an ↑ alveolar concentration of oxygen or volatile anesthetic.
- **Elimination:** Subcutaneous diffusion and biotransformation are insignificant, as most inhalational anesthetics are eliminated by exhalation.
- **Minimum alveolar concentration (MAC):** The alveolar concentration of an inhalational anesthetic that prevents movement in 50% of patients in response to surgical incision.
  - Factors that ↑ MAC: Young age, hypernatremia, hyperthermia, chronic alcohol intoxication, acute cocaine or amphetamine intoxication.
  - Factors that ↓ MAC: Old age, $PaCO_2 < 40$ or > 95 mmHg, hyponatremia, hypothermia, pregnancy, intravenous anesthetics (opioids, ketamine, bar-

biturates, benzodiazepines), local anesthetics, lithium, acute alcohol intoxication.
  - Factors that **do not affect** MAC: Hypo- or hyperthyroidism.
- Theories on how inhalational anesthetics work:
  - **Agent-specific theory:** Each agent works on different receptors and mechanism.
  - **Unitary hypothesis:** All inhalational anesthetics share a common mechanism: **Meyer Overton rule:** Anesthesia results from agents dissolving at specific sites. **Critical volume hypothesis:** Anesthesia results from expanding lipid bilayer.

Specific Inhalational Anesthetics

- **Nitrous oxide** (N_2O): The only inorganic anesthetic. MAC 105%; blood/gas: 0.47. See Table 1-1 for effects of N_2O on organ systems.
- **Key points:** N_2O does not exhibit much muscle relaxation properties and is known to cause significant postoperative nausea and vomiting. N_2O is known to oxidize cobalt in vitamin B_{12}. It inhibits B_{12}-dependent enzymes, such as **methionine synthetase** (necessary for myelin formation) and **thymidylate synthetase** (necessary for DNA synthesis). May see pernicious anemia. Should be avoided in pregnancy.
  - **Contraindications:** Air embolus, pneumothorax, gastrointestinal obstruction, tympanoplasty, and intracranial air.
- **Isoflurane:** Nonflammable and pungent gas. MAC 1.2; vapor pressure: 240; blood/gas: 1.4. See Table 1-2 for effects of isoflurane on organ systems.
- **Desflurane:** Similar chemical structure to isoflurane. MAC: 6; vapor pressure: 681; blood/gas: 0.42. Desflurane's low solubility results in rapid wash-in and wash-out of the anesthetic. A special vaporizer is used due to its high vapor pressure. Degradation in dried carbon dioxide (CO_2) absorbent can cause carbon monoxide poisoning. See Table 1-3 for effects of desflurane on organ systems.
- **Sevoflurane:** Nonpungent and less soluble gas. MAC 2.0; vapor pressure: 160; blood/gas: 0.65. Widely used for inhalational induction along with N_2O. See Table 1-4 for effects of sevoflurane on organ systems.
  - Degradation of sevoflurane in the presence of CO_2 absorbent creates **compound A** (associated with renal toxicity); levels of compound A are ↑ with: Low fresh gas flow, ↑ respiratory temperature; dried baralyme; high sevoflurane concentration; long duration of exposure to sevoflurane.

CO_2 absorbent and volatiles: Desflurane associated with carbon monoxide poisoning Sevoflurane associated with compound A and fires

TABLE 1-1. Effects of Nitrous Oxide on Organ Systems

Cardiovascular	Minimal changes in blood pressure, heart rate, and cardiac output. Known to cause pulmonary hypertension.
Pulmonary	Results in rapid shallow breaths without changes in minute ventilation. Therefore, no changes in resting carbon dioxide (CO_2). Depressed peripheral chemoreceptor sensitivities to hypoxia.
Cerebral	Like other inhalational agents, cerebral blood flow, intracranial pressure, and cerebral metabolic rate of oxygen (O_2) are ↑.
Renal/Hepatic	↓ blood flow.

TABLE 1-2. Effects of Isoflurane on Organ Systems

Cardiovascular	Minimal cardiac depression, systemic vascular resistance ↓. CO maintained by an ↑ in heart rate (HR) via baroreceptor reflex and mild β-adrenergic stimulation. Rapid ↑ in isoflurane can result in transient ↑ in HR and blood pressure. Theoretically can cause coronary steal syndrome.
Pulmonary	Rapid shallow breaths that result in ↓ minute ventilation and ↑ resting partial pressure of carbon dioxide (pCO_2). Normal response to hypoxia and hypercapnia are abolished.
Cerebral	↑ cerebral blood flow and intracranial pressure (ICP). Hyperventilation can prevent ↑ in ICP.
Renal	↓ blood flow.

TABLE 1-3. Effects of Desflurane on Organ Systems

Cardiovascular	Significant ↓ in systemic vascular resistance and ↑ in heart rate (HR). Cardiac output is generally well maintained. Rapid ↑ in concentration results in transient ↑ in catecholamine levels with ↑ in HR and blood pressure (BP), more pronounced than isoflurane.
Pulmonary	Rapid shallow breaths that result in ↓ in minute ventilation and ↑ in resting partial pressure of carbon dioxide (pCO_2). Pungency can be responsible for coughing, breath holding, and laryngospasm.
Cerebral	↑ cerebral blood flow and intracranial pressure, which can be prevented with hyperventilation (cerebral vessels still respond to changes in pCO_2). Cerebral metabolic rate of O_2 is ↓.
Renal	Reduces blood flow proportionate to ↓ in BP.

- Degradation of sevoflurane, especially with desiccated CO_2 absorbent, can also lead to fires in the anesthesia circuit (isolated incident).

- **Halothane:** MAC: 0.75%; vapor pressure: 243; blood/gas: 2.4. See Table 1-5 for effects of halothane on organ systems.
  - **Key points:** Two types of hepatotoxicity: **Self-limiting disease:** With hypoxia and a ↓ in hepatic blood flow, halothane undergoes reduction, which results in mild elevation in liver function tests. **Halothane hepatitis:** Rare, immune-mediated disease. ↑ risks are seen in middle-aged, obese patients who are exposed to halothane multiple times along with phenobarbital for induction. ↑ aspartate transaminase (AST), alanine transaminase (ALT), bilirubin, encephalopathy, and centrilobular necrosis are often seen.
- **Methoxyflurane:** Most lipid-soluble agent. MAC: 0.16%; vapor pressure: 22.5; blood/gas: 12. See Table 1-6 for effects of methoxyflurane on organ systems.
  - **Key points:** The oxidative metabolite fluoride is responsible for causing vasopressin-resistant high-output renal failure. Toxicity is proportional to plasma fluoride level (2–3 MAC hr).
- **Enflurane:** MAC: 1.7 vapor pressure 175, blood/gas: 1.7. See Table 1-7 for effects of enflurane on organ systems.

TABLE 1-4. Effects of Sevoflurane on Organ Systems

Cardiovascular	Slight ↓ in myocardial contractility, systemic vascular resistance, and blood pressure (BP). Minimal changes in heart rate; therefore, cardiac output is mildly ↓.
Pulmonary	Potent bronchodilator, second to halothane. Rapid, shallow breaths that result in ↓ in minute ventilation and ↑ in resting partial pressure of carbon dioxide (pCO_2).
Cerebral	↓ in cerebral vascular resistance causes ↑ in cerebral blood flow and intracranial pressure.
Renal	Reduces blood flow proportionate to ↓ in BP. Sevoflurane is metabolized by the liver's P-450 system similar to but 10 times less than methoxyflurane. Serum fluoride level is ↑, but there is no clinical evidence of renal failure.

TABLE 1-5. Effects of Halothane on Organ Systems

Cardiovascular	Direct myocardial depressant, coronary blood flow ↓ but adequate perfusion due to less O_2 demand. Depresses baroreceptor reflexes; therefore, no compensatory tachycardia with hypotension. Cardiac output ↑ by two mechanisms: (1) myocardial depression, and (2) ↓ in heart rate. **Unlike most other agents, there is no change in systemic vascular resistance.** Halothane is known to sensitize the heart to catecholamines.
Pulmonary	Rapid, shallow breaths, but unlike N_2O, minute ventilation ↓ as well. Apneic threshold ↑; therefore, resting partial pressure of carbon dioxide (pCO_2) ↑. By inhibition of intracellular calcium mobilization, it is a highly potent bronchodilator.
Cerebral	↑ cerebral blood flow and intracranial pressure (ICP). However, the ↑ in ICP can be blunted by preinduction hyperventilation.
Renal	↓ renal blood flow.

TABLE 1-6. Effects of Methoxyflurane on Organ Systems

Cardiovascular	Like halothane, ↓ cardiac contractility. Blood pressure and cardiac output ↓. Unlike halothane, heart rate usually rises.
Pulmonary	Rapid, shallow breaths resulting in lowered minute ventilation. Resting partial pressure of carbon dioxide (pCO_2) is ↑.
Cerebral	↑ cerebral blood flow and intracranial pressure. Cerebral metabolic rate of oxygen (O_2) is ↓.
Renal	↓ in renal blood flow and glomerular filtration rate.

TABLE 1-7. Effects of Enflurane on Organ Systems

Cardiovascular	Direct myocardial depressant and ↓ systemic vascular resistance. Heart rate rises moderately, but cardiac output ↓ significantly.
Pulmonary	Rapid, shallow breaths; ↓ in minute ventilation; and rise in resting partial pressure of carbon dioxide (pCO_2). Responses to hypercapnia and hypoxia are abolished.
Cerebral	↑ cerebral blood flow and intracranial pressure. Deep anesthesia level and hypocapnia are known to cause electroencephalographic changes from high voltage, high frequency to spike-and-wave pattern, and eventually result in **epileptic form.**
Renal	Reduces blood flow proportionate to ↓ in blood pressure.

NONVOLATILE ANESTHETIC AGENTS

See Table 1-8 for effects of nonvolatile anesthetic agents on organ systems.

- **Absorption:** Actual amount of drug in the bloodstream.
- **Bioavailability:** Amount of drug that is effective at the site of action.
- **Distribution:** Affected by organ perfusion, protein binding, degree of ionization, and lipid solubility. Vessel-rich groups (brain, heart, lungs, kidney, and liver) comprise 10% of body mass but receive 75% of blood flow. Lipid-soluble and nonionized molecules pass freely through lipid membranes.
- **Biotransformation:** Primarily by liver.
  - Oxidation: Removing electrons.
  - Reduction: Adding electrons.
  - Hydrolysis: Adding H_2O.
  - Hepatic clearance depends on hepatic blood flow, fraction of drug extracted by the liver, and amount of drug that is not bound by serum protein.
- **Excretion:** Primarily by kidneys.
- **Clearance:** Rate of elimination.
- **Elimination:** Biotransformation + excretion. The body can be conceptualized into two compartments: central and peripheral. After an IV bolus, a drug first undergoes a distribution phase (α phase), which is equivalent to the distribution of a drug from plasma and vessel-rich groups to nonactive tissues and vessel-poor groups. Thereafter, the drug undergoes an elimination phase (β phase).
- **Efficacy:** 100% of drug effect.
- **Potency:** Effect relative to dosage.
- **Therapeutic index:** LD50/ED50.
- **Barbiturates**: Thiamylal (3–6 mg/kg), phenobarbital, thiopental (3–6 mg/kg), methohexital (1–2 mg/kg).
  - Mechanism of action: Presynaptically inhibits excitatory neurotransmitters; postsynaptically $\uparrow$ sensitivity to γ-aminobutyric acid (GABA); direct stimulation of GABA receptors.
  - Duration of action is mostly determined by distribution to nonactive sites. Low serum albumin level and hypovolemia may result in a higher brain concentration. Lower induction doses are required for the elderly due to (1) $\downarrow$ binding protein, (2) $\downarrow$ volume of central compartment, and (3) $\downarrow$ hepatic blood flow.
  - Biotransformation = hepatic oxidation to inactive water-soluble metabolites.
  - Drug interactions: Central nervous system (CNS) depressants such as alcohol, H_1 blockers, narcotics, and benzodiazepines can potentiate its effects.
- **Benzodiazepines:** Mainly used for premedication and sedation. Diazepam (IV and oral), midazolam (IV and IM), and lorazepam (IV, oral, and IM).
  - Mechanism of action: Facilitates GABA binding and hyperpolarization by $\uparrow$ chloride conductance.
  - Diazepam and lorazepam are well absorbed from the gastrointestinal tract. Midazolam and lorazepam are well absorbed via the IM route. Diazepam has a long duration of action due to its high protein binding and slow hepatic extraction. Lorazepam has a long duration of action due to its high affinity for the receptors. Midazolam has a half-life of 2 hr and is the shortest-acting drug in the group.

TABLE 1-8. Effects of Nonvolatile Agents on Organ Systems

	CV	Pulmonary	Cerebral	Other
Barbiturates	Depression of central vasomotor center in the brain stem: vasodilation and ↑ HR.	V_T and MV ↓. Normal response to hypoxia and hypercapnia is blunted.	Vasoconstriction, ↓ CBF and ICP. $CMRO_2$ ↓: protection to global ischemia.	Known to induce aminolevulinic acid synthetase, may precipitate acute intermittent porphyria.
Benzodiazepines	Minimal change in BP, HR, and CO. However, with combination of opioids, SVR and BP can ↓.	Normal response to CO_2 is minimally affected.	$CMRO_2$ is mildly depressed along with CBF and ICP, much less than barbiturates. Not known to be significantly neuroprotective.	
Opioids	Do not tend to be myocardial depressants. Histamine release associated with morphine and meperidine.	Depress ventilation. Normal response to CO_2 is blunted, ↑ resting pCO_2. Associated with chest wall rigidity.	Cause ↓ in $CMRO_2$, CBF and ICP activation of chemoreceptor trigger zone induces nausea and vomiting.	Gastrointestinal: Slows gastric emptying and peristalsis. Ileus and constipation.
Ketamine	BP, HR, and CO are all ↑ due to CNS sympathetic stimulation.	Normal ventilation and airway reflexes are sustained. Potent bronchodilator.	$CMRO_2$, CBF, and ICP are all ↑, unlike other nonvolatile agents.	Shares similar structure as phencyclidine, therefore similar psychomimetic effects including ↑ in ICP and hallucinations.
Etomidate	No significant effects on myocardial contractility, BP, and CO.	Ventilation is maintained. Induction typically does not cause apnea.	Like barbiturates, CBF, ICP, and $CMRO_2$ are all ↓.	Inhibition of B-11 hydroxylase and adrenocortical suppression are seen.
Propofol	Direct myocardial depressant, vasodilator.	Normal response to hypoxia and hypocapnia are blunted.	CBF and ICP are ↓.	Antipruritic and antiemetic properties. Effective in terminating status epilepticus.

BP, blood pressure; CBF, cerebral blood flow; $CMRO_2$, cerebral metabolic rate of O_2; CO, cardiac output; HR, heart rate; ICP, intracranial pressure; MV, minute ventilation; pCO_2, partial pressure of carbon dioxide; V_T, tidal volume.

Opioid lipid solubility:

Morphine (least)

Meperidine

Methadone

Alfentanil

Fentanyl

Sufentanil (most)

Histamine-releasing opioids:

Morphine

Meperidine

Codeine

Fentanyl

- Benzodiazepines are metabolized by the liver into water-soluble glucuronides. The end product of diazepam is pharmacologically active, the enterohepatic circulation of diazepam produces a secondary peak in 6–10 hr after its administration. The conjugated metabolite of midazolam is a mild CNS depressant, and its effect is more prominent in patients with renal failure.

- **Opioids:** A mild sedative effect but a potent analgesic.
  - Relative potency: Morphine, 1; meperidine, 0.1; fentanyl, 100; sufentanil, 1000; alfentanil, 20; and remifentanil, 200.
  - Mechanism of action: Binding to mu 1,2; kappa; delta; and sigma receptors throughout the CNS system. Analgesia is achieved by:
    - Inhibiting presynaptic release of substance P, dopamine, and acetylcholine (ACh).
    - Pain impulse transmission is interrupted at the level of dorsal horn.
    - Descending inhibitory impulses are accentuated.
    - Intrathecal opiates bind to opioid receptors at the substantia gelatinosa in the spinal cord.
    - All can be given intravenously. Morphine and meperidine are absorbed reliably via the IM route. Fentanyl can be administered transdermally via patch, which provides reservoir of drug in the upper dermis. Higher lipid solubility results in a faster onset of action.
    - Lower fat solubility of morphine slows passage through blood-brain barrier.
    - Alfentanil has a rapid onset of action due to its high nonionized fraction.
    - Many lipid-soluble drugs can undergo first-pass uptake by the lungs and then diffuse back to systemic circulation later. Biotransformation primarily depends on hepatic metabolism.
    - Morphine 6-glucuronide is pharmacologically active.
    - Normeperidine (metabolite of meperidine) is associated with seizure activity. The effects are more pronounced in renal failure patients.
    - Remifentanil has a unique ester structure that allows for hydrolysis by nonspecific esterases in the blood. Context-sensitive half time is approximately 3 min, regardless of duration of infusion.
- **Ketamine:** Interacts with multiple receptors, but primarily works by serving as an antagonist at the *N*-methyl-D-aspartate (NDMA) receptors. Unlike barbiturates, ketamine does not depress the reticular activating system (RAS). Ketamine dissociates the thalamus from the cerebral cortex (patients appear conscious but cannot process sensory input). Ketamine is administered IV 1–2 mg/kg, IM 3–5 mg/kg. High lipid solubility and an ↑ in cardiac output (CO) result in faster uptake by the brain. Duration of action is terminated by redistribution. Biotransformation is primarily by liver. Its metabolite, norketamine, possesses anesthetic activity.
- **Etomidate:** Depresses the RAS by enhancing GABA affinity to its receptors. Similar to midazolam, it contains an imidazole ring that gives water solubility and lipid solubility at physiologic pH.
  - IV administration. IV induction: 0.2–0.4 mg/kg. Duration of action terminated by redistribution. Biotransformation is obtained by (1) hepatic metabolism and (2) plasma esterase hydrolysis.
- **Propofol:** Modulates GABA receptors. Contains egg lecithin, which is commonly found in egg yolk. Most people allergic to eggs are sensitive to egg white. IV induction 1–2 mg/kg. Awakening from a single dose is about

3–5 min mostly due to redistribution. Less negative side effects on awakening than barbiturates or etomidate. Has intrinsic anti-emetic effects. Biotransformation is primarily by the liver. Inactive conjugated metabolite is excreted by the kidneys.

NEUROMUSCULAR BLOCKING AGENTS

- The neuromuscular junction (Figure 1-1) contains the motor neuron and the muscle endplate separated by the synaptic cleft.
- Mechanism of muscle contraction: Ach → nicotinic receptors → depolarization → release of Ca^{2+} from sarcoplasmic reticulum (SR) → actin and myosin interaction → muscle contraction.

Depolarizing Neuromuscular Blockers

Succinylcholine: A depolarizing muscle relaxant, similar in structure to ACh, activates the cholinergic receptor but is not hydrolyzed by acetylcholinesterase. Prolonged activation of the receptor inhibits repolarization and the action potential disappears. Phase I block results in muscle relaxation.

- Rapid onset of action (within 30–60 sec) and short duration of action (< 10 min). Most is metabolized by pseudocholinesterase, and only a fraction of the drug reaches the effect site. Blockade terminated by diffusion away from site.
- Duration of action is prolonged by:
  - Conditions that result in lower serum enzyme levels such as pregnancy and liver or renal failure.
  - Abnormal enzymes as seen in atypical plasma cholinesterase or pseudocholinesterase deficiency.
  - Neostigmine, which inhibits pseudocholinesterase.
  - Hypothermia.

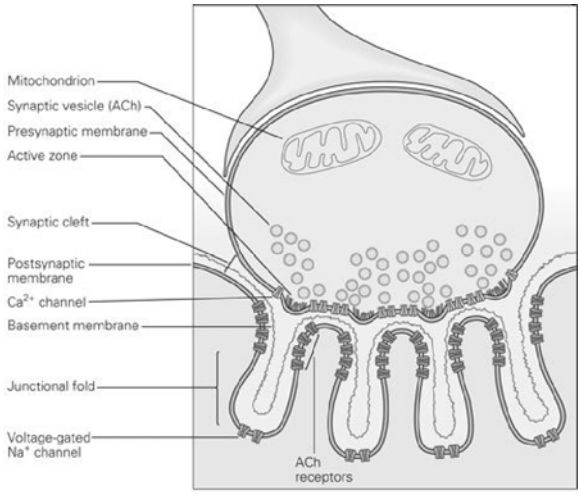

FIGURE 1-1. Adult neuromuscular junction.

(Reproduced, with permission, from Kandel ER, Schwartz JH, Jessell TM. *Principles of Neural Science.* New York: McGraw-Hill, 2000: 188.)

Dibucaine number reflects quality, not quantity, of plasma cholinesterase.

- Administration: 1–1.5 mg/kg. Higher dose is required in pediatric patients due to ↑ extracellular space.
- Atypical plasma cholinesterase:
  - Heterozygote (one abnormal gene): 20–30 min of relaxation. Dibucaine inhibits 40–60% atypical plasma cholinesterase.
  - Homozygote (two abnormal genes): Relaxation > 6 hr. Dibucaine inhibits < 20% of atypical plasma cholinesterase.
- Cardiovascular: Succinylcholine activates all ACh receptors of the autonomic system, and its activity on the muscarinic receptors on the SA node can cause bradycardia (more commonly seen in children and upon administration of second dose).
- Hyperkalemia: Normal muscle contraction releases potassium, and serum level is ↑ by 0.5 mEq/L. This effect is significantly pronounced in trauma, burn injury, and denervation injury due to a proliferation of extrajunctional receptors. In severe cases, hyperkalemia can cause refractory cardiac arrest. Risk period: burn injury: 6–8 hr after the injury until last eschar is healed; denervation injury: peaks after 7–10 days and duration varies. **Hyperkalemia is not reliably blocked by pretreatment.**
- Fasciculations: Stimulation of presynaptic ACh receptors results in generalized motor unit contractions. **This can be prevented by a small dose of nondepolarizing neuromuscular blocking agent.**
- Muscle pain: Highest risk in healthy females having outpatient surgeries. **Not reliably blocked by pretreatment.**
- ↑ intragastric pressure: Thought to be caused by muscle fasciculation. However, this effect is offset by an ↑ in lower esophageal sphincter tone. **Pretreament abolishes this ↑ in pressure.**
- Intraocular pressure ↑: Thought to be caused by extraocular muscle contractions. Should be avoided in patients with open globe injury. **Not reliably blocked by pretreatment.**
- Malignant hyperthermia: Known trigger in susceptible patients. May present with paradoxical contraction of jaw muscles.
- ↑ intracranial pressure (ICP): Generalized muscle fasciculations stimulate muscle stretch receptors along with rise in partial pressure of carbon dioxide (pCO_2), which ↑ cerebral activity, cerebral blood flow (CBF), and ICP. **This can be prevented by pretreatment with nondepolarizing neuromuscular blocker.**

Nondepolarizing Neuromuscular Blocking Agents (Table 1-9)

- Two main categories:
  - **Steroidal:** Tend to be vagolytic (pancuronium).
  - **Benzylisoquinolines:** Tend to release histamine (tubocuraine, metocurine, and atracurium) and may cause hypotension, tachycardia, flushing, bronchospasm.
- Prolonged in liver failure: Pancuronium, rocuronium, and vecuronium.
- Prolonged in renal failure: Metocurine, gallamine, pancuronium, and vecuronium. (Rocuronium, atracurium, and mivacurium are independent of renal function.)
- **ICU neuropathy** is associated with pancuronium and vecuronium. Long-term use in intensive care unit (ICU) patients is associated with prolonged neuromuscular blockade possibly from accumulation of active metabolites, changing drug clearance, and the development of polyneuropathy. A higher incidence is seen with sepsis, renal failure, and the use of long-term

or high-dose corticosteroids. Prolonged lack of ACh binding at postsynaptic ACh receptors can also mimic a chronic denervation state, leading to receptor dysfunction and prolonged paralysis. Patients present with muscle weakness and an inability to wean from the ventilator **without any sensory deficits.** Motor axonal damage and myopathy are seen.

- Generally, peripheral paralyzing diseases demonstrate hypersensitivity to nondepolarizing neuromuscular blockers. These include amyotrophic lateral sclerosis, familial periodic paralysis, Guillain-Barré syndrome, muscular dystrophy, and myasthenia gravis.

TABLE 1-9. Nondepolarizing Neuromuscular Blockers

	Intubation Dose	Metabolism	Clearance	Key Note
Tubocurarine	0.5 mg/kg	Not significantly metabolized.	Mostly excreted by the kidneys: Prolonged in renal failure.	Associated with significant histamine release.
Metocurine	0.3 mg/kg	Not significantly metabolized.	Mostly excreted by the kidneys.	Histamine release is half as much as tubocurarine.
Atracurium	0.5 mg/kg	Two-thirds by nonspecific esterases; one-third by Hoffman elimination.	Laudanosine, metabolite of Hoffman degradation, is associated with CNS stimulation and precipitation of seizures.	Associated with significant histamine release.
Cisatracurium	0.1 mg/kg	Mostly degraded via Hoffman elimination.	Same as atracurium.	No association with histamine release.
Mivacurium	0.2 mg/kg	Only nondepolarizing neuromuscular blocker that is metabolized by pseudocholinesterase.	Renal excretion.	Associated with similar histamine release as atracurium.
Vecuronium	0.1 mg/kg	Limited metabolism by the liver.	75% biliary excretion; 25% renal excretion.	Liver metabolite has 60% of parent drug action.
Rocuronium	0.5 mg/kg	Undergoes no metabolism.	Primarily biliary excretion with minimal renal excretion.	Longer duration of action is seen in female and elderly. Shorter in pediatric population.
Pancuronium	0.1 mg/kg	Some degree of metabolism by the liver.	Excretion is primarily renal.	Hypotension and tachycardia are caused by vagal blockade.

CHOLINESTERASE INHIBITORS

- Cholinesterase inhibitors are **competitive antagonists** of acetylcholinesterase, while organophosphates are **noncompetitive inhibitors** (irreversible bonds). Also, neostigmine may have some direct agonist effect at the nicotinic receptors, and presynaptically blocks a feedback mechanism, resulting in an ↑ mobilization of acetylcholine. See Table 1-10 for characteristics of cholinesterase inhibitors.
- General side effects: Vagal stimulation causes bradycardia, which can progress to sinus arrest. Therefore, the cholinesterase inhibitors are usually given with anticholinergic drugs. Muscurinic activation can cause bronchospasm, peristaltic activity, ↑ salivation, nausea, and vomiting.

AUTONOMIC MODULATORS

The autonomic nervous system is comprised of the sympathetic nervous system and the parasympathetic nervous system (Figure 1-2):

- **Sympathetic nervous system:** Arises from T1–L2, L3. Short myelinated preganglionic fibers synapse at the respective ganglions, which send unmyelinated fibers to the effector organs. Preganglionic fibers release ACh at the synapse, while postganglionic fibers release epinephrine (epi) and norepinephrine (NE).
  - Synthesis of NE: (1) In the liver: phenylalanine → tyrosine; (2) in the presynaptic cytoplasm: tyrosine → dopa → dopamine; (3) in the vesicles: dopamine → NE → epi.
  - Metabolism depends on reuptake, degradation by monoamine oxidase or catechol-*O*-methyltransferase (COMT), and diffusion into the circulation.
  - **Activating receptors:**
    - α_1: Activation ↑ intracellular calcium which results in smooth muscle contraction: bronchoconstriction, vasoconstriction, uterine contraction, and sphincter contraction. Mild ↑ in cardiac contractility and predisposes myocardium to ventricular arrhythmia. ↑ secretion of insulin and lypolysis.
    - α_2: Mostly located at the presynaptic nerve terminal and works via negative feedback mechanism resulting in ↓ exocytosis of neurotransmitters. Activation causes smooth muscle relaxation and vasodilation. CNS: sedation.
    - β_1: Most important; located on postsynaptic cardiac membranes. Activation via stimulation of adenylate cylase has positive chronotropic

TABLE 1-10. Cholinesterase Inhibitor Characteristics

Neostigmine	Does not cross blood-brain barrier (BBB). Onset = 5–10 min. Duration = around 1 hr. Prolongation of action with liver and kidney failure. Can cross placenta and cause fetal bradycardia.
Pyridostigmine	One-fifth as potent as neostigmine. Slightly longer onset and duration of action than neostigmine.
Edrophonium	One-tenth as potent as neostigmine. More rapid onset and shorter duration of action than neostigmine. Recommended to be given with atropine due to its rapid onset.
Physostigmine	Crosses BBB. Effective in treatment of central anticholinergic syndrome or delirium caused by benzodiazepines. Completely metabolized by plasma esterases.

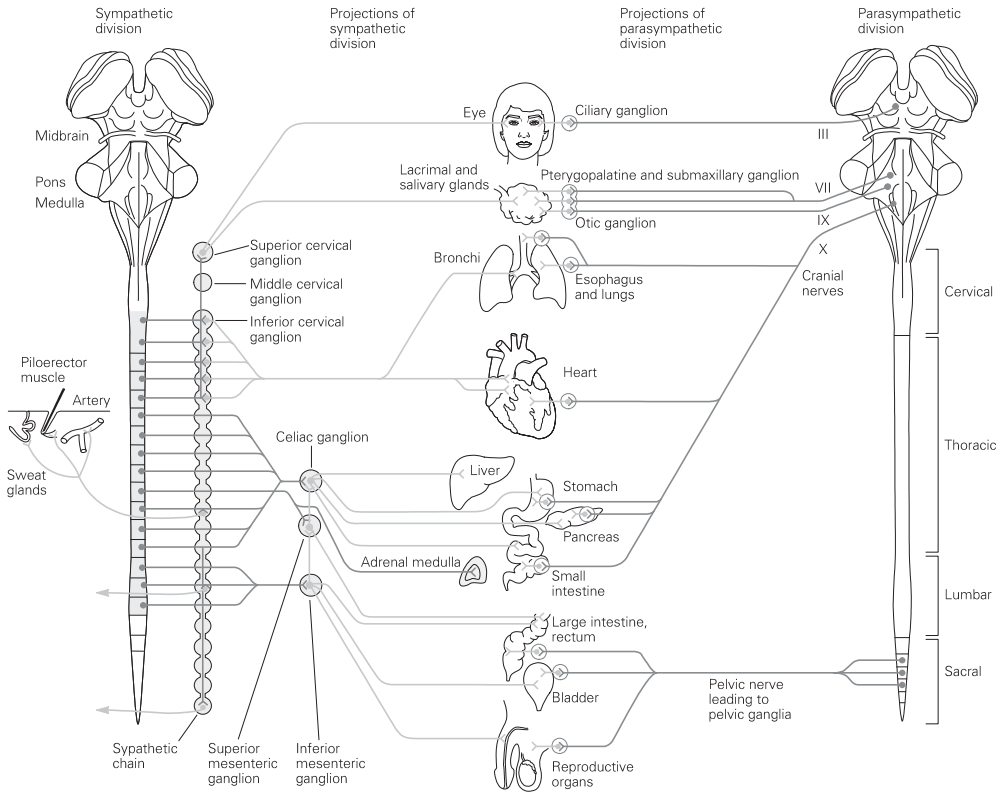

FIGURE 1-2. Autonomic nervous system.

The distribution of the sympathetic system is thoracoabdominal (T1–L3), while the parasympathetic system is craniosacral. (Reproduced, with permission, from Kandel ER, Schwartz JH, Jessell TM. *Principles of Neural Science*. New York: McGraw-Hill, 2000: 964.)

(↑ HR), positive inotropic (↑ contractility), and positive dromotropic (↑ conduction) effects.
    - β_2: Primary postsynaptic adrenoceptors located on smooth muscle and gland cells. Activation relaxes smooth muscle, resulting in bronchodilation, vasodilation, uterine relaxation (tocolysis), and relaxation of bladder and gut. Also can cause glycogenolysis, lipolysis, gluconeogenesis, and insulin release.
  - **Endogenous catecholamines** (see Table 1-11): Epinephrine, norepinephrine, dopamine.
  - **Synthetic adrenergic agonists** (see Table 1-12): Ephedrine, isoproterenol, dobutamine, phenylephrine, methyldopa, clonidine, dexmedetomidine.
- **Parasympathetic nervous system:** Arises from the brain stem and sacral plexus. Vagus nerve is responsible for 75% of all parasympathetic activity. Preganglionic fibers synapse with cells located near the effector organ, which sends postganglionic fibers to the effector organs (heart, lungs, gastrointestinal organs, liver, and genitourinary system). ACh released.

TABLE 1-11. Endogenous Catecholamines

	Activating Receptors	Clinical Effects
Epinephrine	α_1, β_1 and β_2	Inotropic, ↑ cardiac output (CO) with ↑ myocardial O_2 demand. β_2 stimulates ↓ in systemic vascular resistance. ↑ cerebral and coronary perfusion. ↓ splanchnic and renal perfusion.
Norepinephrine	α_1 and β_1	Inotropic with ↑ contractility. At higher doses vasoconstriction >> β_1.
Dopamine	< 2 μg/kg/min: dopaminergic effects	↑ urine output by (1) renomesenteric dilation, (2) ↓ level of aldosterone, (3) ↑ CO.
	2–10 μg/kg/min: β-adrenergic effects	Cardiac contractility is ↑. Coronary O_2 demand usually higher than supply.
	> 10 μg/kg/min: α-adrenergic effects	Vasoconstriction, bronchoconstriction, uterine constriction. Predisposes myocardium to ventricular arrhythmia.

TABLE 1-12. Synthetic Adrenergic Agonists

	Activating Receptors	Clinical Effects
Ephedrine	Weak epinephrine	Longer duration of action. ↑ cardiac output (CO), heart rate (HR), and blood pressure (BP). Indirectly releases norepinephrine (NE) at the receptors.
Isoproterenol	β_1 and β_2	↑ HR and contractility. β_2-mediated ↓ in SVR. O_2 demand ↑, while supply falls due to ↓ in diastolic BP.
Dobutamine	β_1	Primarily ↑ CO and contractility.
Phenylephrine	α_1	Primary action is peripheral veno- and vasoconstriction. BP and afterload ↑, while reflex bradycardia could result in ↓ in CO.
Methyldopa	α_2	Enters the synthesis pathway of NE and epinephrine (Epi) as mentioned above. Produces false neurotransmitters: methylepinephrine and methynorepinephrine. These false neurotransmitters activate alpha-2 receptor which causes ↓ sympathetic nervous system stimulation (vasodilation and lowered BP).
Clonidine	α_2	Direct α_2 agonist, which results in lowered BP, HR, and vasodilation. Sedating properties are effective for lowering anesthetic requirements intraoperatively. Used as an adjunct in regional anesthesia to ↑ the quality and duration of nerve blocks.
Dexmedetomidine	α_2	More potent α_2 agonist than clonidine. Possesses sympatholytic properties as mentioned above. Also possesses sedating and analgesic properties. Known to lower anesthetic requirement. Initial hypertension is soon followed by hypotension and bradycardia during the ongoing therapy.

DIRECT VASODILATORS

- **Sodium nitroprusside:** Potent vaso- and venodilator; metabolized to nitric oxide.
  - Results in ↓ afterload, preload, and BP. CO and renal blood flow are well maintained. ↓ cardiac oxygen demand due to ↓ afterload and preload could be beneficial in ischemic cardiac disease. Reflex tachycardia might be seen.
  - Cerebral vasodilation results in ↑ CBF and ICP.
  - Pulmonary vessels are also dilated and pulmonary arterial pressures are lowered. Hypoxic pulmonary vasoconstriction is compromised, which can ↑ the shunt fraction.
  - Metabolism: Sodium nitroprusside receives an electron from hemoglobin, which results in the formation of methemoglobin and a nitroprusside free radical, which in turn produces cyanide.
  - Formed cyanide can (1) bind with methemoglobin to form cyanmethemoglobin, (2) bind with thiosulfate to form thiocyanate, (3) bind with cytochrome oxidase to interfere with Kreb cycle and oxygen utilization.
  - Cyanide toxicity results from an inability to utilize oxygen and is characterized by arrhythmia, metabolic acidosis, and ↑ mixed venous oxygen content. Treatment consists of:
    - Mechanical ventilation with 100% O_2.
    - Sodium nitrate, which produces methemoglobin to bind cyanide.
    - Sodium thiosulfate to bind extra cyanide.
- **Nitroglycerin:** Similar mechanism of action to sodium nitroprusside in that the drug is metabolized to nitric oxide. However, it is mostly a venodilator, which can cause some reduction in afterload.
  - ↓ in preload leads to lowered left ventricular end-diastolic pressure and myocardial oxygen demand. It can reverse coronary vasospasm and redistribute coronary blood supply to ischemic areas. It is also known to possess some antiplatelet effects.
  - Highly effective for ischemic cardiac disease or relieving cardiogenic pulmonary edema.
  - Similar to sodium nitroprusside, pulmonary and cerebral vasculature are dilated and CBF and ICP are ↑.
  - Metabolism: The metabolite nitrate is formed, which receives an electron from hemoglobin to form methemoglobin. Severe methemoglobinemia can be treated with methylene blue (1–2 mg/kg).
- **Hydralazine:** Dilates resistant arterioles.
  - Significant reduction in afterload, ↑ HR, contractility, and CO are seen. Must be cautious in patients with preexisting coronary artery disease, but can be beneficial in patients with congestive heart failure.
  - Cerebral vasodilation results in ↑ CBF and ICP.

Nitroglycerin is a venodilator, while nitroprusside is both a vaso- and venodilator.

ANTICHOLINERGICS

- Anticholinergics competitively block ACh and its action on the receptor.
- At clinical doses, only the muscarinic receptors are blocked.
- See Table 1-13 for effects of anticholinergics on organ systems.
- **Atropine:** A tertiary amine that crosses the blood-brain barrier (BBB) and placenta. Usual dose is 0.5 mg; maximum up to 2 mg. Potent treatment of bradyarrhythmias and bronchial smooth muscle relaxation. CNS stimulation may result at toxic doses (hallucination, irritability, and nervousness).

TABLE 1-13. Effects of Anticholinergics on Organ Systems

Cardiovascular	Action on sinoatrial node results in tachycardia by blocking vagal stimulation. Shortened PR interval is observed via ↑ AV nodal conduction.
Pulmonary	↓ airway secretion and relaxes bronchial smooth muscle.
Cerebral	May cause excitation (restless, hallucination) or depression (sedation and amnesia).
Gastrointestinal	↓ salivation, intestinal motility, and peristalsis, which results in prolonged gastric emptying time.
Genitourinary	Urinary retention may result by relaxation of smooth muscle.
Secretory glands	↑ body temperature by inhibition of sweat glands.

Glycopyrrolate is the only anticholinergic that does not cross the BBB.

However, CNS effects are minimal at the usual clinical doses. Known to cause fever. Caution in patients with coronary artery disease, narrow-angle glaucoma, and bladder neck obstruction.

- **Scopolamine:** Unlike atropine, scopolamine can cause CNS depression (sedation and drowsiness). Commonly used for premedication for sedation and amnesia without cardiovascular effects. Also effective for motion sickness.
- **Glycopyrrolate:** Usual dose: 0.2–0.3 mg. Quaternary structure prevents this drug from crossing the BBB. Potent inhibitor of salivation and respiratory secretion. Clinical profile is otherwise similar to atropine.

LOCAL ANESTHETICS

- Nerve cells have resting membrane potential of –70mV, which is maintained by an unequal exchange of sodium and potassium. With impulse propagation, depolarization results in sodium channels opening and an influx of sodium ions.
- Repolarization is achieved by outflow of potassium ions, and resting potential restored.
- Local anesthetics are thought to bind to inactivated sodium channels. Subsequently, further depolarization is prevented and impulse propagation is disrupted.
- Local anesthetics have a lipophilic aromatic ring linked to a hydrophilic tertiary amine by an amide or ester linkage.
- **Potency:** More lipophilic (more alkyl groups or halides on benzene ring), ester bond, more protein binding (specifically the sodium channel).
- **Duration of action:** Associated with binding to α_1 acid glycoprotein. Higher affinity = more clearance.
- **Effect of pH:** Due to the amine group, local anesthetics tend to be basic. If injected into an acidic environment, the drug will become ionized and demonstrate poor penetration to nerve cells along with a slower onset of action. Commonly, sodium bicarbonate is added for neutralization to improve onset, quality, and duration of the nerve block.
- **Systemic absorption:** Depends on the vascularity of the site: intravenous > tracheal > intercostals. Then, CPEBS (caudal > paraspinal > epidural > brachial plexus > sciatic).
- There are two categories of local anesthetics:
  - **Esters** (maximum dose): Chloroprocaine (12 mg/kg), procaine (12 mg/kg), cocaine (3 mg/kg), tetracaine (3 mg/kg). Metabolized by plasma

cholinesterase. Prolonged effects are seen in patients with genetically abnormal enzymes or liver disease.

- **Amides** (maximum dose), in order of duration: Prilocaine (8 mg/kg), lidocaine (4.5 mg/kg, with Epi 7 mg/kg), mepivacaine (4.5 mg/kg, with Epi 7 mg/kg), ropivacaine (3 mg/kg), bupivacaine (3 mg/kg). Bupivacaine has intrinsic vasoconstrictive effects, while lidocaine has intrinsic vasodilatory effects.

- Complications:
  - Local toxicity
    - Cardiovascular: ↓ myocardial contractility (hypotension), ↓ cardiac conduction (bradycardia and heart block) could lead to cardiac arrest. Due to strong protein binding, IV injection of bupivacaine has resulted in severe cardiotoxic events. Ropivacaine has shown to be less cardiotoxic.
    - Lidocaine has been shown to depress hypoxic drive and cause apnea in extreme situations.
  - CNS: Commonly eyes (blurred vision), ears (tinnitus), and mouth (tongue paresthesia) are affected. Also, CNS excitatory symptoms include restlessness, nervousness, and agitation, which could lead to tonic-clonic seizure (treated with benzodiazepine or barbiturates).
  - Management:
    - Supportive care: Airway, breathing, and circulation.
    - Lipid emulsion (Intralipid) has been shown to reverse local anesthetic toxicity by binding and extracting the lipophilic local anesthetic from the tissue and preventing local anesthetic inhibition of myocardial function.
  - **Cauda equina syndrome:** Permanent neurologic damage seen after continuous intrathecal infusion of local anesthetics.
  - **Transient neurologic symptoms:** Pain in lower extremities including the buttocks. Patient also complains of shooting pain down the leg, while motor is intact. Obese outpatients in lithotomy positions are at higher risk. Symptoms last over 1 week and can be treated with nonsteroidal anti-inflammatory drugs (NSAIDs).

Systemic absorption of local anesthetics:

IV > tracheal > intercostal > caudal > paraspinal > epidural > brachial plexus > sciatic

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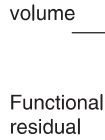
CHAPTER 2

Physiology and Anesthesia

- **Pulmonary function testing:** Lung volumes and capacities (see Figure 2-1).
  - **Functional residual capacity (FRC)** is the lung volume at the end of normal exhalation.
  - FRC = expiratory reserve volume (ERV) + residual volume (RV).
  - Factors that decrease FRC include pregnancy, obesity, supine position, and general anesthesia.
  - **Closing capacity (CC)** is the volume at which small airways in the lungs begin to close.
  - Factors that increase CC include:
    - Age
    - Bronchitis
    - Left ventricular failure
    - Surgery
    - Smoking
    - Obesity
  - If CC is greater than FRC, shunting will occur during tidal breathing.

The inward recoil of the lungs = the outward recoil of the chest at FRC.

- **Pulmonary function tests:**
  - Forced vital capacity (FVC) is the volume of gas that can be expired as forcefully and rapidly as possible after maximal inspiration.
  - The ratio of the forced expiratory volume in 1 sec (FEV_1) to the FVC is proportional to the degree of airway obstruction.
  - Normally, FEV_1/FVC is > 80%.
  - Both FEV_1 and FVC are effort dependent, while forced midexpiratory flow (FEF25–75%) is effort independent and is a more reliable measurement of obstruction.
  - Carbon monoxide diffusing capacity (DL_{CO}) collectively measures all the factors that affect the diffusion of gas across the alveolar-capillary membrane.
  - DL_{CO} is ↓ with the loss of parenchyma (eg, in emphysema) or alveolar fibrosis (eg, in sarcoidosis, asbestosis, berylliosis, oxygen toxicity, and pulmonary edema).



Diffusion capacity is affected by not only the permeability of the alveolar-capillary membrane but also the pulmonary blood flow.

FIGURE 2-1. Spirogram.

(Reproduced, with permission, from Lumb A, Nunn JF. *Applied Respiratory Physiology.* Butterworth-Heinemann, 2000.)

- **Restrictive disease** is a proportional ↓ in all lung volumes; FVC, FEV_1, FRC, and total lung capacity (TLC) are all reduced, but FEV_1/FVC and FEF25–75% remain normal.
- **Obstructive disease** is associated with small airway obstruction to expiratory flow; FEV_1/FVC and FEF25–75% are reduced, FVC, FEV_1, FRC, and TLC are normal or slightly ↑.

Decreased TLC and RV is the hallmark of restrictive lung disease.

- **Lung mechanics:**
  - The chest has a tendency to expand outward, while the lung has a tendency to collapse. At FRC, the outward and inward forces on the lung are equal.
  - **Laplace's law:**

$$\frac{\text{Pressure} = 2 \times \text{Surface tension}}{\text{Radius}}$$

Surfactant synthesis begins at approximately 34 weeks' gestation by ***type II pneumocytes****. Steroid administration can increase the number of cells and therefore surfactant production. The ability of surfactant to lower surface tension is directly proportional to its concentration.*

  - Alveolar collapse is proportional to surface tension but inversely proportional to alveolar size.
  - **Surfactant** lowers surface tension, especially on smaller alveoli. Therefore, it keeps smaller alveoli from collapsing.
- **Compliance:**
  - Lung compliance (CL) = $\dfrac{\text{Change in lung volume}}{\text{Change in transpulmonary pressure}}$
  - Chest wall compliance (CW) = $\dfrac{\text{Change in chest volume}}{\text{Change in transthoracic pressure}}$
  - $\dfrac{1}{\text{Total compliance } (C_{total})} = \dfrac{1}{CW} + \dfrac{1}{CL}$
  - Normal CW and CL are 200 mL/cm H_2O each, and C_{total} is 100 mL/cm H_2O.
- **Control of ventilation:**
  - Central response:
    - Chemosensitive areas are located in medulla, near the origin of cranial nerves IX and X.
    - CO_2 passes through the blood-brain barrier, H^+ is formed in the CSF, and CSF acidosis occurs.
    - ↑ **$Paco_2$** is a very potent stimulus for ventilation, increasing both tidal volume and respiratory rate.
  - Peripheral response:
    - **Carotid bodies** are located at the bifurcation of the common carotid arteries, and they impact predominantly ventilation.
    - **Aortic bodies** are located in the aortic arch, and they impact primarily in circulatory changes.
    - Both respond to **Pao_2** levels < 60 mmHg.
    - They do not respond to low oxygen content or low oxygen saturation.

Central chemoreceptors are extremely sensitive to hydrogen ions, or $Paco_2$ while peripheral chemoreceptors are sensitive to Pao_2.

- **Ventilation:**
  - Ventilation is described by $PaCO_2$.
  - $PaCO_2 = \dfrac{VCO_2}{VE - VD}$

$VCO_2 = CO_2$ production
VE = total minute ventilation
VD = dead space ventilation

Expired $Paco_2$ ($Peco_2$) is the average $Paco_2$ in an expired gas sample and NOT the same as end-tidal $Paco_2$ ($Etco_2$).

Hypoxemia can be caused by:

1. *Alveolar hypoventilation*
2. *Diffusion impairment*
3. *Ventilation-perfusion mismatch*
4. *Right-to-left shunt*

***Bohr effect:** Oxy-Hb dissociation curve shift with changes in carbon dioxide.*

***Haldane effect:** Oxygenation of hemoglobin lowers the affinity for carbon dioxide.*

- **VE** = Tidal volume (VT) × Respiratory rate (RR)
- Alveolar ventilation (VA) = (VT – VD) × RR
- **Physiologic dead space (VD)** = anatomic dead space (VD(an)) + alveolar dead space (VD(al))
- Normal **dead space to tidal volume fraction** (VD/VT) is 0.3.
- $\frac{VD}{VT} = \frac{PaCO_2 - PeCO_2}{PaCO_2}$
- $PeCO_2$ = mixed expired carbon dioxide tension.
- Causes of increased alveolar dead space include:
  - Pulmonary embolism.
  - Pulmonary vascular disease.
  - Vasculitis.
  - Chronic obstructive pulmonary disease (COPD).
  - Acute respiratory distress syndrome (ARDS).
  - Pulmonary fibrosis.
  - Shock.

- **Oxygenation:**
  - Arterial oxygen content ($CaCO_2$) = (1.34 × Hb × SaO_2) + (0.003 × PaO_2).
  - Most efficient way to increase $CaCO_2$ is to increase hemoglobin.
  - Oxygen delivery (DO_2) = CI × $CaCO_2$ (mL/min/m²).
  - **Mixed venous SaO_2 (SvO_2)** = $\frac{VO_2}{CO \times Hb \times 13}$
  - Normal SvO_2 = 65–75%; PvO_2 = 35–45 mmHg.
  - P50 is the partial pressure of oxygen at which hemoglobin is 50% saturated (see Figure 2-2).
  - Normal P50 in adults is about 27 mmHg and in infants about 19 mmHg.
  - A **right shift** in this curve results in increased unloading of oxygen at the tissue level resulting in increased temperature, ↑ 2,3 DPG, acidosis (↓ pH), pregnancy, ↑ pCO_2, adult hemoglobin.

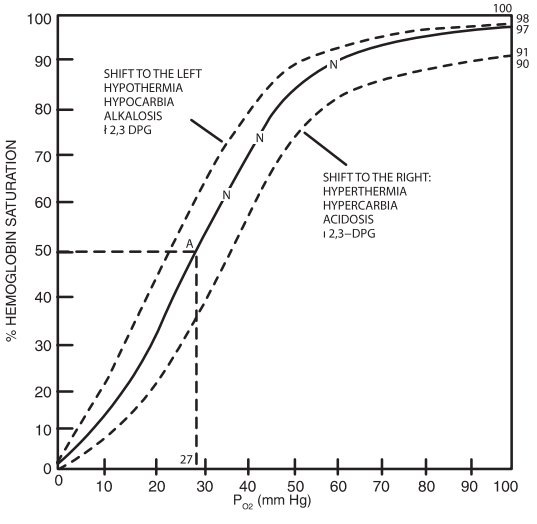


FIGURE 2-2. Oxygen-hemoglobin dissociation curve.

Oxyhemoglobin dissociation curve. N = normal curve; A = hemoglobin 50% saturated with O_2.

- A **left shift** in this curve results in decreased unloading of oxygen at the tissue level, ↓ temperature, ↓ 2,3 DPG, alkalosis (↑ pH), ↓ pCO_2, fetal hemoglobin.

- **Ventilation-perfusion (V/Q) ratio:**
  - Distribution of ventilation (Figure 2-3):
    - Right lung receives more ventilation than the left one (53% vs. 47%).
    - Ventilation per unit lung volume is smallest at the highest portion.
  - Distribution of pulmonary blood flow:
    - Pulmonary perfusion is approximately 5 L/min.
    - Two major determinants of distribution are gravity and hypoxic pulmonary vasoconstriction.
  - Normal V/Q is 1. If there is ventilation but no perfusion, the V/Q ratio is infinity and this is dead space. If the V/Q ratio is zero, perfusion but no ventilation, this is defined as absolute shunt.
  - Shunt fraction (QS/QT) = $\frac{Cc\text{O}_2 - Ca\text{O}_2}{Cc\text{O}_2 - C\bar{v}\text{O}_2}$

    $Cc\text{O}_2$ = content pulmonary capillary blood
    $Ca\text{O}_2$ = content arterial blood
    $C\bar{v}\text{O}_2$ = content mixed venous blood
  - Normal QS/QT = 0.1
- **Alveolar-arterial gradient:**
  - $PA\text{O}_2 = (PB - P\text{H}_2\text{O}) \times Fi\text{O}_2 - (Pa\text{CO}_2/0.8)$
  - A-a gradient = $PA\text{O}_2 - Pa\text{O}_2$ normal 10–20 mmHg or ¼ age
    - Normal $Pa\text{O}_2$ decreases with age and can be approximate by **102 – (age/3)**
    - Another approximation: $\mathbf{Pa\text{O}_2 = 5 \times Fi\text{O}_2}$
  - **Normal A-a gradient:** Hypoventilation and low $Fi\text{O}_2$

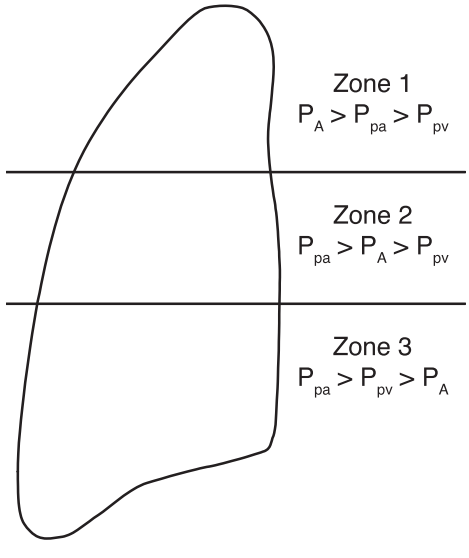


FIGURE 2-3. Zones of the lung.

$PA\text{O}_2$ is dependent on barometric pressure and is therefore influenced by altitude.

- **Increased A-a gradient:** V/Q mismatch, diffusion impairment and right-to-left (intracardiac) shunt.

- **Effects of anesthesia on respiratory function:**
  - General anesthesia increases VD and VD/VT partially due to moderate pulmonary hypotension, loss of skeletal muscle tone, or loss of bronchoconstrictor tone.
  - General anesthesia also increases shunt as the result of atelectasis and airway collapse in dependent areas of the lung.
  - When patients are mechanically ventilated, best perfusion is in the dependent lung and best ventilation is in the nondependent lung, creating V/Q mismatch.
  - Volatile anesthetics and potent vasodilators inhibit hypoxic pulmonary vasoconstriction, a protective mechanism for diverting blood flow to ventilated areas of the lung.

CARDIAC PHYSIOLOGY

Hemodynamics

Components of the Vasculature

CO – (MAP – CVP)/SVR

SVR = 80 × (MAP – CVP)/CO

PVR = 80 × (PAP – LAP)/CO

- **Arteries:** Thick walled with elastic tissue; under high pressure.
- **Arterioles:** Site of highest resistance. α_1-adrenergic receptors (splanchnic circulation) and β_2 adrenergic receptors (skeletal muscle) that are innervated by the autonomic nervous system (ANS). Resistance is regulated by the ANS.
- **Capillaries:** Largest cross-sectional area where nutrient, gas, and water exchange occur.
- **Venules, veins:** Low-pressure vessels innervated by the ANS. Blood flow is analogous to Ohm's law for electrical circuits: **I = V/R** (current I is analogous to flow, voltage is analogous to pressure).

$$Q = P/R$$

Coronary perfusion pressure (CPP) is determined by the difference between aortic diastolic pressure (AoDP) and end-diastolic ventricular pressure (LVEDP).

Coronary Blood Flow

- Myocardial wall tension during systole can completely stop coronary blood flow to the left heart. The left ventricle is perfused during diastole.
- The right ventricle has lower intramural pressure and is perfused during systole and diastole.
- Sixty to seventy percent of O_2 is extracted by the heart.
- **Coronary reserve** is the ability of coronary flow to increase circulation over baseline state.
- Endogenous regulators of cerebral blood flow (CBF) are adenosine, nitric oxide, and adrenergic stimulation.
- CBF becomes exhausted when 90% of flow is blocked by stenosis of a coronary artery.

The endocardium is most vulnerable to ischemia since it is subjected to the highest pressure during systole.

Resistance

- Factors that change resistance of blood vessels: $\mathbf{R = 8\eta l/r4}$
- Resistance is directly proportional to the viscosity of blood. ↑ hematocrit will ↑ viscosity.

- Resistance is inversely proportional to the fourth power of the radius of the vessel (↑ resistance ↓ radius)
- Resistance is directly proportional to the length of the vessel.

Compliance

- **C = V/P,** where compliance = volume / pressure.
- Inversely related to elastance and describes the distensibility of blood vessels.
- Much greater for veins than for arteries, which explains why more blood volume is in the veins than the arteries.
- As a person ages, arteries become stiffer and less distensible, leading to a ↓ compliance.

Pressure

- Pressure is highest in the aorta and lowest in the vena cava.
- The largest pressure drop in the systemic circulation is seen in the arterioles because they are the site of highest resistance (**Q = P/R**).
- **Arterial pressure:**
  - Systolic pressure: Measured after the heart contracts.
  - Diastolic pressure: Measured when the heart is relaxed and blood is returning to the heart via the veins.
  - Pulse pressure (PP): Difference between systolic and diastolic pressure. A ↓ in compliance will lead to an ↑ pulse pressure.
  - Mean arterial pressure = DBP + 1/3 PP.
  - Venous pressure: Veins have high compliance and can hold a large volume of blood at low pressure.
- **Atrial pressure:** LAP is estimated by pulmonary capillary wedge pressure (PCWP) (see Chapter 5).

MAP = (Systolic BP + 2 Diastolic BP) / 3.

Patients with reduced ventricular compliance are most affected by loss of atrial systole ("atrial kick").

The Cardiac Cycle (Figure 2-4)

- **Atrial systole (phase 1):**
  - The P wave precedes atrial contraction and represents electrical activation of the atria.
  - Contributes to 25% of ventricular filling.
  - The ↑ in atrial pressure caused by atrial systole is the a wave on the venous pulse curve.
- **Isovolumetric ventricular contraction (phase 2):**
  - Begins after onset of QRS wave, which represents electrical activation of the ventricles.
  - When ventricular pressure becomes greater than atrial pressure, the AV valves close. Closure corresponds to the first heart sound (HS). The first HS may be split because the mitral valve closes before the tricuspid valve.
  - The aortic valve is closed during this phase so no blood leaves the ventricle
- **Rapid ventricular ejection (phase 3):**
  - When ventricular pressure becomes greater than aortic pressure, the aortic valve opens and rapid ejection of blood into the aorta occurs.
  - Most of the stroke volume is ejected during this phase, causing the ventricular volume to ↓.

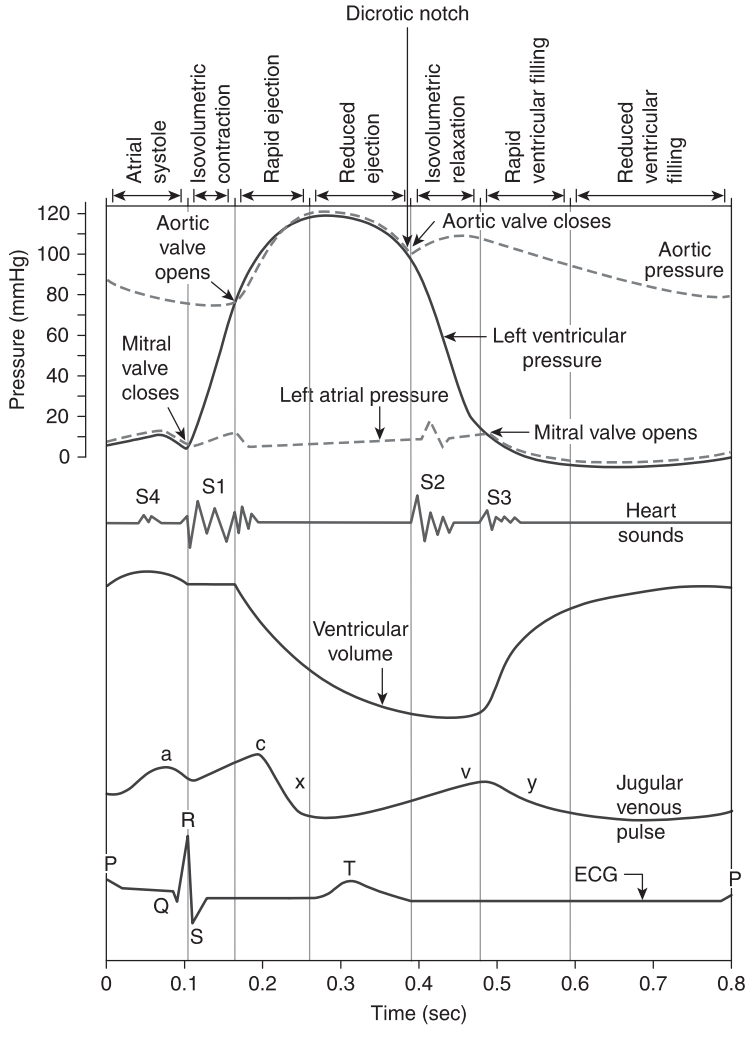


FIGURE 2-4. The cardiac cycle.

(Reproduced, with permission, from Le T et al. *First Aid for the Basic Sciences: Organ Systems.* New York: McGraw-Hill, 2009: 29.)

In contrast to skeletal muscles and neurons, action potential in cardiac muscle is due to opening of both fast sodium channels and slower calcium channels.

- The T wave represents ventricular repolarization and marks the end of ventricular contraction and rapid ejection.

- **Reduced ventricular ejection (phase 4):**
  - Ejection of blood from the ventricles continues at a slower rate.
  - Ventricular and aortic pressure ↓ as the runoff of blood into the smaller arteries occurs.
- **Isovolumetric ventricular relaxation (phase 5):**
  - Repolarization is complete, corresponding to the end of the T wave.
  - The aortic and pulmonic valves close.
  - The dicrotic notch, or incisura, is seen in the aortic pressure tracing after closure of the aortic valve.
  - Ventricular pressure ↓ rapidly because the ventricle is relaxed.
  - All valves are closed, which makes ventricular volume constant in this phase.

- When ventricular pressure becomes less than atrial pressure, the mitral valve opens.
- **Rapid ventricular filling (phase 6):**
  - The ventricular filling from the atrium begins.
  - Rapid passive filling of blood into the ventricles from the atria causes the third HS (normal in peds, abnormal in adults).
- **Reduced ventricular filling (phase 7):**
  - Ventricular filling continues at a slower rate.
  - Longest phase of the cardiac cycle.
  - Dependent on the heart rate (HR).

CO = HR × SV

Cardiac index = CO / body SA

Cardiac Output

Contractility

- Also known as ionotropism.
- The ability of the cardiac muscle to develop a force at a given muscle length.
- Can be estimated by ejection fraction (EF).
- EF = Stroke volume (SV) / End-diastolic volume (EDV).
- EF = 60–70% within normal limits (WNL).
- Positive ionotropes: ↑ HR ↑ the force of contraction in a stepwise fashion with sympathetic stimulation (catecholamines) via β_1 receptors.
- Negative ionotropes: Parasympathetic stimulation via muscarinic receptors.

Major factors that affect stroke volume:

1. *Preload*
2. *Afterload*
3. *Contractility*
4. *Wall motion abnormalities*
5. *Valvular dysfunction*

Preload

- Normally equal to EDV, right atrial pressure (RAP), PCWP, pulmonary artery (PA) diastolic pressure, central venous pressure (CVP).
- When venous return increases, end-diastolic volume increases and stretches the ventricular muscle fibers.
- ↓ preload could be secondary to hypovolemia from hemorrhage or fluid losses, venodilation from general anesthesia or neuraxial anesthesia, tension pneumothorax and pericardial tamponade, which ↓ venous return secondary to ↑ pressure around the heart.

Svo_2 is a good determinant of the adequacy of cardiac output.

$Svo_2 = Sao_2 - (VO_2 / (CO \times Hb \times 1.34)$

Svo_2 - mixed venous saturation

Sao_2 - arterial oxygenation

CO - cardiac output

VO_2 - oxygen consumption

Afterload

- Left ventricular afterload = aortic pressure.
- Right ventricular afterload = pulmonic pressure.
- Frank-Starling forces (Figure 2-5).
  - ↑ in stroke volume or cardiac output in response to an ↑ in venous return or EDV.
  - Changes in contractility shift the curve either upward or downward.

Cardiac output is inversely related to afterload.

Autonomic Effects on Heart

- **Chronotropic effects:**
  - Sympathetic nervous system (SNS) ↑ HR by acting on β_1 receptors via norepinephrine on the sinoatrial (SA) node.
  - Parasympathetic nervous system (PNS) ↓ HR by acting on muscarinic receptors via acetylcholine (ACh) on the SA node.

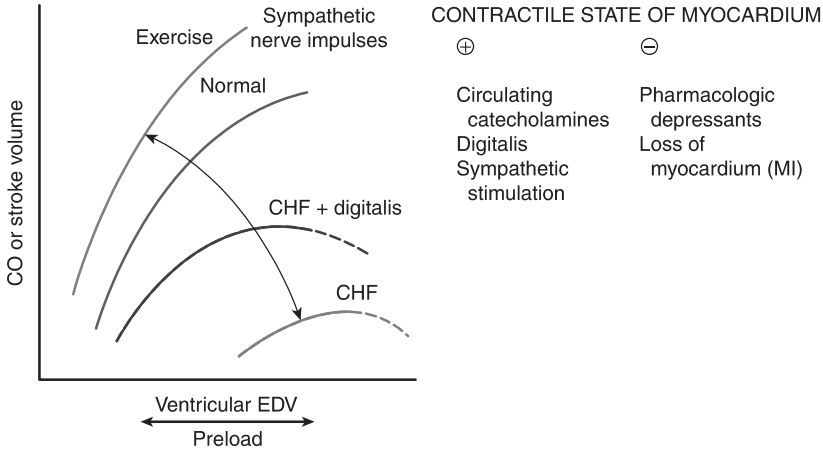


FIGURE 2-5. Frank-Starling curves.

(Reproduced, with permission, from Le T et al. *First Aid for the Basic Sciences: Organ Systems.* New York: McGraw-Hill, 2009: 24.)

The main neurotransmitter of the sympathetic system is norepinephrine, which provides positive chronotropic (heart rate), inotropic (contractility), and lusitropic (relaxation) effects.

The major neurotransmitter of the parasympathetic system is acetylcholine, which has more direct inhibitory effects on the heart, mainly the atria.

The major resting tone of the heart is parasympathetic.

- **Dromotropic effects:**
  - SNS works on β_1 receptors via ACh in atrioventricular (AV) node to ↑ conduction velocity, ↑ the conduction of action potentials from the atria to the ventricles and ↓ the PR interval.
  - PNS works on muscarinic receptors via ACh in the AV node to ↓ conduction velocity of action potentials and ↑ the PR interval.
- **Ionotropic effects:**
  - SNS works on β_1 receptors to ↑ contractility.
  - PNS works on muscarinic receptors to ↓ contractility. The SA node, atria, and AV node have PNS innervation, but the ventricles do not.

Neural Effects on Heart

- **Baroreceptor reflex:** Responsible for minute-to-minute regulation of blood pressure (BP) by a negative feedback mechanism.
- Baroreceptors are stretch receptors located in the wall of the carotid sinus near the bifurcation of the common carotid arteries.
- ↓ in arterial BP ↓ stretch on walls of carotid sinus.
- ↓ stretch ↓ firing rate of glossopharyngeal nerve, which travels to the vasomotor center of the brain stem.
- The vasomotor center reacts by ↓ PNS outflow to the heart and ↑ SNS outflow to heart and vessels, resulting in ↑ HR, contractility, and CO.
- ↑ venoconstriction → ↑ venous return → ↑ in BP.
- Baroreceptors are also located in the aortic arch but respond to ↑ but not ↓ in BP.
- Many anesthetic drugs blunt cardiac reflexes. The SNS response to ↓ BP is reduced.

Hormonal Effects on Heart

- **Renin-angiotensin-aldosterone system:**
  - Long-term BP regulation by adjusting blood volume.
  - Angiotensin-converting enzyme (ACE), renin.

- Angiotensinogen → angiotensin I (inactive) → angiotensin II (active).
  - A ↓ in renal perfusion causes juxtaglomerular (JG) cells of afferent arteriole to secrete renin.
  - ACE converts angiotensin I to angiotensin II in the lungs.
  - Angiotensin II stimulates synthesis and secretion of aldosterone by adrenal cortex. Aldosterone ↑ sodium reabsorption at renal tubule, → ↑ in blood volume.
  - Angiotensin II also → widespread vasoconstriction of arterioles, → ↑ in total peripheral resistance (TPR) and mean arterial pressure (MAP).
- **Vasopressin (ADH):**
  - Atrial receptors respond to a ↓ in blood volume and cause release of ADH from the posterior pituitary.
  - ADH is a vasoconstrictor that ↑ TPR by acting on arterioles.
  - ADH ↑ water reabsorption at the distal tubules and collecting ducts.
- **Atrial natriuretic peptide (ANP):**
  - Released from atria in response to ↑ atrial stretch.
  - Causes dilation of arterioles.
  - Causes excretion of salt and water by kidney, which reduces blood volume and ↓ BP.
  - Inhibits renin secretion.

Hormones affecting the heart can be produced by the cardiomyocytes or delivered to the heart from other tissues.

Cardiac Reflexes

- **Carotid sinus reflex:** Used to treat supraventricular tachycardia (SVT) through vagal stimulation by carotid massage.
- Also, arterial hypoxemia is sensed by chemoreceptors in the carotid sinus, which activated the SNS, causing ↑ HR.
- **Bainbridge reflex:** Atrial stretch causes ↑ HR, leading to ↑ CO.
- **Oculocardiac reflex:** ↓ HR with ocular pressure or stretching of ocular muscles.
- **Abdominal reflex:** ↓ HR with stretch of abdominal viscera.
- **Cushing reflex:** ↑ ICP compresses cerebral blood vessels → cerebral ischemia. → ↑ MAP (SNS activation) with ↓ HR
- Peripheral vasoconstriction causes blood flow to peripheral organs to ↓ in an attempt to preserve blood flow to brain.

Cardiovascular Effects of Opioids

- Opioids are often used in cardiac anesthesia for hemodynamic stability.
- May cause dose-dependent bradycardia possibly secondary to central stimulation of vagal nucleus.
- Morphine and codeine cause a histamine release that can → vasodilation and hypotension.

Cardiovascular Effects of Volatile Agents

See Table 2-1.

TABLE 2-1. Circulatory Effects of Volatile Anesthetics

	Isoflurane/Desflurane	Sevoflurane	Halothane	Nitrous Oxide
Cardiac output	0	0	-*	+
Heart rate	++/0	0	0	+
Blood pressure	--*	--*	-*	0
Stroke volume	-*	-*	-*	-
Contractility	--*	--*	--*	-*
Systemic vascular resistance	--	--	0	0
Pulmonary vascular resistance	0	0	0	+
Coronary blood flow	+	+	0	0
Cerebral blood flow	+	+	++	0
Muscle blood flow	+	+	-	0
Catecholamine levels	0	0	0	0

*, dose dependent; +, increase; ++, large increase; 0, no change; -, decrease; --, large decrease.
(Reproduced, with permission, from Duke J. *Anesthesia Secrets,* 3rd ed. Elsevier, 2006: 73.)

In contrast to most other anesthetics, ketamine causes central stimulation of the sympathetic nervous system and inhibition of norepinephrine reuptake leading to increases in blood pressure, heart rate and cardiac output.

Cardiovascular Effects of Induction Agents

- **Propofol:**
  - Peripheral vasodilation causes up to 40% ↓ in MAP.
  - Diminished baroreceptor response to hypotension.
  - ↓ CO.
  - No reflex tachycardia.
- **Etomidate:**
  - Mild ↓ in MAP.
  - CO and contractility maintained.
- **Thiopental:**
  - Peripheral vasodilation causes ↓ in MAP.
  - Maintained CO secondary to reflex tachycardia.
- **Ketamine:** Stimulation of SNS → ↑ HR, MAP, and CO.
- **Midazolam:**
  - Minimal myocardial depression.
  - Cardiac output and MAP maintained.
  - Possible slight ↑ in HR.

Hepatic Anatomy

Liver Structure

- The falciform ligament separates the smaller left lobe from the larger right lobe of the liver.
- The right lobe is further divided into two smaller lobes, caudate and quadrate.
- The liver is made up of 50–100,000 lobules.
- Made of hexagonal plates of hepatocytes and portal triads surrounding a central hepatic vein.
- Acinus: Functional unit of the liver.
  - Zone 1: Cells closest to the portal tract that are well oxygenated.
  - Zone 3: Cells closest to the centrilobular veins receive the least oxygen (most susceptible to injury).
- Venous drainage from the central veins of hepatic lobules coalesce to form the hepatic veins, which empty into the inferior vena cava (IVC).

The liver has dual oxygen supply: 45–50% from hepatic artery and 50–55% from portal vein.

Liver Blood Supply

- The liver receives 20–25% of total CO.
- The liver has a dual blood supply from the hepatic artery and the portal vein.
  - Total hepatic blood flow is autoregulated such that a ↓ in either blood supply results in a compensatory ↑ in the other.
  - Hepatic artery flow is inversely related to portal blood flow and directly related to adenosine-induced arteriole dilation.
  - Splanchnic nerve stimulation results in hepatic artery vasoconstriction and therefore ↓ hepatic blood flow.
  - Hepatic circulation is supplied with β receptors. β stimulation vasodilates the hepatic artery. β blockers reduce hepatic blood flow.
- The portal vein has only α and dopaminergic receptors.

Liver Function Tests

Serum Bilirubin

- Normal total bilirubin (conjugated + unconjugated): < 1.5 mg/dL.
- Normal unconjugated bilirubin: 0.2–0.8 mg/dL.
- Unconjugated bilirubin:
  - Has minimal urinary excretion.
  - Water soluble.
  - Neurotoxic.
- Normal conjugated bilirubin: 0.0–0.3 mg/dL.
- Conjugated bilirubin:
  - Reflects balance between production and biliary excretion.
  - Water soluble.
  - Excreted in urine.
  - Jaundice obvious when total bilirubin > 3 mg/dL.
- Conjugated hyperbilirubinemia associated with ↑ urinary urobilinogen and may reflect:
  - Hepatocellular dysfunction.
  - Intrahepatic cholestasis.

Serum albumin, prothrombin time, cholesterol, and pseudocholinesterase are good measures of hepatic synthetic function.

- Extrahepatic biliary obstruction (unconjugated hyperbilirubinemia) may be seen with:
  - Hemolysis.
  - Congenital or acquired defects in conjugation.

Serum Transaminases

- Released into circulation as a result of hepatocellular injury or death.
- Aspartate transaminase (AST [SGOT]): Present in many tissues including liver, heart, skeletal muscle, and kidneys.
- Alanine transaminase (ALT [SGPT]): Primarily located in the liver.
- Normal levels for AST and ALT are between 10 and 32 units/L.
- Circulating half-life ($t_{1/2}$) for AST and ALT are 18 and 36 minutes, respectively.
- Mild elevations can be seen with cholestasis or metastatic liver disease (< 300 units/L).

Serum Alkaline Phosphatase

- Produced by kidneys, small bowel, liver, bone, and placenta and excreted into bile.
- Normal levels are between 45 and 125 units/L.
- Circulating $t_{1/2}$ is about 7 days.
- ↑ levels can be seen in:
  - Hepatocellular injury.
  - Hepatic metastatic disease.
  - Intrahepatic cholestasis.
  - Biliary obstruction
  - Pregnancy.
  - Bone disease (Paget's disease or bone metastases).
- Combination of elevated alkaline phosphatase and serum γ-glutamyl transpeptidase activity is the most sensitive indicator of hepatobiliary disease.

Decreased albumin levels can lead to an ↑ fraction of unbound and therefore more pharmacologically active levels of drugs that are highly protein bound (ie, diazepam, warfarin, barbiturates).

Serum Albumin

- Normal level is between 3.5 and 5.5 g/dL.
- $t_{1/2}$ is 2–3 weeks; may initially be normal in acute liver disease.
- < 2.5 g/dL usually indicates chronic liver disease and malnutrition.
- Hypoalbuminemia can also be caused by increased losses such as nephritic syndrome, protein-losing enteropathies, burns, and ascites.
- Total body albumin can be elevated in cirrhotic patients who have low serum albumin levels but a large amount of albumin in ascetic fluid.

Blood Ammonia

- Normal levels range from 47 to 65 μmol/L.
- Significant elevations usually reflect disruption of hepatic urea synthesis and can lead to severe hepatocellular damage.
- Rough correlation between arterial ammonia and hepatic encephalopathy.

Prothrombin Time (PT)

- Normal levels range from 11 to 14 sec.
- PT is the most important qualitative measure of the liver's function and ability to synthesize proteins.
- Measures not only PT but entire extrinsic coagulation pathway including fibrinogen, prothrombin, and factors V, VII, and X.
- PT's $t_{1/2}$ is 6 hours; therefore, it can reflect acute hepatic injury.
- Prolongation of PT > 3–4 sec corresponds to an International Normalized Ratio (INR) > 1.5.
- Causes of prolongation of PT include:
  - Drug effects.
  - ↓ plasminogen levels.
  - Fibrinolysis.
  - Disseminated intravascular coagulation (DIC).
  - Vitamin K deficiencies (ie, malnutrition, cystic fibrosis).

All coagulation factors, except factor VIII and von Willebrand factor, are produced by the liver.

Effects of Anesthesia on Hepatic Function

Hepatic Blood Flow

- All anesthetic agents indirectly reduce hepatic blood flow in proportion to a ↓ in MAP or CO.
- **Volatile anesthetics:**
  - All volatile anesthetics reduce portal hepatic blood flow.
  - Halothane causes greatest reduction in blood flow.
  - Isoflurane causes the least reduction in blood flow.
    - Only agent that causes a significant direct arterial vasodilation that can ↑ hepatic arterial blood flow.
    - Total hepatic blood flow ↓, even with isoflurane, because the ↓ in portal blood flow offsets an ↑ in hepatic artery flow.

Hepatic blood flow = hepatic perfusion pressure – splanchnic vascular resistance.

Most volatile anesthetics reduce hepatic perfusion pressure.

Ventilation

- Positive pressure ventilation with high mean airway pressures ↓ venous return and CO, which can compromise hepatic blood flow.
- ↓ venous return ↑ hepatic venous pressure. Positive end-expiratory pressure (PEEP) can accentuate these effects.
- Surgical procedures near the liver can reduce hepatic blood flow by up to 60%.
- Intraoperative medications:
  - β blockers, α agonists, H_2 blockers, and vasopressin reduce hepatic blood flow.
  - Low-dose dopamine may ↑ liver blood flow.

Metabolic Functions

- Endocrine stress response secondary to fasting and surgical trauma is generally observed.
  - Elevated catecholamines, glucagon, and cortisol → hyperglycemia and negative nitrogen balance.
  - May be partly blunted by deep anesthesia, regional anesthesia, or pharmacologic blockage of the SNS.

- **Drug metabolism:**
  - Halothane directly inhibits the metabolism of phenytoin, warfarin, and ketamine.
  - Halothane ↓ hepatic blood flow, which alters pharmacokinetics of other drugs such as fentanyl, verapamil, and propranolol.

Biliary Function

- All opioids can cause spasm of the sphincter of Oddi and ↑ biliary pressure (fentanyl > morphine > meperidine). Can → false-positive cholangiograms.
- Halothane and enflurane may blunt the ↑ in biliary pressure following opioid administration.
- Naloxone and glucagon may relieve opioid-induced spasms.

Liver Function Tests (LFTs)

- Mild postop liver dysfunction in healthy patients is not uncommon. Can be secondary to ↓ blood flow from anesthesia, sympathetic stimulation, and the surgical procedure.
- Significant postop elevations in LFTs are the result of underlying liver disease or the surgical procedure itself.
- Postop jaundice is most commonly due to overproduction of bilirubin because of resorption of a large hematoma or red cell breakdown following transfusion.
- Postop jaundice can also be secondary to three causes:
  1. Prehepatic (↑ bilirubin production): Transfusions.
  2. Hepatic (hepatocellular dysfunction): Underlying liver disease, ischemic or hypoxic injury, drug-induced, or intrahepatic cholestasis.
  3. Posthepatic (biliary obstruction): Postop cholecystitis, postop hepatitis, retained common bile duct stone.

Hepatic Dysfunction Associated with Halogenated Anesthetics

Halothane Hepatitis

Risk factors associated with halothane hepatitis include middle age, obesity, female sex, and repeat exposure to halothane (within 28 days).

- Several mechanisms have been described:
  - Formation of hepatotoxic metabolic intermediates and immune hypersensitivity.
  - Antibodies directed against hepatocyte components.
  - Genetic susceptibility.
- Diagnosis of exclusion, therefore viral hepatitis, cytomegalovirus, Epstein-Barr virus, and herpesvirus should be excluded.
- Severity can range from an asymptomatic elevation of LFTs to hepatic necrosis.
- Incidence of a mild form of syndrome as high as 20% in adults following a second exposure to halothane; incidence of fatal hepatic necrosis is approximately 1:35,000.

Hepatitis from Other Halogenated Agents

This has been seen in enflurane and isoflurane; estimated at 1:500,000–1:300,000.

Functions of the Kidney

- Acid-base balance.
- Excretion and detoxification of blood.
- Endocrine function: Vitamin D conversion, calcium and phosphate homeostasis, erythropoietin secretion.
- Regulation of body fluid volume and composition.
- Regulating renin.

Renal blood flow is autoregulated between mean arterial blood pressures of 80 and 180 mmHg.

Renal Anatomy

- The kidneys receive 20–25% of total CO.
- The renal arteries are branches of the aorta, which take off below the superior mesenteric artery.
- The renal veins drain into the IVC.
- Sympathetic stimulation is via the celiac and renal plexuses and cause constriction; there is no parasympathetic innervation to the kidney.
- Pain fibers from the renal pelvis and upper ureter enter the spinal cord via splachnic nerves.

The Nephron

- Each kidney is composed of 1 million nephrons.
- Each nephron contains glomerulus, proximal tubule, loop of Henle, distal tubule, collecting tubule, and juxtaglomerular apparatus.
  - **Glomerular capillaries:** Connected to Bowman's capsule; has a large surface area for filtration of blood.
  - **Bowman's capsule:** At proximal end where ultrafiltrate is formed.
  - **Proximal convoluted tubule:** The ultrafiltrate is reabsorbed here. Na^+ and other solutes (H_2O, bicarbonate, glucose, proteins, magnesium, calcium, urea) are also reabsorbed. Ammonia is produced here, and organic anions and cations are secreted.
  - **Loop of Henle:** Reabsorption of Na^+, Cl^-, H_2O, K^+, Ca^+, and Mg^+ occurs here. This loop is responsible for maintaining hypertonic medullary interstitium and indirectly provides the collecting tubules with the ability to concentrate urine.
  - **Distal convoluted tubule:** Responsible for additional reabsorption of NaCL, H_2O, K^+, Ca^+, and bicarbonate. Also secretes hydrogen ion, K^+, and Ca^+.
  - **Collecting tubule:** Reabsorbs NaCl, H_2O, K^+, and bicarbonate, and secretes K^+ and hydrogen ion. Ammonia is also produced here.
  - **Juxtaglomerular apparatus:** Secretes renin.

Renal Filtration and Autoregulation

- The two components of glomerular filtration rate: MAP and afferent arteriole diameter.
- ↑ MAP → ↑ GFR. GFR describes the flow rate of filtered fluid through the kidney and this flow is directly related to the MAP, as an ↑ in pressure → an ↑ in flow to the kidney.
- GFR is directly proportional to efferent arteriole tone and inversely proportional to afferent arteriole tone.

GFR = (140 – age) (body weight in kg) / (serum creatinine)(72)

- Normal GFR is 120 +/– 25 mL/min in men and 95 +/– 20 mL/min in women.
- Glomerular filtration produces about 180 L of fluid per day. The majority of the fluid is reabsorbed, so approximately only 1 L is finally secreted.
- Glomerular filtrate may either be reabsorbed or secreted from the tubules either by active transport or passive transport.
- Renal blood flow (RBF) is autoregulated at blood pressures of 80–180 mmHg.
- RBF regulation is to maintain GFR.
- Renin-angiotensin-alderosterone axis has an effect on RBF, with angiotensin II ultimately causing renal vasoconstriction.

Acid-Base Physiology and Balance

- Infusion of electrolyte-containing solutions, changes in ventilation and perfusion affect the acid-base balance; therefore, understanding of the physiologic effects is essential for treatment of acid-base disturbances.
- Acid-base disorders can be put into different categories (Table 2-2).
- To determine if a patient has an acid-base disorder, the pH can be calculated from the Hendersen-Hasselbalch equation (HH):

$$pH = 6.1 + \log \frac{HCO_3}{0.03 \times PaCO_2}$$

Buffers

The body's initial response to an acid-base disturbance is an immediate **buffer**. The next response of **compensation** occurs over a longer period of time. The last phase is **correction.**

- **Buffer:** Immediate reaction by whichever buffers predominate in a particular body fluid compartment.
- Physiologically important buffers include:
  - Bicarbonate (H_2CO_3/HCO_3^-).
  - Hemoglobin (HbH/Hb–).
  - Intracellular proteins.
  - Phosphates.
  - Ammonia.

TABLE 2-2. Defining Acid-Base Disorders

Disorder	Primary Change	Compensatory Change
Respiratory		
Acidosis	↑ $Paco_2$	↑ HCO_3
Alkalosis	↓ $Paco_2$	↓ HCO_3
Metabolic		
Acidosis	↓ HCO_3	↓ $Paco_2$
Alkalosis	↑ HCO_3	↑ $Paco_2$

(Reproduced, with permission, from Morgan GE, Mikhail MS, Murray MJ. *Clinical Anesthesiology,* 4th ed. New York: Lange Medical Books/McGraw-Hill, 2006: 710.)

Compensation

Respiratory disturbances are compensated by changes in the renal system, and renal system acid-base disturbances are compensated by changes induced in the respiratory system (see Table 2-2).

Correction

Occurs when the cause of the acid-base disturbance has been treated. Common causes for unbalance include:

- Respiratory acidosis: COPD, muscle relaxants, narcotics.
- Respiratory alkalosis: Mechanical ventilation, hyperventilation.
- Metabolic alkalosis: Nasogastric suction, vomiting, diuretics.
- Metabolic acidosis: Diabetes mellitus, renal failure, shock
- Metabolic acidosis can be broken down into two categories: Anion gap and nonanion gap.
  Anion gap = [Na+] – ([Cl^-] + [HCO_3^-]) where normal AG = 7–14 mEQ/L

↑ Anion gap acidosis—

SLUMPED

Salicylates
Lactic acidosis
Uremia
Methanol
Paraldehyde
Ethylene glycol
Diabetes mellitus

Ventilation and Oxygenation

- Normal $PaCO_2$ = 36–44. One can determine if changes in pH are based on respiratory changes by the golden rule: **For every 10 torr change in $PaCO_2$, the pH will change 0.08 unit in the opposite direction.**
- If the $PaCO_2$ change can be accounted for (pH) with the golden rule, then there is no metabolic component to the acid-base disturbance.
- If the $PaCO_2$ cannot account for all of the changes in pH, then there is a metabolic component to the disturbance.
- The alveolar air equation can be used with results of an arterial blood gas (ABG) to determine causes of hypoxemia:
  $PaO_2 = (FiO2 \times P_{barometric}\ pressure) - PaCO2/0.8$ where $P_{barometric}$ pressure = 713
  - ↑ A-a gradient: V/Q mismatch, shunting, diffusion barrier.
  - Normal A-a gradient: Hypoventilation, ↓ FiO_2.

Predictors of Postoperative Renal Failure

- ↑ blood urea nitrogen (BUN) and creatinine.
- Left ventricular dysfunction.
- Diabetes.
- Advanced age.
- Jaundice.
- Cardiac or aortic surgery.

Acute Renal Failure (ARF)

- Defined as a significant ↓ in GFR over 2 weeks or less or inadequate urine output of < 0.5 mL/kg/hr (see Table 2-3).
- A good urine output does not rule out ARF.
- Elevated serum creatinine is a late sign of renal failure.
- GFR may be reduced by 50% or more before abnormal elevation in renal labs is observed.
- When muscle wasting has occurred, creatinine levels may be normal despite reduced GFR (creatinine production is proportional to muscle mass).

TABLE 2-3. Tests of Renal Function

Test	Normal Value	Abnormal Value	Comments
Specific gravity	1.01–1.03	> 1.03 prerenal azotemia.	Nonspecific; affected by glucose, diuretics, radiocontrast.
BUN	10–20 mg/dL	> 50 associated with renal impairment.	Nonspecific.
Creatinine	0.8–1.3mg/dL men 0.6–1mg/dL women	Doubling of creatinine. Associated with 50% reduction in GFR.	Late indicator of renal failure.
Fractional excretion of sodium (FE_{Na})	1–3%		FE_{Na} < 1% prerenal. FE_{Na} > 3% ATN.
Creatinine clearance	100–125 mL/min	↓ renal.	Good test for measuring GFR. Reserve 60–100; mildly impaired 40–60. RF < 25.

Creatinine clearance = ([Creatine]$_{urine}$ – Urinary flow rate) / [Creatinine]$_{plasma}$

- Creatinine clearance is the most sensitive test for renal failure but is limited by 24-hr urine collection.
- **Prerenal azotemia** accounts for 60% of cases of ARF:
  - ↓ blood flow to kidney.
  - Hypovolemia.
  - Myocardial dysfunction.
  - Sepsis with shunting of blood away from kidney.
- **Renal azotemia** accounts for 30% of cases of ARF:
  - Acute tubular necrosis (ATN): Leading cause and may be due to ischemia or toxins.
  - Nephrotoxins: Radiocontrast media, aminoglycosides, fluoride associated with volatile anesthetic metabolism.
  - Muscular injury causing rhabdomyolysis.
  - Hemolysis causing hemoglobinuria.
- **Postrenal azotemia** accounts for 10% of cases of ARF: Obstructive nephropathy.
- Nonrenal causes of elevated BUN and creatinine:
  - ↑ nitrogen absorption.
  - Diabetic ketoacidosis.
  - Hematoma reabsorption.
  - Gastrointestinal bleeding.
  - Hypercatabolism.
  - Hepatic disease.
  - Drugs.

Sevoflurane can produce compound A metabolites, which can, associated with low gas flows of < 2 L/min, cause renal failure (in animal studies only, not in humans).

Anesthesia Effects on Renal Function

- Volatile agents temporarily depress renal function.
- ↓ in urine output, GFR, renal blood flow (RBF), and electrolyte excretion.

- Usually reversible and short lived.
- Neuraxial anesthesia depresses renal function but not to same extent as general anesthesia.
- Muscle relaxation:
  - Atracurium and cisatracurium undergo Hoffman degradation and ester hydrolysis and therefore can be used in patients with renal failure.
  - Succinylcholine can be used in patients with renal failure as long as their K^+ is < 5.0 mEq/L. Some studies note that the duration of succinylcholine may be prolonged in renal failure patients on dialysis.

Elimination of pancuronium, curare, pipecuronium, and doxacurium are primarily renal and neuromuscular effects that can be prolonged with renal insufficiency.

Renal Failure and Perioperative Management

- Preoperative goals in this patient population:
- Euvolemic (no signs of active fluid overload/congestive heart failure [CHF]).
- Normotensive.
- Electrolytes WNL (K < 5.0).
- Platelets (> 75,000).
- Acid-base balance should be within normal range.
- Indications for dialysis (which many patients have prior to OR):
  - Uremic platelet dysfunction.
  - Uremic symptoms.
  - Pericardial tamponade.
  - Bleeding.
  - Hypervolemia.
  - CHF.
  - Severe acidosis.
  - Hyperkalemia.
- CaC_{l2} temporizes cardiac effects of K^+ until further measures can be taken to shift K^+ intracellularly (glucose and insulin, hyperventilation, $NaHCO_3$, potassium-binding resins).

AUTONOMIC NERVOUS SYSTEM

- Comprised of sympathetic and parasympathetic nervous systems, each having opposite effects on end organs (see Chapter 1, Figure 1-2).
- Network of nerves and ganglia that control involuntary physiologic parameters.
- Exerts partial control over systemic BP, HR, urinary bladder emptying, sweating, body temperature, and gastrointestinal motility and secretions.
- Reflexes:
  - Arterial baroreceptors:
    - Located in the carotid sinus and aortic arch.
    - Respond to alterations in stretch caused by changes in blood pressure.
  - Volatile anesthetics interfere with baroreceptor function (halothane > isoflurane).
  - Venous baroreceptors: Located in right atrium and great veins.
  - Bainbridge reflex: ↑ in HR when right atrium is stretched by ↑ filling pressures.

SYMPATHETIC NERVOUS SYSTEM (SNS)

- Also known as the thoracolumbar nervous system.
- Activation of the SNS produces a diffuse physiologic response.
- **Preganglionic sympathetic neurons:**
  - Originate from the intermediolateral columns of the thoracolumbar spinal cord.
  - Synapse with postganglionic fibers in paravertebral sympathetic ganglia as unpaired prevertebral ganglia or with a terminal ganglion.
  - Exit via the ventral root of the spinal cord (myelinated).
  - Twenty-two pairs of paravertebral ganglia lie on both sides of the vertebral column.
  - Located closer to the spinal cord than innervated organ.
  - Stimulate nicotinic cholinergic postganglionic neurons by releasing ACh.
  - May ascend or descend in sympathetic chain before synapsing.
- Postganglionic neurons.
- Synapse at targeted end organs.
- Release norepinephrine (NE).
- **Sympathetic ganglia:**
  - Paravertebral ganglia:
    - Twenty-two pairs are connected to spinal nerves by white and gray rami communicantes and are connected by nerve trunks to form the lateral chains.
    - The paravertebral ganglia include cervical ganglia; stellate ganglia (cervical + T1 ganglia); thoracic, abdominal, and pelvic sympathetic trunks.
  - Prevertebral ganglia: Celiac, superior and inferior mesenteric, and renal ganglia.
  - Terminal ganglia lie near the innervated organs (ie, bladder, rectum).
- Postganglionic adrenergic receptors and end-organ effects (see Table 2-4):
  - α_2 receptors:
    - Presynpatic stimulation.
    - Inhibits release of norepinephrine; termed *negative feedback*.
  - α_1, β_1, and β_2 receptors:
    - Postsynaptic
    - Stimulated by norepinephrine.

Acetylcholine is the neurotransmitter of all preganglionic sympathetic fibers and postganglionic parasympathetic fibers.

PARASYMPATHETIC NERVOUS SYSTEM (PNS)

- Activation of the PNS produces a discrete response (ie, vagal stimulation produces bradycardia).
- Preganglionic parasympathetic neurons:
  - Originate from cranial nerves III, VII, IX, and X and sacral segments 2–4.
  - Synapse with postganglionic neurons close to the end organ.
    - Preganglionic and postganglionic neurons release ACh.
    - Cholinergic receptors are classified as nicotinic or muscarinic (see Table 2-5).

Norepinephrine is the neurotransmitter of nearly all postganglionic sympathetic fibers.

TABLE 2-4. Adrenergic Receptors and End-Organ Effects

Receptor	Organ	Response
β_1 (B1)	Heart	Increases heart rate, contractility, and conduction velocity.
	Fat cells	Lipolysis.
β_2 (B2)	Blood vessels	Dilation.
	Bronchioles	Dilation.
	Uterus	Relaxation.
	Kidneys	Renin secretion.
	Liver	Gluconeogenesis, glycogenolysis.
	Pancreas	Insulin secretion.
α_1 (A1)	Blood vessels	Constriction.
	Pancreas	Inhibits insulin release.
	Intestine, bladder	Relaxation but constriction of sphincters.
α_2 (A2)	Presynaptic nerve endings	Inhibits norepinephrine release.
Dopamine-1	Blood vessels	Dilates renal, coronary, and splanchnic vessels.
Dopamine-2	Presynaptic endings	Inhibits norepinephrine release.
	Central nervous system	Psychic disturbances.

(Reproduced, with permission, Miller RD, Stoelting RK. *Basics of Anesthesia,* 4th ed. New York: Churchill Livingstone, 2000: 36.)

TABLE 2-5. Cholinergic Receptor Stimulation and End-Organ Effects

Muscarinic	Heart	↓ heart rate, contractility, conduction velocity.
	Bronchioles	Constriction
	Salivary glands	Stimulates secretion.
	Intestine	Contraction and relaxation of sphincters, stimulates secretions.
	Bladder	Contraction and relaxation of sphincters.
Nicotinic	Neuromuscular junction	Skeletal muscle contraction.
	Autonomic ganglia	Sympathetic nervous system stimulation.

(Reproduced, with permission, Miller RD, Stoelting RK. *Basics of Anesthesia,* 4th ed. New York: Churchill Livingstone, 2000: 36.)

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CHAPTER 3

Preoperative Evaluation

The Mallampati classification has a low positive predictive value if used alone, which makes a thorough airway examination necessary to minimize unexpected difficult intubations.

AIRWAY EVALUATION

History

Ask patient about previous experience, and review previous records for difficult intubation and laryngoscopy grades.

Physical Exam

- The Mallampati classification system (Figure 3-1 and Table 3-1) evaluates tongue size relative to oral cavity. Although it has low positive predictive value by itself, when combined with a thorough examination, it can minimize unexpected difficult intubations.
- A thorough airway examination includes multiple components (Table 3-2) in addition to the Mallampati classification, including: incisor length, overbite presence, interincisor distance, palate shape, mandibular space compliance, thyromental distance, neck length and thickness, and range of motion of head and neck.
- Evaluate neck flexion and extension. Caution must be applied to trauma patients or patients with Down syndrome or rheumatoid arthritis (atlantoaxial instability).
- According to the ASA Task Force on Managing Difficult Airway, multiple examinations yield a better chance of identifying possible difficult airway.

MANAGING DIFFICULT AIRWAY

- There are four types of management problems that can be managed with the difficult airway algorithm (Figure 3-2):
  1. Difficult to ventilate
  2. Difficult to intubate
  3. Difficulty with cooperation
  4. Difficult tracheostomy
- Based on the above four problems, two paths can be chosen:
  1. Awake intubation, which can be noninvasive or surgical.
  2. Intubation after induction of general anesthesia:
     - If unsuccessful, attempt mask ventilation and try other modalities (different laryngoscopy blades, fiber-optic, or glidescope).

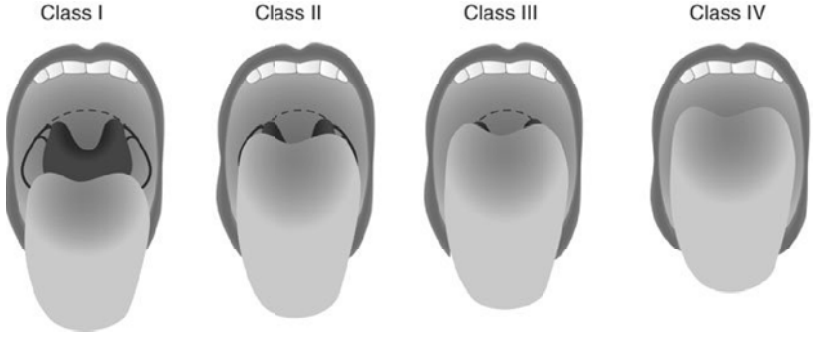

FIGURE 3-1. Mallampati classification.

(Reproduced, with permission, from Longnecker DE et al. *Anesthesiology*, New York: McGraw-Hill, 2008: 125.)

TABLE 3-1. Mallampati Classification

Class	Structures Identified when Patient Seated
I	Soft and hard palate, uvula, tonsillar pillars
II	Soft and hard palate, parts of uvula
III	Soft and hard palate, base of uvula
IV	Hard palate only

- If unable to mask ventilate, consider LMA. If LMA is not adequate or feasible, either emergency noninvasive airway ventilation (combitube, rigid bronchoscope, transtracheal jet ventilation) or emergency invasive airway access is indicated.

PREOPERATIVE TESTING

- Routine testing of patients with no clinical risk factors can result in high number of false positives. Initiation of treatment based on the "abnormal" results can yield more harm than good for the patient. Obtaining tests therefore should be carefully selected (Table 3-3).

TABLE 3-2. Physical Exam for Potentially Difficult Airway

Airway Examination Component	Nonreassuring Findings
1. Length of upper incisors	Relatively long
2. Relation of masillary and mandibular incisors during normal jaw closure	Prominent "overbite" (maxillary incisors anterior to mandibular incisors)
3. Relation of maxillary and mandibular incisors during voluntary protrusion of cannot bring	Patient mandibular incisors anterior to (in mandible front of) maxillary incisors
4. Interincisor distance	Less than 3 cm
5. Visibility of uvula	Not visible when tongue is protruded with patient in sitting position (*eg,* Mallampati class greater than II)
6. Shape of palate	Highly arched or very narrow
7. Compliance of mandibular space	Stiff, indurated, occupied by mass, or nonresilient
8. Thyromental distance	Less than three ordinary finger breadths
9. Length of neck	Short
10. Thickness of neck	Thick
11. Range of motion of head and neck	Patient cannot touch tip of chin to chest or cannot extend neck

(Practice guidelines for management of the difficult airway, *Anesthesiology* 2003; 98:1269–1277.)

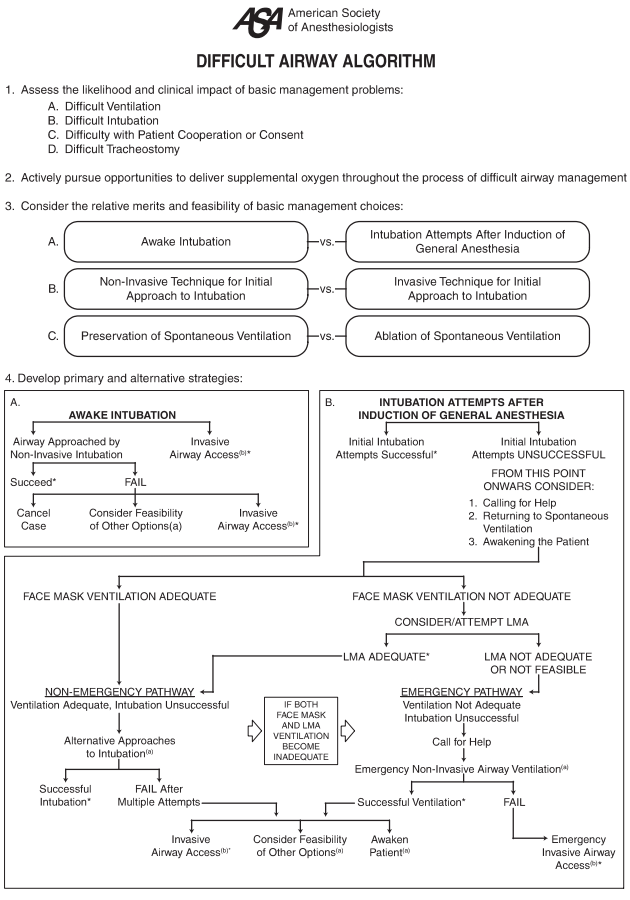

FIGURE 3-2. Difficult airway algorithm.

(Reproduced, with permission, from Practice guidelines for management of the difficult airway. *Anesthesiology* 2003; 98:1269–1277.)

- Patients are classified into the following ASA categories at the end of preoperative evaluation:

ASA Class	Disease State
1	No organic, physiologic, or psychiatric disturbance
2	Mild to moderate systemic disease
3	Severe systemic disturbance may or may not be related to the reason for surgery
4	Severe systemic disease that is life threatening with or without surgery
5	Moribund patient with little chance of surviving
E	Any patient presenting for emergency operation

- Pulmonary function test (PFT): For nonthoracic surgery, it has a limited role, since PFTs rarely provide additional information. Might be useful in situations where responsiveness to bronchodilator therapy is needed.

- Routine testing of patients with no clincial risk factors can result in a high number of false positives. Initiation of treatment based on the "abnormal" results can yield more harm than good for the patient. Therefore, preoperative tests should be carefully selected (Table 3-3).

COEXISTING CONDITIONS

Evaluation of Patients with Cardiovascular (CV) Disease

HISTORY

- Check for hypertension, congestive heart failure (CHF), myocardial infarction (MI) or angina, and any valvular heart disease. Ask about symptoms of certain CV disease.
- Keep in mind that elderly, women, and diabetic patients might have atypical presentation of symptoms.

Cardiovascular-Related Disease	Symptoms
Uncontrolled hypertension	Headache, blurred vision, chest pain, nausea and vomiting, and changes in mental status
Coronary artery disease	Angina, fatigue, ↓ exercise tolerance, previous history of myocardial ischemia or infarction
Congestive heart failure	Orthopnea, dyspnea, lower extremity edema, fatigue

TABLE 3-3. Preoperative Tests

TESTS	INDICATIONS
CBC: Suggested as the only necessary test for elective surgery.	Neonates, age > 75, patients with renal disease and malignancy, procedures with large expected blood loss.
Electrolytes: Indicated in patients with systemic diseases or medications that affect the kidneys.	Patients with renal disease, diabetes, and diuretic use.
Coagulation studies: Abnormal results in patients with no risk factors minimally affects anesthetic plan.	Patients with bleeding disorder or anticoagulant use, hepatic disease, chemotherapy.
Pregnancy test	Any female patients of childbearing age since patient's history is unreliable.
Electrocardiogram (ECG): Can provide information regarding patient's status of myocardium and coronary circulation. Combined with clinical risk factors, ECG can provide vital information.	Males > 40 years old, females > 50 years old with systemic disease; patients with cardiopulmonary risk factors, diabetes, digoxin use, radiation therapy.
Chest x-ray: Useful to identify pneumonia, pulmonary edema, atelectasis, and pulmonary nodules only in populations at risk (active pulmonary disease or intrathoracic surgery).	Age > 75, patients with cardiopulmonary disease, malignancy, and tobacco use > 20 years.

HYPERTENSION (HTN)

- Two types:
  1. Essential HTN (95% of cases).
  2. Secondary HTN: Due to other identifiable sources (Table 3-4).
- Three stages:
  Stage 1: 140–160/90–100
  Stage 2: 160–180/100–110
  Stage 3: > 180/> 110
- HTN indicates ↑ risk of coronary artery disease (CAD) and cerebral vascular disease.
- Patients with stage 3 HTN have ↑ risk of labile blood pressure (BP) intraoperatively and ischemic cardiac events. However, there is no absolute evidence to suggest delaying the surgery will reduce risk. It is appropriate to delay the surgery if evidence of end-organ damage is present (new-onset headache, blurred vision, renal insufficiency, and left ventricular hypertrophy with strain).
- Current recommendation is to hold angiotensin-converting enzyme inhibitors (ACEIs). Although β blockers are helpful, acute use to bring the BP down prior to surgery is not recommended.

CORONARY ARTERY DISEASE (CAD)

Leading cause of death in United States among both men and women.

HISTORY

- Crucial to the discovery of cardiac or comorbid diseases. Identify serious cardiac conditions such as unstable angina, recent or past MI, CHF, severe valvular disease, and significant arrhythmia. Also, elicit presence of pacemaker or ICD.
- Patient may report history of percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG).
- Patient might present with symptoms of angina. Other coexisting diseases such as diabetes mellitus or peripheral vascular disease are associated with CAD.

PHYSICAL EXAMINATION

- BP measurement (preop BP is the best predictor of response to laryngoscopy).
- Auscultation of heart: Check for rhythm, rate, and murmurs (consider endocarditis prophylaxis if murmur present). Gallops could indicate CHF. Check for bruits over the carotid arteries to minimize risk of stroke.
- Check for jugular vein distention, lower extremity edema, and peripheral pulses.

TABLE 3-4. Identifiable Sources of Secondary Hypertension

Renal	Glomerulonephritis, renal artery stenosis, polycystic kidney.
Endocrine	Cushing's syndrome, congenital adrenal hyperplasia, pheochromocytoma, Conn's syndrome.
Neurogenic	Acute porphyria, anxiety, ↑ intracranial pressure.
Others	Coarctation of aorta, polyarteritis nodosa, hypercalcemia.

Summary of 2007 Guidelines on Perioperative CV Evaluation (Figure 3-3)

- Clinical risk factors for postoperative complications:
  1. CAD.
  2. History of compensated/decompensated heart failure.
  3. Cerebrovascular disease.
  4. Diabetes.
  5. Renal insufficiency.
- Cardiac conditions that may delay surgery:
  1. Unstable coronary syndrom: Unstable angina, recent MI (within 4 weeks).
  2. Decompensated heart failure.
  3. Significant arrhythmia: Mobitz II, 3° AV block, SVT, symptomatic bradycardia.
  4. Severe valvular disease:
     a. Severe aortic stenosis: Aortic valve area < 1 cm^2; pressure gradient > 40 mmHg.
     b. Symptomatic mitral stenosis.
- Risk stratification for noncardiac procedures:
  1. Vascular surgery (**> 5%**): Aortic, peripheral vascular.
  2. Intermediate-risk surgery (cardiac risk of 1–5%): Carotid endarterectomy, intraperitoneal/thoracic head/neck surgery, orthopedic, prostate.
  3. Low-risk surgery (< 1%): Endoscopic, cataract, breast, ambulatory.
- Cardiac testing: Routine ECG is recommended for males > 40 years old and females > 50 years old. Any patients with major clinical predictors of high perioperative CV risk should have surgery delayed until the conditions are optimized.

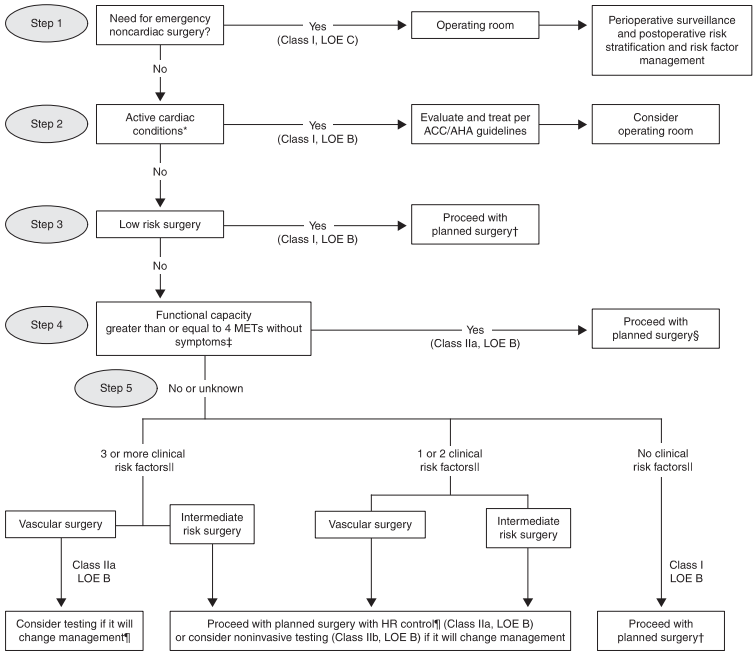

FIGURE 3-3. Perioperative cardiovascular evaluation for noncardiac surgery.

(Reproduced, with permission, from Fleisher L et al. ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery. *Circulation* 2007; 116: e418–e500.)

Elective surgeries should be postponed for 4–6 weeks after bare-metal stent placement and 12 months after drug-eluting stent placement.

- Patients who have undergone recent coronary revascularization are at risk for stent thrombosis if their dual antiplatelet therapy (aspirin and clopidogrel [Plavix]) is discontinued for surgery. Risk of thrombosis is highest 4–6 weeks after bare-metal stents and 12 months after drug-eluting stents; consequently, elective surgeries are postponed until after this time when Plavix can temporarily be discontinued (Figure 3-4).

Evaluation of Patients with Pulmonary Disease

- Pulmonary complications are more frequent than cardiac complications after major operations. These complications include atelectasis, pneumonia, bronchospasm, hypoxemia, and respiratory failure requiring mechanical ventilation.
- Factors for pulmonary complications:
  - Strongest prediction depends on site of the surgery: Upper abdominal surgery associated with highest risk. Type of surgery is important as well: abdominal aortic aneurysm (AAA) repair > thoracic > upper abdominal surgery > neck/peripheral vascular > neurosurgery.
  - Hypoxemia is mainly caused by (1) ↓ in functional residual capacity (FRC) and vital capacity (VC), and (2) diaphragmatic dysfunction (persists even with adequate analgesia).
- Pulmonary changes associated with general anesthesia:
  - ↑ in dead space.
  - ↑ in intrapulmonary shunt.
  - ↓ in FRC.

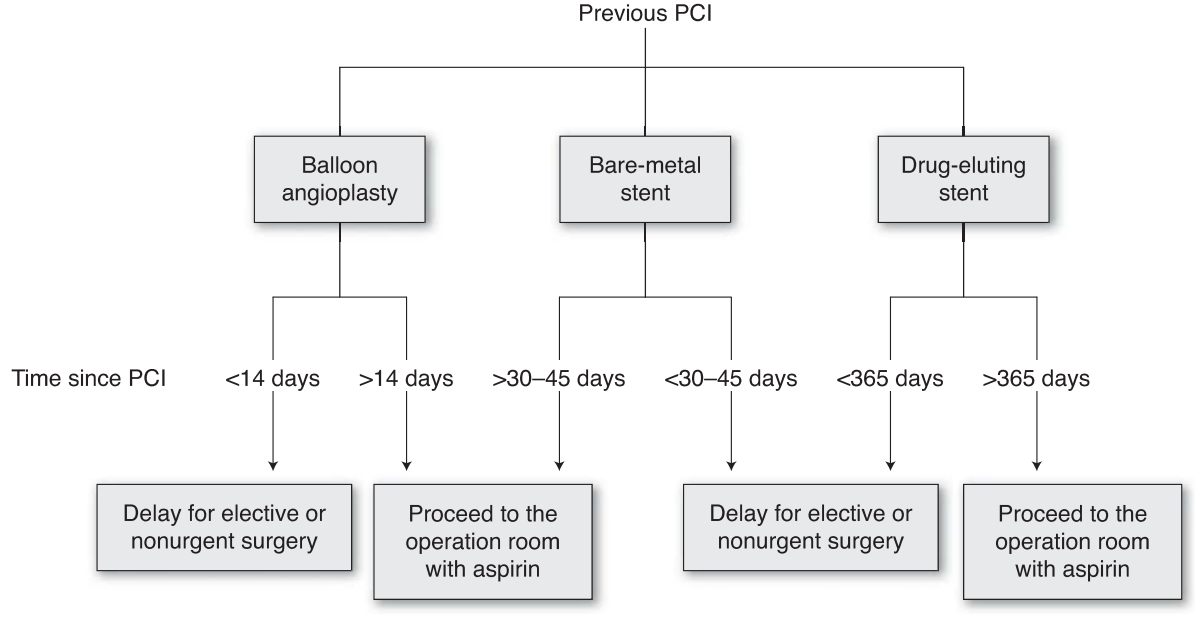

FIGURE 3-4. Management of patients with previous percutaneous coronary intervention (PCI) who require noncardiac surgery.

(Reproduced, with permission, from Fleisher L et al. ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery. *Circulation* 2007; 116: e418–e500.)

- ↓ in surfactant production.
- ↑ in alveolar permeability.

- Pulmonary complications ↑ with duration of surgery: Significant after 2-3 hours.
  - Tobacco use: Important risk factor. Cessation of smoking for 2 days can improve carboxyhemoglobin levels but increases mucus clearance. A true reduction of pulmonary complications is significant only after cessation of smoking for more than 8 weeks.
  - Asthma: Disease seen very commonly among surgical patients. Thorough history regarding frequency of attacks, previous history of intubation, steroid use (current or previous), current medications, and triggering factors. Perioperative bronchodilator use should be considered, as well as hydrocortisone 100 mg every 8 hr for anyone with severe asthma or taking steroids.

Reduction in postoperative pulmonary complications occurs after 8 weeks of smoking cessation.

History

Ask patients about tobacco use, asthma, sleep apnea, recent upper respiratory infection, shortness of breath, and chronic cough.

Physical Exam

- Thorough physical exam including auscultation (presence of bilateral and clear breath sounds); check for stridor, rales, or especially wheezing if history of asthma is present.
- Assessment of respiratory rate and chest excursion, and ability to complete sentences should be performed.

Evaluating Patients with Neurologic Disease

History

Ask patients about previous history of stroke, seizures, depression, nerve injury and review medications (antiseizure medications, monoamine oxidase inhibitors [MAOIs], tricyclic antidepressants [TCAs], Plavix [clopidogrel], aspirin or other blood-thinning medications). Ability to answer questions adequately indicates intact mental status.

Physical Examination

Must evaluate preop strength and sensation if postop changes are expected.

Seizure Disorders

- Characterized by paroxysmal, synchronous discharges by group of neurons in the brain.
- Preop considerations:
  - Review medications: Many antiepileptic medications have additive effect with sedatives.
  - Monitor end-organ impact of the medications: Concentrate on renal and hepatic function.
  - Avoid medications (methohexital) that can activate seizure foci.

Neuromuscular Diseases

Preop considerations: Anticipate concomitant involvement of other organs.

- Associated cardiomyopathy (Duchenne's) and conduction abnormalities (myotonic dystrophy) are common (review ECG).
- Myotonic dystrophy patients show ↑ sensitivity to respiratory depressants.
- Dysmorphic features could present difficult intubation.
- Review preop motor strength prior to administration of muscle relexant.
- Consider ↑ chance of malignant hyperthermia in myopathy patients.

Evaluation of Patients with Endocrine Disease

Diabetes Mellitus

- History should be obtained with regard to any end-organ damage (retinopathy, neuropathy, nephropathy).
- Diabetics are more prone to develop CAD with atypical presentations, perioperative MI, HTN, CHF, peripheral and cerebrovascular disease.
- Tight glucose management during perioperative period is important. Also, symptoms of hypoglycemia must be evaluated (sweating, tremors, agitation, and mental status change).
- Preop considerations: Evaluate end-organ damage (cardiovascular, pulmonary, renal). Beware silent myocardial ischemia/infarction. Must keep in mind that diabetics are more prone to:
  - Labile BP.
  - Painless myocardial ischemia.
  - Orthostatic hypotension.
  - Lack of heart rate variability.
  - Reduced heart rate response to atropine or propranolol.
  - Neurogenic, atonic bladder.
  - Gastroparesis with delayed emptying. Consider H_2 blocker or metoclopramide premed.
  - Limited-mobility joint syndrome (stiff-joint syndrome): Atlanto-occipital joint joint may be involved, which could lead to difficult intubation.
  - Difficult intubation: About 30% incidence.

Thyroid Disease

Preoperative evaluation should focus on signs and symptoms of hyper- or hypothyroidism.

- **Hyperthyroidism:** Weight loss, heat intolerance, tachycardia, atrial fibrillation, nervousness, tremor, muscle weakness, bone resorption, and diarrhea.
  - Preop considerations:
    - May postpone surgery until euthyroid.
    - Resting HR < 85.
    - Continue antithyroid medications until the morning of surgery.
- **Hypothyroidism:** Weight gain, cold intolerance, bradycardia, slow mentation, pleural/pericardial effusion.
  - Preop considerations:
    - May result in hypothermia, hypoglycemia, hypoventilation, and hyponatremia.
    - ↑ sensitivity to respiratory depressant.
    - Slow gastric emptying.
    - Must continue thyroid medications until the morning of surgery.

- **Physical exam:** Should focus on size of the thyroid gland and its effect on the airway. Large mass may distort the upper airway, especially in supine position. Chest x-ray or computed tomography (CT) scan needs to be reviewed.

- **Pheochromocytoma:**
  - Triad of symptoms: (1) headache, (2) diaphoresis, (3) tachycardia.
  - Preop considerations:
    - Vitals signs must be checked closely, and severity of symptoms must be adequately evaluated preoperatively.
    - Check for orthostatic hypotension.
    - Check for signs of myocardial ischemia.
    - More prone to be hypovolemic preoperatively.
    - β blocker only after initiation of α blockade.

Evaluation of Patients with Renal Disease

History

- Obtain etiology of renal dysfunction:
  - Primary: Glomerulonephropathy, polycystic kidney disease.
  - Secondary: Cardiac, pulmonary, hepatic-related disease.
    - Obtain information on daily urine output and fluid restriction.
    - Identify comorbidities (HTN, CAD, cerebrovascular accident [CVA]).
    - Review laboratory to determine renal reserve and metabolic derangement.
- If on dialysis, determine when the last day of dialysis was:
  - Hemodialysis better than peritoneal dialysis.
  - Dialysis on the day of surgery is optimal.
- It is important to group patients into renal function rather than renal disease:

Renal Function	Creatinine Clearance
Normal	100–120
↓ renal reserve	60–100
Mild renal impairment	40–60
Moderate renal insufficiency	25–45
Renal failure	< 25
End-stage renal disease (ESRD)	< 10

Physical Exam

- Identify signs of fluid overload:
  - Dyspnea
  - Hypoxemia
  - Generalized edema
  - Ascities
  - Juglular vein distention
- Identify signs of hypvolemia: Common in dialysis patients.
  - Tachycardia
  - Hypotension

- Narrow pulse pressure
- Dry mucous membranes
- ↓ capillary refill

- ABG: To determine degree of acidosis or severity of hypoxemia.
- ECG: To determine ischemia, hyperkalemia, or conduction abnormalities.
- Planning anesthetic plan: Characteristics of patients with renal dysfunction.
  - Less protein bounding of anesthetics.
  - More pronounced systemic effects due to azotemia.
  - Less clearance of renally excreted anesthetics.

CHAPTER 4

Anesthesia Machines

COMPONENTS

- All anesthesia machines have the same basic components (regardless of manufacturer or year of make).
- The machines consist of breathing systems, compressed gases, flow meters, vaporizers, ability to eliminate carbon dioxide, and scavenging.

ANESTHESIA CIRCUITS AND SAFETY SYSTEMS

A full O_2 cylinder is 660L at 2200 psi
A full N_2O cylinder is 1590L at 745 psi

For nonliquefied gases such as O_2, the pressure declines proportionally to the amount withdrawn. For liquefied gases such as N_2O, pressure remains constant until all the liquid has vaporized, at which point the pressure starts to drop.

*The **O_2 analyzer** in the inspiratory limb is necessary to prevent the delivery of a hypoxic gas mixture to the patient.*

- The anesthesia machine is divided into three circuits:
  - High pressure: Consists of the cylinders and cylinder primary pressure regulators.
  - Intermediate pressure: Starts at the regulated cylinder supply source, includes the pipeline supply source, and ends at the flow control valves.
  - Low pressure: Begins at the flow control valves and and includes the flow tubes, vaporizers, and one-way check valve (Datex-Ohmeda).
- The **Diameter Index Safety System (DISS)** provides threaded body fittings that are noninterchangeable connections for the gas pipelines.
- The hanger yoke assembly orients and supports the cylinder and ensures unidirectional flow of gases into the machine. This is also a color-coded system: green = oxygen, blue = nitrous oxide, gray = carbon dioxide, and yellow = air. This is the color-coded system in the United States; the colors differ internationally.
- The **Pin Index Safety System (PISS)** eliminates the possibility of placing the gas cylinder on the wrong yoke.
- **Pressure regulators:**
  - Each gas cylinder has a pressure regulator that reduces the high pressure in the gas cylinder to one that is suitable for use in the anesthesia machine. The highest pressure possible in the O_2 cylinder at 2200 psi is reduced to 45 psi. The highest pressure possible in the N_2O cylinder at 745 psi is reduced to 45 psi.
  - Second-stage pressure regulators for oxygen further reduce pressure to 12–19 psi (see Figure 4-1).
- Drager machines have a first-stage pressure regulator and a pressure relief valve. Omeda machines have a first and second stage pressure regulator as well as a pressure relief valve.
- **Check valve** is located downstream of the fresh gas inlet. It prevents reverse flow of gases from the machine to the atmosphere.
- **Fail-safe valve:**
  - Ensures that the supply pressure of all gases other than oxygen are decreased or turned off if the oxygen supply pressure ↓; an alarm is sounded to alert the anesthesiologist.
  - Remember that this does not prevent a hypoxic gas mixture from being delivered (this is why an O_2 analyzer is necessary).
- **Oxygen flush valve:** Allows direct communication between the high-pressure circuit and the low-pressure circuit. The oxygen flush valve can be used to provide jet ventilation on the condition that a one-way check valve be present between the vaporizers and the oxygen flush valve as well as a pressure relief valve upstream of the check valve. Hazards of the oxygen flush valve include:
  - Overuse can dilute the anesthetic gases and potentially result in awareness.
  - Use of the oxygen flush valve can result in barotrauma to patients' lungs.

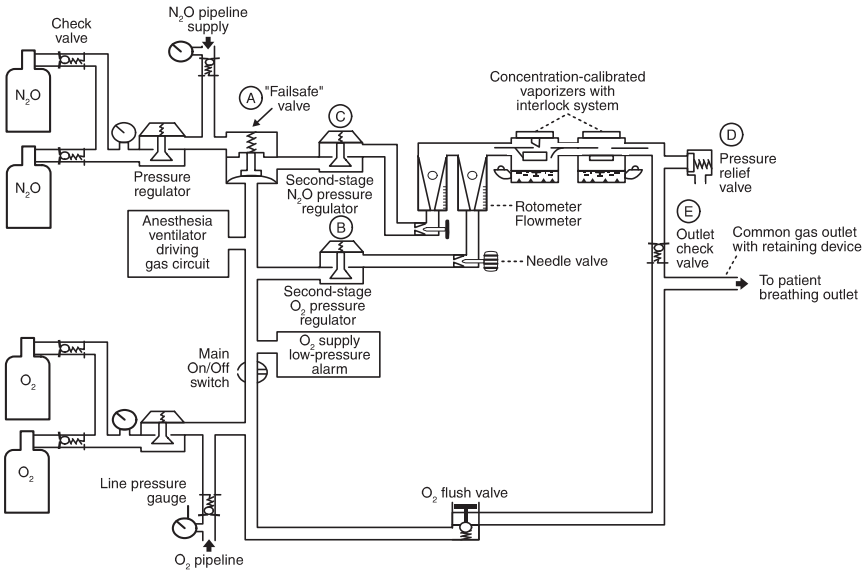

FIGURE 4-1. Internal Circuitry of Anesthesia Machines

(Reproduced, with permission, Longnecker DE et al. *Anesthesiology*. New York: McGraw-Hill, 2008: 769.)

- **Note:** Use of the oxygen flush valve to evaluate the low-pressure circuit can be misleading since closure of the one-way check valve can → undetected leaks in the low-pressure circuit.

BREATHING SYSTEMS

- Anesthesia breathing systems are classified into four categories: closed, semiclosed, open, and semiopen. These systems are comprised of materials needed to deliver volatile anesthetics, air, and oxygen from the machine to the patient.
- There are certain advantages and disadvantages of each type of breathing system based on scavenging ability/rebreathing, heat/humidity conservation, dead space, resistance, and presence or absence of valves.

Mapleson Circuits

- Mapleson circuits (semiopen) are made up four components:
  1. Breathing tubes and face mask
  2. Fresh gas inlet
  3. Pressure-limiting valve (pop-off valve)
  4. Reservoir bag
- Mapleson circuits are subdivided into letters A–F.
  - Mapleson A circuits have the pop-off valve located near the face mask and the fresh gas flow comes from the back of the circuit close to the reservoir bag.
  - Mapleson B and C circuits have the pop-off valve close to the face mask and the fresh gas inflow close to the patient.
- Mapleson D, E, and F circuits have the fresh gas inflow close to the patient and the pop-off valve back close to the reservoir bag. Mapleson F (Jackson-Rees) is

*For spontaneous ventilation: A > DEF > CB = (least rebreathing) **A**ll **D**ogs **C**an **B**ite (most rebreathing).*

*For mechanical ventilation: DEF > CB > A = (least) **D**og **B**ites **C**an **A**che (most).*

an Arye-T piece with reservoir bag and overflow valve, which is best used for transport of intubated patients and in pediatric patients.

- Different Mapleson circuits are best for either spontaneous or mechanical ventilation. Mnemonics can help to remember:
  - For spontaneous ventilation: A > DEF > CB = (least rebreathing) ***A**ll **D**ogs **C**an **B**ite* (most rebreathing).
  - For mechanical ventilation: DEF > CB > A = (least) ***D**og **B**ites **C**an **A**che* (most).
- Semiopen systems carry the *advantage* of small resistance/small dead space with an absence of valves. They have the *disadvantage* of poor scavenging ability/high flows necessary to prevent rebreathing and loss of heat and humidity. See Figure 4-2.

Bain System

- Represents a variant form of the Mapleson D circuit.
- The fresh gas inlet flows within a tube in the outer corrugated tube. Exhaled gas goes through the outer corrugated tubing and is vented by the expiratory valve located near the reservoir bag.
- Advantages of the Bain system include:
  - The fresh gas is warmed by the exhaled gas due to a countercurrent heat exchange.
  - Easy scavenging of gases due to location of pop-off valve away from the patient and close to the reservoir bag.
- Disadvantages of the Bain system include difficulty in ascertaining kinking or disconnection in the fresh gas outflow tract (the outer corrugated tubing must be transparent for this reason).

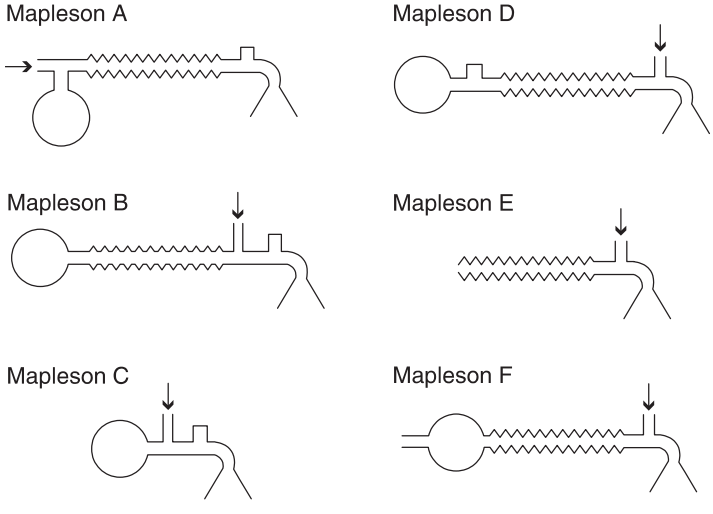

FIGURE 4-2. Mapleson circuits.

(Reproduced, with permission, Longnecker DE et al. *Anesthesiology*. New York: McGraw-Hill, 2008: 793.)

Circle System

- Components:
  - Fresh gas inflow source.
  - Inspiratory and expiratory unidirectional valves.
  - Inspiratory and expiratory corrugated tubes.
  - Y-piece connector.
  - Adjustable pressure-limiting (APL) valve.
  - Reservoir bag.
  - CO_2 absorbent.
- Rules of the circle system:
  - Unidirectional valve located between the patient and the reservoir bag on both expiratory and inspiratory limbs of the circuit.
  - Fresh gas flow inlet cannot enter the system between the patient and the expiratory limb.
  - APL valve cannot be located between the patient and the expiratory limb.
- Types of circle systems:
  - Semiclosed: Most commonly used, partial rebreathing of exhaled gas.
  - Closed: Complete rebreathing of gas after CO_2 has been reabsorbed. APL valve remains closed. Inflow gas matches that being consumed by the patient.
- Advantages of the circle system:
  - Maintenance of relatively stable inspired gas concentrations.
  - Conservation of respiratory moisture/humidity or heat.
  - Prevention of operating room (OR) pollution.

Bellows

Bellows may be classified according to their direction of movement during the expiratory phase. This direction can be ascending or descending. The ascending bellows design is the safer of the two since it does not fill if a complete disconnect occurs.

Bellows are classified according to their movement during expiration. If there is a disconnect or major leak, ascending bellows will collapse while descending bellows continue to move upward and downward.

FLOW METERS

- The purpose of flow meters is to measure gas flow to the common gas inlet as well as to control those flows.
- Physics of flow meters:
  - The space between the flow tube and the float is the annular space. Flow meters are characterized as variable orifice since they are tapered in such a way that the smallest diameter is at the bottom and the largest diameter is at the top.
  - The float is in equilibrium in the position where the downward force of gravity is equaled by the upward force of the gas flow.
  - Viscosity of the gas determines the laminar flow and density of the gas determines the turbulent flow. **Low flow → viscocity; high flow → density.**
  - When a single gas has two flow tubes, they are arranged in series and controlled by a single flow control valve.
  - Flow is read at the top of most floats with the exception of ball floats, which are read at the center of the ball.

At low flows, gas flow is dependent on viscosity; at high flows, gas flow is dependent on density. Length of the flowmeter tube makes NO difference in accuracy.

- Hazards of flow meters:
  - Leaks in the flow meter are hazardous due to their location downstream from all safety devices with the exception of the oxygen analyzer. The oxygen flow meter is located downstream of all other flow meters and closest to the common gas outlet in order to minimize the possibility of delivering a hypoxic gas mixture in the event of a leak (located on the right of other gases in the United States).
  - Inaccuracy due to sticking of the floats to the flow meter (more common in low-flow states), damaged floats, or flow meters that are not perfectly aligned in the vertical position.
  - Proportioning system: Oxygen and nitrous oxide are linked mechanically or pneumatically to have a flow rate ratio of 3:1 to prevent delivery of a hypoxic mixture.

VAPORIZERS

Physics of Vaporization

- Vapor pressure: Most volatile agents exist in the liquid state below 20°C. Vapor pressure is directly proportional to temperature and independent of atmospheric pressure.
- Desflurane is unique in that its boiling point is close to that encountered in the OR, necessitating special vaporizer designs.
- The latent heat of vaporization is the number of calories required to convert 1 gram of a liquid to the gaseous phase. Since this energy comes either from an outside source or the gas itself, the remaining liquid tends to cool due to the energy required to convert it to the gaseous phase.
- The specific heat of a substance is the number of calories required to ↑ the temperature of 1 g of a substance by 1°C. Vaporizers are constructed of metals that have high specific heat in order to minimize the ↓ in temperature (due to the latent heat of vaporization) associated with vaporization.
- Thermal conductivity is the speed at which heat travels through a substance. The metals that compose a vaporizer have high thermal conductivity and maintain a constant temperature.

Measured Flow Vaporizer

- The copper kettle is an example of a measured flow vaporizer. Copper is used since it has a high specific heat and thermal conductivity, thus keeping the temperature of the vaporizer constant.
- Vaporizer output is controlled by a dedicated flow meter. When the flow meter valve is turned on, all the carrier gas enters the vaporizer, becoming saturated with anesthetic vapor.
- This saturated vapor that leaves the copper kettle is then combined with fresh gas downstream so the concentration of the inhalation anesthetic is diluted to reach clinical levels.
- The disadvantage of the copper kettle is that if the total gas flow were to fall, the anesthetic concentration could reach dangerous levels.

Variable Bypass Vaporizer

- The vaporizers that are specific for the delivery of halothane, enflurane, isoflurane, and sevoflurane are referred to as variable bypass.

- There are two routes in these vaporizers for gas flow (splitting of total fresh gas flow): the bypass chamber and the vaporizing chamber.
- Turning the dial on the vaporizer alters the proportion of gas channeled into these chambers. The gas going into the vaporizing chamber becomes saturated with anesthetic gas.
- These vaporizers are characterized by:
  - Method of vaporization.
  - Temperature compensation.
  - Agent specific (equipped to accommodate one type of gas).
  - Out of circuit (located outside the breathing circuit).
  - Flow over.

Desflurane Tec 6 Vaporizer

- Desflurane has the following features:
  - A boiling point of 22.8°C (close to room temperature).
  - Vapor pressure is three to four times greater than that of other inhaled anesthetics.
  - Miminum alveolar concentration (MAC) of 6.6% (low potency).
  - Low blood gas solubility coefficient (0.45).
- For this reason, the desflurane vaporizer is pressurized and heated.
- The desflurane Tec 6 vaporizer has two gas circuits arranged in parallel: a fresh gas circuit and a vapor circuit.
- In contrast to variable bypass vaporizers, the desflurane Tec 6 requires manual adjustments in the concentration control dial to maintain a constant concentration of gas at altitudes other than sea level.

The desflurane Tec 6 vaporizer warms and pressurizes liquid desflurane to 1500 mmHg.

Factors Influencing Vaporizer Output

- Low flow rates (< 250 mL/min) cause vaporizer output to be less than the dial setting due to insufficient turbulence in the vaporizing chamber.
- High flow rates (> 15 L/min) result in the vaporizer output's being less than the dial setting due to incomplete mixing of the carrier gas with the volatile anesthetic in the vaporizing chamber.
- Temperature is compensated for in most vaporizer designs. The higher the temperature, the more carrier gas is shunted through the bypass chamber.
- Positive pressure ventilation or oxygen flushing can cause the vaporizer output to be greater than the dial setting due to intermittent back pressure. This is known as the **pumping effect.**
- Vaporizer output is influenced by carrier gas composition.
- Tipping of the vaporizers can cause an ↑ vapor concentration, though tipping is unlikely because the vaporizers are secured to the anesthesia machines themselves.

One way check valves between vaporizers and O_2 flush valve limit the pumping effect.

E CYLINDERS

- Anesthesia machines are equipped with E cylinders of compressed gases for use in case the wall (central) supply fails or malfunctions.
- One can calculate the amount of gas present in the tanks by virtue of knowing the pressure and vice versa. One is also able to perform additional calculations with regards to length of time and amount of gas a tank has left (see Table 4-1).

TABLE 4-1. E Cylinders

	Oxygen	Nitrous Oxide	Air	Carbon Dioxide
Contents of cylinder (L)	625	1590	625	1590
Pressure in cylinder/ full (psi)	2000	750	1800	750

CO_2 ABSORBERS

- Types of absorbents:
  - Soda lime (most commonly used).
  - Baralyme.
  - Calcium hydroxide/calcium chloride (Amsorb).
- Soda lyme composition: 80% calcium chloride, 15% water, 4% sodium hydroxide, and 1% potassium hydroxide.
  - Small amounts of silica added for hardness (and minimize alkaline dust).
  - Chemical neutralization equations:

$$CO_2 + H_2O \rightarrow H_2CO_3$$
$$H_2CO_3 + 2NaOH \rightarrow Na_2CO_3 + H_2O + Heat$$
$$H_2CO_3 + Ca(OH)_2 \rightarrow CaCO_3 + 2NaOH$$

- Baralyme composition: 20% barium hydroxide and 80% calcium hydroxide.
  - Baralyme may produce fires when used with sevoflurane.
  - Chemical neutralization equations:

$$CO_2 + H_2O \rightarrow H_2CO_3$$
$$H_2CO_3 + Ba(OH)_2 \rightarrow BaCO_3 + 2H_2O + Heat$$
$$H_2CO_3 + CA(OH)_2 \rightarrow CaCO_3$$

Carbon monoxide formation is a result of volatile agent degradation by strong bases present in carbon dioxide absorbents.

- Amsorb consists of calcium hydroxide and calcium chloride.
  - Primary advantage lacks strong bases sodium and potassium hydroxide. This eliminates production of carbon monoxide, compound A, and possibility of fire in breathing circuit.
  - Disadvantages are less absorptive capacity and higher cost.
- Carbon monoxide production is a hazard when using desiccated CO_2 absorbent.
- Factors that ↑ the risk of carbon monoxide production are:
  - Desiccated CO_2 absorbent.
  - High temperatures in the CO_2 absorbent.
  - The type of inhalational agent used ↑ carbon monoxide production in the following order: desflurane > enflurane > isoflurane > halothane = sevoflurane.
- Low fresh gas flows.
- The type of absorbent (Baralyme ↑ the amount of CO produced).

- The concentration of the volatile agent (the greater the concentration, the greater the CO produced).
- Compound A is a nephrotoxic degradation product produced when sevoflurane reacts with CO_2 absorbent (found in animal studies). Factors that ↑ the production of compound A are:
  - Use of Baralyme.
  - High temperatures in the CO_2 absorbent.
  - Dehydration of Baralyme (**Note:** Dehydration of soda lime ↓ Compound A production).
  - Low flow of fresh gas (< 2 L/min).
  - High sevoflurane concentration in the anesthetic circuit.
- Factors that can ↑ efficiency of CO_2 absorbents include:
  - Granule size: Optimal size is 4–8 mesh. Absorptive capacity ↑ as granule size ↓. If granules are too small, resistance ↑.
  - pH sensitive dye: This dye is added to the lime so that when exhaustion of soda lime or Baralyme occurs, the anesthesiologist is alerted (by violet color) and will change the container as to prevent rebreathing of exhaled gases.
  - Channeling: This is the passage that gases pass through in the canister (path of least resistance), though the bulk of CO_2 bypasses the granules. Firm packing of the granules in the canister prevents channeling.

Large granules cause channeling and ↓ capacity for absorption.
Small granules ↑ resistance to CO_2 absorption.

ALARMS

Anesthesia machines have alarms that are activated when the O_2 pressure ↓ below a certain level, such as 30 psi. Other alarms include:

- Disconnect alarms.
- Low- and high-pressure ventilator alarms.
- Alarms connected to patient monitors (end-tidal carbon dioxide [$ETCO_2$], pulse oximetry, blood pressure, etc.).

*The low-pressure monitor alarm will be **first** to alarm with disconnect since critical airway pressures will not be reached with ventilatory cycle.*

MECHANICAL VENTILATION

- Positive-pressure ventilation works by applying positive pressure to the lungs, either indirectly through a mask or directly through an endotracheal tube or tracheotomy tube.
- This creates a periodic pressure gradient between the ventilator and the alveoli that results in inspiration. Expiration occurs passively.
- Positive-pressure ventilation has several disadvantages, including:
  - ↑ in ventilation-perfusion (V/Q) mismatch due to ventilation favoring the more compliant nondependent areas of the lung and perfusion favoring the dependent areas of the lung.
  - ↓ in cardiac output due to a ↓ in venous return to the heart during the inspiratory phase.
  - The possibility of barotrauma.

VENTILATION MODES

- **Controlled mechanical ventilation (CMV):**
  - Provides fixed respiratory rate (RR) and tidal volume (V_T).
  - Mode is reserved for patients who are not capable of respiratory effort.

- If a patient is capable of inspiratory effort, sedation and paralysis are necessary.

- **Assist control (AC) ventilation:**
  - Pressure sensor is incorporated that senses a minimum inspiratory effort by the patient.
  - When patient effort is sufficient, the ventilator delivers a fixed V_T.
  - If patient does not have sufficient inspiratory effort, controlled mechanical ventilation is delivered.
- **Synchronized intermittent mechanical ventilation (SIMV):**
  - Allows the patient to take spontaneous breaths while giving a minimum number of mechanical breaths.
  - SIMV also synchronizes the mechanical breaths to coincide whenever possible with a spontaneous inspiratory effort.
  - Prevents the juxtaposition of spontaneous and mechanical breaths (stacking), which would result in large tidal volumes.
  - Advantages of SIMV are greater ease of weaning and patient comfort.
  - The disadvantage is that at low respiratory rates, patients with poor inspiratory effort can be underventilated.
- **Pressure support ventilation (PSV):**
  - Allows the patient to breathe spontaneously while augmenting the patient's tidal volume with a predetermined positive pressure.
  - ↓ the work of breathing and overcomes the resistance of the circuit and the ventilator.
  - Disadvantages of PSV include the inability to vary the respiratory rate (which is entirely dependent on the patient's own respiratory effort). Therefore, a weak patient who cannot initiate sufficient numbers of breaths will be underventilated.
- **Pressure control ventilation (PCV):**
  - Similar to PSV in that the patient's V_T is supplemented by set positive pressure; however, the RR may be adjusted in PCV.
  - Peak airway pressures may therefore be limited and the likelihood of barotrauma is ↓.
  - Disadvantage is that the V_T is dependent on the compliance of the lungs.
- **Inverse I:E ratio ventilation (IRV):**
  - Reverses the inspiratory-to-expiratory ratio from 1:3 to 1:1 by adding an inspiratory pause.
  - Air trapping produced by incomplete emptying of the lungs on expiration ↑ the functional residual capacity (FRC) and producing an intrinsic positive end-expiratory pressure.
- **Airway pressure release ventilation (APRV):**
  - Alternates between a high and low PEEP setting and permits the patient to breathe spontaneously.
  - Advantages of APRV are less incidence of barotrauma and a limitation of circulatory depression and the use of sedation.
- **High-frequency jet ventilation (HFJV):**
  - High-pressure gas is delivered through cannula at a rate of 120–600 times/min, producing a V_T at or below anatomic dead space.
  - HFJV is used in laryngeal, tracheal, or bronchial procedures as well as instances in which the airway could not be secured by conventional methods.
  - Disadvantages include inadequate heating or humidification of inspired gases during prolonged use of HFJV.

Drawbacks associated with high-frequency ventilations:

Barotrauma

Poor humidification of gases

No scavenging of anesthetic agents

Difficulty in monitoring ventilation

PEEP AND CPAP

- Main indications for positive airway pressure therapy are to ↑ the FRC, to improve V/Q mismatch, and to ↑ lung compliance.
- Two modalities are generally used to achieve this:
  - **Positive end-expiratory pressure (PEEP):** Applied during the expiratory phase of a mechanically delivered breath.
  - **Continuous positive airway pressure (CPAP):** Applied to both the inspiratory and expiratory phases during spontaneous breathing.
- If CPAP is applied to a patient without an advanced airway, the pressure should be set at < 15 cm H_2O and only in patients with intact respiratory reflexes due to the risk of aspiration.
- The advantage/disadvantages of CPAP over PEEP include:
  - CPAP results in lower peak inspiratory pressures than PEEP.
  - The disadvantage of CPAP is that it provides less support.
- There is a higher incidence of barotrauma when CPAP and PEEP levels exceed 20 cm H_2O.
- Other risks include an ↑ in dead space, an ↑ in pulmonary vascular resistance, and a concomitant ↑ in right ventricular afterload.
- This ↑ in right ventricular afterload together with a ↓ in left ventricular preload results in a ↓ in cardiac output.
- An ↑ in central venous pressure also ↑ intracranial pressure.

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CHAPTER 5

Monitoring and Equipment

PULSE OXIMETRY MONITORING

Deoxyhemoglobin absorbs more light in the red band (660 nm). Oxyhemoglobin absorbs more light in the infrared band (940 nm). The red-to-infrared ratio is used to calculate SpO_2.

- Measurement of peripheral oxygen saturation of hemoglobin and pulse rate.
- **Technique:** Plethysmography and spectrophotometric analysis is combined to measure the relative absorption of red and infrared light wavelengths. Absorption of specific wavelengths of light relative to the pulsatile oxyhemoglobin signal is transmitted to a photo detector. The oxygen saturation (the ratio of oxyhemoglobin to deoxyhemoglobin) is calculated from absorption curves programmed into the device.
- Accuracy is affected by multiple variables:
  - Absence of pulsatile waveform.
  - ↑ carboxyhemoglobin.
  - ↑ methemoglobin.
  - Shivering.
  - Bright external light.
  - Methylene blue.
  - Fingernail polish (blue/dark red).
  - Hypoxia.
  - Hypotension.
  - Hypothermia.
  - Hypovolemia.
- The relationship that exists between hemoglobin saturation and arterial oxygen tension (mmHg) is reflected in the oxyhemoglobin dissociation curve, as seen in Chapter 2, Figure 2-2.

CarboxyHb is viewed as oxyhemoglobin and therefore SpO_2 = 100%. Methemoglobin absorbs red and infrared light at 1:1 ratio; therefore the SpO_2 = 85%.

ELECTROCARDIOGRAM (ECG)

- Used for detection of dysrhythmias, myocardial ischemia, electrolyte imbalance, and pacemaker function.
- A 12-lead system can be used pre- and postoperatively to detect areas of ischemia:
  - II, III, aVF (inferior leads): Right coronary artery.
  - V_2, V_3, V_4 (anterior leads): Left anterior descending artery.
  - I, aVL, V_5, V_6 (lateral leads): Circumflex artery.
  - V_1, V_2 (septal leads): Right coronary and left anterior descending arteries.
- **A 5-lead system with both lead II and V_5 provides 80–96% sensitivity for detection of intraoperative ischemic events.**
  - Lead II (level of atria): Detection of dysrhythmias, inferior ischemia, P wave abnormalities, electrolyte imbalance.
  - Lead V_5 (5th intercostal space, anterior axillary line): Detection of anterior or lateral wall ischemia.
- ST-segment analysis: Criteria for ischemia:
  - Flat or downsloping of ST depression.
  - 1–2 mm and > 60–80 msec after the end of QRS with or without T-wave inversion in V5.
  - ST segment elevation with peaked T waves.
- ST segment analysis is not useful in:
  - Wolff-Parkinson-White (WPW) syndrome.
  - Bundle branch blocks.
  - Extrinsic pacemaker capture.
  - Digoxin therapy.

NONINVASIVE BLOOD PRESSURE MONITORING (NIBP)

- The cuff is inflated greater than systolic pressure to stop arterial blood flow and then slowly deflated.
- The cuff should cover the two-thirds of the upper arm or thigh; the width of the cuff should not be 50% greater than the diameter of the limb.
  - A narrow cuff may give falsely high values.
  - A wide cuff may give falsely low values.
- **Techniques:**
  - Auscultation: **Korotkoff sounds** are audible through a stethoscope placed under or just beyond the distal third of an inflated blood pressure.
  - Automated devices can measure oscillations in cuff pressure (oscillotonometry).
  - Mean pressure correlates well with the pressure at which the maximal oscillation occurs. Venous congestion may occur if the instrument is set to cycle too frequently.

Mean arterial pressure correlates with the pressure at which maximal oscillations occur.

TEMPERATURE MONITORING

- Temperature monitoring sites:
  - Esophageal: Distal one-third to one-fourth of esophagus.
  - Nasopharynx: Adjacent to nasopharyngeal mucosa, reflecting carotid blood passing nearby.
  - Tympanic membrane: Blood supply to this area is derived from posterior auricular and internal maxillary arteries, which are direct branches of external carotid artery.
  - Pulmonary artery: Reflects core temperature.
  - Rectal, oral, axillary, bladder, and skin: Temperatures are not indicators of core temperature.
- Hyperthermia occurs infrequently:
  - Exposure to endogenous pyrogens.
  - Thyrotoxicosis.
  - Pheochromocytoma.
  - Malignant hyperthermia.
  - Anticholinergic blockade of sweating.
- Perioperative hypothermia commonly occurs:
  - Anesthetic-induced inhibition of thermoregulation.
  - Cold ambient environment of the operating room.
  - Heat loss due to surgical exposure of tissues.
  - Risk of hypothermia ↑ in elderly, neonates, burn patients, and patients with spinal cord injuries.
- Phases of heat loss during anesthesia:
  - **Phase I: Redistribution phase:**
    - Core temperature ↓ 1–2°C during first hour under general anesthesia. Heat is redistributed from warm central compartments (abdomen, thorax) to peripheral compartments (arms and legs).
    - Anesthetic medications cause direct peripheral vasodilatation and inhibit centrally mediated thermoregulatory constriction.
  - **Phase II: Heat loss phase:**
    - Three- to four-hour linear ↓ in core temperature.
    - Results from heat loss exceeding metabolic heat production.
  - **Phase III: Steady-state equilibrium phase:** Core temperature plateau that usually develops after 4 hours of anesthesia that is maintained passively or actively:
    - Passive plateau results when metabolic heat production equals heat loss.

Mechanisms of heat loss:
Radiation—dissipation of heat to cooler surroundings.
Largest component.
Conduction—direct skin contact.
Convection—heat loss to air flow.
Evaporation—heat of vaporization.

  - Active mechanism causes vasoconstriction to ↓ heat loss and to alter distribution of heat within the body.
- Effects of hypothermia:
  - **33–35°C:**
    - Significant protection against cerebral ischemia and hypoxemia.
    - Slows metabolic rate and metabolism of drugs.
    - Inhibits immune function and macrophage phagocytosis and impairs wound healing.
  - 28–33°C:
    - Reversibly ↑ prothrombin time (PT) and partial thromboplastin time (PTT).
    - Bleeding time is prolonged due to local cutaneous hypothermia.
  - < 28°C:
    - ↑ blood glucose.
    - ↑ hematocrit and blood viscosity (3% ↑ in viscosity per degree ↓).
    - ↑ hemoglobin binding to oxygen (left shift).
    - ↓ in antidiuretic hormone (ADH) and intravascular volume.
    - ↓ in oxygen consumption by about 50%.
    - ↓ in carbon dioxide (CO_2) production.
    - ↓ cerebral metabolic rate of oxygen ($CMRO_2$) (10% ↓ per degree) and cerebral blood flow (CBF).
    - ↓ platelet count and platelet function.
- Prevention of intraoperative hypothermia:
  - Much more effective than treatment.
  - Prewarming (prior to induction) will ↓ initial temperature drop.
  - Active airway humidification.
  - Covering layers of blankets to prevent evaporation can reduce heat loss by 30% (single layer) to 50% (three layers). The total body surface of covering is more important than type of covers.
  - Warmed IV fluids.
  - **Forced-air convective warming is most effective.**

PULMONARY MONITORING

End-Tidal Carbon Dioxide ($ETCO_2$) Monitoring

- Capnography is the measurement of CO_2 in each breath of the respiratory cycle.
  - The capnograph displays a waveform of CO_2 and the value of the CO_2 at the end of expiration, known as the end-tidal CO_2.
  - Assesses the adequacy of ventilation, detects esophageal intubation, indicates disconnection of the breathing circuit, helps to diagnose circulatory problems and malignant hyperthermia.
  - If the patient has a stable cardiac status, stable body temperature, absence of lung disease, and a normal capnograph trace, $ETCO_2$ approximates the partial pressure of CO_2 in arterial blood ($PaCO_2$).
  - The normal gradient between $ETCO_2$ and $PaCO_2$ is 5–10 mmHg and reflects the alveolar dead space (alveoli that are ventilated but not perfused).
- ↑ $ETCO_2$:
  - Hypoventilation.
  - Hyperthermia.
  - Malignant hyperthermia.
  - Hypermetabolic states.

- Sepsis.
- Rebreathing.
- ↑ skeletal muscle activity.

- ↓ $ETCO_2$:
  - Hyperventilation.
  - Hypothermia.
  - Hypoperfusion.
  - Pulmonary embolism.
  - Cardiac arrest.
  - Disconnect, leakage, or partial obstruction (sudden drop).

In most cases, $ETCO_2$ will closely follow $Paco_2$ with a gradient of < 5.

Causes of increase $Paco_2$ to $ETCO_2$ gradient:

1. *↓ dead space ventilation.*
2. *Esophogeal intubation.*
3. *Decreased cardiac output.*
4. *Disconnect or leak.*

MEASUREMENT TECHNIQUES

- **Infrared absorption spectroscopy** is the most commonly used. Gases of molecules that contain at least two dissimilar atoms absorb infrared radiation. CO_2 absorbs infrared radiation at a wavelength of 4.3 mm. The amount of infrared radiation absorbed is proportional to the number of CO_2 molecules (partial pressure of CO_2) present in the chamber, according to the Beer-Lambert Law. This allows the calculation of CO_2 values.
- **Photo-acoustic spectroscopy** irradiates the gas sample with pulsatile infrared radiation. The periodic expansion and contraction produces a pressure fluctuation of audible frequency that can be detected by a microphone.
- **Capnography tracing** (as seen in Figure 5-1):
  - Phase I: Inspiratory baseline (anatomic dead space), beginning of expiration.
  - Phase II: Expiratory upstroke.
  - Phase III: Expiratory plateau (highest point is $ETCO_2$), #4 in figure.
  - Phase IV: Inspiratory downstroke.
- Abnormal traces:
  - **Rebreathing** (Figure 5-2): Waveform does not return to baseline.
    - Fresh gas flow too low.
    - Incompetent expiratory valve.
    - Exhausted CO_2 absorbent.
  - **Obstructive** (Figure 5-3): No plateau is reached. Obstructive airway disease.
  - **Oscillations** (Figure 5-4): Cardiac impulses transmitted to capnograph.
  - **Curare cleft** (Figure 5-5): Deep indentation during plateau phase. Spontaneous respiratory effort.

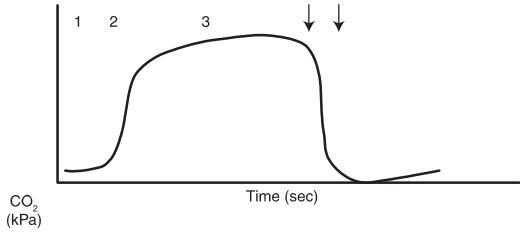

FIGURE 5-1. Capnography tracings.

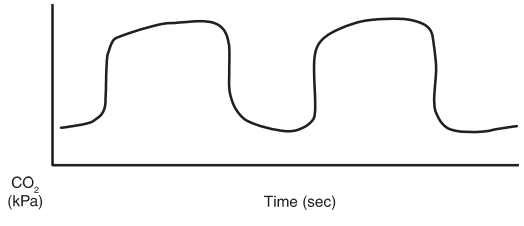

FIGURE 5-2. Capnograph (rebreathing).

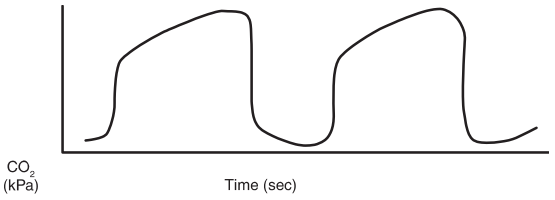

FIGURE 5-3. Capnograph (obstructive)

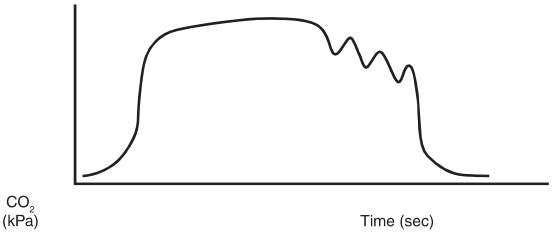

FIGURE 5-4. Capnograph (cardiac oscillation).

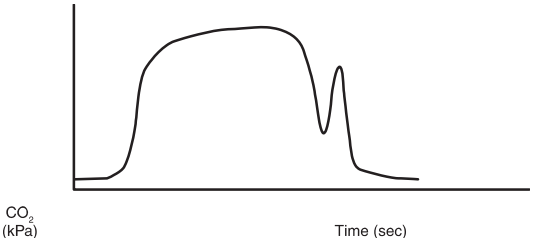

FIGURE 5-5. Capnograph (curare cleft).

NEUROMUSCULAR BLOCKADE TWITCH MONITORS

- Assesses neuromuscular function while using intermediate-acting muscle relaxants.
- Peripheral nerve stimulator delivers a current with variable frequency and amplitude.
- Common sites:
  - Ulnar nerve stimulation for adductor pollicis muscle.
  - Facial nerve stimulation for orbicularis oculi muscle (more closely reflects blockade of the larynx).
- **Train of four (TOF):**
  - Height of the fourth twitch is measured against that of the first twitch.
  - > 0.7 correlates with adequate return of muscular function.
  - Does not diminish with use of succinylcholine (no fade).
  - **Tetanus:**
    - Continuous stimulation for 5 seconds at 50 Hz.
    - Response fades only with nondepolarizing drugs.
    - Sustained tetanus is present at a TOF of > 0.7.

INVASIVE MONITORS

Invasive Arterial Blood Pressure Monitoring (IABP)

INDICATIONS

- Rapid BP changes are anticipated (major fluid shifts).
- Need for multiple arterial blood gases.
- Failure of indirect monitoring (burns).
- Intra-aortic balloon counterpulsation.
- Induced hypotension.
- Cardiac surgery.
- Major vascular surgery.

Contraindications

- No collateral blood flow.
- Local infection.
- Vascular insufficiency.
- Raynaud's phenomenon.

An underdamp waveform is hyperresonant and therefore → falsely high systolic BP and falsely low diastolic BP. An overdamp waveform → falsely low BP.

Waveform

- Periodic complex wave reproduced by Fourier analysis.
- Damping prevents the system from overshooting after responding to a change.
  - Overdamping: Slurred upstroke, absent dicrotic notch, and loss of fine detail. Causes include blood clots, air bubbles in the tubing, and kinked catheters.
  - Under damping: Exaggerated peaks and troughs in the waveform, falsely high systolic pressures and low diastolic pressures. Causes include long connecting lines (> 1.4 mm), small tubing (< 1.5 mm internal diameter), or the catheter occluding the vessel.
- Rate of upstroke indicates contractility.
- Rate of downstroke indicates peripheral vascular disease.
- Variations during respiratory cycle indicate hypovolemia.
- Artery selection (as pulse moves peripherally, systolic and pulse pressures are more exaggerated).
  - Radial: Preferred because of its superficial location and collateral flow. Must check ipsilateral ulnar artery flow before cannulation.
  - Ulnar: Deeper, more tortuous course. Must check ipsilateral radial artery flow before cannulation.
  - Brachial: Large and easily identifiable, but close to the elbow making catheters more prone to kinking and to thrombosis.
  - Femoral: Prone to pseudo-aneurysm and atheroma formation, ↑ incidence of infection and arterial thrombosis.
  - Dorsalis pedis and posterior tibial: Most distorted waveforms.
  - Axillary: Nerve damage can result from a hematoma or traumatic cannulation.

***Allen's test** is a simple but not reliable method of determining adequacy of ulnar collateral circulation.*

Complications

- Hematoma
- Thrombosis
- Vasospasm
- Air emboli
- Skin necrosis
- Infection
- Loss of digits
- Intra-arterial injection

CENTRAL VENOUS CATHETER

Central venous pressure (CVP) is measured at the junction of the vena cava and the right atrium. Approximates the right atrial pressure, which determines the right ventricular end-diastolic volume.

Locations

- Subclavian vein
- Internal jugular vein.

Indications

- Fluid management
- Rapid infusion of large volumes
- Parenteral nutrition
- Aspiration of air emboli
- Transvenous pacing
- Temporary hemodialysis
- Chemotherapy

Giant "A" waves result from atrial contraction against a closed tricuspid valve seen in heart block, junctional rhythms, or tricuspid stenosis. Large "V" waves result from tricuspid regurgitation.

Contraindications

- Tricuspid valve vegetations.
- Renal cell tumor in right atrium.
- Ipsilateral carotid endarterectomy.
- Waveform (corresponds with cardiac events; see Figure 5-6).
  - **A wave:** Atrial contraction. Absent in atrial fibrillation. Larger in tricuspid stenosis, right ventricular hypertrophy, pulmonary hypertension, heart block, junctional rhythms.
  - **C wave:** Early right ventricular contraction, tricuspid valve elevation.
  - **V wave:** Atrial filling against a closed tricuspid valve. Larger in tricuspid regurgitation, constrictive pericarditis, tamponade.
  - **X descent:** Atrial relaxation.
  - **Y descent:** Tricuspid valve opening.

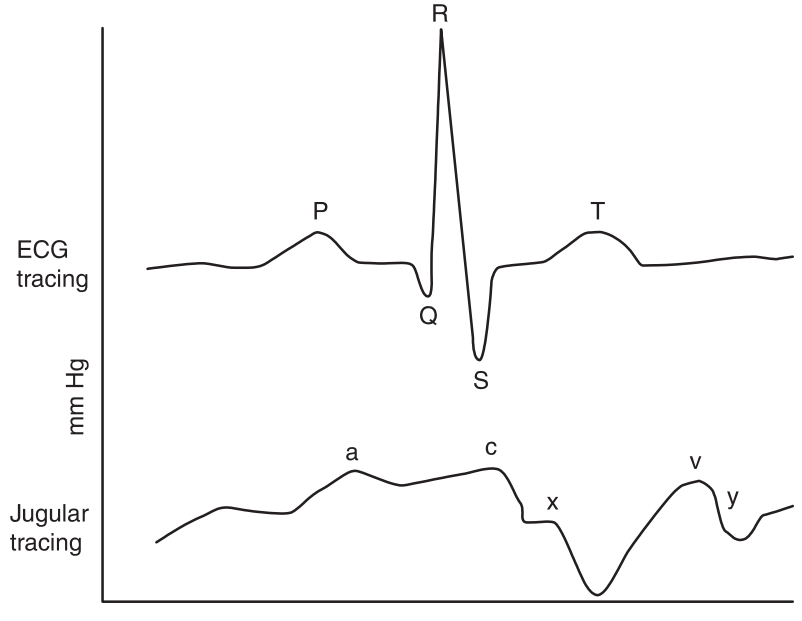

FIGURE 5-6. CVP waveform.

Complications

- Infection.
- Air emboli.
- Dysrhythmias.
- Cardiac perforation.
- Thrombosis.
- Trauma to adjacent arteries and nerves.
- Pneumothorax.
- Chylothorax.
  - Cardiac tamponade.
- **CVP values:**
  - Used for measurement of right ventricular end-diastolic pressure (RVEDP).
  - Venous pressure should be measured during end expiration. A normal value in a spontaneously breathing patient is approximately 5–10 cm H_2O. This rises about 3–5 cm H_2O during controlled ventilation.
  - Can be used to differentiate types of shock.
    - Hypovolemic: Low CVP, low cardiac index (CI), high systemic vascular resistance (SVR).
    - Cardiogenic: High CVP, low CI, low SVR.
    - Vasogenic: Low CVP, high CI, high SVR.

PA catheterization can cause right bundle branch block (RBBB) → complete heart block in patients with LBBB.

Pulmonary Artery Catheter (PAC)

Used to indirectly assess left ventricular end-diastolic pressure (LVEDP) in patients whose CI, preload, volume status, or mixed venous saturation need to be known.

Indications

- **Cardiac disease:** Coronary artery disease (CAD) with left ventricular dysfunction, recent myocardial infarction (MI), valvular disease, heart failure.
- **Pulmonary disease:** Acute respiratory distress syndrome (ARDS), severe chronic obstructive pulmonary disease (COPD).
- **Fluid management:** Shock, trauma, acute renal failure, burns, hemorrhagic pancreatitis.
- **Surgical procedures:** Coronary artery bypass graft (CABG), valve repair, aortic aneurysm repair, major vascular surgery, sitting craniotomies, portal systemic shunts.
- **Obstetrics:** Placental abruption, severe toxemia.

There is a good correlation between the CVP and PCWP with good LV function, no wall motion abnormalities, and no pulmonary disease (normal PVR).

Contraindications

- Left bundle branch block (may need a pacing catheter).
- WPW or Ebstein's malformation.
- Bacteremia.
- Right heart mass.
- Tricuspid or pulmonary mechanical valves.
- Frequent arrhythmias.
- Pulmonic stenosis
- Newly inserted transvenous pacemaker.
- Bifascicular heart block.
- Coagulopathy.

Complications (Risk ↑ with ↑ Catheter Duration):

- Infection
- Thrombosis
- Endocarditis
- Conduction abnormalities
- Pulmonary infarction
- Pulmonary artery rupture:
  - Sudden onset of hemoptysis.
  - Incidence 0.2% (risk ↑ if balloon is left in permanently wedged position).
  - Risk factors include pulmonary hypertension, age > 60, anticoagulation therapy.
  - Management includes left lateral decubitus positioning, intubation with double lumen endotracheal tube (ETT), positive end-expiratory pressure (PEEP), embolization via bronchoscopy or lobectomy.
  - Mortality rate is 50-70%.

Waveform

- Pulmonary artery pressure (PAP):
  - Initial positive upstroke: RV systole.
  - Dicrotic notch: Pulmonary valve closes.
  - Normal PA systolic pressure 20–30 mmHg.
  - Elevated PAP: Hypervolemia, LV failure, pulmonary hypertension, mitral valve (MV) disease, vasoactive medications, cross-clamp of aorta, light anesthesia.
- Pulmonary capillary wedge pressure (PCWP):
  - Nonpulsatile tracing in smaller branch of PA occluding blood flow.
  - Tip should be in zone 3 of the lung for most accurate pressures ($P_{alv} < P_{pa}$ and P_{pv}).
  - Reflection of left atrial pressure, which reflects left ventricular end-diastolic volume (LVEDV) or LVEDP.
  - Not accurate with MV disease, aortic valve (AV) disease, atrial myxoma, tamponade, right-to-left shunts.
  - Measurements should be obtained at end expiration (intrathoracic pressure is closest to 0).
  - For PEEP > 10 cm H_2O, PCWP = measured PCWP – ½ PEEP/1.36.

Cardiac Output (Normal 3.3–3.5)

Cardiac output (CO) equals oxygen consumption per minute (VO_2) divided by arterial oxygen content (Ca_{O2}) minus mixed venous oxygen content (Cv_{O2}).

- **Techniques:**
  - **Indicator dilution:** A known amount of dye (indocyanine green) is injected into the PA. Arterial blood is withdrawn from the aorta as the dye circulates, and a concentration-versus-time curve is derived. The first-pass curve is used to determine CO.
  - **Thermodilution:** A saline bolus of known volume (5–10 cc) and temperature (usually ≤ 25°C) is injected through the proximal (RA) lumen. The thermistor at the end of the PAC monitors the change in blood temperature, and a temperature-versus-time curve is generated. May be inaccurate in tricuspid regurgitation, intracardiac shunts, atrial fibrillation
- Cardiac index (CO normalized for patient size): CI = CO / BSA.

Mixed Venous Oximetry

- Estimates tissue oxygen balance.
- Normal value is 75%, indicating tissue extraction of oxygen of 25%.
- $SvO_2 = SaO_2 - Vo_2/(Hb \times 13.8) \times CO$.

CARDIAC MONITORS

Transesophageal Echocardiography (TEE; Figure 5-7)

- May be used to determine the etiology of acute hypotension such as LV dysfunction, hypovolemia, peripheral vasodilatation, pulmonary embolism, aortic dissection.
- Provides information on function and structure of cardiac valves.
- Ischemia manifests within seconds on TEE, before changes occur on ECG or PA catheter monitoring.

The transgastric short axis view is most optimal to monitor intraoperative myocardial ischemia since it provides a view of all three coronary artery distributions.

Indications

- Acute hemodynamic disturbances.
- Cardiac valve repair.
- Congenital heart surgery.
- Hypertrophic obstructive cardiomyopathy (HOCM) repair.
- Thoracic aortic aneurysm.

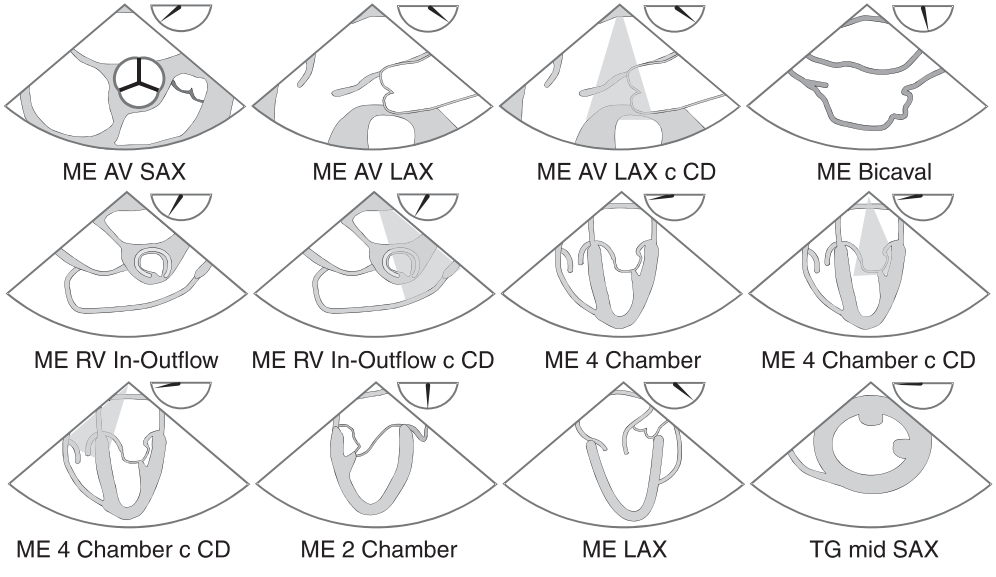

FIGURE 5-7. TEE Views

The eight basic cross-sections in TEE and four cross-sections with color Doppler (CD): ME, midesophageal; TG, transgastric; AV, aortic valve; SAX, short axis; LAX, long axis; RV, right ventricle; In-Outflow, inflow and outflow; mid SAX, midventricular short axis. (Reproduced, with permission, from Shanewise JS et al. ASE/SCA IOE TEE Guidelines. *Anesth Analg.* 1999; 89: 870–884.)

Bispectral Index (BIS)

- Method of electroencephalogram (EEG) analysis validated as an objective measure of sedation depth in the operating room with inhalational or IV anesthetics using superficial scalp electrodes.
- Unitless scale from 0 to 100:
  - 0: Coma
  - 40–60: General anesthesia
  - 60–90: Sedated
  - 100: Awake
- Not reliable in predicting movement to a noxious stimulus.
- Suggests high or low probability of intraoperative awareness.

EEG

↓ amplitude or slowing of the EEG may be a sign of CNS hypoxia.

- **Waveforms:**
  - Delta: ≤ 3 Hz, seen during deep sleep.
  - Theta: 3.5–7.5 Hz, seen during sleep.
  - Alpha: 8–13 Hz, most prominent with relaxation, disappear with attention.
  - Beta: > 13 Hz, augmented by barbiturates and benzodiazepines.
- Stages with general anesthesia:
  - Premeds: ↑ beta activity and then ↑ slowing.
  - Induction: Frontal intermittent rhythmic delta activity (FIRDA) often is observed, then diffuse faster activity is seen, typically slowing from beta to alpha frequencies, superimposed on variable theta and delta.
  - Deep: Burst suppression.
- **Changes:**
  - Diffuse: Change in anesthetic depth, blood pressure, or cerebral blood flow.
  - Localized: Focal ↓ in cerebral blood flow resulting from either acute change in vessel caliber or hypotension in the setting of a fixed stenosis.
- **Clinical uses:**
  - Carotid endarterectomy: Guides to acute changes in cerebral blood flow that occur during carotid cross-clamping. These usually are seen within 30 sec and indicate a need for shunting.
  - Aneurysm repair.
  - Hypothermic circulatory arrest for cardiac surgery.

Electromyography (EMG)

Indicates function of innervating nerve of muscle fibers.

- **Technique:** Multiple sterile, stimulating EMG needles are placed into the muscles to be examined. Spontaneous or induced EMG activity is recorded continually with a low noise amplifier.
- **Indications:**
  - Facial nerve repair.
  - Selective dorsal rhizotomy.
  - Tethered spinal cord release.
  - Pedicle screw placement.

Evoked Potentials

Signals generated after electrical stimulation that allow for assessment of neural pathways intraoperatively.

Types

- **Somatosensory evoked potential (SSEP):**
  - Scalp electrodes stimulate peripheral afferent nerves and record response from sensory pathway.
  - Median nerve at the wrist: Upper extremity monitoring.
  - Posterior tibial nerve posterior to the medial malleolus: Lower-extremity monitoring.
  - **Clinical uses:**
    - **Spinal surgery:** SSEP monitors the integrity of dorsal columns. Inability to test motor pathways, which are more important clinically than dorsal column integrity, is a significant limitation of the technique in spinal surgery.
    - **Carotid surgery:** Sensitive for detection of cerebral ischemia and the need for shunting.
    - **Cerebral aneurysm surgery:** Changes may indicate occlusion of parent vessel branches, which could be reversed by repositioning of aneurysm clips.
    - **Aortic cross-clamping:** Immediate changes indicate high risk for neurological injury.
- **Brainstem auditory evoked response (BAER):**
  - Records cortical responses to auditory stimuli (monitor acoustic nerve, brain stem, and cerebral cortex).
  - **Clinical uses:** Cerebellopontine angle surgery: Acoustic neuroma or meningioma, or for microvascular decompression for tic douloureux or hemifacial spasm.
- **Motor evoked potential (MEP):**
  - Electrical or magnetic stimulation of the motor cortex or the spinal cord.
  - Neurogenic potentials in the distal spinal cord or peripheral nerve.
  - Myogenic potentials from the innervated muscle.
  - **Clinical uses:** Spinal surgery—as adjunct to SSEPs.
- **Visual evoked potential (VEP):**
  - Stimulation by flashing light-emitting diodes or strobe lights.
  - Assess integrity of visual pathways (including optic nerves).
  - Cannot detect visual field defects.
  - **Clinical uses:** Resection of craniopharyngiomas, pituitary adenomas, and suprasellar meningiomas that arise in the parasellar region.
- **Anesthetics effects:**
  - Volatile anesthetics:
    - Depression of evoked potentials and prolongation of latencies.
    - Affect cortically evoked responses more than subcortical, spinal, or peripherally evoked responses.
  - Barbiturates:
    - ↓ evoked potential amplitude and lengthen latency, but typically recordings can be obtained despite high doses.
    - ↑ beta frequency activity.
    - Fast-acting barbiturates (methohexital) sometimes can ↑ epileptiform spikes.

Factors affecting SSEPs and MEPs:

- *Anesthetics*
- *Blood pressure*
- *Temperature*
- *Arterial blood gas tension*
- *Extreme hemodilution*
- *Hypocarbia*

- Etomidate:
  - Low doses: ↑ evoked potential amplitude but prolong latencies.
  - Induction doses: Amplitude may be reduced.
- Ketamine: Either does not affect or may ↑ evoked potential amplitude.
- Narcotics:
  - Mild reduction in amplitude of evoked potentials but usually allow consistent monitoring.
  - Remifentanil (often used in total intravenous anesthesia) also causes mild changes.
- Benzodiazepines:
  - ↓ amplitude with little effect on latencies.
  - ↑ beta activity.
  - ↓ epileptiform activity.
- Neuromuscular blockers: No significant effect on evoked potentials. Suppresses evoked muscular responses.
- Propofol: ↑ latency and ↓ amplitude of cortical SSEPs.

CHAPTER 6

Airway Management

ANATOMY

The superior laryngeal nerve provides sensation to all areas from the epiglottis to the vocal cords and innervates the cricothyroid muscle.

The recurrent laryngeal nerve provides sensation to all areas below the vocal cords and innervates all laryngeal muscles except the cricothyroid muscle.

- Nasopharynx: Extends from the nose to the pharynx.
- Oropharynx: Extends from the mouth to the pharynx.
- Pharynx: Begins at the junction of the nasopharynx and oropharynx and extends to the cricoid cartilage.
- Larynx: A group of cartilages and muscles that act to protect the vocal cords. These cartilages are: thyroid, cricoid, epiglottic, and paired arytenoid, corniculate, and cuneiform.
- The nerve supply of the airway and associated functions are detailed in Table 6-1.
- Effects of damage to the nerve supply:
  - Bilateral superior laryngeal nerve: Hoarseness.
  - Unilateral recurrent laryngeal nerve: Ipsilateral vocal cord paralysis with voice change and ↓ ability to prevent aspiration.
  - Bilateral recurrent laryngeal nerve: Stridor, respiratory distress acutely. If the palsy is chronic, then compensatory actions by other muscles prevent the respiratory distress.
  - Bilateral vagus nerve: Flaccid, midline vocal cords, prevents phonation.
- Blood supply to larynx is from thyroid arteries.

Management of the Normal Airway and Equipment

PREOXYGENATION

- First step in administration of general anesthesia (GA).
- Five minutes of breathing 100% O_2 or four vital capacity breaths will significantly ↑ the time until desaturation occurs during apnea.
- Also known as **denitrogenation** because the process replaces nitrogen with O_2 in the lungs.

TABLE 6-1. Nervous System Supply of the Airway

NERVE	FUNCTION
Lingual nerve	Sensation to the anterior two-thirds of the tongue.
Glossopharyngeal nerve	Sensation to posterior one-third of tongue, roof of pharynx, tonsils, underside of soft palate.
Superior laryngeal nerve (external branch)	Motor to cricothyroid muscle, sensation to anterior subglottic mucosa.
Superior laryngeal nerve (internal branch)	Sensation from the epiglottis to the vocal cords.
Recurrent laryngeal nerve	Motor to intrinsic muscles of the larynx (except cricothyroid).

FACE MASK

- Used for preoxygenation and for assisting with or providing ventilation after apnea due to the induction of GA.
- Creates an airtight seal with the patients face.

POSITIONING

- Head in sniffing position.
- Mask held with left hand; right hand is used to squeeze the anesthesia bag.
- Thumb and forefinger are positioned in a "C" around the orifice to the O_2 supply and push the mask down onto the face.
- Third and fourth fingers lift the mandible toward the mask.
- Fifth finger lifts the angle of the mandible and creates an anterior jaw thrust.
- Head straps can be used to help keep the mask in place.
- In patients with a large face, facial deformities, or facial hair, two hands may be needed to create a tight seal with the mask. A second person can then squeeze the bag.
- Only 20–25 cm H_2O pressure should be used during positive pressure ventilation via mask.
- Prolonged mask ventilation and head strap use are associated with pressure injuries to the facial and trigeminal nerve branches.
- Corneal abrasions can occur if the eyes are not protected.

NASAL AND ORAL AIRWAYS–CONSIDERATIONS

- After induction of anesthesia, the tongue, epiglottis, and soft tissues in the oropharynx relax and may fall back onto the posterior pharyngeal wall and cause obstruction of the airway.
- Oral and nasal airways create an opening between the tongue and posterior pharyngeal wall so that air can pass through.
- Oral airways can cause laryngospasm when placed in an awake or lightly anesthetized patient.
- Measuring the length from the patient's nose to the meatus of the ear can approximate the appropriate length of the nasal airway.
- The nasal airway must be lubricated and, in an awake patient, the nares should be anesthetized with lidocaine prior to placement.
- Contraindications to nasal airways include bleeding disorders, basilar skull fractures, or significant nasal deformities.

LARYNGEAL MASK AIRWAY (LMA)

The LMA does not fully protect the airway from gastric regurgitation.

- A supraglottic airway used for ventilation, to provide GA, or as a conduit for passing a bronchoscope or endotracheal tube (ETT).
- Sizes are based on weight and body size and can be used for neonates to adults (Table 6-2).
- It consists of a wide tube with a large distal cuff intended to sit in the hypopharynx.
- Protects airway (at least partially) from secretions.
- May be used with positive-pressure ventilation at < 20 cm H_2O for 2–3 hours.

TABLE 6-2. LMA SIZES AND CUFF INFLATION VOLUMES

SIZE	PT. WEIGHT (KG)	INFLATION VOLUME (ML)
1	< 5 kg	4
1.5	5–10	7
2	10–20	10
2.5	20–30	14
3	> 30	20
4	< 70	30
5	> 70	40

PLACEMENT

- The cuff is partly or completely deflated, flattened, and lubricated.
- The nondominant hand is placed behind the occiput to create head extension and neck flexion.
- The dominant hand holds the LMA with the index finger between the cuff and the tube. The index finger then advances the superior surface of the device in an upward direction along the palate and into the pharynx until the resistance of the upper esophageal sphincter is encountered.
- The index finger of the dominant hand is removed while the nondominant hand holds the tube in place.
- The cuff is inflated to a maximum of 60 cm H_2O pressure.

Use of an LMA may be associated with less bronchospasm compared to ETT, which is placed in the trachea.

COMPLICATIONS

- Gastrointestinal reflux and aspiration can occur since there is no seal of the esophagus.
- Laryngospasm.
- Coughing.
- Bronchospasm.
- Sore throat.
- Hoarseness.
- Trauma.
- Nerve injury.

CONTRAINDICATIONS

- Full stomach (or equivalent, such as pregnancy, morbid obesity, hiatal hernia, or significant gastroesophageal reflux disease).
- Pharyngeal pathology or obstruction.
- Poor oral opening.
- Poor pulmonary compliance.

Other Types of LMA

LMA Flexible

Has a reinforced, flexible tube that can be manipulated without kinking. It has been used in oropharyngeal surgery and when there may need to be head movement.

LMA Proseal

Has a gastric drain to help prevent aspiration and confirm placement.

LMA Fastrach

Designed to be a conduit for passing an ETT either blindly or using a fiber-optic bronchoscopy.

High-pressure cuffs have a higher risk of ischemic damage to tracheal mucosa and are less suitable for prolonged intubation.

Uncuffed ETTs are used in the pediatric population to minimize pressure injury and postintubation croup (see Chapter 9).

LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

- **Endotracheal tubes (ETTs):**
  - Usually made of polyvinyl chloride.
  - Adult tubes have an inflatable cuff that is either high pressure/low volume or low pressure/high volume (most commonly used because they cause less mucosal ischemia).
  - A pilot can estimate the amount of pressure in the cuff and should be monitored because cuff pressure can ↑ during GA; especially if nitrous oxide is used.
  - A flexible stylet can be used to modify the shape of the ETT, though there are also preformed tubes (oral or nasal RAE tubes and wire-reinforced tubes).
  - Double-lumen ETTs are used when lung isolation is necessary.
- **Laryngoscopes:**
  - Consist of a handle and a blade. The handle contains a battery that supplies a light bulb or fiber-optic bundle at the end of the blade.
  - Blades come in two general designs: curved (eg, Macintosh) and straight (eg, Miller). They are both available in multiple sizes.

Normal Laryngoscopy

Necessary preparation and equipment (is noted in Table 6-3).

TABLE 6-3. Equipment for General Anesthesia

Oxygen source and bag (anesthesia machine or ambu bag)
Facemask
ETTs of multiple sizes with stylets that have had their cuffs checked for leaks
Oral and nasal airways
Suction equipment (no induction without suction)
Functioning laryngoscope handle with blades of different sizes and shapes
Stethoscope and end-tidal carbon dioxide monitor
Pillow and/or blankets for head positioning

Position

- The patient should be supine, with the bed at the level of the laryngoscopist's xyphoid process.
- The patient should be in the "sniffing position" with the head on a pillow 5–10 cm above the bed (creating flexion of the neck) and the neck extended backwards.
- This creates a straighter plane of view from the mouth to the larynx.
- With morbidly obese patients, a "ramp" may be necessary below the upper back, shoulders, neck and head to displace the mass of their chest.

"Feeling" the pilot balloon is not a reliable indicator of determining adequacy of cuff pressure.

Procedure

- After preoxygenation and induction of anesthesia, the laryngoscope is held with the left hand, and the right hand opens the mouth (most effectively with a scissoring motion between the upper and lower teeth). The blade is inserted into the right side of the mouth, and the tongue is swept toward the left. The blade is advanced into the vallecula (with a curved blade) or under the epiglottis (with a straight blade) and then pulled up and in a caudal direction (always being careful of the teeth and lips). This should bring the vocal cords into view so that the ETT can be taken with the right hand and advanced through them. The laryngoscope is removed, and the cuff is inflated.
- If the vocal cords cannot be seen, an assistant can apply the **BURP** maneuver: **B**ackward (posterior), **U**pward, **R**ightward (lateral), **P**ressure (to the larynx, which often improves the view).
  - **Confirmation of ETT placement:** Consistent end-tidal CO_2 is the best way to confirm placement. Auscultation of breath sounds, watching for chest rise and condensation in the ETT, and chest x-ray are also used for confirmation.
  - **Grades of laryngoscopy:**
  - **I:** Full view of the glottic opening.
  - **II:** Only the posterior part of the glottic opening can be seen.
  - **III:** Only the tip of the epiglottis can be seen.
  - **IV:** Only the soft palate can be seen.

BURP maneuver:

Backward (posterior)
Upward
Rightward (lateral)
Pressure (to the larynx, which often improves the view).

Persistent detection of $ETCO_2$ is the best indicator of tracheal placement of a ETT.

Rapid Sequence Intubation

Used in patients who have "full stomachs." The induction agent and muscle relaxant are given almost simultaneously, and the ETT is inserted as soon as relaxation has occurred without mask ventilation.

- **Sellick's maneuver** (cricoid pressure) is applied from induction until confirmation of ETT placement to prevent esophageal reflux after induction of GA. It is performed by pushing the cricoid cartilage downward (posterior) onto the vertebrae to occlude the esophagus.
- If the ETT cannot be placed and mask ventilation is required before a second attempt at laryngoscopy, cricoid pressure should be maintained and positive pressure ventilation can be performed.

Nasotracheal Intubation

Preparation

The nostril to be used should be prepped with phenylephrine drops to provide vasoconstriction and ↓ membrane swelling. If the patient will be awake, then local anesthesia or airway nerve blocks should be performed. The ETT is sometimes softened in warm water and is lubricated.

For a nasotracheal intubation, the ETT should be introduced along the floor of the nose at an angle perpendicular to the face.

Insertion

The prepared ETT is inserted and advanced along the floor of the nostril. Direct laryngoscopy is performed to visualize the ETT in the oropharynx, at which point it is advanced through the vocal cords. If it does not pass through easily, Magill forceps can be used to help guide the ETT.

Complications

- **Incorrect placement:** It can be esophageal (breath sounds over the stomach but not in the lung fields) or endobronchial (may have high inspiratory pressures, low oxygen saturation or unilateral breath sounds). The ETT cuff position can be checked by squeezing the pilot balloon while feeling for the cuff in the sternal notch.
- **Trauma:** Damage to teeth is the most common cause of claims. Lips can be cut if caught between the teeth and the blade. Sore throat is a common complaint after intubation. Multiple attempts at intubation or a significant amount of airway manipulation can cause tissue edema.

Physiologic Effects

Hypoxia and hypercarbia can occur. Hypertension and tachycardia are normal responses to laryngoscopy. They can be ↓ or prevented by IV lidocaine (1–1.5 mg/kg) or narcotics prior to laryngoscopy. Antihypertensive medications can also be used. Arrhythmias can also occur.

Laryngospasm

A reflexive closure of the vocal cords due to stimulation by secretions, suctioning, an ETT (or anything that stimulates the superior laryngeal nerve). Rapid identification of the problem is imperative. Treatment involves positive pressure ventilation via face mask, stimulation of the patient, deepening the level of anesthesia, IV lidocaine (1–1.5 mg/kg) or IV succinylcholine (usually only 20–40 mg is necessary). Negative-pressure pulmonary edema can develop because of the large negative intrathoracic pressure that occurs from attempting to breathe against a closed glottis.

Bronchospasm

Asthmatic patients may develop bronchospasm after endotracheal intubation. It can be treated with albuterol through the ETT or epinephrine (if it is very severe). The anesthetic gases will cause some degree of airway relaxation as well.

Aspiration

After induction of GA and abolition of the normal gag reflex, gastric contents can be aspirated into the lungs, causing severe pneumonitis. Keeping patients NPO and cricoid pressure are used to try to prevent this.

Management of the Difficult Airway

- "Difficult airway" means a trained anesthesiologist has difficulty with mask ventilation and/or tracheal intubation.
- The American Society of Anesthesiologists (ASA) has created practice guidelines including the Difficult Airway Algorithm for the management of the anticipated or unanticipated difficult airway (see Figure 3-2).
- **History:** An airway history to detect past history of difficult airway or medical/surgical conditions that may suggest difficulty.
- **Physical:** An airway physical exam may improve detection of a difficult airway. It is most helpful if multiple features are assessed together, as reviewed in Table 6-4.

Preparation for a Difficult Airway

- There should be a "difficult airway cart" with specialized equipment for the management of the difficult airway. It should include laryngoscope blades of different shapes/sizes, ETT stylets/guides, LMAs, intubating LMAs, fiberoptic bronchoscope, esophageal tracheal combitube, equipment for transtracheal jet ventilation, and equipment for invasive airway access.
- The patient should be adequately preoxygenated, and supplemental O_2 should be given throughout the airway management process.
- The patient should be informed if difficulty is suspected regarding the risks and the possible procedures that may be necessary (including the possibility of awake intubation).
- The anesthesiologist should make sure that assistants are available if help is needed.

TABLE 6-4. Physical Airway Assessment

Length of upper incisors
Presence of an overbite
Interincisor distance
Mallampati classification
Shape of the palate
Compliance of the mandibular space
Thyromental distance
Length of the neck
Thickness of the neck
Range of motion of the head/neck

Strategy for Managing the Difficult Airway

A plan should include:

- Assessment of the likelihood of difficult ventilation, intubation, patient cooperation, or tracheostomy.
- Consideration of the risks/benefits of options including awake vs. asleep intubation, the use of noninvasive vs. invasive techniques, and maintaining spontaneous ventilation vs. abolishing it.
- Deciding on a preferred approach to awake intubation; to managing a patient who can be ventilated but not intubated or a patient who cannot be intubated or ventilated.
- A plan for alternative approaches should the primary approach fail or should it not be feasible in a particular patient.

The Unanticipated Difficult Airway (GA Induced)

- Always call for help from another practitioner (if possible).
- Laryngoscopy should be attempted by the most experienced person available. Any further attempts at direct laryngoscopy should be performed after some change has been made (eg, patient's position, blade type or size).
- If mask ventilation is possible, then other airway tools can be tried or the patient can be ventilated until they wake up and an awake technique can be used.
- If mask ventilation is not possible, an LMA can be inserted, which usually makes ventilation possible. The patient can be intubated via the LMA or can be ventilated until they wake up and an awake technique can be used.
- An emergent invasive/surgical airway may be necessary if ventilation is still not possible with an LMA and the patient is becoming hypoxic.

The Anticipated Difficult Airway

- An awake intubation technique is probably the safest, assuming it is possible (the patient is able to cooperate). It may involve a blind technique, direct laryngoscopy, or the use of a fiber-optic bronchoscope or other airway tool.
- An invasive airway may be necessary, and supplies should be easily accessible.

Awake Intubation

- Light sedation may be given to calm patient anxiety.
- An antisialagogue (such as glycopyrrolate) should be given to ↓ secretions.
- Local anesthetics must be used to anesthetize the airway to make the procedure more tolerable for the patient. (See Regional Anesthesia for descriptions of these blocks)
- ETT can be inserted via nasal or oral route.
- Once the ETT is in place, GA can be induced.

Fiber-Optic Bronchoscope–Guided Intubation

- Can be performed before or after the induction of GA (with or without muscle relaxation) and via either the nasal or oral route.

Nerve blocks that aid in an awake fiberoptic intubation are:

Glossopharyngeal nerve (sensation to posterior third of tongue and oropharynx).

Superior laryngeal nerve block.

Recurrent laryngeal nerve block (transtracheal block).

- There is usually a side port to which an oxygen supply or suction can be attached to help prevent hypoxia and clear out secretions for a better view.
  - An ETT is loaded onto the bronchoscope and can be advanced once the distal end is inside the trachea.
- Other intubating tools:
  - Rigid fiber-optic scopes such as the Bullard and WuScope laryngoscopes. They have laryngoscope-type blades with a light and an optical source that allows direct visualization of the airway.
  - Video laryngoscopes, such as the Glidescope and Video-Macintosh laryngoscope, are shaped like normal Macintosh laryngoscopes but have a camera at the distal end that projects their view onto a screen. An ETT can be inserted alongside like normal.

Esophageal Tracheal Combitube

A supraglottic airway that is inserted blindly. It has two lumens, a large oropharyngeal balloon and a smaller distal cuff designed to isolate the airway from the esophagus even if it is positioned within the esophagus (as it most often is).

Retrograde Intubation

- Can be performed in an awake patient after proper skin and airway local anesthesia have been placed.
- An 18-gauge IV catheter is inserted into the trachea through the cricothyroid membrane (confirmed by aspiration of air from the trachea). A wire is passed through the catheter and is advanced until it comes out of the mouth or the nose. An ETT can then be advanced over the wire into the trachea.
- Once the ETT is in the trachea, the wire can be removed.

Transtracheal Jet Ventilation

- Used when a patient cannot be intubated or ventilated.
- A 12- to 16-gauge IV catheter (or specialized catheter) is inserted into the trachea via the cricothyroid membrane.
- O_2 can be supplied via a jet ventilator (attached by a connecter with a luer lock) or a low pressure O_2 flow meter.

Cricothyroidotomy

- Usually a last resort during a cannot intubate or ventilate situation.
  - Commercial kits are available with a catheter, wire, and tube.
  - Once in place, O_2 can be supplied by an ambu bag or anesthesia circuit.

Extubation

- Complications can occur during or just afterward, including respiratory failure and hypoxia, airway obstruction, laryngospasm, aspiration, bronchospasm, hypertension, tachycardia, ↑ intracranial pressure, and ↑ intraocular pressure.
- All patients should receive 100% O_2 after extubation until adequate ventilation is confirmed. Many patients are given supplemental O_2 during transportation to the postanesthesia care unit (PACU).

Deep Extubation

- ETT is removed while the patient is still deeply anesthetized but breathing adequately.
- All muscle relaxants should be reversed or should have worn off.
- This approach may be better in patients who are at risk for bronchospasm or those in whom coughing or bucking on the ETT would be detrimental.
- Contraindications include patients with difficult airways and those at risk for aspiration or airway obstruction.

Awake Extubation

- ETT is removed once the patient is awake and able to follow simple commands. Muscle relaxation should be reversed or should have worn off.
- Most patients will cough or buck on the ETT and will respond to oropharyngeal suctioning. Opiates or IV lidocaine may ↓ these responses somewhat.
- After the oropharynx is suctioned and the ETT cuff is deflated, the ETT is removed gently and swiftly with positive pressure on the anesthesia bag to help clear secretions.

Extubation of the Difficult Airway

- A plan should be in place for extubating and the possible need for reintubation.
- Considerations should include possible causes of post-extubation ventilatory problems and a plan for managing the airway after extubation.
- Awake extubations are usually preferred.
- Extubation may be performed using an ETT exchanger (or a similar tool) that can facilitate reintubation should it be necessary.

Laryngospasm is a forceful involuntary spasm of the laryngeal muscles caused by sensory stimulation of the superior laryngeal nerve. Any airway stimulation such as secretions, ETT passage, suctioning, oral/nasal airway placement can trigger laryngospasm. Treatment includes: Positive-pressure ventilation with 100% O_2. IV lidocaine or propofol. Succinylcholine (0.25-1 mg/kg) to relax laryngeal muscles.

Large negative intrathoracic pressures generated during laryngospasm can result in negative-pressure pulmonary edema, especially in young healthy patients.

Extubation during light phase of anesthesia is avoided due to ↑ risk of laryngospasm. The presence of an ETT in an awake asthmatic patient may trigger bronchospasm.

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CHAPTER 7

Regional Anesthesia

- Done as single injection or with insertion of a catheter for a continuous infusion.
- This type of block can be the sole source of anesthesia, done in conjunction with general anesthesia or for postop analgesia.
- Uses include abdominal, thoracic, lower-extremity, and pelvic surgeries as well as most obstetric procedures.

Anatomy

Boundaries of epidural space: Foramen magnum, sacral hiatus, dura, ligamentum flavum.

- **Vertebral column:** Surrounds and protects the spinal cord and the spinal nerve roots.
  - Seven cervical vertebrae (C1–C7), 12 thoracic vertebrae (T1–T12), 5 lumbar vertebrae (L1–L5), 5 sacral vertebrae (S1–S5) and 4 fused coccygeal vertebrae.
  - Characteristics of vertebrae include a vertebral body, two pedicles, two laminae, a spinous process, two transverse processes, and two superior and two inferior articulating processes.
  - The anterior and posterior longitudinal ligaments, the ligamentum flavum, the interspinous ligaments, and supraspinous ligaments help to stabilize the vertebral column (Figure 7-1).

Spinal cord terminates at L1 in adults and L3 in children. Subarachnoid space terminates at S2 in adults and S3 in children.

- **Meninges:** The three layers of tissue that surround the spinal cord. Pia mater is the innermost layer followed by the arachnoid mater and finally the dura mater.
  - Between the pia and arachnoid is the subarachnoid space that contains the cerebrospinal fluid (CSF).
  - Between the dura and arachnoid is the subdural space (a potential space).
  - Just outside the dura, between that and the ligamentum flavum is the epidural space.
- **Spinal cord:** Runs from the base of the brain through the foramen magnum down to the L1 vertebrae in adults (L3 in children).

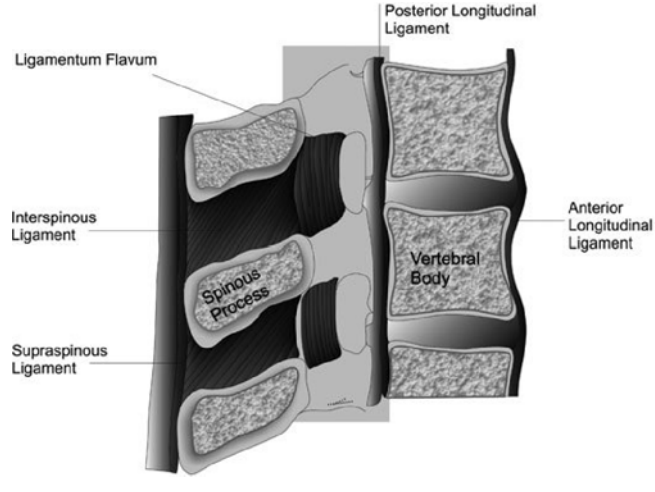

FIGURE 7-1. Ligaments of the vertebral column.

(Reproduced, with permission, from Hadzic A, ed. *Textbook of Regional Anesthesia and Acute Pain Management.* New York: McGraw-Hill, 2007: 234.)

- The anterior and posterior nerve roots come together and travel out through the intervertebral foramen at each spinal level from C1 to S5, creating spinal nerves.
- Cauda equina is the continuation of the spinal nerves below L1.
- One anterior spinal artery (which originates from the vertebral artery) and two posterior spinal arteries (which originate from the posterior inferior cerebellar arteries) provide the blood supply to the anterior two-thirds and posterior one-third of the spinal cord, respectively. There is also contribution of blood supply from the intercostal arteries and the lumbar arteries, including the artery of Adamkiewicz.

- Surface anatomy: Spinous processes = midline of back (unless scoliosis). Handy landmarks (Figure 7-2):
  - Most prominent spinous process: C7.
  - Inferior edge of the scapula: T7.
  - Level of the iliac crests: L4–5.
  - Level of the posterior superior iliac spines: S2.

Physiology

- The main goal of neuraxial anesthesia is to block the spinal nerve roots by bathing them in local anesthetic.

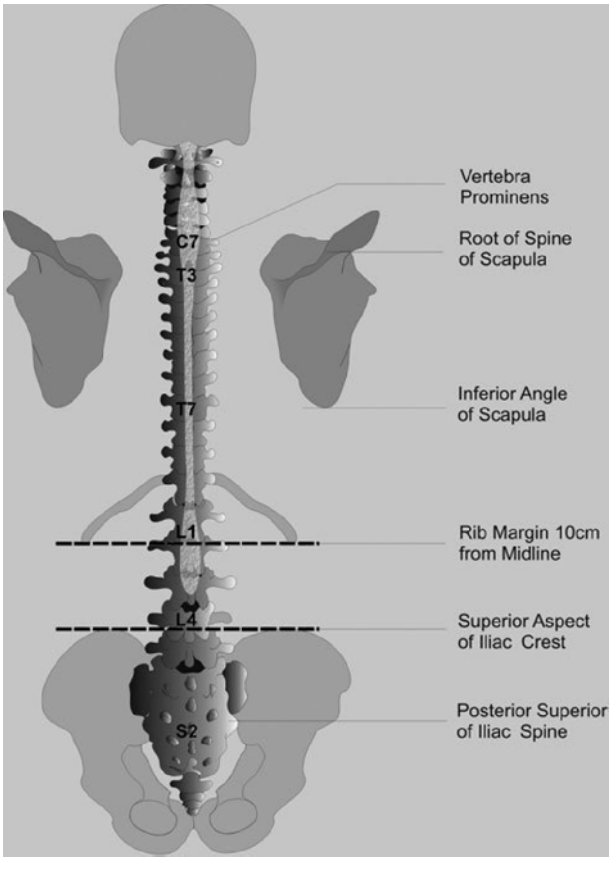

FIGURE 7-2. Surface landmarks for various levels of epidural blockade.

(Reproduced, with permission, from Hadzic A, Ed. *Textbook of Regional Anesthesia and Acute Pain Management.* New York: McGraw-Hill, 2007: 236.)

- Anterior nerve fibers: Efferent motor and autonomic outflow.
- Posterior nerve fibers: Somatic and visceral sensation.

- **"Differential blockade":** Because different types of nerve fibers require different concentrations of local anesthetic to be blocked, there will often be different characteristics of the block at various levels. In general, the further (ie, more cephalad) from the needle insertion site, the lower the concentration, so that the block appears as follows:
  - Motor blockade will be present where the concentration is highest (the extent of this depends on the dose).
  - Sensory blockade spreads about two levels higher than motor blockade.
  - Sympathetic blockade spreads about two levels higher than sensory blockade (where the concentration is the lowest).
- **Autonomic blockade:**
  - Sympathetic fibers travel with the T1–L2 nerve roots. Parasympathetic fibers travel with cranial and sacral nerves (therefore not as commonly blocked).
  - **Cardiac effects:** ↓ BP, +/– ↓ HR and contractility, vasodilation, ↓ venous return (prehydration may help compensate). Treat with fluids and vasopressors as needed.
  - **Gastrointestinal effects:** ↓ ileus due to sympathectomy and relatively greater parasympathetic input, +/– ↓ hepatic blood flow.
  - **Urological effects:** ↓ autonomic control of bladder (→ urinary retention), no change in renal blood flow due to autoregulation.
  - **Endocrine effects:** ↓ neurohumoral stress response; adrenal pathways can be blocked (with a T11 level block).
  - **Pulmonary effects:** Significant in patients with chronic lung disease who use accessory muscles of respiration, which may be blocked with sufficiently high levels of somatic blockade.

Neuraxial structures encountered in midline vs. paramedian approaches:
Midline: Skin → subcutaneous tissue → supraspinous ligament → interspinous ligament → ligamentum flavum → [dura for spinals]
Paramedian: Skin → subcutaneous tissue → paraspinal muscles → ligamentum flavum → [dura for spinals]

Pre-procedure Considerations

- Contraindications to neuraxial anesthesia:
  - Absolute: Patient refusal, infection at the site of injection, severe hypovolemia, ↑ intracranial pressure (ICP).
  - Relative: Coagulopathy/bleeding disorder, severe ankylosing spondylitis (AS) or multiple sclerosis (MS), sepsis, preexisting neurological deficit, severe spinal deformities, an uncooperative patient (eg, dementia).
- Neuraxial blocks can be performed in the sitting, lateral decubitus positions or prone positions.
  - **Sitting:** Easiest to find the midline. The patient leans forward with the lumbar spine maximally flexed (to "open" the intervertebral spaces).
  - **Lateral decubitus:** Good option for patients who cannot sit up. The patient lies on his side with the head and neck flexed downward and the hips and knees flexed (the "fetal position").
  - **Prone:** Used mostly with spinal anesthesia for anorectal procedures because this is the position in which the surgery will take place (technically the jackknife position).
- Midline vs. paramedian approach:
  - **Midline:** The needle is inserted in the midline in the space between two palpated spinous processes +/– a slight cephalad angle. For an epidural, the needle will pass through the skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, and finally the ligamentum flavum. For a spinal, one more layer, the dura, must be passed through.

- **Paramedian:** This approach is good for patients who have spinal abnormalities (arthritis, scoliosis, past surgery) because it provides easier access to the spinal canal. The needle is inserted 1–2 cm lateral to the spinal process and is directed at a 10- to 25-degree angle toward the midline. The needle will pass through skin, subcutaneous tissue, paraspinal muscles, and then ligamentum flavum and dura as per the midline approach.
- Surgical procedure location: See Table 7-1.

Spinal Anesthesia

- Spinal anesthesia causes a very dense block of motor and sensory fibers.
- Catheters can be inserted for a continuous spinal block (2–3 cm into the subarachnoid space). Very small gauge catheters have been associated with cauda equina syndrome and were withdrawn from the market in the 1990s.

Needle Types

- **Quincke:** Sharp, beveled tipped with an end injection site.
- **Whitacre:** Blunt, rounded point (pencil point) with a side injection site.
- **Sprotte:** A pencil point with a long side injection site (gives better free flow of cerebrospinal fluid [CSF]).
- **Greene:** Sharp, beveled tipped with an end injection site

Technique

Performed below L1 in adults and L3 in children. The needle is inserted using any of the three approaches mentioned above, through the ligamentum flavum (which will feel like a "pop") and the dura-arachnoid membrane (may or may not feel a second "pop"). The position can be confirmed by free flow of CSF (aspiration may be necessary if a very small gauge needle is used).

Baricity

Hyperbaric: Heavier than CSF, local + glucose

Hypobaric: Lighter than CSF, local + sterile water

Block-Level Determinants

Baricity: The specific gravity of the local anesthetic vs. CSF. This is the most important determinant of block height.

- **Hyperbaric solution** is heavier than CSF and therefore will travel to dependent areas, with gravity. It is created by the addition of glucose to the local anesthetic.
- **Hypobaric solution** is lighter than CSF and therefore will travel to non-

TABLE 7-1. Surgical Procedure Locations

Surgical Site	Block Level Needed	Surface Anatomy Landmark
Upper abdomen	T4	Nipple line
Lower abdomen	T6	Xiphoid process
Pelvis	T8–10	Umbilicus (or just above)
Lower extremity	T10	Umbilicus
Hemorrhoids	S2	Perineum

dependent areas. It is created by the addition of sterile water to the local anesthetic.
- **Isobaric solution** has the same specific gravity as CSF and therefore will stay close to the level at which it was injected.
- Patient position (due to baricity).
- Level of injection.
- Dose of local anesthetic used: Higher amounts = higher block level.
- Spinal abnormalities such as kyphoscoliosis can affect the spread of the local anesthetic.
- CSF volume: ↓ CSF volume = ↑ level of blockade. This can occur during pregnancy or with a large abdominal tumor (due to epidural vein engorgement) or with ↑ age. Lower volumes of local anesthetic should be used in these cases.
- Direction of the needle injection site (this is controversial).
- Patient height: At the extremes of height, dose adjustments may be necessary.

Use smaller spinal local volumes in pregnancy, large abdominal tumors, elderly, and short patients.

Medications for Spinal Anesthesia

- Local anesthetics: Preservative free only. Hyperbaric tetracaine and bupivacaine are most common. Tetracaine causes greater motor block, but they are similar in onset (5–10 min) and duration (90–120 min). Ropivacaine, lidocaine, mepivacaine, chloroprocaine, and procaine can also be used.
- Vasoconstrictors (epinephrine): Often added to prolong the duration of the local anesthetic. It has a minimal effect on bupivacaine, but ↑ the duration of a block with tetracaine by about 50%.
- Narcotics: Can be added to help improve the quality of the block.
- Other adjuvants: Clonidine and neostigmine have analgesic qualities and have been added to spinal blocks.

Epidural Anesthesia

Location

- **Cervical:** Used mostly for pain management.
- **Thoracic:** Usually used for analgesia in conjunction with general anesthesia.
- **Lumbar**: Most common. Can be used as the primary anesthesia for any surgery below the diaphragm.
- **Sacral** (also known as a caudal block).
- Catheters are usually inserted (2–6 cm into the epidural space), although single-shot techniques can be done as well.
- Epidural space: Lies outside of the dura. Contained within it are the nerve roots, fatty connective tissue, lymphatics, and Batson's venous plexus.
- Compared to spinal anesthesia, there is slower onset of the block (10–20 min); it is less dense and can have varied motor blockade.

Needle Types

Most are 17- to 18-gauge and 9–10 cm long (even longer ones exist and are sometimes needed with larger patients). The diameter is larger than for a spinal needle so that loss of resistance can be obtained.

- **Tuohy:** Most common. It has a blunt bevel and a curve at the tip.
- **Crawford:** Straight-tipped needle. It is easier to pass a catheter through, but it is also easier to go through the dura with this needle vs. the blunt-tipped one.
- **Hustead:** Has a less curved tip than a Tuohy.

Technique

- **Loss of resistance:** Most commonly used. The needle is inserted using the midline or paramedian approaches with the stylet in place. Once the needle reaches the interspinous ligament (identified by a greater tissue "grasp" on the needle), the stylet is removed and a glass or plastic syringe filled with about 2 mL of saline or air is attached to the end of the needle. The needle is then further advanced slowly, with repetitive attempts at injecting the contents of the syringe. The injection will finally be successful when the needle has passed into the epidural space (the resistance to injection will be lost).
- **Hanging drop:** This starts out the same way, up until the removal of the stylet. Once the stylet has been removed, the needle is filled with saline (or other fluid) so that a drop just hangs off the end of the needle. The needle is then slowly advanced into the epidural space. Once there, the fluid will be drawn into the epidural space by negative pressure.

Test Dose

Epinephrine concentrations: 1:200,000 → 1000 mg = 1,000,000 μg per 200,000 mL → 5 μg/mL.

Injection of a small amount of local anesthetic with epinephrine (usually 3 mL of 1.5% lidocaine with 1:200,000 epinephrine, which amounts to 45 mg of lidocaine and 15 μg of epinephrine) to detect subarachnoid or intravascular location of the needle or catheter. If it is intrathecal, there will be an apparent spinal-type anesthesia. If it is intravascular, the heart rate and systolic blood pressure should ↑ by 20 beats per minute and 15 mmHg above their respective baselines. False positives and negatives are possible.

Incremental Dosing

Even if a test dose was performed and was negative, and if aspiration of the epidural needle or catheter produced neither CSF nor blood, it may still be intrathecal or intravascular. By injecting the total amount of local anesthetic to be used in about 5-mL aliquots, it is possible to detect mild symptoms of either of these unintended placements.

Block-Level Determinants

1–2 mL of local expands level of epidural block by one dermatome.

- Amount of local anesthetic: 1–2 mL of local anesthetic are required to expand the level of the block by one dermatome (less is needed for analgesia).
- Age: ↑ age = ↓ dose of local anesthetic required.
- Patient height: ↑ height = ↑ dose of local anesthetic required.
- Patient position: Plays a small role in the spread of local anesthetic.
- Needle insertion site: Important when planning level of block. For example, if knee arthroscopy (L4/L5 dermatomes), a lumbar epidural is appropriate. If abdominal surgery is planned with the incision centered around T8, the epidural should be placed at T8. This practice allows for the least amount of local anesthetic (↓ toxicity) and side effects (unwanted dermatomes/autonomic nerves blocked) while matching the analgesia to the precise location of the surgical insult. Placing a lumbar epidural for an abdominal case and using large doses of local anesthetic to "push up" the level is poor practice and results in frozen legs, urinary retention, and often poor pain control.

Medications for Epidural Anesthesia

Chosen based on the duration of the procedure and whether the epidural is the primary anesthetic, a supplement to other anesthesia, or for analgesia.

- Local anesthetics: Preservative free only.
  - Short- to medium-acting: Lidocaine (1.5–2%), chloroprocaine (3%), and mepivacaine (2%).
  - Long-acting: Bupivacaine (0.5–0.75%), ropivacaine (0.5–1%), and etidocaine.
  - Repeat dosing is usually done based on duration of action of the particular agent used. About one-third of the original dose is used for the repeat dose.
- Vasoconstrictors (epinephrine): ↑ duration and quality of block.
- Narcotics: May be added to the local anesthetic. This is commonly done using fentanyl with dilute bupivacaine for labor analgesia.
- Sodium bicarbonate: 1 mEq/10 mL of local anesthetic is mixed in just prior to injection to hasten the onset of the blockade. By ↑ the pH of the local anesthetic, it ↑ the nonionic form, which can better penetrate the lipid membrane of the nerve cells. It is used with lidocaine, mepivacaine, and chloroprocaine, but not with bupivacaine (because they precipitate when mixed together).

Infusion Regimens for Epidural Anesthesia

- Can be used as background infusion only or patient-controlled epidural analgesia (PCEA).
- Lumbar epidural: 8–12 mL/hr good starting point (may require fine-tuning).
- Thoracic epidural: 6–8 mL/hr good starting point (may require fine-tuning).
- PCEA: 3-mL bolus every 15 min (start background infusion at lower end of suggested scale).

Causes of Inadequate Epidural Anesthesia

- Improper injection location (false loss of resistance).
- Unilateral block: Occurs when the catheter is located lateral to the midline or has traveled out of the epidural space. It can often be remedied by pulling the catheter out 1–2 cm.
- Segmental sparing: May occur due to septa that may exist within the epidural space.
- Sacral sparing: Due to an inadequate block of the very large nerve roots L5, S1, and S2.

Caudal Anesthesia

A very common technique used in children.

- **Uses:** Procedures below the diaphragm. Mostly used as a supplement to general anesthesia or for analgesia. In adults, it can be used for anorectal procedures.
- **Technique:** In children, it is usually performed in the lateral or prone position after general anesthesia has been induced. A needle is inserted into the sacral hiatus at a 45-degree cephalad angle. Once the sacrococcygeal ligament is penetrated (heralded by a "pop"), the angle of the needle is ↓ and it is advanced (Figure 7-3). If there is negative aspiration for CSF or blood then either medication can be injected or a catheter can be inserted.
- **Medication:** 0.5–1.0 mL/kg of 0.125–0.25% bupivacaine (with or without epinephrine) or 0.2% ropivacaine is commonly used in children. Opioids can be added as well. In adults, 15–20 mL of 1.5% lidocaine (with or without epinephrine and fentanyl) is commonly used.
- **Contraindication:** Patients with pilonidal cysts should not receive caudal blocks because bacteria from the cyst could be transferred into the epidural space by the needle passing through it.

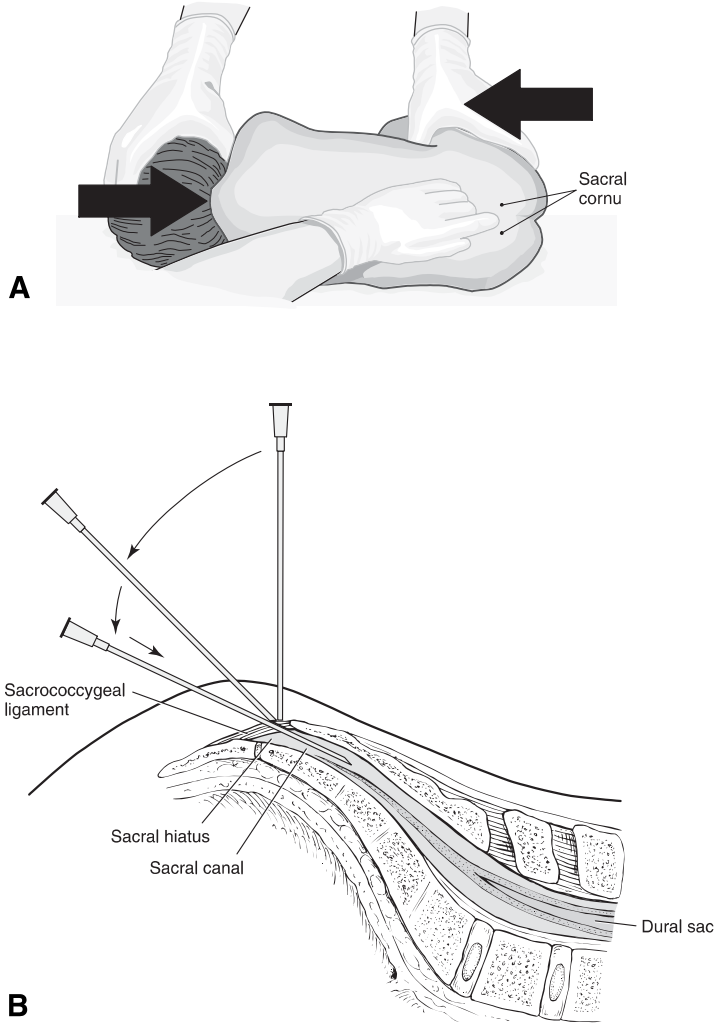

FIGURE 7-3. Caudal blockade.

A: Positioning and palpation of landmarks for caudal block. **B:** Needle insertion through sacrococcygeal membrane. (Reproduced, with permission, from Morgan G et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006: 315.)

Combined Spinal Epidural (CSE)

Combines the fast onset and dense block of spinal anesthesia with the repeat dosing ability of an epidural.

Technique

- An epidural is placed (a normal one or one specific to CSE) in one of the lower lumbar spaces, so as not to risk touching the spinal cord with the spinal needle.
- Once loss of resistance is obtained, a spinal needle is placed through the epidural needle and advanced until there is free flow of CSF.
- At this point, the spinal dose of local anesthetic is injected and the spinal needle is withdrawn. The epidural catheter can then be threaded as with a regular epidural (Figure 7-4).

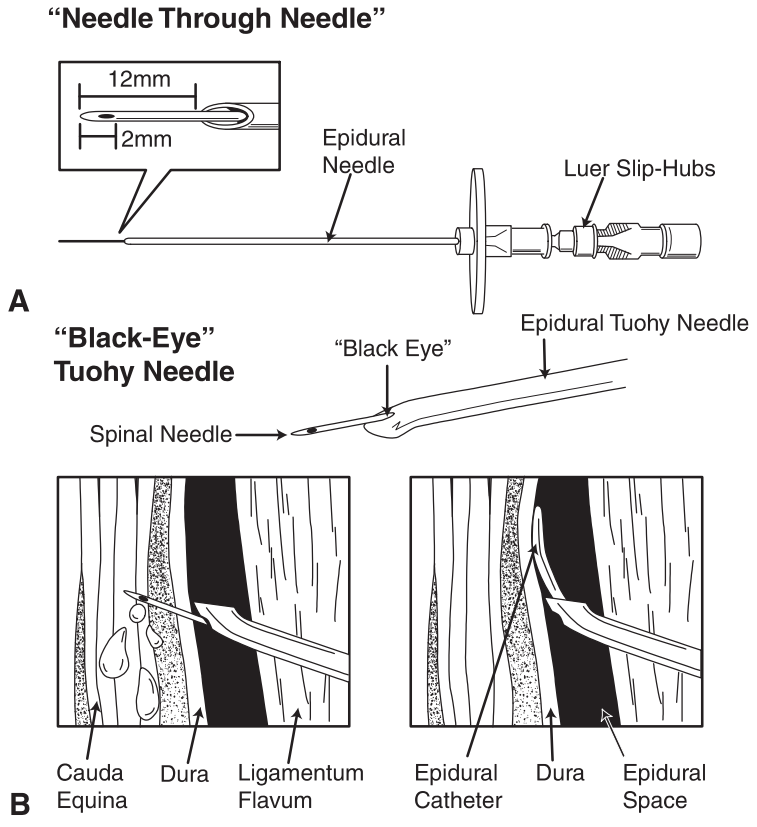

FIGURE 7-4. Combined spinal epidurals.

A: Combined spinal and epidural needles. **B:** Combined spinal-epidural technique. Left panel shows the spinal needle projecting through into the subarachnoid space while the Tuohy needle remains in the epidural space. Right panel shows the spinal needle withdrawn and the flexible epidural catheter advancing cephalad. (Reproduced, with permission, from *Cousins & Bridenbaugh's Neural Blockade In Clinical Anesthesia and Management of Pain*, 4th ed. New York: Lippincott Williams & Wilkins, 2009: 266.)

Delays in neuraxial anesthesia placements with anticoagulants:
Clopidogrel: 7 days
Abciximab: 24–48 hr
Tirofiban and eptifibitide: 4–8 hr
Lovenox: 10 hr (but catheters must be removed 10–12 hr after last dose and 2 hr before next dose).

Neuraxial Anesthesia and Anticoagulation

- NSAIDS and subcutaneous unfractionated heparin are not contraindications for neuraxial anesthesia.
- Antiplatelet medications (eg, clopidogrel or glycoprotein IIb/IIIa inhibitors) are considered contraindications for neuraxial anesthesia, and the drug effects have worn off when using these techniques. Should wait:
  - Clopidogrel: 7 days
  - Abciximab: 24–48 hr
  - Tirofiban and eptifibitide: 4–8 hr
- Neuraxial anesthesia should be performed no less than 10 hr after a dose of low-molecular-weight heparin.
- A patient who is receiving low-molecular-weight heparin should have an indwelling epidural (or spinal) catheter removed 10–12 hr after the previous dose and at least 2 hr before the next dose.
- Patients who are taking long-term warfarin must stop the medication and have a normal International Normalized Ratio (INR) prior to performance of neuraxial anesthesia.

COMPLICATIONS

- **Postdural puncture headache (PDPH):** Can occur anytime the dura is punctured.
  - Can occur after a "wet tap" (when the dura is punctured unintentionally during an epidural), an uneventful epidural, spinal anesthesia, a lumbar puncture, or a myelogram.
  - The headache is usually a throbbing or constant pain that is bilateral, frontal or behind the eyes, occipital, and extends into the neck. There may be photophobia or nausea/vomiting, but it is characterized by its positional nature. The headache is relieved by lying down and worsened by sitting up or standing.
  - Onset: Usually 12–48 hr after the procedure.
  - ↑ risk with young, female patients who are pregnant and with the use of larger, cutting needles.
  - PDPH is believed to be due to a continuous CSF leak through the punctured dura causing a ↓ in ICP.
  - Treatment:
    - Initially, conservative: Bed rest, analgesics (nonsteroidal anti-inflammatory drugs [NSAIDs], acetaminophen, vigorous hydration (IV or PO).
    - Cerebral vasoconstrictors (caffeine, sumatriptan, and methergine) have been widely used but with variable success.
    - Epidural blood patch: More invasive treatment but with 90% effectiveness. An epidural is performed at, or one level below, the original injection site and 15–20 mL of autologous blood is inserted (aseptically) into the epidural space. If done within 24 hr of the puncture, the success rate drops dramatically.
- **Backache:**
  - Occurs more commonly with neuraxial anesthesia than with general anesthesia.
  - Usually self-limited or requires treatment with NSAIDs.
  - Due to localized tissue trauma, muscle spasm, ligament strain, or irritation from the local anesthetics.
- **Urinary retention:**
  - Most common in men, due to blockade of the S2–S4 nerve roots.
  - Treat with bladder catheterization acutely, but it usually resolves as the block wears off.
- **High spinal or total spinal:**
  - Occurs when the block extends into the cervical levels and brain stem.
  - Signs include significant hypotension, bradycardia, respiratory insufficiency (→ apnea), and unconsciousness.
  - Treatment: Airway and ventilatory support, IV fluid hydration, pressor support (ephedrine, phenylephrine, epinephrine, and/or dopamine), and atropine (for bradycardia).
- **Subdural injection:**
  - Subdural space is located between dura and arachnoid mater and extends intracranially.
  - Small amounts of local anesthetic injected can cause a profound sympathetic block.
  - Presents with sensory block with mild motor block.
  - Onset is 10 – 15 min longer than total spinal.
  - Treatment: Same as total spinal.
- **Cardiac arrest:** Can occur with spinal anesthesia.
  - Due to ↑ vagal response to ↓ preload, usually in young healthy patients.
  - May be prevented with prehydration. Bradycardia should be treated with atropine (early), and may require ephedrine or epinephrine.

ABGs are often unchanged in high spinals.

- **Systemic toxicity of local anesthetic:** Occurs mostly with epidural anesthesia.
  - Due to inadvertent intravascular injection.
  - Early symptoms: Tinnitus, tongue numbness, light-headedness. With higher levels of local anesthetics, there can be seizures, unconsciousness, hypotension, cardiac arrhythmias, and complete cardiovascular collapse.
  - Severe systemic toxicity can usually be avoided by the use of a test dose and by incremental dosing of local anesthetic.
- **Transient neurologic symptoms (TNS):**
  - Back pain with radiation to the legs that resolves within a few days to a week. Patients with TNS do not suffer from sensory or motor deficits. In particular, if there is motor involvement, this must be investigated immediately.
  - Risk factors: Lidocaine spinal anesthesia for knee arthroscopy or a procedure done in the lithotomy position, ambulatory surgical status, obesity.
- **Neurologic injury:**
  - Peripheral neuropathies (with paresthesia and limited motor weakness) are most common.
  - May be due to needle trauma to the nerve roots sometimes heralded by a pain or paresthesia during performance of the block.
  - Paraplegia can occur if local anesthetic is injected into the spinal cord, just as function of the nerve can be destroyed during any intraneural injection.
  - Local anesthetics themselves can be neurotoxic.
  - Not all postoperative neurologic injury that occurs during neuraxial anesthesia is necessarily due to the block (positioning or preexisting medical conditions may be the cause).
  - **Cauda equina syndrome (CES):** Injury to multiple nerve roots causing bowel and bladder dysfunction, paresis of the legs, sensory deficits, and pain. May be caused by neurotoxicity due to inadequate distribution of hyperbaric lidocaine (most commonly) with pooling around the cauda equina.
- **Infection:**
  - Meningitis and arachnoiditis: Due to contamination of the needle, medication being injected, or from bacteria on the skin.
  - Epidural abscess: A rare complication of (typically) epidural anesthesia. Four clinical stages: (1) back pain with tenderness; (2) nerve root or radicular pain develops; (3) motor and/or sensory deficits develop, or there may be sphincter dysfunction; (4) paraplegia or paralysis.
  - Diagnosis: Culture of epidural catheter tip, blood cultures, magnetic resonance imaging (MRI) or computed tomography (CT) scan.
  - Treatment: Neurosurgical intervention is often required, but some cases may be treated conservatively with antibiotics.
- **Spinal or epidural hematoma:**
  - Usually occurs in patients with coagulopathies or bleeding diatheses.
  - Symptoms are rapid in onset and are due to mass effects. They start as back and leg pain but advance to numbness, weakness, and possibly bowel dysfunction.
  - Diagnosis: MRI, CT, or myelogram.
  - Treatment: Emergent surgical decompression (if done within 8–12 hr, then recovery is better).
  - Prevention: Avoidance of neuraxial techniques in patients with coagulopathy, bleeding diathesis, thrombolytic therapy, platelet dysfunction, and thrombocytopenia.

Indications

- Sole technique for surgical anesthesia (+/– light sedation).
- Nerve block for postop pain combined with general anesthesia.
- Many of these (especially many of the upper- and lower-extremity blocks) can be performed with the placement of a catheter for a continuous nerve block rather than just a single injection.
- Alternative for patients for whom general anesthesia is not ideal (such as with severe postoperative nausea and vomiting, risk or history of malignant hyperthermia, or hemodynamically unstable or very sick patients.

Risks

All are rather rare, but are common to all peripheral blocks.

- Local anesthesia toxicity (intravascular injection) including seizures and cardiovascular collapse.
- Nerve damage with short- or long-term paresthesias.
- Infection.

Contraindications

- Uncooperative patient or patient refusal.
- Systemic infection (for catheter placement).
- Preexisting peripheral neuropathy (controversial).
- +/– performing a block on a patient under general anesthesia (although this is routinely done in children).

Preparation

- **Equipment and monitoring:**
  - ASA monitors should be applied.
  - Resuscitation equipment should be available along with supplemental oxygen (for management of local anesthetic toxicity should there be intravascular injection).
  - Intralipid 20% should always be immediately available wherever blocks are performed, for management of cardiac toxicity.
- **Premedication:** Benzodiazepines +/– opiates, to help patient tolerate the procedure (although only enough so that communication can be maintained).

Techniques

- **Field block:** Performed by injecting a large volume of local anesthetic in the area around cutaneous nerves (eg, superficial cervical plexus block).
- **Use of fixed anatomic relationships:** Needle position and insertion site are determined by constant relationships between anatomic structures (eg, transarterial approach to the axillary block).
- **Paresthesia:** The paresthesia that is produced in the distribution of a sensory nerve is used to identify when the needle makes contact with that nerve. Since deliberate needle-nerve contact is sought, there is a theoretically higher chance that the needle is intraneural and not perineural, as desired. For this reason, care must be taken while injecting the local anesthetic, while monitoring for signs of intraneural injection such as high pressures during injection.

- **Nerve stimulator:** A nerve stimulator connected to an insulated needle applies a low-level electrical current from its tip, producing motor nerve stimulation which → muscle contractions. The particular muscles that contract indicate which nerve is being stimulated. As the needle gets closer to the nerve, less current is required for stimulation, and muscle contractions get stronger. A current of ≤ 0.5 mA is desirable.
- **Ultrasound guidance:** An ultrasonographic image of the nerve or plexus of interest in displayed on a screen and the needle advanced in real-time toward the target. Local anesthetic can be visualized spreading around the nerves, and structures such as blood vessels and pleura can be avoided.
- **Combination of techniques:** Many practitioners choose to combine ultrasound (which gives anatomical confirmation) with nerve stimulation (which gives functional confirmation).

Specific Nerve Blocks

- Nerve stimulation remains the primary mode of locating nerves or plexi when performing peripheral nerve blocks, although ultrasound-guided blocks are becoming more and more popular.
- The two methods are fundamentally different in their approach. In general, the principles that apply to performing one ultrasound-guided block can be (with a few exceptions) applied to all ultrasound-guided blocks.
- For this reason, each of the following blocks will be considered individually with the nerve stimulation method, followed by a general discussion of ultrasound-guided regional anesthesia techniques.

Brachial plexus mnemonic:

Randy Travis Drinks Cold Beer.

Upper-Extremity Blocks

- Used for procedures of the upper extremity or shoulder (see Table 7-2).
- Brachial plexus anatomy (Figure 7-5).
  - Ventral rami (roots) of C5–T1 → trunks → divisions → cords → terminal branches.
  - Anatomic relationships:
    - Trunks: Lateral aspect of transverse processes to the clavicle.
    - Divisions: Lie under the clavicle.

TABLE 7-2. Innervation of Upper-Extremity Muscles

Nerve	Origin	Muscles Controlled
Musculocutaneous	C5–C7	Anterior arm flexors
Axillary	C5–C6	Deltoid, teres minor
Radial	C5–T1	Posterior muscles of arm/forearm/hand
Median	C5–T1	Anterior forearm flexors
Ulnar	C8–T1	Anterior forearm flexors, intrinsic hand muscles

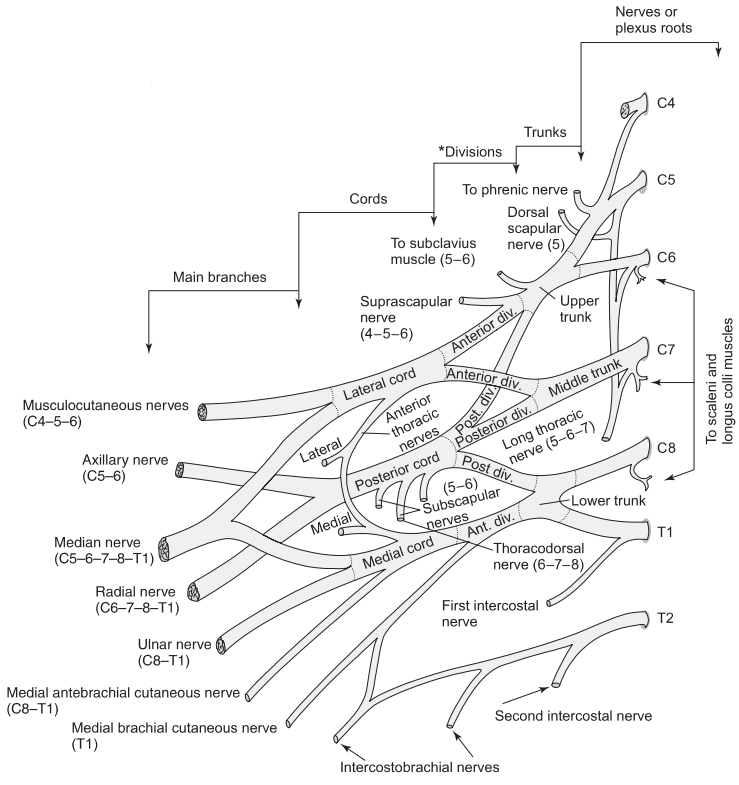

FIGURE 7-5. Anatomy of the brachial plexus.

(Reproduced, with permission, from Morgan G et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006: 330.)

- Cords: Formed just below the clavicle and are named for their position relative to the axillary artery.
- Branches: Begin at the lateral border of pectoralis minor.

- The nerves of the brachial plexus are contained within a compartment that is partly composed of loose fascia and partly by surrounding muscular planes. Injection of local anesthetic into this compartment (or "sheath," as it is commonly called) will spread throughout it, → blockade of all of the nerve roots within it.

Interscalene Block (Brachial Plexus)

Blocks C5–C7 (may miss C8–T1).

Uses

Shoulder, arm, and forearm (although it may miss the distribution of the ulnar nerve if sufficient spread to distal trunks does not occur).

Anatomy

In the groove between the anterior and middle scalene muscles, the nerve roots become trunks. The classic location is within this groove at the level of the cricoid cartilage.

Technique

- Position: Supine with the head turned to the contralateral side.
  - 22-gauge, 1.5-inch needle is inserted perpendicular to the skin and directed slightly medially and caudally (Figure 7-6).
  - A paresthesia in the arm or muscle contraction in the arm, wrist, hand, deltoid, or pectoralis should be obtained.
  - Volume of local anesthetic is dependent on the technique used. If nerve stimulation is used, 30–40 mL of local anesthetic should be injected. However, with ultrasound guidance, less volume may be adequate as long as the trunks are surrounded with local anesthesia.
  - Continuous catheters can be inserted and used for postoperative analgesia.

The phrenic nerve is almost always blocked in interscalene blocks.

Complications and Side Effects

- Phrenic nerve is almost always blocked with this approach, which may cause respiratory distress in patients with preexisting pulmonary disease.
- Recurrent laryngeal nerve block.
- Stellate ganglion blockade and Horner's syndrome.
- Intra-arterial injection into the vertebral artery (→ seizures quickly because it goes directly to the brain).
- Epidural, intrathecal, or subdural injection.
- Pneumothorax.
- Permanent phrenic nerve injury.

Supraclavicular Block (Brachial Plexus)

Blocks all elements of the brachial plexus.

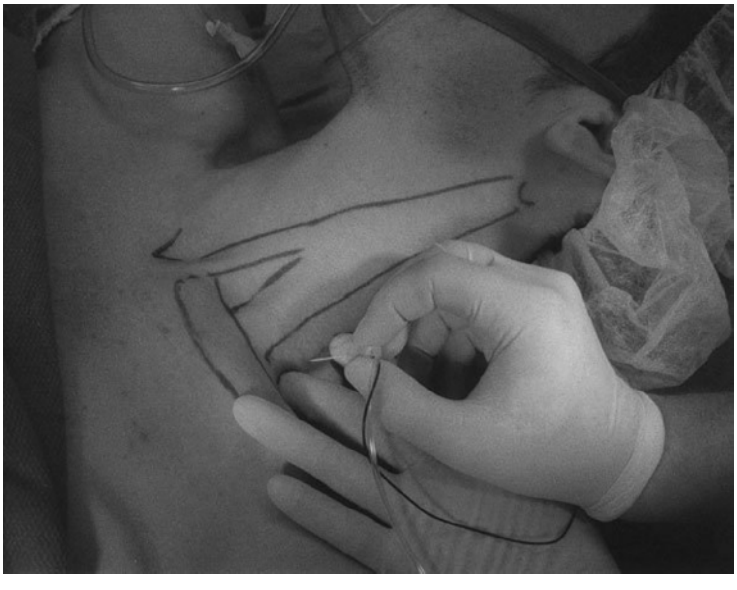

FIGURE 7-6. Needle insertion site and orientation for interscalene brachial plexus block.

(Reproduced, with permission, from Hadzic A. *Textbook of Regional Anesthesia and Acute Pain Management.* New York: McGraw-Hill, 2007: 411.)

Uses

Arm, forearm, and hand anesthesia. Can also be used for shoulder surgery.

Anatomy

The trunks of the brachial plexus run closely together as they become squeezed between the first rib and clavicle. Above the first rib, along the lateral border of the anterior scalene muscle, the brachial plexus can be located as it runs just lateral to the subclavian artery.

Technique

- Position: Supine with the head turned to the contralateral side.
- The lateral border of the sternocleidomastoid muscle is marked where it meets the clavicle. The point of needle entrance is 1 inch (or approximately a thumb's breadth) lateral to this.
- A finger is placed just above the clavicle at the insertion site and a 22-gauge 2-inch needle inserted just cephalad to the finger, aiming caudally at an angle of 45°, in the same plane as the midline (Figure 7-7).
- Twitches should be obtained above the clavicle, at a depth of 1–3 cm.
- 25–40 mL of local anesthetic should be injected when a hand twitch (either flexion or extension) is obtained.

Complications

- Pneumothorax
- Hemothorax
- Horner's syndrome
- Phrenic nerve blockade

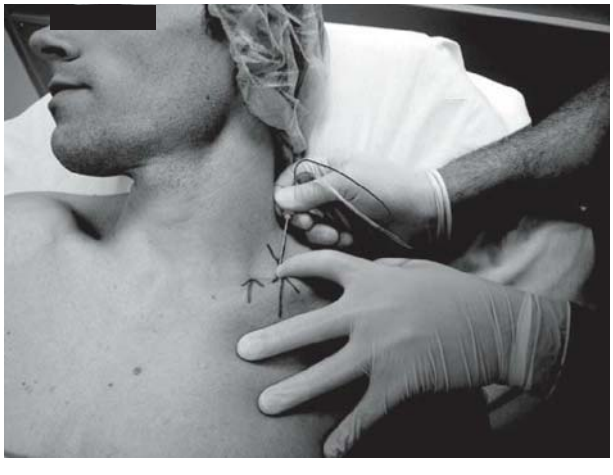

FIGURE 7-7. Landmarks and needle orientation for supraclavicular branchial plexus block.

(Reproduced, with permission, from Hadzic A. *Textbook of Regional Anesthesia and Acute Pain Management*. New York: McGraw-Hill, 2007: 423.)

Infraclavicular Block (Brachial Plexus)

Blocks the brachial plexus at the level of the cords.

Uses

Hand, forearm, elbow and upper arm anesthesia. Good technique for the placement of indwelling catheters.

Anatomy

The brachial plexus has divided into cords as it passes over the first rib and into the axilla.

Techniques

- **Raj approach:** The patient lies supine with his head turned to the contralateral side. The pulse of the axillary artery is identified with the arm abducted at 90 degrees. With a puncture site 2 cm caudal to the midpoint of the clavicle, a 21-gauge, 4-inch needle is inserted at a 45-degree angle toward the arterial pulse (Figure 7-8).
- **Coracoid approach:** With the patient lying supine, the coracoid process is identified. A 21-gauge, 4-inch needle is inserted 2 cm medial to and 2 cm caudal to the coracoid process in an angle that is perpendicular to the floor.
- 30–40 mL of local anesthetic are injected once hand twitches have been obtained.

Complications

- Pneumothorax
- Hemothorax
- Chylothorax
- Intra-arterial injection

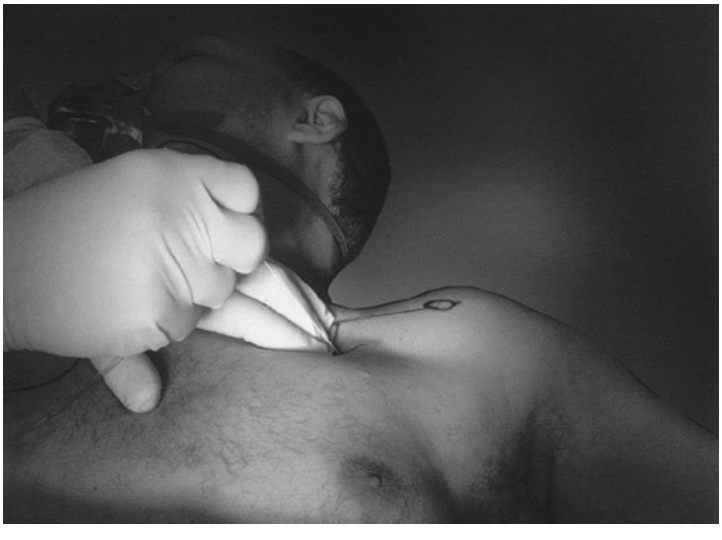

FIGURE 7-8. Needle insertion site and orientation for Raj technique of infraclavicular block.

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 10-12.)

Axillary Block (Brachial Plexus)

Blocks the median, radial, and ulnar nerves.

An axillary block misses the musculocutaneous and intercostobrachial nerves.

Uses

Forearm and hand anesthesia.

Anatomy

- The main landmark for this block is the pulsation of the axillary artery at its highest point within the axilla.
- The median and musculocutaneous nerves lie anterior to the artery, while the radial and ulnar nerves lie posterior to the artery.

Techniques

Both begin with the patient lying supine with the arm abducted and the elbow flexed at 90 degrees.

- **Transarterial approach:** A 22-gauge, 1.5 inch needle is inserted into the pulsation of the axillary artery until blood flows into the needle. The needle is then advanced slowly until blood flow stops. All or part of the 40 mL of local anesthetic can be injected at this posterior location. The needle can then be withdrawn, again looking for arterial blood flow. Once there is blood flow, the needle can be withdrawn slightly until the blood flow again stops and the remainder of the local anesthetic can be injected at this location anterior to the artery.
- **Nerve stimulator technique:** A 22-gauge, 1.5-inch nerve stimulator needle is inserted above and below the pulsation of the axillary artery and redirected as necessary to obtain muscle twitches in the desired brachial plexus distribution in the hand and biceps (Figure 7-9). Again, 40 mL of local anesthetic is injected in separate aliquots at each location, once satisfactory twitches are obtained.

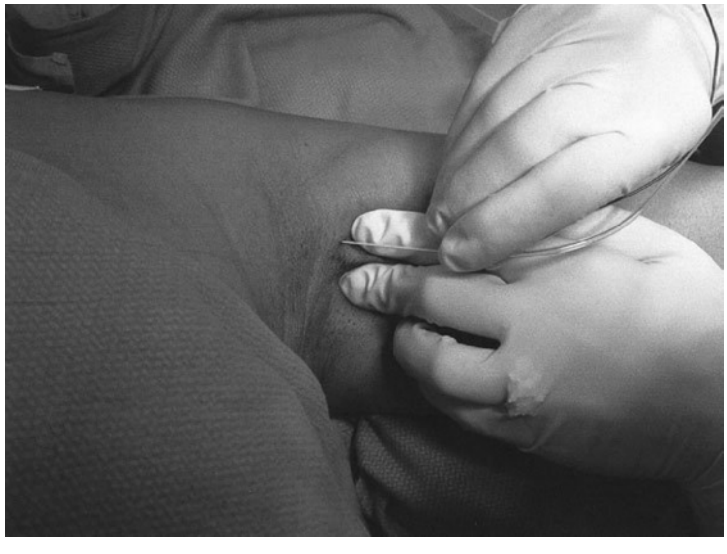

FIGURE 7-9. Needle insertion site and orientation for axillary block.

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 11-1.)

Complications

- Intravascular injection
- Hematoma

Avoid use of bupivicaine for Bier blocks.

Blockade of the Distal Branches of the Brachial Plexus

- **Intercostobrachial and medial brachial cutaneous nerves:** Blocked together with a subcutaneous field block using 5–10 mL of local anesthetic in the anteromedial axilla.
- **Musculocutaneous nerve:** Blocked by injecting about 5–10 mL of local anesthetic into the belly of the coracobrachialis muscle.
- **Radial nerve:**
  - Upper arm blockade: A needle is inserted 4 cm proximal to the lateral epicondyle of the humerus toward the bone; 5 mL of local anesthetic is injected once bone is contacted and the needle has been withdrawn just slightly.
  - Blockade at the elbow: A needle is inserted lateral to the biceps tendon in the antecubital space and directed toward the lateral epicondyle until either bone or a paresthesia/twitch is identified; 5 mL of local anesthetic is injected.
  - Distal forearm blockade: 5–10 mL of local anesthetic is administered as a field block 1–2 cm proximal to the styloid process of the radius, fanning anteriorly and posteriorly.
- **Median nerve:**
  - Blockade at the elbow: 5 mL of local anesthetic is injected just medial to the brachial artery pulsation in the antecubital area, with the needle directed toward the medial epicondyle, just in front of the bone.
  - Blockade at the wrist: About 5 mL of local anesthetic is injected just medial to the palmaris longus tendon and deep to the flexor retinaculum at the level of the proximal crease in the wrist.
- **Ulnar nerve:**
  - Blockade at the elbow: 3–5 mL of local anesthetic is injected just proximal to the medial epicondyle, with care taken not to insert the needle directly into the cubital tunnel (between the medial epicondyle and the olecranon process).
  - Blockade at the wrist: 5 mL of local anesthetic is injected either just medial to the ulnar pulse (if palpable) or just lateral and deep to the flexor carpi ulnaris tendon.
- **Wrist block:** Combination of radial, median and ulnar nerve blocks at the wrist. It provides anesthesia for the hand and fingers.
- **Digital blocks:** About 3 mL (total per digit) of local anesthetic (without epinephrine) is injected medially and laterally into the base of the digit.

IV Regional Anesthesia (Bier Block)

Uses

Procedures lasting ≤ 1 hr on the forearm or hand.

Technique

- An IV catheter is inserted into the surgical hand.
- The surgical arm is exsanguinated by being elevated and wrapped tightly with an elastic bandage. The proximal cuff of a double tourniquet is inflated and the hand is unwrapped. The absence of a radial pulse should

be confirmed (to assure there is no blood flow in or out of the arm, which could → systemic toxicity of local anesthetic).
- Local anesthetic is then injected into the IV catheter (50 mL of 0.5% lidocaine or 20 mL of 2% lidocaine). Onset of the block takes about 10 min.
- The distal cuff of the tourniquet can be inflated, and the proximal cuff deflated once the patient develops tourniquet pain.
- Care must be taken that the cuffs are not deflated too early, which may result in local anesthetic toxicity. A minimum time of 20 min is generally accepted, although the longer the duration before tourniquet deflation, the less the blood level of local anesthetic and the safer the deflation.

Lower-Extremity Blocks

- Innervation is derived from the L1–S3 nerve roots (see Table 7-3). These nerve roots form the lumbar plexus and the sacral plexus (most important component of which is sciatic nerve).
- The lumbar plexus divides the femoral nerve, obturator nerve, and lateral femoral cutaneous nerve, which are contained within a psoas compartment as they travel through the psoas muscle into the thigh.
- In the lower leg, the sciatic nerve divides the common peroneal and tibial nerves.
- These blocks can be used as an alternative to neuraxial blockade for anesthesia of the lower extremity.

Femoral Nerve Block

Uses

Anesthesia of the anterior thigh, knee, and a minor area on the medial aspect of the foot. It is often used for procedures on the knee.

Anatomy

The femoral nerve lies lateral to the femoral artery in the anterior thigh, just below the inguinal ligament.

Technique

- The patient is positioned supine and the femoral pulse is identified.
- The needle is inserted about 2 cm lateral to the pulsation of the artery

TABLE 7-3. Innervation of Lower-Extremity Muscles

Nerve	Origin	Muscles Controlled
Femoral	L2–L4	Quads, pectineus, iliacus
Obturator	L2–L4	Adductors, gracilis, obturator externus
Sciatic–tibial	L4–S3	Posterior muscles of thigh/leg/foot
Common peroneal	L4–S2	Short head biceps femoris, peroneal muscles, tibialis anterior, toe extensors

FIGURE 7-10. Needle insertion site and orientation for femoral nerve block.

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 21-13b.)

(about 2 cm below the inguinal ligament) until a paresthesia or quadriceps twitch is obtained (Figure 7-10).
- 15–20 mL of local anesthetic is injected.

Complications

- Nerve injury.
- Sometimes a single, large-volume injection is performed, with the goal of blocking the obturator and lateral femoral cutaneous nerves as well. This is termed a three-in-one block. The efficacy of the three-in-one block has been challenged: It seems unreliable in blocking the obturator nerve.

Lateral Femoral Cutaneous Nerve Block

Uses

Anesthesia of the lateral thigh.

Anatomy

The nerve becomes accessible near the anterior superior iliac spine (ASIS) as it passes below the ligament.

Technique

- The patient lies supine and a needle is inserted 2 cm medial to and 2 cm caudal to the ASIS.
- 10–15 mL of local anesthetic is injected in a fanning pattern once the needle "pops" through the first layer of fascia (fascia lata).

Complications

Few specific to this block.

Lumbar Plexus Block

Uses

- Postop pain control following hip, thigh, and knee procedures.
- Covers femoral, obturator and lateral femoral cutaneous nerves.

Anatomy

- L2–4 branches emerge from foramina into the psoas muscle.
- Within the psoas the nerves divide into anterior (obturator nerve) and posterior (femoral nerve) branches.

Technique

- The patient is placed in a lateral decubitus position.
- Midline and iliac crest identitified and line drawn between the structures.
- Needle is inserted 4 cm lateral to the midline.
- Obtain twitch of quadriceps or patella.

Complications

- Epidural spread causing high neuraxial anesthesia.
- Hypotension.
- Iliopsoas or renal hematoma.

Sciatic block landmarks: Posterior superior iliac spine, greater trochanter, sacral hiatus.

Sciatic Nerve Block

Uses

Anesthesia of the lower leg and posterior aspect of the knee and thigh.

Anatomy

The nerve exits the pelvis through the sciatic notch after passing by the piriformis muscle. It then travels near the lesser trochanter of the femur as it courses down the thigh, splitting into the tibial and common peroneal nerves at a variable (usually 5–12 cm) above the popliteal crease.

Techniques

- **Posterior (Labat) approach:**
  - The patient lies in the lateral decubitus position with the nonoperative leg down. The upper limb (one to be blocked) is positioned with hip and knee partially flexed.
  - The greater trochanter and the posterior superior iliac spine (PSIS) are palpated and marked, and a line drawn between them. The midpoint of this line is found, and the insertion site identified 4 cm distal from the midpoint, on a line 90 degrees from original line.
  - A 10-cm stimulating needle is inserted at right angles to all planes until a hamstring, calf, or foot twitch is obtained (Figure 7-11).
- **Anterior lithotomy approach:**
  - The patient lies supine with the operative leg flexed 90 degrees at the knee and hip (unilateral lithotomy position).

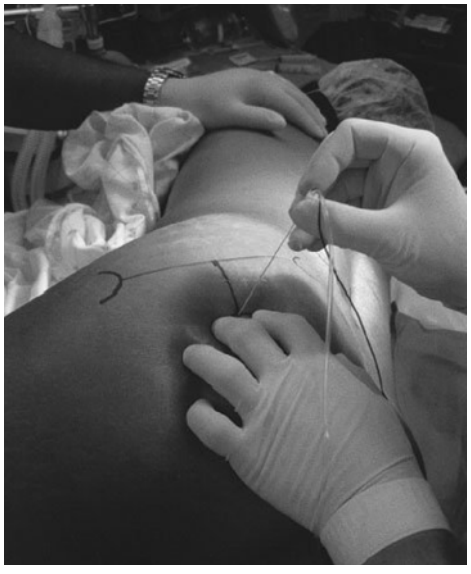

FIGURE 7-11. Needle insertion site and orientation for sciatic nerve block (Labat approach).

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 19-1.)

- A 10-cm stimulating needle is inserted at the midpoint between the greater trochanter and the ischial tuberosity until a hamstring, calf, or foot twitch is obtained.

- **Anterior approach:**
  - The patient lies supine and a line drawn along the inguinal crease.
  - The femoral pulse is then palpated and marked on this line, and a second line extended in a perpendicular fashion 4–5 cm toward the thigh.
  - A 10-cm stimulating needle is inserted and directed straight down toward the floor until a hamstring, calf, or foot twitch is obtained.
  - Occasionally, bone is contacted on the initial needle insertion (lesser trochanter). In this situation, internal rotation of the thigh often allows for displacement of this bony structure out of the way, allowing passage of the needle past the femur towards the sciatic nerve.
- 20 mL of local anesthetic is all that is required for these blocks, as it is a solitary nerve, rather than a plexus.

Efficacy of a popliteal block for surgery can be limited by the use of tourniquets.

Complications

Puncture of the superior or inferior gluteal arteries (posterior approach) or femoral artery or nerve (anterior approach).

Popliteal Block

This is actually a block of the sciatic nerve performed near the popliteal fossa proximal to the nerve's division into two terminal branches.

Uses

Anesthesia/analgesia of the calf, foot, and ankle.

Anatomy

- The sciatic nerve travels down the thigh and splits into the tibial and common peroneal nerves in the proximal aspect of the popliteal fossa.
- The nerves lie superficial (posterior) to the popliteal vessels.
- The tendons of the biceps femoris (laterally) and semimembranosus/semitendinosus muscles (medially) define the sides of the popliteal triangle, and can be palpated while the patient is in the prone position.
- The nerve(s) lie in the middle of this triangle, approximately 7-10 cm proximal to the popliteal crease.

Techniques

- **Prone approach:**
  - The patient is positioned prone and the biceps femoris tendon (laterally) and the semitendinosus tendon (medially) are identified and marked (to outline the popliteal fossa).
  - The popliteal crease is also marked, thereby forming a triangle with the two tendons.
  - The midline of the popliteal crease is located, and a line is drawn perpendicular to it, ending at the point of the triangle.
  - A 10-cm stimulating needle is inserted 1 cm lateral to this line and about 7–8 cm proximal to the crease until an ankle or foot twitch is obtained (Figure 7-12).
- **Lateral approach:**

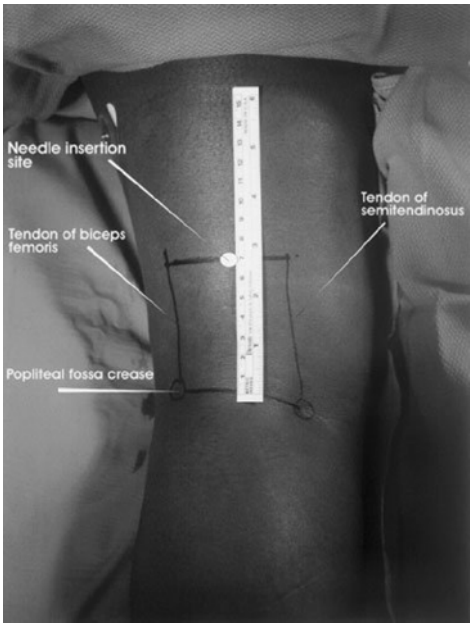

FIGURE 7-12. Needle insertion site for popliteal block (prone approach).

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 22-17.)

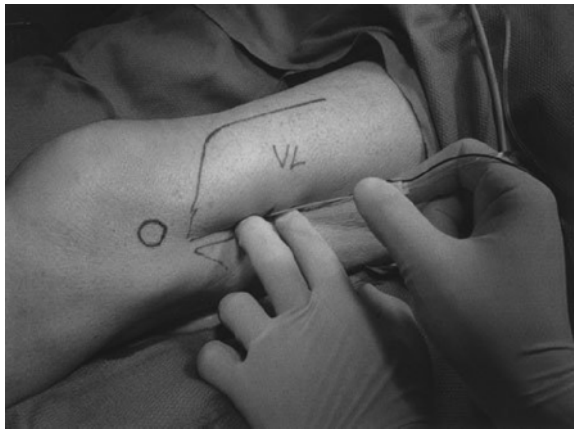

FIGURE 7-13. Needle insertion site and final needle orientation for popliteal block (lateral approach).

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 23-1.)

- The patient lies supine, and the groove between the biceps femoris muscle and the vastus lateralis muscle is identified on the lateral side of the thigh.
- A 10-cm stimulating needle is inserted in this groove 7–8 cm proximal to the popliteal crease at a 30-degree posterior angle and advanced until ankle or foot twitches are obtained (Figure 7-13).
- For either approach, 30–40 mL of local anesthetic should be injected to ensure adequate coverage of both the tibial and peroneal components.

Complications

Arterial or venous puncture.

Ankle Block

Uses

Anesthesia/analgesia of the foot.

Anatomy

The foot is supplied by five nerves, four of which are branches of the sciatic nerve (Figure 7-14 and Table 7-4).

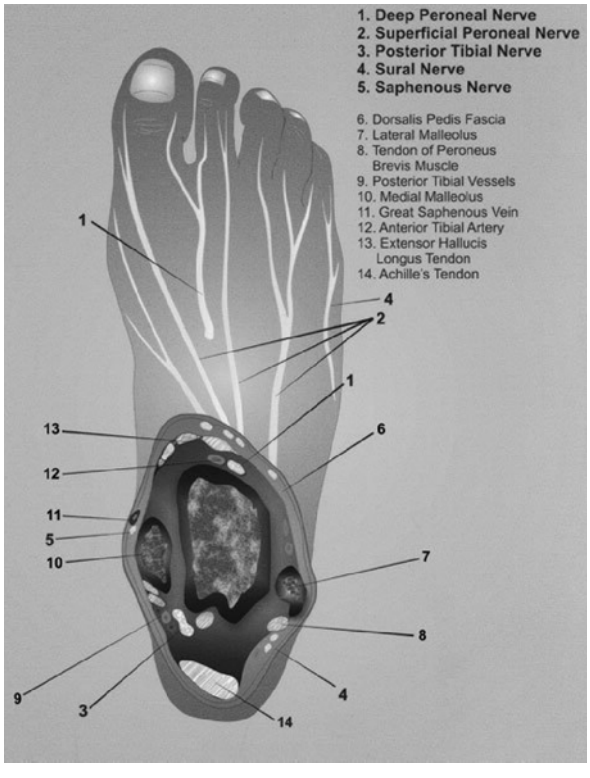

FIGURE 7-14. Cross-sectional anatomy of the ankle at the level of the malleoli.

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 24-2.)

TABLE 7-4. Innervation of Foot

	Location	Innervation
Deep peroneal nerve	Lateral to the flexor hallucis longus at the level of the medial malleolus.	Sensation between the first and second toes and motor function to toe flexors.
Superficial peroneal nerve	Lateral to the extensor digitorum longus at the level of the lateral malleolus, superficial.	Sensation to dorsal aspect.
Posterior tibial nerve	Posterior to the posterior tibial artery at the level of the medial malleolus.	Sensation to planar aspect.
Sural nerve	Between the Achilles tendon and the lateral malleolus.	Sensation to posterolateral leg, lateral foot, and fifth toe.
Saphenous nerve	Anterior to the medial malleolus, originates from the femoral nerve.	Sensation to anteromedial leg and medial foot.

Technique

- The deep peroneal nerve is blocked by inserting a needle between the extensor digitorum longus and extensor hallucis longus tendons at the level of the medial and lateral malleoli. (See Table 7-4.)
- The superficial peroneal nerve is blocked with a subcutaneous ring across the dorsal surface of the ankle. This can be a continuation of the subcutaneous injections made for the sural and saphenous nerves.
- The posterior tibial nerve is blocked by inserting a needle posterior to the medial malleolus, alongside the posterior tibial artery pulse. When bone is contacted, the needle is withdrawn 1–2 mm to prevent subperiosteal injection, and the local anesthetic deposited.
- The sural nerve is blocked with a deep subcutaneous ring between the lateral malleolus and the Achilles tendon.
- The saphenous nerve is blocked with subcutaneous injection anterior to the medial malleolus.
- About 5–8 mL of local anesthetic should be used for each nerve.

Complications

- Hydrostatic damage to small nerves can occur with highly pressured injection.
- Direct nerve damage associated with the use of sharp needles has been reported.

Head and Neck Blocks

Superficial Cervical Plexus Block

Superficial cervical plexus block landmarks:
Mastoid process
Sternocleidomastoid muscle
Sternal notch

Uses

Anesthesia for procedures on the anterior neck (eg, carotid endarterectomy, cervical lymph node biopsy, excision of thyroid nodule), analgesia for clavicle fracture.

Anatomy

- The cervical plexus is formed from the roots of C1–C4, which, after emerging from behind the posterior border of the sternocleidomastoid muscle, send branches superficially in the anterior triangle of the neck, laterally down to the acromion and anteriorly to the skin overlying the clavicle.
- Some fibers cross the midline to innervate the contralateral anterior neck skin.

Technique

- The patient lies supine with the head turned slightly to the contralateral side. 20 mL of local anesthetic is drawn up and attached to a 25-gauge needle.
- The landmarks are the mastoid process, the sternocleidomastoid muscle, and the sternal notch.
- Using the two bony landmarks, the midpoint of the sternocleidomastoid is identified, and 10 mL of local anesthetic deposited at the posterior border of the muscle (Figure 7-15).
- A further 5 mL each is then injected along the posterior border proximally and distally (a distance of 2–3 cm each).

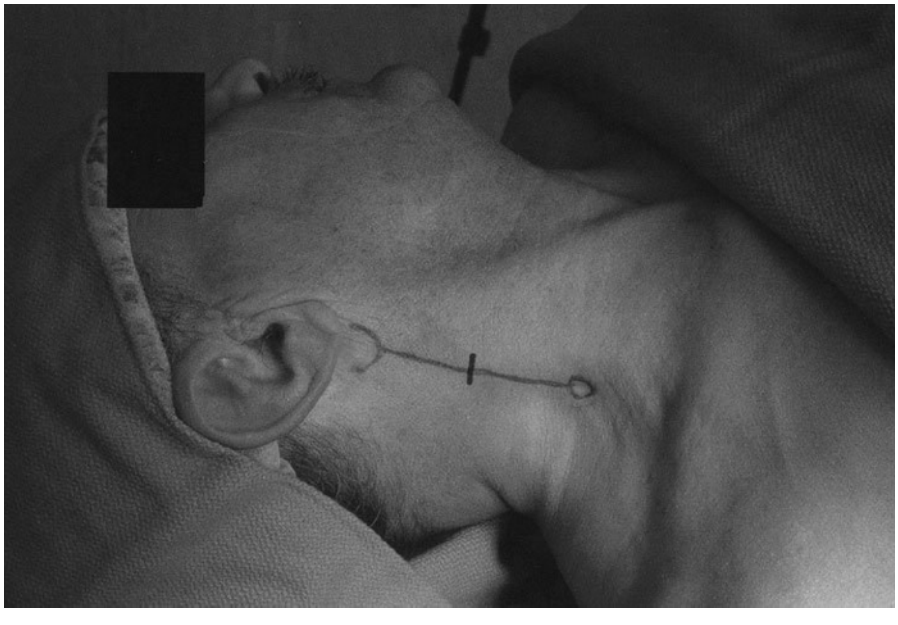

FIGURE 7-15. Needle insertion site for superficial cervical plexus block.

(Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 8-17.)

Complications

- As it is simply a subcutaneous injection of 20 mL of local anesthetic, superficial cervical plexus block is generally very safe.
- However, the use of deep cervical plexus blocks (a different technique involving needle contact with the transverse processes of the cervical vertebrae, and one that will not be described here) carries a significantly higher risk of serious complications, including vertebral artery puncture and injection, nerve root damage, contact with/injection into the cervical spinal cord, and total spinal. The added benefit of providing motor block to the strap muscles of the neck is increasingly thought to not outweigh these risks.
- Superficial cervical plexus block provides equivalent sensory anesthesia/analgesia as deep cervical plexus block, with less risk for complications. These authors, like many others, have abandoned the deep cervical plexus block in favor of the superficial block alone.

Airway Anesthesia

Uses

When used for awake laryngoscopy or fiberoptic intubation, anesthesia of the airway can ↓ gagging, laryngospasm, and unwanted cardiovascular responses associated with these procedures.

Techniques

- Premedication with anticholinergics (eg, glycopyrrolate 0.2 mg IV) to ↓ secretions will help with absorption of local anesthetic in mucous membranes.
- **Topical anesthesia:** Cetacaine spray can be applied to tongue and posterior pharynx. Alternatively, 4% lidocaine can be aerosolized with a nebulizer and inhaled over 15–20 min to anesthetize the entire upper airway.
- **Transmucosal glossopharyngeal nerve block:** 4% lidocaine-soaked pledgets can be grasped with Magill forceps and held for 10–15 sec behind each posterior tonsillar pillar. The glossopharyngeal nerve is located submucosally here and can be blocked easily, providing mucosal anesthesia down to the anterior surface of the epiglottis.
- **Superior laryngeal nerve block:** A needle is made to contact the superior cornu of the hyoid bone and 5 mL injected bilaterally. Provides anesthesia to the vocal cords, arytenoid cartilages, and posterior surface of the epiglottis.
- **Transtracheal anesthesia:** A needle is inserted into the cricothyroid membrane and advanced into the trachea (evidenced by aspiration of air). 5 mL of 2% lidocaine is injected during inspiration. Provides anesthesia to the vocal cords and proximal trachea.

Airway innervation:

Superior laryngeal (CN X)

Internal branch—sensation of epiglottis, arytenoids, vocal cords

External branch—cricothyroid muscle

Inferior laryngeal (CN X)

Sensation below vocal cords

All muscles except cricothyroid

Glossopharyngeal (CN IX)

Sensation of posterior one-third of tongue.

Complications

- Local anesthetic toxicity can occur because of the large amounts used.
- Gastric aspiration can occur because of blockade of the airway reflexes that normally prevent it.

Trunk Blocks

Intercostal Block

Uses

Postoperative analgesia, pain management for rib fractures, herpes zoster, or cancer pain.

Anatomy

- The spinal nerves at each thoracic level travel through the intervertebral foramen into a groove on the inferior side of the corresponding rib.
- The nerves lie within a neurovascular bundle along with the intercostal vein and artery, both of which lie superior to the nerve.

Technique

- The patient is placed in the supine, lateral decubitus, or prone position.
- The inferior level of each rib is marked and a needle is inserted, usually at the angle of the rib (about 7 cm from the spinous process of the corresponding vertebrae). The needle is advanced until contact is made with the rib, at which point the needle is walked off the inferior edge of the rib and advanced about 0.5 cm.
- 3–5 mL of local anesthetic is injected after negative aspiration.

Complications

- Pneumothorax.
- Local anesthetic toxicity is a significant concern because of the high level of absorption from this area.

Paravertebral Block

Uses

Breast surgery, inguinal hernia repair, procedures to the chest or body wall.

Anatomy

- The paravertebral space is bounded by the parietal pleura anterolaterally, the rib and intercostal muscles posteriorly, and the vertebrae and intervertebral foramina medially. Thoracic spinal nerves pass through the intervertebral foramen before dividing into anterior and posterior rami. Also located in this space is the sympathetic chain.
- Anesthesia for breast surgery requires blockade of T2–T6.
- Inguinal herniorraphy requires blockade of T9–L1.

Technique

- The patient is in a sitting position, and the spinous processes of the selected vertebrae are marked at their superior most point (C7 can be used as a convenient guide).
- A second mark is made 2.5 cm lateral to the each of these first points.
- Because of the angulation of the spinous processes, a needle inserted at each of these new points should contact the transverse process of vertebra immediately below (ie, spinous process of T4 should correspond to transverse process of T5).

- A 22-gauge Tuohy needle is advanced perpendicular to the skin until the transverse process is contacted. The needle is then redirected either above or below the transverse process and advanced 1 cm (Figure 7-16).
- 4 mL of local anesthetic is injected at each level after negative aspiration.
- Alternatively, a 20- to 30-mL bolus can be slowly injected at one level in the middle of the intended dermatomal area (single-injection method).
- A catheter can be introduced through the Tuohy needle for continuous postoperative analgesia.

COMPLICATIONS

Pneumothorax, epidural spread bilaterally.

ULTRASOUND-GUIDED NERVE BLOCKS

- Ultrasonography has become a popular and useful technology to guide the placement of nerve blocks.
- Its proposed benefits include visualization of the needle and target structures, thereby reducing the incidence of accidental puncture of nerves, vessels, and other unintended structures; and the ability to witness the expansion of the local anesthetic "pool" as the injectate is delivered, thereby ensuring the target nerve is "soaked" with local anesthetic.

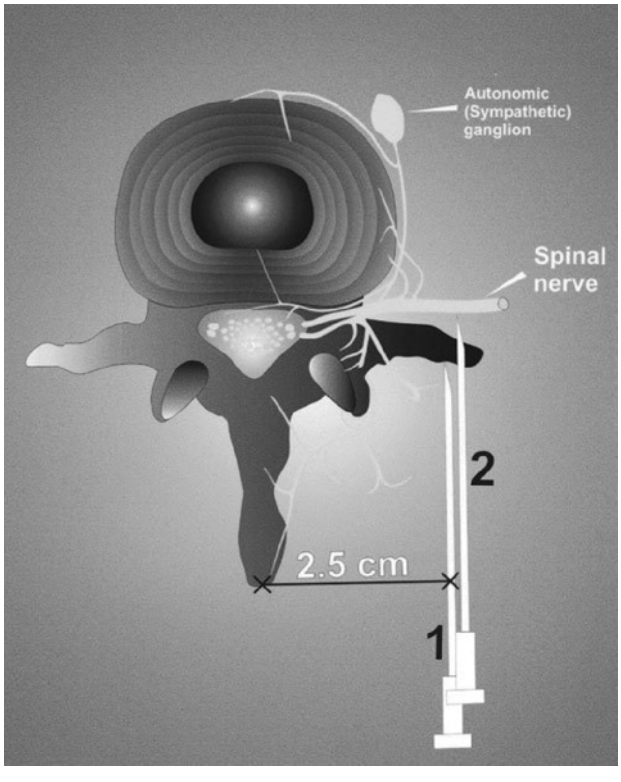

FIGURE 7-16. Needle orientation for the paravertebral block.

The needle tip contacts transverse process, then is withdrawn slightly and walked off the bone a further 1 cm into the paravertebral space. (Reproduced, with permission, from Hadzic A. *Peripheral Nerve Blocks: Principles and Practice.* New York: McGraw-Hill, 2004: Fig. 16-10.)

- Hard evidence of clear benefit over nerve stimulation is still lacking, but smaller studies are accumulating, indicating ↑ speed of block placement, ↑ block success, and fewer complications compared with the traditional methods of block placement.

Ultrasound Basics

- Ultrasound transducers contain piezoelectric crystals, which, when an electric current is applied, vibrate and produce sound in the range of 2–15 MHz (well above the range of human hearing).
- Sound waves enter tissues and reflect back at different rates and angles, depending on the tissue density and reflective properties. The same crystal then converts the sound waves back to an electrical signal, which is interpreted by the computer and shown on the monitor as a two-dimensional image of the underlying anatomical structures.
- *Echogenicity* refers to the property of tissues to reflect sound back to the transducer.
- Tissues that reflect most of the sound waves appear white, and are termed *hyperechoic* (eg, bone, tendon).
- Tissues that reflect little of the sound appear black or dark gray, and are termed *hypoechoic* (eg, most fluid-filled spaces such as blood vessels, cysts).
- Nerves can appear either hyperechoic (eg, sciatic nerve) or hypoechoic (eg, proximal brachial plexus), depending on the amount of connective tissue they have.

The Ultrasound Machine

Many different makes of machines exist, but all share some common features. There are a few settings that are important to be able to manipulate.

- **Frequency:** High-frequency sound waves in general provide a picture with higher resolution, but cannot penetrate very deep. Therefore, high-frequency settings (ie, 10–15 MHz) are appropriate for shallow blocks such as interscalene or femoral nerve blocks. Low frequency has better tissue penetration, but at the cost of reduced resolution. Low-frequency settings (2–8 MHz) are used for deep blocks such as the sciatic or the neuraxial space.
- **Depth:** The depth can be adjusted so that the target structure is seen in the middle of the screen image, and extraneous structures deep to it are not visualized.
- **Gain:** This is the property of the ultrasound machine to "hear" the incoming signal. If the gain is turned up, more of the signal will be shown on the image, and the screen will be in general more "white." Reducing the gain will darken the screen. Gain can be adjusted up and down to heighten the contrast between different structures, improving the visualization of the target nerves.
- **Transducer:** Two basic types of transducers (or "probes") exist, based on their shape and beam pattern:
  - **Linear-array transducers:** The probe surface is flat, or linear, and emits parallel sound waves, producing a rectangular image. Linear-array transducers are of generally higher frequencies and are best for shallow blocks.
  - **Curved-array transducers:** The probe surface is convex and emits sound waves in a fan-shaped pattern, resulting in a fan-shaped screen

Use high-frequency linear-array transducers for superficial blocks and low-frequency curved-array transducers for deeper blocks.

image. These are usually low-frequency probes and are used primarily for deeper blocks. One potential advantage for deeper structures is the wider field view, which can help identify peripheral landmarks that may not appear on a linear-array image.

Ultrasound Imaging and Local Anesthetic Injection

- **Image planes:** There are two principal image planes that are used when identifying a structure such as a nerve or blood vessel:
  - **Short-axis (transverse):** The ultrasound beam is perpendicular to the long axis of the nerve. This view will result in a nerve or vessel appearing like a circle on the image screen (Figure 7-17).
  - **Long-axis (longitudinal):** The ultrasound beam is parallel to the long axis of the nerve. This view will result in a nerve or vessel appearing like a long rectangle (Figure 7-18).
  - Most clinicians prefer to image nerves in the short axis, as there is less chance that slight movements of the transducer will cause the nerve to disappear from the screen.
- **Needle insertion:** Just as there are two orientations for the transducer, there are two markedly different ways to approach the target with the needle:
  - **In-plane approach:** The needle is directed at the ultrasound beam end-on, so that the whole length of the needle can be appreciated on

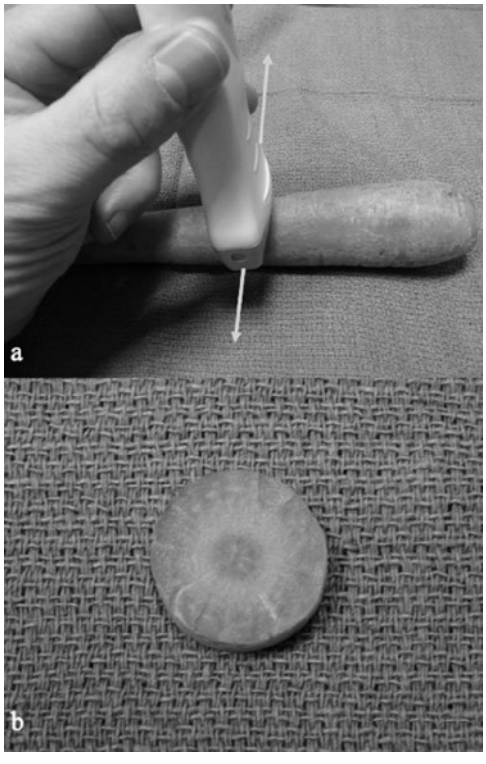

FIGURE 7-17. A: Short-axis orientation of the ultrasound probe. B: Resulting image (ie, a transverse "slice" of the structure").

FIGURE 7-18. A: Long-axis orientation of the ultrasound probe. B: Resulting image (ie, a longitudinal "slice" of the structure).

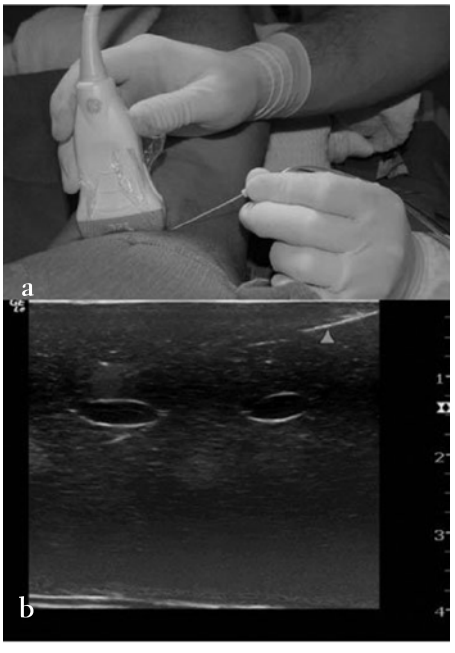

FIGURE 7-19. In-plane needle insertion.

A: Needle orientation relative to probe. **B:** Needle seen in same plane as image (triangle).

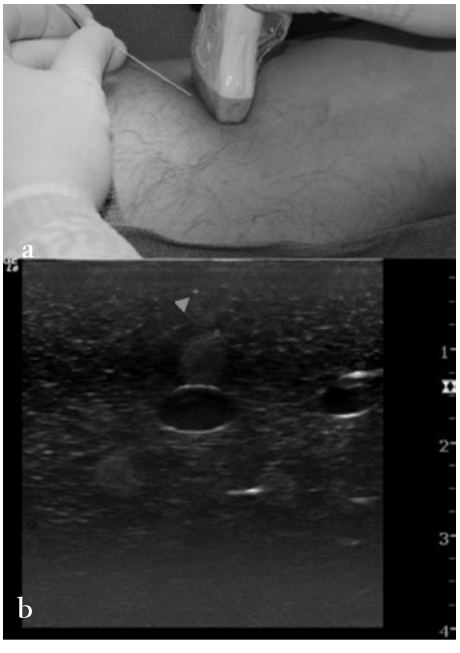

FIGURE 7-20. Out-of-plane needle insertion.

A: Needle orientation relative to probe. **B:** Needle seen as discrete "dot" crossing image plane (triangle).

the screen, provided that the needle and beam planes are lined up exactly (Figure 7-19).

- **Out-of-plane approach:** In this approach, the needle is inserted 90 degrees to the ultrasound beam. When the needle (tip or shaft) crosses the beam, its presence can be detected on the screen only as a discrete "dot" (Figure 7-20).
- Many practitioners prefer to use the in-plane approach, so that the whole needle can be visualized in real-time approaching the target nerve. It is easy to be fooled by the out-of-plane approach into thinking that the dot represents the tip, when in fact it is a portion of the shaft; meanwhile, the tip is dangerously beyond the plane of the image.

- **Local anesthetic injection:**
  - After "parking" the needle next to the nerve, the syringe is aspirated and a slow 1- to 3-mL bolus initiated.
  - A spread of dark (hypoechoic) injectate should be appreciated on the image screen where the needle tip is located. If this spread is *not* seen, the needle tip is not where it is thought to be, and the injection should be halted, as this represents injection into an unknown plane (including possible intravascular injection). The needle is then repositioned and another bolus attempted.
  - The remainder of the local can be injected slowly in increments, with intermittent aspiration.
  - Unlike nerve stimulator techniques, there is no recommended volume of injectate for each block, and simply depends on the visualization of spread around the target nerve or plexus.

Selected Ultrasound-Guided Blocks

The novice ultrasound-guided block practitioner, with some practice, can achieve quite a lot with a few carefully chosen techniques. Below are four ultrasound-guided blocks that every regional anesthesia enthusiast should know.

Ultrasound-Guided Interscalene Block

- The probe is placed on the anterolateral neck between the cricoid cartilage and the clavicle, and the anterior and middle scalene muscles identified lateral to the caroid artery and internal jugular vein. The hypoechoic trunks of the brachial plexus can be seen between the two muscles (Figure 7-21).
- A 5-cm, 22-gauge needle is advanced from the lateral side in-plane (through the middle scalene muscle) and local anesthetic deposited at all three trunks.

Ultrasound-Guided Supraclavicular Block

- The probe is placed just superior to the midclavicle, and the following structures identified: subclavian artery, first rib, pleura, and the brachial plexus lateral and slightly superior to the subclavian artery (Figure 7-22).
- A 5-cm, 22-gauge needle is advanced from the lateral side in-plane and local anesthetic deposited around the margins of the brachial plexus bundle. Often, two injections are sufficient: one below (on top of the rib) and one above.

Ultrasound-Guided Femoral Block

- The probe is placed at the inguinal crease in a transverse orientation, and the femoral artery is identified. Immediately lateral to the pulsating artery is the nerve, which is often seen as a flattened, triangular, hyperechoic area wedged between the iliacus muscle, the femoral artery, and the fascia iliaca (Figure 7-23).

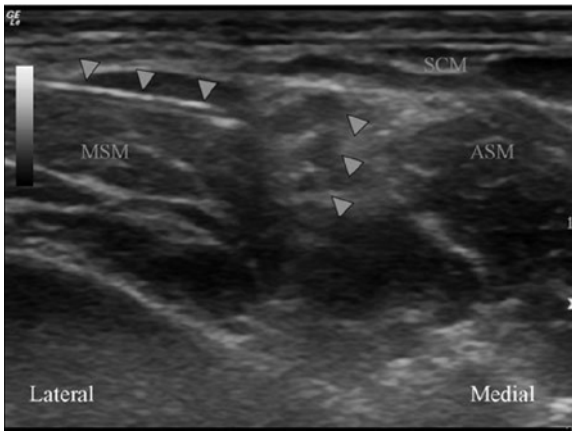

FIGURE 7-21. Ultrasound-guided interscalene block.

ASM, anterior scalene muscle; MSM, middle scalene muscle; SCM, sternocleidomastoid muscle. Orange triangles = brachial plexus. Blue triangles = needle.

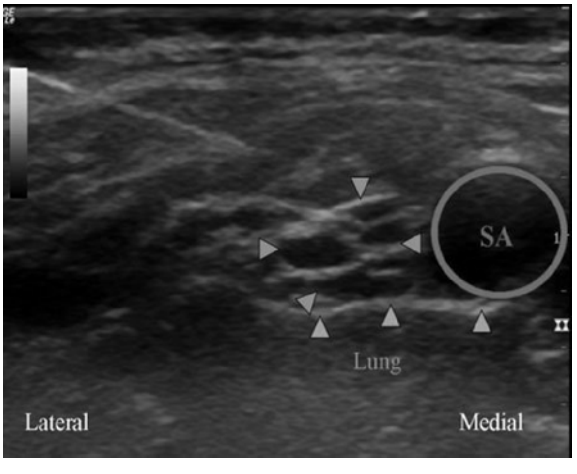

FIGURE 7-22. Ultrasound-guided supraclavicular block.

SA, subclavian artery. Orange triangles =brachial plexus. Green triangles = first rib.

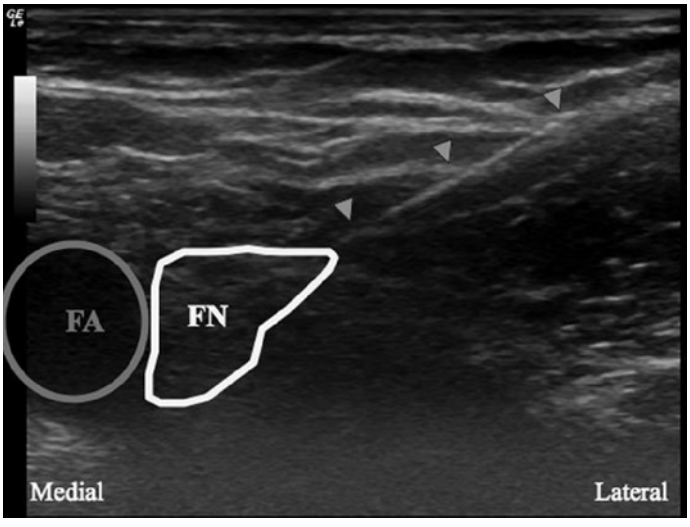

FIGURE 7-23. Ultrasound-guided femoral nerve block.

FA, femoral artery; FN, femoral nerve. Triangles = needle.

- A 5-cm, 22-gauge needle is advanced from the lateral side in-plane and local anesthetic deposited underneath +/– lateral to the femoral nerve, separating it from the iliacus muscle below.

Ultrasound-Guided Popliteal Block (Lateral Approach)

- With the leg raised and resting on a pillow or cushion so that the underside of the knee is free of the bed, the probe is placed in a transverse orientation at the popliteal crease.
- The depth is adjusted until the pulsating popliteal artery is visible on the screen. Just superficial and slightly lateral to the artery should be the tibial nerve, which appears as a hyperechoic round nerve with dark spots on the inside ("honeycomb").
- To achieve a good image, the probe often has to be tilted cephalad or caudad. Once a good image is seen, the probe is slid up the popliteal fossa in a cephalad direction, while carefully watching the tibial nerve. At some point proximal to the popliteal crease, a smaller, shallower nerve will enter from the lateral aspect of the screen and merge with the tibial nerve (Figure 7-24).

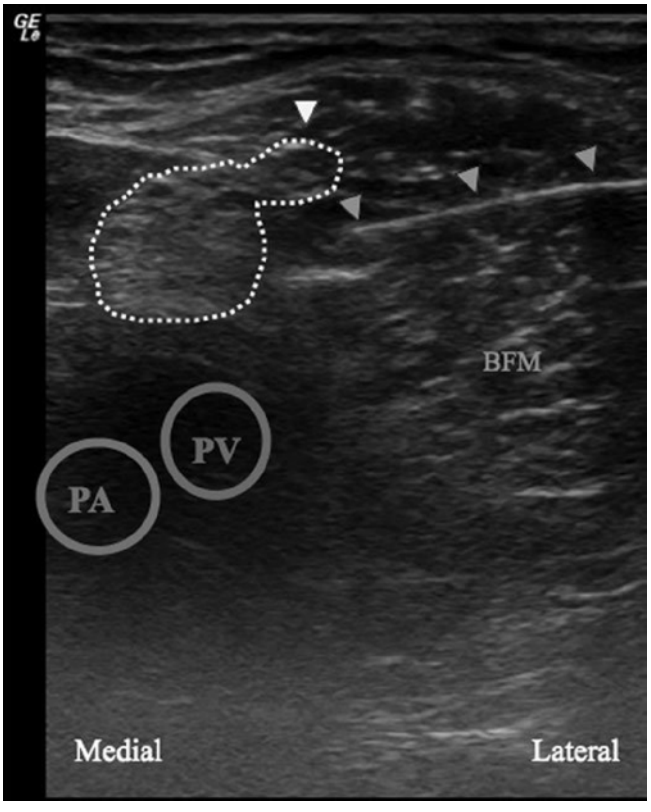

FIGURE 7-24. Ultrasonographic image of the sciatic nerve in the popliteal fossa.

Note peroneal nerve (yellow triangle) separating from tibial nerve as sciatic nerve travels distally. BFM, biceps femoris muscle; PA, popliteal artery; PV, popliteal vein. Triangles = needle.

This is the common peroneal nerve, and, once merged, these two comprise the sciatic nerve. It is at this point that the popliteal block is most effective, as the local is surrounding only one nerve, not two.

- A 10-cm, 21-gauge needle is advanced from the lateral side of the thigh, in-plane with the transducer. When contact is made with the sciatic nerve, local is deposited so that the nerve is completely surrounded. This may require two or more separate redirections and injections above, below, and beyond the nerve to achieve this.

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CHAPTER 8

Postoperative Recovery

Yetunde Orimogunje, MD

PACU Areas

- Phase I: Monitoring and staffing ratios equivalent to intensive care unit (ICU; ratio of 1.5 beds per operating room, ratio of one nurse per two patients); appropriate for patients who undergo neuraxial or general anesthesia until transfer to floor or phase II.
- Phase II: Stabilizes patient for care on surgical floor or discharge home; some patients who undergo monitored anesthesia care (MAC) can be fast-tracked to phase II avoiding phase I.

Standards for Postanesthesia Care

- Readily available anesthesiologist to ensure patient's safe recovery from anesthesia and discharge from the PACU.
- Staff of specially trained nurses.
- Proximity to the operating room.
- Equipment available for routine care: Supplemental oxygen, suction, monitoring of vital signs, pulse oximeter, electrocardiogram, ventilators, transducers for monitoring of intravascular pressures, devices for continuous infusions of medication.

All ambulatory patients who received sedating medications are required to have an adult companion to escort them home.

Transport from the Operating Room to PACU

- Patients should have stable and patent airway, adequate ventilation and oxygenation, and hemodynamic stability.
- Should be transported with oxygen supplementation to avoid transient hypoxemia ($Spo_2 < 90$), which occurs in 30–50% of otherwise healthy patients breathing room air.
- Unstable patients should be kept intubated and transported with a portable monitor, emergency drugs, and intubating equipment.

Admission to PACU

- Anesthesiologist provides nurse with pertinent details of patient's history, medical condition, anesthetic, and surgery.
- Vital signs recorded at least every 15 min.
- Supplemental oxygen based on Spo_2 readings on room air.

Discharge from PACU

- All patients must be evaluated by anesthesiologist prior to discharge.
- Criteria for discharge are established by Department of Anesthesiology, hospital, and medical staff.
- Varies according to patient's disposition to ICU, regular floor, outpatient department, or directly home.
- Minimal discharge criteria include ability to move extremities, ability to maintain and protect airway easily, arousability, stable vital signs for 30–60 min (see Aldrete Score in Table 8-1, where a score of 8–10 indicates readiness to move to next level of care).

TABLE 8-1. Aldrete Score for PACU Discharge

Activity	Moves four extremities	2
	Moves two extremities	1
	Not able to move extremities	0
Respiration	Able to breath and cough	2
	Dyspnea or limited breathing	1
	Apneic	0
Circulation	BP ± 20% of preanesthetic level	2
	BP ± 21-49% of preanesthetic level	1
	BP ± 50% of preanesthetic level	0
Consciousness	Fully awake	2
	Arousable	1
	Not responding	0
O_2 saturation	Maintains O_2 saturation > 92% on room air	2
	Supplemental O_2 to maintain O_2 saturation > 90%	1
	O_2 saturation < 90% with supplemental O_2	0

(Adapted from Duke J. *Anesthesia Secrets,* 3rd ed. Elsevier, 2006: 219.)

- Ideally, discharge criteria also include control of postoperative pain, control of nausea/vomiting, normothermia.
- Following regional anesthesia, patient should show signs of resolution of motor block.
- Lack of resolution of neuraxial block after 6 hr should raise suspicion of spinal or epidural hematoma.
- Causes for admission: Persistent hypoxemia, unresolving block, pain, persistent hypertension/hypotension or hemodynamic instability.
- Urination and drinking/eating before discharge is not required unless patient is diabetic or has history of urinary retention.

6–8 hours = time from onset of epidural hematoma symptoms to surgical decompression before irreversible neurologic injury ensues.

MANAGEMENT OF POSTOPERATIVE PROBLEMS

Upper-Airway Obstruction

CAUSES

- Tongue falling onto the posterior pharynx in unconscious patients.
- Laryngeal obstruction secondary to laryngospasm or edema, indicative of airway injury.
- Secretions, blood, or vomitus in the airway.
- External pressure on the trachea most commonly from a neck hematoma.

PHYSICAL EXAM

- Flaring of the nares.
- Retraction at the suprasternal notch (tracheal tug) and intercostal spaces.
- Vigorous diaphragmatic and abdominal contractions.

Negative pressure pulmonary edema usually occurs in young, muscular men. Treatment is supportive +/- duiretics.

TREATMENT

- **Obstruction:**
  - Head tilt–jaw thrust method: Most effective method of eliminating upper airway obstruction by tongue.
  - Nasopharyngeal airways: Tolerated in the semiconscious patient awakening from anesthesia.
  - Oral airways: May stimulate gagging and vomiting in semiconscious patient, can precipitate laryngospasm.
- **Laryngospasm:**
  - Incomplete: Extension of the head and anterior displacement of mandible and application of positive pressure with a bag and mask delivering 100% oxygen.
  - Complete: Treat with administration of succinylcholine 10–20 mg IV if above method is ineffective; intubation indicated if obstruction persists despite good head positioning and oral airway.
  - Complication: Prolonged laryngospasm can cause negative pressure pulmonary edema. This occurs secondary to high negative intrathoracic pressures generated after patient takes a deep breath against a closed glottis, causing marked ↑ in transmural pressure with subsequent fluid filtration into the lung.
- **Laryngeal edema:**
  - Humidified inhaled gases and nebulized epinephrine (0.25–0.5 mL of 2.25% epinephrine in 5 mL H_2O or normal saline).
  - Dexamethasone 0.15 mg/kg: Efficacy not confirmed.
- **Postoperative wound hematoma:**
  - Seen in head and neck, thyroid, and carotid surgery.
  - Can rapidly compromise the airway.
  - Open the wound to relieve tracheal compression.
  - Retained gauze packing: Rare occurrence seen after oral surgery in the hypopharynx.

Hypoxemia

Defined as $PaO_2 < 60$ mmHg.

CAUSES

- Right-to-left intrapulmonary shunt (atelectasis).
- Ventilation and perfusion (V/Q) mismatch (↓ functional residual capacity [FRC]).
- Alveolar hypoventilation (residual effects of anesthetic or neuromuscular blockers).
- Aspiration.
- Pulmonary embolus.
- Pneumothorax.
- Increased oxygen consumption (shivering).
- Decreased cardiac output.
- Advanced age.
- Obesity.
- Posthyperventilation hypoxia (compensatory hypoventilation replenishes CO_2, which was depleted by intraoperative hyperventilation).
- Postoperative pulmonary edema secondary to left ventricular (LV) failure, acute respiratory distress syndrome (ARDS).

Diagnosis

- Pulse oximetry facilitates early detection of hypoxemia.
- Arterial blood gas (ABG) confirms diagnosis and guides therapy.

Clinical Signs

- Obvious cyanosis may not be detected if hemoglobin concentration is reduced.
- Restlessness, tachycardia, cardiac irritability → obtundation, bradycardia, hypotension → cardiac arrest.

Treatment

- Supplemental O_2: FiO_2 30–60% +/– positive airway pressure (see Table 8-2).
- Naloxone for residual effects of opioids.
- Positive end-expiratory pressure (PEEP) for hypoxemia despite FiO_2 50%; increases FRC and PaO_2, which allows a ↓ in inspired O_2 without a ↓ in PaO_2. PEEP can impede venous return and occlude pulmonary capillary beds, causing a decreased CO and V/Q mismatch.
- Intubation and mechanical ventilation if hypoxemia persists despite FiO_2 100% or if hypercapnea accompanies supplemental O_2.
- Chest tube for symptomatic pneumothorax > 15–20% of lung field.

Nasopharynx acts as reservoir enabling a maximum of 40% O_2 to be delivered.

Hypoventilation

Defined as $PaCO_2 > 45$ mmHg.

Causes

- Drug-induced central nervous system (CNS) depression (volatiles, opioids).
- Residual effects of neuromuscular blocking drugs (overdose, inadequate reversal, drug interactions with -mycins or magnesium, hypothermia, renal/hepatic dysfunction).
- Suboptimal ventilatory muscle mechanics (obesity, patient position, surgical site).
- Increased production of CO_2 (shivering, malignant hyperthermia, sepsis).
- Coexisting chronic obstructive pulmonary disease (COPD).
- Splinting due to pain.

TABLE 8-2. Predicted Fio_2 with Supplemental Oxygen and Various Systems

System	Delivery Flow (L/min)	Predicted Fio_2
Nasal cannula	2	0.3
Nasal cannula	4	0.4
Face mask	6	0.5
Partial rebreathing mask	6	0.6
Partial rebreathing mask	8	0.8
Nonrebreather mask	10–15	0.8+

(Adapted from Duke J. *Anesthesia Secrets,* 3rd ed. Elsevier, 2006: 221.)

Diagnosis

- Measurement of $PaCO_2$.
- Nerve stimulator or sustained head lift of 5+ sec to confirm reversal of paralysis.

Clinical Signs

HTN and tachycardia not specific as not always present in postoperative patients.

Obesity-hypoventilation syndrome (Pickwickian syndrome) may cause respiratory acidosis → arterial hypoxemia → cor pulmonale.

Treatment

- Intubation required if there is obtundation, circulatory depression, or pH < 7.15.
- Drug-induced hypoventilation (see Table 8-3 for differential of drug-induced hypoventilation).
- Inhaled anesthetics:
  - Continue spontaneous emergence if patient maintains patent upper airway.
  - Controlled ventilation via endotracheal tube (ETT) is necessary to allow for elimination of inhaled anesthetic if patient is unable to maintain airway.
- Residual opioid effect:
  - Naloxone 40 μg IV every 2 min up to 200 μg. Titrate to avoid precipitous reversal of opioid-induced analgesia and activation of sympathetic nervous system. This can → pulmonary edema, hypertensive crisis, myocardial ischemia, and cardiac dysrhythmias, including ventricular fibrillation. Shorter duration than most opioids, so watch for re-narcosis of patient. Also can cause nausea and vomiting.
  - Doxapram 60–100 mg followed by 1–2 mg/min IV does not reverse analgesia but can cause HTN and tachycardia.
- Residual neuromuscular blockade:
  - Additional anticholinesterase drugs.
  - Mechanical ventilation of lungs until neuromuscular blocking drug dissipates.

TABLE 8-3. Differential of Drug-Induced Hypoventilation

Problem	Signs/Symptoms	Treatment
Inadequate reversal of neuromuscular block	Uncoordinated, ineffectual respiration effort.	Neostigmine 0.05 mg/kg IV. Possible mechanical ventilation.
Narcosis	Slow ventilation, sedated.	Naloxone 0.04–0.4 mg IV. Respiratory support.
Residual inhalation anesthesia	Shallow breathing, sleepy.	Encouragement of deep breathing and stimulation.

(Adapted from Duke J. *Anesthesia Secrets,* 3rd ed. Elsevier, 2006: 219.)

Hypotension

Opioids and the resolution of pain also may cause hypotension, especially in susceptible individuals.

Causes

- Hypovolemia (most common—blood loss, third-space sequestration, ongoing hemorrhage, inadequate volume replacement).
- ↓ myocardial contractility (residual anesthetics, heart failure, ischemia, hypocalcemia).
- ↓ systemic vascular response (SVR; residual anesthetics, sepsis).
- Cardiac tamponade.
- Cardiac dysrhythmias.
- Hypoxemia.
- Pulmonary embolus.
- Pneumothorax.
- Acidosis.

Diagnosis

- Central venous pressure monitoring in patients with normal LV function to assess intravascular fluid volume.
- Pulmonary artery catheter in patients with LV dysfunction or COPD assists in assessing etiology of hypotension (see Table 8-4).
- ↓ hematocrit with evidence of bleeding at operative site suggests inadequate surgical hemostasis.
- Electrocardiogram (ECG): Assess for ischemia or dysrhythmias.
- Oliguria: Urine output < 0.5 mL/kg/hr suggests hypovolemia.
- Fluid challenge with 250–500 mL crystalloid or 100–250 mL colloid.

Treatment

Twenty to thirty percent reduction of blood pressure from patient's baseline requires treatment, which depends upon etiology.

Hypovolemia

- Volume expansion: Crystalloid as first line and then consider colloid and packed red blood cells.
- Elevate legs and place in Trendelenberg position.

TABLE 8-4. Interpretation of Pulmonary Artery Catheterization Readings

Hypovolemia	↓ Myocardial Contractility	Sepsis
↓ pulmonary artery occlusion pressure (< 10 mmHg) ↓ cardiac index (< 2.5 L/min/m^2) ↑ SVR (normal 900–1400 dynes/s/cm^5)	↑ pulmonary artery occlusion pressure (> 15 mmHg) ↓ cardiac output	↓ pulmonary artery occlusion pressure ↑ cardiac output ↓ SVR

- ↓ myocardial contractility.
- Inotropic drugs after optimization of intravascular fluid volume.
- Sepsis.
- Maintain coronary perfusion pressure with IV α-agonist such as phenylephrine.
- Metabolic acidosis: Bicarbonate IV.

Hypertension

Causes

- Noxious stimuli (incisional pain, endotracheal intubation or bladder distention).
- Enhanced sympathetic nervous system activation as part of a neuroendocrine response to surgery or secondary to hypoxemia, hypercapnea, or metabolic acidosis.
- Essential HTN.
- Pheochromocytoma.
- Hyperthyroidism.
- Hypervolemia or intracranial HTN.
- Hypoxemia.
- Anemia.
- Hypoglycemia.
- Tachydysrhythmias.
- Myocardial ischemia.
- Withdrawal.
- Hypothermia with shivering.
- Fever.
- Malignant hyperthermia.

Treatment

- Blood pressure elevation > 20–30% of patient's normal baseline or patients with myocardial ischemia, heart failure, or bleeding should be treated.
- Adequate pain control.
- Mild to moderate HTN:
  - IV β blockers: Labetalol, esmolol, propranolol.
  - Calcium channel blockers: Nicardipine or nitroglycerin paste.
  - Nifedipine and hydralazine: Associated with reflex tachycardia and myocardial ischemia.
- Marked HTN:
  - Direct intra-arterial pressure monitoring.
  - Nitroprusside, nitroglycerin, nicardipine. or fenoldopam infusion.

Systolic blood pressure is falsely ↓ 0.7 mmHg every 1 cm the transducer is elevated above the right atrium.

Complications

- Postoperative bleeding.
- Myocardial ischemia.
- Heart failure.
- Intracranial hemorrhage.
- Dysrhythmias.

Dysrhythmias

Causes

- **Bradycardia:** β blockers, α adrenergics, neostigmine/edrophonium, opioids, succinylcholine, hypoxemia, hypothermia.
- **Tachycardia:** Atropine, pancuronium, meperidine, albuterol, hydralazine, pain, fever, hypovolemia, anemia, hypoxemia, acidemia, full bladder, malignant hyperthermia.
- **Premature atrial contractions (PACs)/premature ventricular contractions (PVCs):** Digitalis toxicity, electrolyte abnormalities (hypokalemia, hypomagnesemia), ↑ sympathetic tone, hypoxemia, respiratory acidosis, myocardial ischemia, HTN.

Treatment

- Most cardiac arrhythmias do not require treatment but need correction of underlying cause.
- First priority is to ensure patency of upper airway and adequacy of arterial oxygenation.
- Atropine (3–6 μg/kg IV) to ↑ heart rate.
- Verapamil (75 μg–150 μg/kg IV over 1–3 min) to ↓ heart rate.
- Lidocaine (1–1.5 mg/kg IV) to suppress PVCs.
- Cardioversion for hemodynamically significant tachyarrhythmias unresponsive to drug therapy.

Hypothermia

- Shivering is a compensatory mechanism by the body to offset heat loss and may be associated with vasoconstriction.
- Shivering is prevented by anesthetics and neuromuscular blockers.
- Causes a 300–400% ↑ in oxygen consumption by skeletal muscle activity.

Hypothermic infants undergo nonshivering thermogenesis, which increases O_2 consumption.

Causes

- Intraoperative hypothermia.
- Anesthetic gas effect (volatiles ↓ shivering threshold and normal vasoconstrictive response to hypothermia).
- Sepsis.
- Drug allergy or transfusion reaction.

Complications

- Myocardial infarction (MI)/arrhythmias/↑ duration of muscle relaxant.
- Rise in O_2 consumption, CO_2 production, and cardiac output.
- Nonspecific neurologic signs (posturing, clonus, Babinski).
- Metabolic acidosis.

Treatment

- Forced air warming.
- Warming lights or heating blankets.
- Meperidine 10–50 mg IV.

Pain Control

- **Mild to moderate pain:**
  - Tylenol plus codeine/hydrocodone/oxycodone PO.
  - Ketorolac 30 mg IV: Orthopedic and gynecologic procedures.
  - Opioid agonists/antagonists: Butorphanol 1–2 mg, nalbuphine 5–10 mg.
- **Moderate to severe pain:** Analgesic effect peaks 4–5 min; maximal respiratory depression not seen until 20–30 min later.
  - Meperidine 10–20 mg IV.
  - Hydromorphone 0.25–0.5 mg IV.
  - Morphine 2–4 mg IV.
  - IM opioids: Delayed and variable effects; onset 10–20 min; delayed respiratory depression seen after 1 hr.

Agitation

Causes

- Pain.
- Young and elderly patients.
- Systemic disturbances (hypoxemia/hypercapnia, acidosis, hypotension).
- Bladder distention.
- Gastric dilation.
- Surgical complication (eg, occult abdominal hemorrhage).
- Preoperative anxiety.
- Adverse drug effects (central anticholinergics, phenothiazines, ketamine).

Treatment

Agitation has a higher incidence with the use of short-acting volatile anesthetics.

- Physostigmine 1–2 mg IV for delirium secondary to atropine or scopolamine.
- Midazolam 0.5–1 mg for persistent agitation if pain and systemic disturbances can be ruled out.

Delayed Awakening

Defined as failure to regain consciousness 30–60 min after general anesthesia.

Causes

- **Residual drug effect:** Opioids, benzodiazepines, anticholinesterases.
- **Increased drug sensitivity:**
  - Age.
  - Hypothermia.
  - Renal/hepatic disease.
  - Drug interactions (monoamine oxidase inhibitors (MAOIs) + narcotics/barbiturates/sedatives, furosemide/-mycins + muscle relaxants).
- **Metabolic:**
  - Renal/hepatic disease.
  - Hypothyroidism.
  - Adrenal insufficiency.
  - Hypoxemia/hypercarbia.
  - Hyper/hypoglycemia.
  - Hyperosmolar hyperglycemic nonketotic coma.

- Electrolyte abnormalities (hyponatremia/hypomagnesemia, hyper/hypocalcemia).
- Malignant hyperthermia.
- Sepsis.
- Prior alcohol ingestion.

- **Neurologic:**
  - Hypoperfusion (cerebrovascular ischemia, embolism).
  - Hyperperfusion (HTN → intracranial hemorrhage).
  - Elevated intracranial pressure (subdural hematoma, cerebral edema, pneumocephalus)

Factors Affecting Emergence from Anesthetics

- **Inhaled anesthetics:**
  - Duration of anesthesia.
  - Alveolar ventilation.
  - Blood and lipid solubility.
  - Magnitude of metabolism.
  - Speed of emergence of inhaled anesthetics is directly proportionate to alveolar ventilation but inversely proportional to blood solubility.
- **Intravenous anesthetics:**
  - Dose administered.
  - Time of last injection.
  - Lipid solubility.
  - Hepatic inactivation.
  - Renal excretion.
  - Emergence from IV anesthetics is dependent on redistribution rather than elimination half-life.

Diagnosis and Treatment

- Naloxone 0.04 mg increments for opioids.
- Flumazenil 0.2 mg increments for benzodiazepines.
- Physostigmine 1–2 mg for anticholinergics.
- Forced-air warming devices to raise core body temperature.
- Electrolyte labs and ABG to rule out metabolic disturbances.
- Radiologic studies and neurologic consults to rule out intracranial causes.

Nausea and Vomiting

Factors Associated with ↑ Incidence

- History of postoperative nausea and vomiting.
- History of motion sickness.
- Female gender—worse during menstruation or first trimester of pregnancy.
- Younger age.
- Obesity.
- Nonsmokers.
- Postoperative pain.
- Swallowed blood.
- Type of surgery (eye muscle surgery, middle ear surgery, laparoscopic surgery, gynecologic surgery).
- Anesthetic drugs (opioids, nitrous oxide, volatiles, neostigmine).
- ↑ vagal tone commonly proceeds or coincides with emesis.

Generally, children < 2 years of age are at low risk for PONV.

TREATMENT

- Supplemental O_2.
- Serotonin antagonists:
  - Ondansetron 4–8 mg: Effective immediately.
  - Dolasetron 12.5 mg: Requires 15 min for effects.
  - Tropisetron 5 mg.
  - Granisetron 3 mg.
- Perphenazine 5 mg.
- Droperidol 0.625–1.25 mg: Side effects—associated with prolonged QT interval and fatal arrhythmias, sedation, extrapyramidal symptoms.
- Metoclopramide 0.15 mg/kg: Side effects—extrapyramidal symptoms; contraindicated in small bowel obstruction, Parkinson's, tricyclic antidepressants, or MAOIs.
- Scopolamine 1.5 mg transdermal: Side effects—dry mouth, somnolence, blurred vision, fever.
- Dexamethasone 4–8 mg: Combine with another antiemetic; effective for 24 hr; side effects—impaired wound healing and high blood glucose levels at higher doses.
- Propofol 10–20 mg bolus followed by 10 μg/kg/min infusion.

Incidental Trauma

Treatment for corneal abrasions:

- *PO NSAIDs for pain*
- *Eyedrops*

- **Corneal injury:**
  - Caused by dryness or accidental eye contact during airway management.
  - Presents as tearing, pain, and photophobia.
  - Abrasions heal within 72 hr.
- **Oral soft tissue trauma:** Caused by laryngoscopy, indwelling airway, biting during awakening.
- **Loosened or damaged teeth:** Dental consult.
- **Pharyngitis and hoarseness:** Caused by drying from unhumidified gases or trauma from pharyngeal suctioning.
- **Nerve injury:** Caused by positioning and poor padding of affected limb.

SECTION II

Anesthesia for the Subspecialties

- Pediatric Anesthesia
- Obstetric Anesthesia
- Cardiac Anesthesia
- Thoracic Anesthesia
- Anesthesia for Vascular Surgery
- Neurosurgical Anesthesia
- Anesthesia for Trauma
- Critical Care
- Pain Management
- Anesthesia for Ophthalmic and ENT Surgeries
- Anesthesia for Genitourinary Surgery
- Anesthesia for Patients with Liver Disease
- Anesthesia for Patients with Endocrine Disease
- Anesthesia for Organ Transplantation
- Anesthesia for Laparoscopic Surgery
- Anesthesia for Orthopedic Surgery
- Anesthesia Outside of the Operating Room
- Special Considerations in Anesthesia Practice

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CHAPTER 9

Pediatric Anesthesia

Milestones

- Newborn: Neonatal through 1 month of age.
- Infant: 1 month of age through the first year of life.
- First 24–72 hr: Most significant physiologic and hemodynamic transition.
- At 3 months, circulatory and ventilatory adaptation are completed, and thermoregulation is altered to a more adult state.

Fetal Circulation

- Placenta and three shunts:
  - **Placenta:** Oxygenates blood from inferior vena cava (IVC) to right atrium (RA).
  - **Foramen ovale:**
    - Remains open due to low pressure in left atrium (LA) and high pressure in RA: closed, fluid-filled alveoli, compressed blood volume, low Pa_{O_2} and pH.
    - RA divided by crista dividends.
    - Oxygenated blood shunted RA to LA (coming from IVC).
    - Bypass right ventricle (RV) and pulmonary vascular blood.
    - Open to low LA pressures and high RA pressures/high pulmonary vascular resistance (PVR).
  - **Ductus arteriosus:**
    - Low-resistance due to low Pa_{CO_2} to descending aorta (where arch becomes descending aorta, in proximity of left subclavian artery takeoff).
    - Shunts blood (90%) pulmonary artery (PA) to descending aorta.
  - **Ductus venosus:** Shunts 50% of umbilical blood flow straight to IVC–heart, bypasses liver.
- **First several hours of life:**
  - Clamp cord and initiate ventilation.
  - Reduction of PVR and ↑ systemic vascular resistance (SVR).
  - Alveoli expand, → decreased PVR due to increased to Pa_{O_2} → ductus arteriosus (DA) constricts → ↑ pulmonary blood flow → ↑ LA pressure ↑ functional closure of foramen ovale, though 50% of 5-year-olds have patent foramen ovale.
  - Physiologic closure of DA—due to ↓ in prostaglandins, ↑ in pO_2.
  - Initially reversible:
    - Stressors: Hypoxemia, hypothermia, hypervolemia, sepsis, acidemia.
    - Prematurity: High pulmonary flow from left-to-right shunting → congestive heart failure (CHF).
    - Patency retained with prostaglandin E_1 (PGE_1).
  - Anatomic closure of DA takes 4–10 days (rarely more than 2 weeks).
  - Foramen ovale closes. Incidence probe: Patent foramen ovale 30% at 30 years old and 20% at 30+ years old.

Normal ABG for healthy full-term neonate:

pH: 7.21

Pa_{O_2}= 50 mm Hg

Pa_{CO_2}= 40–50 mm Hg

Neonatal Cardiopulmonary Physiology

Cardiac Output (CO)

- Fixed stroke volume: Noncompliant ventricles.
- CO dependent on heart rate (HR): CO ↑ 30–60% in neonates. ↓ ability to handle volume or pressure change.

- Immature baroreceptors with hypovolemia = hypotension *without* tachycardia.
- Presence of fetal hemoglobin → left shift of oxyhemoglobin dissociation curve with ↑ hematocrit (Hct) (fetus has higher Hct).
- In fetus, lower 2,3-diphosphoglycerate (2,3-DPG) and presence of hemoglobin F → higher affinity for O_2, compensates for the fact that umbilical venous blood PaO_2 is approximately only 35 mmHg.
- Normalized by 4–6 months of age.
- P50: 19–26 mmHg and lower Hct.
- Hypoxia → bradycardia. See Table 9-1 for normal neonatal to adult vital signs.

Signs of PDA

- *Machine-like continuous murmur*
- *Wide pulse pressure*
- *Bounding peripheral pulses*

Persistent Pulmonary Hypertension

Can happen anytime when transitional circulation persists.

- Elevated pulmonary artery (PA) pressure as a result of:
  - Hypoxia, acidosis, inflammatory mediators—prostaglandins.
  - Extension of smooth muscle into distal respiratory units.
  - Right-to-left shunt through foramen ovale or DA (right-to-left shunt happens as a result of elevated PA pressure; ↑ PVR → PAP → ↓ pulmonary blood flow → right to left shunt via PFO/PDA).

Etiology of PPH

- Primary: Unknown.
- Secondary: Meconium aspiration, sepsis, pneumonia, respiratory distress, congenital diaphragmatic hernia, hyaline membrane disease.

Treatment

Surfactant, high-frequency ventilation, inhaled nitric oxide, extracorporeal membrane oxygenation (ECMO), isoproterenol, tolazoline, and supportive care.

TABLE 9-1. Weight-Based Normal Vital Sign Values

	Neonate	Infant	5-Year-Old	Adult
Weight (kg)	3	4–10	18	70
VO_2 (mL/kg/min) (7–9)	5	4	3	
SBP (mmHg)	65	< 90–95	95	120
HR	130	120	90	80
EBV (mL/kg)	90	80	75	65
Hb (g/dL)	17	11–12	13	14

EBV, estimated blood volume; Hb, hemoglobin; HR, heart rate; SBP, systolic blood pressure; VO_2, oxygen uptake.

Brown fat differentiates at 26–30 weeks. Premature infants born prior may not undergo nonshivering thermogenesis.

Meconium Aspiration

- Chronic fetal hypoxia in third trimester → passage of meconium in utero.
- Meconium enters the pulmonary system → pulmonary HTN.
- Cause respiratory failure: Start with suction, then intubation (if severe meconium staining, newborn is not vigorous, or low APGARs).

Pulmonary System

- Change from fluid-filled alveoli to air-filled with ventilation.
- First 5–10 min of extrauterine life: Normal ventilation volume and tidal ventilation develops.
- 10–20 min: Full functional residual capacity (FRC) developed (C-section babies have a harder time—there is no squeezing of fluid out of lungs when passing through birth canal).
- Respiratory muscles:
  - Type I: Slow-twitch, high-oxidative—sustained muscle activity (only 25% of those while adults have 50%).
  - Type II: Fast-twitch, low-oxidative: immediate short activity → easy fatigue.
- High oxygen consumption: 7–9 mL/kg/min vs. 3 mL/kg/min (adult).
- High closing volume (capacity, which is close to FRC): If tidal volumes are ↓, → shunting → rapid desaturation with laryngospasm.
- High ratio of minute ventilation to FRC:
  - 5:1 vs. 1.5:1 (adult).
  - Rapid induction of inhalational anesthesia, rapid awakening.
- Pliable ribs: Inefficient ventilation.
- Total lung capacity (TLC)/FRC ratio (about 40%)—same as in adults.

Renal System

- By 1 month, kidneys are 70% mature—all components are incompletely developed; by 1 year, fully matured.
- "Obligate sodium loser":
  - Normal renin-angiotensin-aldosterone system.
  - Cannot completely conserve sodium due to immature distal tubules: Inadequate tubular function, ↓ glomerular filtration rate (GFR).
  - Normalize by 1–2 years.
  - Prematurity associated with ↓ creatinine clearance; impaired sodium retention, glucose excretion, and bicarbonate resorption.
  - ↓ concentrating ability in premature infants and at birth.

Metabolism

- Temperature regulation:
  - Via metabolism of brown fat vs. shivering in adults.
  - ↑ body surface area (BSA): ↑ metabolism and ↓ body fat → prone to hypothermia.
- ↓ minimum alveolar concentration (MAC) (at birth).
- Immature nervous system, maternal progesterone, elevated endorphins, immature blood-brain barrier.
- Infants: Neonates = lowest MAC, 2–3 months of age = highest MAC.

Gastrointestinal System

High incidence of gastric reflux (GERD)—very common:

- Delayed gastric emptying.
- Lower esophageal sphincter (LES) incompetence.
- Usually, infant "grows out of it."
- Aspiration precaution: Consider based on severity. Consider severe if history of aspiration pneumonias, unexplained wheezing, frequent choking, desaturations, if ENT diagnosed changes in either hypopharynx or area above vocal cords related to GERD.

Central Nervous System

- At birth: Least mature major organ system.
- Myelination continues up until 2 years of age.
- **Complications:** Intraventricular hemorrhage (IVH), seizures, respiratory depression, and retinopathy.

Epiglottis in infants is long, stiff, and obstructive to laryngoscopy view. Blade of choice: Miller 0 or 1.

Physiologic Anemia of Infants

Hemoglobin F disappears and adult hemoglobin ↑ to 10–11 g/dL by 8–12 weeks of age in term infant, 7–10 g/dL by the age of 6 weeks for premature.

Airway

- "Obligate nose breathers": < 6 months.
- Choanal atresia: Causes respiratory distress, desaturations when feeding.
- Short trachea, neck.
- Hyoid bone not yet calcified.
- Large occiput → flexion with head rest—shoulder role and padding under head may be helpful with airway management.
- Larynx is more anterior and superior and "travels" down and back during development (see Table 9-2).
- During mask ventilation, it is important to keep fingers strictly on the mandibular ramus—no pressure on submental soft tissues (otherwise, obstruction happens, with difficult ventilation).
- Endotracheal tube (ETT) size: Uncuffed vs. cuffed ETT—if cuffed, choose 0.5 size smaller (cuff should be fully deflated upon insertion). Refer to Table 9-3.

TABLE 9-2. Comparisons of Neonatal and Adult Airways

	Neonatal	Adult
Glottis	C4	C5–C6
Narrowest at	Cricoid ring	Vocal cords
Larynx	Funnel-shaped, cephalad	Cylindrical
Epiglottis	Long, stiff	Flat, flexible
Tongue	Larger	

TABLE 9-3. Endotracheal Tube Size Given Age and/or Weight

Age	Inner Diameter
Premature < 2.5kg	2.5
Term neonate	3 (small baby)–3.5
2–8 months	3.5
8–12 months	4
18–24 months	4.5
> 24 months	Age/4 + 4

For children > 2 years of age, estimate of ETT size: (age in years/4) + 4 or (age in years + 16)/4.

- ETT position:
  - 1-2-3 kg/7-8-9 cm at lip.
  - Inner diameter (ID) × 3 (at lip).
  - Add 2–3 cm for nasotracheal ETT.
- Most common morbidity: Tight ETT fit.
- Air leak of 20–30 cm H_2O (or lower) ↓ risk of postextubation croup.
- Currently more liberal use of cuffed ETT (very practice dependent):
  - Eliminates need for ETT exchange if a large air leak is present with uncuffed tube → inflation just to the point when leak is ↓ to allow for delivery of adequate tidal volume.
  - Uncuffed still preferred in premature infants and newborns.

PHARMACOLOGY AND PHARMACODYNAMICS

Preanesthetic Medications

- ↓ level of anxiety.
- Better cooperation with mask induction, or IV placement if IV is needed to induce.
- May delay emergence if case is short.
- Oral medication: Preferable (see Table 9-4).
- Nasal, intramuscular (IM), and rectal routes also available (see Tables 9-4 and 9-5).
- Rectal: Methohexital and thiopental 25 mg/kg, onset 10 min; monitor for respiratory depression and O_2 desaturation.
- Chloral hydrate (not used very often anymore):
  - Used for sedation or premedication.
  - Given orally or rectally.
  - Tolerance easily develops if frequent use.
  - Might cause respiratory depression, apnea—unpredictable dose-response relation.
  - Very unpredictable length of sedation, often lasts very long.

Inhalation Agents

All inhalational agents may be very poorly tolerated in premature infants and newborns, if cardiac depression/failure is present.

TABLE 9-4. Premedication Routes, Doses, and Side Effects

	Midazolam	Ketamine	Fentanyl
Route	PO	PO (available but not often used)	Oral, transmucosal
Dose	0.5–0.75 mg/kg (20 mg maximum)	5–6 mg/kg	10–15 μg/kg
Onset	20–30 min	20 min	10–20 min
Duration	30 min		30 min
Side effects	Loss of balance, head control Dysphoria, paradoxical agitation (when given, or even upon emergence later)	Nystagmus ↑ salivation ↑ nausea/emesis ↑ Heart rate/blood pressure Dysphoria	↑ perioperative emesis ↑ O_2 desat Intensive monitor

If moderate to severe hypovolemia is present (dehydration, bleeding, severe vomiting); a fentanyl/muscle relaxant anesthetic may be appropriate (though recall of events is more probable in older children).

Halothane

- Least noxious smell: Used primarily as induction agent.
- Halothane is more soluble than isoflurane: Longer emergence, longer effect, longer induction time.
- May sensitize myocardium to catecholamines → arrhythmia (maximum epinephrine dose: 7–10 μg/kg).
- Causes significant bradycardia, myocardial depression, much less vasodilatation → ↓ blood pressure in younger children, hypovolemic. This is advantage in management of HISS.

Isoflurane

- Pungent smell, → laryngospasm during induction (not for inhalation induction).
- Used as maintenance.
- Less myocardial depression than halothane.
- ↓ BP, ↓ SVR (mainly due to vasodilatation—less myocardial depression).

Child's anxiety: Midazolam + parental presence on induction = midazolam alone.

TABLE 9-5. Pros and Cons of Nasal Route Premedications

Pro	Con
Rapid absorption	Lack of patient cooperation
Bypass first-pass hepatic metabolism	Stinging sensation
Midazolam 5 mg/mL Onset: 10 min (short onset)	Bitter taste

Desflurane

- Induction: ↑ laryngospasm, coughing, secretions—not used for induction.
- Used as maintenance.

Sevoflurane

- Suitable for smooth mask induction: Nonpungent smell (nonirritable, but still unpleasant smell). Rapid induction and emergence: low blood-gas solubility.
- Low level of myocardial depression.
- Fewer dysrhythmias than halothane.
- Possible toxic metabolite with low flow (< 2 L) in a rebreathing circuit.
- With soda/baralyme forms nephrotoxic compound A; higher gas flow is required to minimize this, especially if long exposure.
- With desiccated soda/baralyme, CO is formed (carboxyhemoglobin).

Nitrous Oxide

- N_2O is often used with sevoflurane and oxygen to produce a more potent inhaled anesthetic, especially for induction in children (though not a potent anesthetic on its own).
- ↑ respiratory rate in spontaneously breathing patients.
- Minimal cardiovascular effects.
- By oxidizing cobalt in vitamin B_{12}, N_2O inactivates methionine synthetase, which affects DNA synthesis and has been implicated as a cause of anemia in lab rat studies.

Intravenous Agents

Sedative-Hypnotics

Etomidate lowers seizure thresholds. Avoid in kids with seizure disorders.

- Used after mask induction to deepen the anesthesia.
- All except for etomidate and sodium thiopental have wide usage.
- Larger doses required for pediatric population in general.
- Much higher doses needed for those on antiseizure medication.
- **Propofol:**
  - Induction doses: < 2 years: 2.5–3 mg/kg; > 2 years: 2–2.5 mg/kg.
  - Pain on injection: Reduce pain with prior use of IV lidocaine 0.2–1 mg/kg.
  - Maintenance: 150–200 μg/kg/min. ↓ postoperative vomiting.
- **Etomidate:**
  - Induction dose: 0.2 mg/kg.
  - Stable hemodynamics, if propofol is contraindicated.
  - Even a single dose can cause adrenal suppression.
- **Midazolam, ketamine:** Also good induction agents; good for maintenance as well.

Opioids

- Adjuncts to inhalation agents = balanced anesthetic technique or used alone (↑ risk of recall if not combined with amnestic).
- Blunt hemodynamic response to intubation (in adequate doses).

- ↓ MAC for maintenance.
- Postoperative analgesia.
- Bradycardia.
- Chest wall rigidity.
- Even single dose ↑ nausea and vomiting.
- Respiratory depression: Neonates and infants < 6 months:
  - An ↑ medication amount can cross the immature blood-brain barrier.
  - Morphine: Most lipophobic; administer with caution.
- **Fentanyl:** 1–5 μg/kg, 10 μg/kg and more in cardiac cases.
- **Morphine:** 0.05–1 mg/kg for case (if longer case 2–3 hr and more, can give more, also possible to give more if titrating to respiratory rate—patient breathing spontaneously).
- **Sufentanil:** 1–2 μg/kg.
- **Alfentanil:** 50–100 μg/kg.
- **Hydromorphone:** 1:5 ratio to morphine dose (1:7.5 in adults), less itching, less postoperative nausea and vomiting.

Muscle Relaxants

- Neonate:
  - Variable response to nondepolarizing muscle blockers (NDMBs).
  - Intermediate long acting more like long acting.
  - High volume of distribution → higher induction dose needed; maintenance dose reduced.
- Infant/child: Similar response to NDMB based on weight.
- **Succinylcholine:**
  - Infant: Lower sensitivity requiring higher dose (neonate double dose) 2–3 mg/kg, 4 mg/kg dose for IM administration; for laryngospasm, IV dose is one-tenth of IV induction dose.
  - Bradycardia.
  - Vagally mediated, pretreat with atropine/glycopyrrolate.
  - Controversial usage in children, especially male child < 8 years old.
    - Muscular dystrophy: 1/250,000.
    - Epinephrine 5–10 μg/kg is first-choice resuscitation med—important to correct significant acidosis, though epinephrine does not work well in acidosis.
    - Usually replaced with rocuronium 1 mg/kg (double intubation dose).
    - Consider risk/benefit if needed to use it; always watch for complications (electrocardiogram). Succinylcholine still remains the fastest and most reliable-acting muscle relaxant.
  - **Complications:**
    - **Malignant hyperthermia:** Triggering agent. May be associated masseter spasm. High end-tidal CO_2; tachycardia, HTN.
    - **Hyperkalemic cardiac arrest** with undiagnosed myopathies due to acute rhabdomyolysis (more often) or ↑ release of K^+ as a result of proliferation of extrajunctional acetylcholine (ACh) receptors (separate entity than malignant hyperthermia).
  - **Treatment of malignant hyperthermia:**
    - Cooling measures, dantrolene.
    - Cardiopulmonary resuscitation.
    - Calcium, bicarbonate, insulin/glucose.

Malignant hyperthermia ABG: metabolic and respiratory acidosis.

Reversal Agents

- **Edrophonium:**
  - 1 mg/kg.
  - Rapid 90% reversal in 2 min; fewer muscarinic side effects.
  - Requires advance dosage of atropine 0.01–0.02 mg/kg.
- **Neostigmine:**
  - 0.05–0.075 mg/kg.
  - Ninety percent reversal in 10 min.
  - Glycopyrrolate 0.01 mg/kg; or 0.2 mg for 1 mg of neostigmine.

Antinausea Medications

- Procedures with higher risk of postoperative nausea and vomiting:
  - Tonsillectomy.
  - Strabismus, middle ear surgery.
  - Orchiopexy.
- **Droperidol:** 0.1 mg/kg max. May cause a delay in discharge with higher doses (sedation).
- **Metoclopramide:** 0.15 mg/kg.
- **Ondansetron:** 0.05–0.15 mg/kg.
- **Dolansetron:** 0.35 mg/kg.
- **Dexamethasone:** 0.5 mg/kg (max. 10 mg), consider H_2 blocker with it.

ANESTHETIC MANAGEMENT IN PEDIATRIC PATIENTS

If child chews gum and spits it out, proceed with anesthestic without delay.

If swallows gum, cancel elective case as gum is difficult to extract from bronchus/ trachea if aspirated.

Preoperative Fasting Period

- Clears: 2 hr.
- Breast milk: 4 hr.
- Formula or light meal: 6 hr.
- Fatty, solid meal: 8 hr.
- 1:5000–10,000 incidence of aspiration.
- Inhalation/mask induction.
- Most commonly practiced in pediatric population.

Complications

- Breath-holding, laryngospasm, dysrhythmias, stomach distension.
- Infant < 1 year at higher risk of bradycardia, hypotension, cardiac arrest.
- Halothane > sevoflurane: Faster inhalational uptake due to ↑ alveolar ventilation-to-FRC ratio (5:1).
  - Different distribution of cardiac output.
  - Myocardial depression is similar in equipotent concentrations of sevoflurane and halothane.
- Intracardiac shunts:
  - Right-to-left: Slower rate of inhalation induction. Due to slower rate of ↓ in arterial anesthetic concentration—dilution by shunted blood.
  - Left-to-right: Faster induction—blood immediately recirculated in the lung.
  - Careful if failing heart—rapid cardiac depression.
- Anesthetic dose requirements:
  - Infants 2–3 months: Highest MAC.
  - Neonates/premature infants: Lower MAC.

Regional Anesthesia

- Spinal cord ends at L3 in neonates.
- Beneficial in ex-premature infants (up to 60 weeks' PCA): Less postop apnea/bradycardia/hypoxia.
- In infants, very short duration, rapid CNS turnover: tetracaine 90 min, bupivicaine 60–75 min.
- Hypotension after spinal: Sign of toxicity of local anesthetic. Sympathetic system not fully developed = there is no sympathectomy.
- There are also other commonly used regional anesthetic techniques used in the pediatric population, as listed in Table 9-6.

Monitoring

- Standard monitors similar to those of adults.
- Continuous precordial or esophageal stethoscope (in cases when end-tidal CO_2 is not possible to monitor).
- Case- or patient-dependent circumstances determine the use of invasive monitors.
- Pediatric cardiac: Preductal (right arm, right ear) and postductal SpO_2.

Intravenous Fluid Therapy

- High metabolic demand.
- High BSA-to-weight ratio.
- Deficit (maintenance × hours NPO). Give one-half in the first hour and remaining in the next 2 hr.
- Maintenance fluid requirement: 4-2-1:
  - < 10 kg: 4 mL/kg.
  - 11–20 kg: 2 mL/kg.
  - > 20 kg: 1 mL/kg.
  - Third-space replacement/insensible losses.
  - Intra-abdominal: 6–15 mL/kg/hr
  - Intrathoracic: 4–7 mL/kg/hr
- End point of fluid therapy:
  - Hemodynamic stability.
  - Adequate urine output: 0.5–1 mL/kg/hr.

Blood Loss

- MABL = maximum allowable blood loss.
- MABL = estimated blood volume (EBV) × (starting Hct – target Hct) / starting Hct.

TABLE 9-6. Commonly Used Pediatric Regional Blocks

Block	Procedures
Penile	Circumcision, hypospadias
Ilioinguinal + iliohypogastric	Inguinal hernia
Brachial plexus	Arm and wrist
Caudal epidural	Abdominal, thoracic

- Preterm: 100–110 mL/kg
- Term: 90 mL/kg
- 3–12 months: 80 mL/kg
- > 1 year: 70 mL/kg

- Replaced with 1:3 lactated Ringer's, 1:1 colloid.
- Transfuse if estimated blood loss > 30% or final hemoglobin < 8 g/dL (not a rule—individual based on patient's comorbities, age, and if hemodynamic instability present).
- ↑ apnea in neonates and premature infants if Hct < 30%.
- Packed red blood cells
  - Hct 55–65%.
  - 1 mL/kg ↑ Hct 1.5% (usually given 10–20 mL/kg at a time).
  - Infuse at a slower rate to lessen hypocalcemia and hypothermia.

Postanesthetic Care

Caudal blocks have been shown to reduce incidence of emergence delirium.

- Standard monitors.
- Analgesia.
- Minimal separation from parents.
- Nonopioid analgesics for mild to moderate pain relief:
  - Acetaminophen +/– codeine: PO (10–15 mg/kg), PR (30–40 mg/kg). Maximum dose: 30-60-90 mg/kg/day (newborn-infant-child < 12 years old).
  - Ketorolac: 0.5 mg/kg every 6 hr (try limit to 3 days, not for < 6 months old, watch for ↑ blood urea nitrogen [BUN]/creatinine [Cr]).
  - Ibuprofen: 10 mg/kg.
- Regional techniques.
- Patient-controlled analgesia for older children (even for infants, consider different combinations of continuous infusion/bolus by nurse/demand dose or patient triggered based on patient's maturity).

Postextubation Croup

- Most common complications of ETT intubation.
- Associated with tight ETT fit.
- ↓ with air leak at 20–25 cm H_2O.
- **Risk factors:**
  - Early childhood: 1–4 years.
  - Repeated intubation attempts.
  - Large ETT.
  - Prolonged surgery.
  - Head/neck procedures.
  - Excessive tube movement, coughing, bucking on ETT.

Subglottic Edema

- Even small edema creates high resistance to airflow. Manifests upon arrival at postanesthesia care unit, with 2–4 hr postop.
- Stridor, suprasternal retractions, tachypnea, labored breathing.
- **Treatment:**
  - Mild: Conservative management with humidified air, fluid hydration.
  - Severe: Nebulizer treatment with racemic epinephrine.

- Need to be observed for 4 hr before discharge to home.
- Need to be admitted if requiring second treatment.
- Rebound possible after epinephrine has worn off.

Laryngospasm

- Superior laryngeal nerve stimulation ↑ forced, involuntary spasm of laryngeal muscles.
- **Risk factors:** Recent upper respiratory infection, secondhand smoke, light anesthesia, secretions, extubation in stage 2.
- **Treatment:**
  - Jaw thrust.
  - Positive pressure ventilation.
  - Deepening of anesthesia (if time to do so, patient not severely hypoxic/bradycardic): Propofol bolus, IV lidocaine: 1–1.5 mg/kg.
  - Succinylcholine: Small doses up to one-tenth of intubation dose); 4 mg/kg IM (no IV access, give with atropine).
  - Transport patients in lateral position to drain oral secretions to try prevent laryngospasm.

Apnea of Prematurity

- The risk of apnea, especially postoperatively, is ↑ in premature infants defined as < 44 weeks' PCA. This risk is significantly ↓ after 60 weeks' PCA.
- After an anesthetic, in-hospital respiratory monitoring is required for at least 12 hr for infants at risk.
- Apnea spells not only manifest as cessation of breathing (for 15–20 sec), but also as bradycardia.

Treatment of AOP:

- *Monitoring*
- *IV caffeine*
- *Prevention of anemia/hypovolemia*
- *Respiratory support*

SELECT NEONATAL CASES

Tracheoesophageal Fistula (TEF)

- One in 3000–4000 live births.
- Types:
  - Esophageal atresia only; no communication with trachea.
  - Distal esophageal atresia, proximal esophageal fistula with trachea.
  - Proximal esophageal atresia, distal esophageal fistula with trachea—most common (90%).
  - Proximal and distal fistula with trachea.
  - Continuous esophageal with additional fistula to trachea.
  - Esophageal stenosis; no communication with trachea.
- Endoderm fails to divide into trachea and esophagus at fourth to fifth week.
- At birth: Excessive drooling, cyanotic episodes, coughing, inability to pass nasogastric tube (NGT) to stomach, air in stomach on x-ray.
- Diagnostic contrast medium is contraindicated due to risk of aspiration.
- Associated with prematurity, congenital heart defect (20–25%).

Perioperative Management

- ECHO: Detect heart defect.
- Consider awake intubation, inhalational induction with maintaining spontaneous ventilation until fistula is ligated.

***VATER** syndrome:*

- ***V**ertebral defects*
- ***A**nus (imperforate)*
- ***T**racheo-esophageal fistula*
- ***E**sophageal atresia*
- ***R**adial and/or **R**enal anomalies*

***VACTERL** syndrome = VATER syndrome plus:*

- ***C**ardiac defects*
- ***L**imb abnormalities*

- Ketamine/midazolam/inhalation agent technique = spontaneous ventilation friendly.
- Avoid N_2O and excessive positive pressure ventilation, which may ↑ gastric distention.
- If positive pressure ventilation: Identify the correct ETT position: ETT just distal to defect with preserved equal bilateral breath sounds, if fistula location allows (sometimes at carina). If this not possible, then use low pressures to limit gastric insufflation.
- After fistula is ligated, positive pressure ventilation is no issue.

Postoperative Complications

- Tracheomalacia
- GERD
- Esophageal stricture
- Persistent tracheal diverticulum

Omphalocele vs. Gastroschisis (Table 9-7)

- Both are true surgical neonatal emergencies
- Congenital defects of the anterior abdominal wall permitting external herniation of abdominal viscera.
- **Anesthetic management:**
  - Decompress stomach prior to induction.
  - Awake (risk of intraventricular hemorrhage, especially in premature infants) vs. inhalation induction vs. rapid sequence induction.
  - Adequate muscle relaxation.
  - Manage perioperative fluid (third-space) loss: Critical to prevent hemodynamic instability (too little fluid).
  - Avoid tissue swelling—abdominal compartment syndrome (too much fluid).
- Gastroschisis: "Bag" neonate to prevent hypothermia and dehydration. Prevent hypoglycemia (maintenance solution with D10).
- Omphalocele: Blood loss is more significant due to extensive adhesions.

TABLE 9-7. Comparisons of Omphalocele vs. Gastroschisis

Omphalocele	Gastroschisis
Develops earlier in fetal life: ~ 10th week.	Later in fetal life.
Intestinal contents fail to return to abdominal cavity.	Interruption of the omphalomesenteric artery → ischemia → tissue defect → allowing herniation (R to the umbilicus).
Covered by the amnion.	Not covered → higher risk for infection and fluid shifts.
Umbilical cord at apex of the sac.	Umbilical cord normally situated.
In 50% associated with other congenital defects: cardiac (tetralogy of Fallot), T21, Beckwith-Wiedemann syndrome (microcephaly, macroglossia, hypoglycemia, visceral macrosomia).	Prematurity, other congenital abnormalities not often.

- Ventilation:
  - Avoid N_2O.
  - Might become difficult after abdominal contents reduced to abdominal cavity and defect closed. At the same time hypotension caused by IVC compression may occur.
  - Consider keeping baby intubated postoperatively.
- If defect too big, two stages:
  1. Silo placed to cover up insides and to allow for slow (over 7–10 days) reduction of abdominal contents.
  2. Abdominal wall closure.

PEDIATRIC NEUROMUSCULAR DISORDERS

Duchenne's Muscular Dystrophy

Duchenne's muscular dystrophy and malignant hyperthermia are two separate disease processes with no evidence-based associations.

- One in 3500 male births.
- X-linked recessive.
- Abnormalities of dystrophin protein in muscle membrane.
- Muscle weakness not apparent till toddler.
- Avoid SUX in DMD patients.
- Prolonged response to NDNB.
- If SUX is given (to a patient with undiagnosed DMD):
  - Hyperkalemic cardiac arrest: Due to acute rhabdomyolysis as the main mechanismus, less as a result of ↑ release of K^+ due to ↑ amount of extra-junctional ACH receptors:
    - Hyperkalemia
    - ↑ creatine kinase
    - Cardiac arrest
  - Certain myopathies have higher risk.
  - Similar reactions possible with exposure to inhalation agents.
- **Signs/symptoms of DMD:**
  - Respiratory muscle weakness.
  - Cardiomyopathy, arrhythmia.
  - Calf pseudohypertrophy, difficulty climbing stairs, waddling gait.
- Postopertively, risk for hypoventilation/pulmonary insufficiency.

Myelomeningocele

- Neural defects involving meninges and neural elements.
- May be associated with hydrocephalus and Arnold-Chiari malformation.
- Often prematurity, associated congenital malformations (heart, brain, gastrointestinal).
- Abnormal dysfunction below level of defects: Bowel, bladder, neuromuscular.
- Need to close defect to prevent infection.
- Usually requires multiple surgeries. Latex-free environment recommended, as there is potential for developing latex allergy.
- **Intraoperatively:**
  - No hyperkalemia from SUX.
  - At risk for aspiration from GERD and vocal cord motility abnormality.
- **Postoperatively:** At risk for apnea.

Cerebral Palsy

- Unknown etiology: Current theory questions hypoxic injury.
- Occurrence: 2 in 1000.
- Varied clinical manifestations, nonprogressive, subject to multiple surgeries.
- Intraoperative anesthetic concerns:
  - Possible gastric aspiration due to GERD, bulbar palsy.
  - Might be difficulty with clearance of secretions.
  - Hypothermia.
  - Slow emergence.
  - No hyperkalemia from SUX.

Physical exam diagnosis: Olive-shaped mass in upper abdomen and projectile vomiting.

Pyloric Stenosis

- **Background info:**
  - Obstruction at the gastric outlet.
  - Usually presents at 2–6 weeks full term infant.
  - Postprandial persistent, bile-free, projectile vomiting → weight loss, dehydration.
  - Hypokalemic, hypochloremic metabolic alkalosis → oliguric.
- **Medical emergency, not surgical:** First, correct electrolytes, acid-base, rehydrate before proceeding to operating room.
- **Anesthetic course:** Pyloromyotomy:
  - Careful IV fluids, electrolyte repletion.
  - Full stomach: At risk for aspiration of gastric contents.
    - Suction prior to induction.
    - Rapid-sequence induction (RSI) or modified RSI or awake oral endotracheal intubation. Inhalation induction = aspiration risk.
  - May consider caudal for both intraoperative and postoperative pain control.
  - Extubate when fully awake.
  - Begin small feedings 4–6 hr postop.

Congenital Diaphragmatic Hernia (CDH)

Background

- 1 in 2000–5000 births.
- Surgical urgent though not emergent: Surgery can be delayed for medical stabilization—correct acidosis, reduce pulmonary HTN (prostaglandin, NO) → improvement in pulmonary blood flow and consequently oxygenation
- Some patients require ECMO (severe pulmonary HTN, severe hypercarbia).
- Respiratory distress, cyanosis.
- Hypoplasia of the lung parenchyma and pulmonary vasculature.
- **Associated with other congenital problems:**
  - Polyhydramnios.
  - Central nervous system anomalies: Spina bifida, hydrocephalus, encephalopathy.
  - Gastrointestinal anomalies: Intestinal atresia, malrotation.
  - Genitourinary: Hypospadias.
  - Congenital heart disease: Atrial septal defect, ventricular septal defect, tetralogy of Fallot, coarctation.
- **Cause of death:** Progressive hypoxia and acidosis. Average mortality rate 50%.
- **Long-term sequelae:** Bronchopulmonary dysplasia, pulmonary hypoperfusion.

Classification

- Based on the site of defect. Most common: posterior lateral aspect of diaphragm via the foramen of Bochdalek (75–85%). Left-sided > right.
- Hernias through the esophageal hiatus are small; pulmonary vasculature not compromised.

Diagnosis

- Can be diagnosed in utero due to association with polyhydramnios.
- Severe CDH: Within 6 hr of birth, presents with respiratory distress and cyanosis.

Physical Examination

- Shifted cardiac sounds, dextrocardia.
- Scaphoid abdomen and bulging chest.
- Diminished breath sounds on the affected side, bowel sounds in the chest.
- NGT in the chest.

Management

- Immediate endotracheal intubation (unless there is no respiratory distress), NGT.
- Sedation/analgesia/paralysis.
- Monitor pre- and postductal saturation: Assesment of right-to-left shunting, reflects pulmonary pressures.
- Persistent fetal circulation due to hypoxemia and acidosis.
  - Right-to-left shunt
  - High pulmonary pressure
  - PDA ligation is controversial due to possible acute RV failure and death.
- **Anesthetic management:**
  - Avoid positive-pressure ventilation to avoid gut distention (if intubated, ↓ risk of gastric distention).
  - Central line, A-line.
  - No N_2O: Inhalational agents are poorly tolerated most times.
  - Use low inspiratory pressures, avoid barotrauma, never try to "reexpand" hypolastic lung or a contralateral pneumothorax may occur—sudden bradycardia, hypotension, PaO_2, lung compliance. Consider prophylactic chest tube.
  - Maintain PaO_2 90–100 mmHg, $PaCO_2$ 25–35 mmHg to ↓ PVR.
  - Rapid intraoperative deterioration: Pulmonary HTN crisis, contralateral pneumothorax.
  - Sometimes small abdominal cavity does not accommodate hernia content; repair in stages.

CDH lung hypoplasia is associated with ↓ type 2 pneumocytes and ↓ surfactant.

Postoperative Care Considerations

- Muscle relaxant, hyperventilation.
- ECMO:
  - Consider if poor response to conventional therapy.
  - Hemodynamic instability.
  - Persistent acidosis.
  - Severe barotrauma.
  - Severe pulmonary HTN refractory to pharmacologic treatment.

- **Contraindications:**
  - Gestational age < 35 weeks.
  - Weight < 2000 g.
  - Preexisting intracranial hemorrhage.
  - Congenital or neurological anomalies incompatible with viable outcome.
  - > 1 week of aggressive ventilatory therapy.
  - Congenital heart disease.
- Overall survival rate: 50–87%.

Meconium Aspiration

Background

- Leading cause of death in full-term newborn.
- With meconium-stained amniotic fluid, 55–60% of infants have meconium in tracheas, 35–50% chance of meconium aspiration syndrome with mortality 3.3%.
- Associated findings:
  - Uteroplacental insufficiency, late decelerations.
  - Post-term pregnancy.
  - Maternal HTN.
  - Placental previa.
  - Maternal pulmonary disease.
  - Placental abruptions.
  - Cord prolapse and cord compression.

Meconium aspiration is a major cause of persistent pulmonary HTN in the newborn. Also a cause of peristent fetal circulation.

Pathophysiology

- Hypoxia + ↑ vagal tone → intestinal ischemia.
- ↑ hyperperistalsis and rectal sphincter relaxation → meconium passage.
- Temporary compensated fetal distress → peripheral ischemia.
- Umbilical cord compression also can → meconium passage. This is normal physiology in mature fetus of ≥ 42 weeks.
- Meconium aspiration syndrome:
  - Meconium particles cause partial airway obstruction.
  - Large amount → atelectasis → hypoxia and may → rapid death.
  - Small amount can cause peripheral distal airway obstruction.
  - Partial airway obstruction would allow inspiration, not expiration, with associated air trapping → pneumothorax.
- Chest x-ray: Infiltrates, hyperexpansion, extra-alveolar air.
- Higher risk of persistent fetal circulation/pulmonary HTN.

Treatment

- Best way to prevent aspiration: Early suction of fetal mouth and pharynx before delivery of the shoulders.
- Intubation and tracheal suction if infants are depressed and exposed to thick particulate matter on emergence.
- Provide oxygen support if infant is hypoxic and bradycardic.
- Orogastric tube suction when breathing spontaneously.
- Mechanical support: Avoid air trapping with short inspiratory time.
- Surfactant is effective if given within 6 hr of birth.

Congenital Heart Defects (CHDs)

- CHDs can be classified anatomically (obstructive vs. nonobstructive), shunt (restrictive vs. nonrestrictive), or by clinical manifestations.
- Some well characterized CHDs include:
  - Tetralogy of Fallot.
  - Ventricular septal defect.
  - Pulmonary stenosis.
  - Right ventricular hypertrophy.
  - Overriding aorta.
  - Coarctation of the aorta.
  - Transposition of the great vessels.
  - Pulmonary stenosis with atrial septal defect.
  - Eisenmenger's syndrome.
  - Chronic left-to-right shunting → ↑ PVR, → eventual right-to-left shunting.
- Important to identify any other abnormalities, as several CHDs are associated with congenital syndromes.

Croup vs. Epiglottitis

See Table 9-8.

Nectrotizing Enterocolitis (NEC)

Umbilical vessel cannulation is also associated with NEC risk.

- Primarily affects premature infants (very low birth weight = 5–15%; < 1500 g = 75%).
- ↑ risk with large, early feedings, PDA, polycythemia, acidosis, shock, bowel ischemia.

Etiology

Immature intestine → ↑ stasis, which → ↑ bacterial proliferation, ↑ local infection and ischemic event (low cardiac output, asphyxia, apnea, heart failure, bradycardia), → necrosis and perforation with gangrene, fluid loss, peritonitis, septicemia, disseminated intravascular coagulation, shock, cardiovascular and respiratory collapse.

Signs/Symptoms

- Abdominal distention, bloody feces, irritability, metabolic acidosis, glucose instability, apnea, feeding intolerance, multisystem failure, lethargy.
- Pneumatosis intestinalis.

Treatment

- **Medical:**
  - Cessation of oral intake (NGT to decompress intestines), total parenteral nutrition (TPN).
  - Antibiotics.
  - Ventilatory support, fluid resuscitation, adequate perfusion, correct metabolic acidosis, inotropic agents if needed, coagulopathy correction.

TABLE 9-8. Comparisons of Croup and Epiglottitis

CROUP	EPIGLOTTITIS
Laryngotracheobronchitis—subglottic	Supraglottic inflammation
6 months–5 years	3–7 years
Self-limited	Rapid ↑ cardiorespiratory arrest due to severe airway obstruction
Gradual onset	Abrupt
Viral etiology	*Haemophilus influenzae*, type B
Febrile, no acute distress	Febrile, respiratory distress
Hoarseness with inspiratory stridor	Inspiratory stridor
Physical exam: Benign	Physical exam: ■ Pharyngitis ■ Marked salivation ■ Inflamed epiglottis
X-ray: ■ Narrow epiglottis ■ Subglottic narrowing = Steeple's sign	X-ray: ■ Englarged epiglottis ■ Tracheal narrowing
Treatment: ■ Mild: Conservative management with humidified air, fluid hydration, fever control ■ Severe: Nebulizer treatment with racemic epinephrine ■ Rare intubation	Treatment of acute epiglottitis: ■ Supine poorly tolerated ■ To OR for intubation ■ Induce in sitting position ■ Avoid muscle relaxants and barbiturates ■ Atropine IV: Attenuate reflex bradycardia ■ Lidocaine IV: Minimize coughing and laryngospasm ■ Extubate when fever resolves and presence of air leak around endotracheal tube

- **Surgical:**
  - For perforation, gangrene, presence of portal gas, or clinical deterioration.
  - Peritoneal drain placement at the bedside.
  - Exploratory laparotomy/bowel resection/ileostomy.

Anesthetic Management

- Aspiration precautions.
- Routine monitors (temperature, blood gases, electrolytes, glucose (A-line ideal—usually umbilical artery).
- Continue all principles of medical management in the OR.
- Significant fluid and blood losses need replacement.
- Abdominal relaxation.
- Careful titration of anesthesia (low tolerance for potent agents)—narcotic/muscle relaxant technique.

- Ketamine 0.5–1 mg/kg every 20–30 min.
- Fentanyl 2–3 mg/kg to max 10–12 mg/kg.
- Avoid N_2O.

- Postoperatively in neonatal intensive care unit (NICU) on ventilator till stable, TPN, fluid replacement.

Complications After NEC

- Abdominal adhesions → bowel obstruction.
- Short bowel syndrome—TPN dependent.

NEWBORN RESUSCITATION

- Six percent require resuscitation (< 1500 g, 80%).
- **Risk factors:** Drugs, trauma of precipitate labor and obstetrics, asphyxia, prematurity.

Fetal Asphyxia and Etiology

- Tight nuchal cord, prolapsed cord.
- Premature separation of placenta = abruption, uterine rupture.
- Uterine hyperactivity.
- Maternal hypotension, cardiovascular collapse (amniotic fluid embolization).

Pathophysiology

Acidosis and hypoxia → myocardial depression, shift of oxyhemoglobin dissociation curve to right, ↑ in PVR.

Recognition and Treatment of Primary and Secondary Apnea

- **Primary apnea:** When asphyxiated, the infant responds with an ↑ respiratory rate. If the episode continues, the infant becomes apneic, followed by a drop in heart rate and a slight ↑ in blood pressure. The infant will respond to stimulation and O_2 therapy with spontaneous respiration.
- **Secondary apnea:** When asphyxia is allowed to continue after primary apnea, the infant responds with a period a gasping respiration, falling heart rate, and falling blood pressure. The infant takes a last breath and then enters the secondary apnea period. The infant will not respond to stimulation, and death will occur unless resuscitation begins immediately.
- Because after delivery of an infant it is impossible to differentiate between primary apnea and secondary apnea, assume the infant is in secondary apnea and begin resuscitation immediately.

APGAR

See Table 9-9.

Epidemiology

- Five million neonatal deaths per year worldwide.
- Birth asphyxia accounts for 19% of neonatal deaths.
- Newborns requiring respiratory assistance at birth: 10%.

TABLE 9-9. APGAR Scores

	0	1	2
Appearance	Pale, blue	Body pink, extremities blue	Pink
Pulse	Absent	< 100 bpm	> 100 bpm
Grimace	No response	Grimace	Cough, sneeze, cry
Activity/tone	Limp	Some flexion	Active motion
Respiratory	Absent	Slow, irregular	Good, crying

Epinephrine

Hypotension: 1 μg/kg IV

Cardiac arrest: 10 μg/kg IV

Repeat q 3–5 min

- Newborns requiring extensive resuscitation at birth: 1%.
- Early resuscitation is key (intervene at primary apnea).
- Evaluate respirations.
- If spontaneous respirations → HR.
  - HR < 100: PPV 40 bpm × 15–30 sec.
    - If drug-depressed: Narcan 0.1 mg/kg → evaluate HR.
  - HR > 100 → color:
    - Blue: Give O_2.
    - Pink or acrocyanosis: Observe and monitor.
- No respirations → PPV 15–30 sec.
  - HR < 60:
    - Continue ventilation + chest compression (120/min).
    - Initiate medications if HR < 80 after 30 sec, continue CPR.
  - HR 60–100:
    - HR not ↑: Continue CPR if HR < 80.
    - HR ↑: Continue ventilation.
    - HR > 100: Watch for spontaneous respirations, then discontinue ventilation efforts.

CHAPTER 10

Obstetric Anesthesia

MORTALITY

$$\text{Mortality} = \frac{\text{\# pregnancy-related deaths}}{\text{\# of live births}}$$

- Women > 35 years old.
- African-American.
- Patients without prenatal care

Causes of Death

- Pulmonary embolus: 21%.
- Pregnancy-induced hypertension (HTN): 19%.
- Other causes include amniotic fluid embolization, intracranial hemorrhage (ICH), etc.
- Anesthesia accounts for 2–3% of maternal deaths, most occurring during or after C-sections.
  - General anesthesia: 32 per 1 million.
  - Regional anesthesia: 2 per 1 million.

When Do They Die?

- Thirty-four percent died within 24 hr of delivery.
- Fifty-five percent 1–42 days after delivery.
- Eleven percent 43 days to 1 year after delivery.

MATERNAL PHYSIOLOGIC CHANGES

Central Nervous System

- Minimum alveolar concentration (MAC) ↓:
  - ↓ 40% with normalization around the third day after delivery.
  - Due to ↑ progesterone and β-endorphin levels.
- ↑ sensitivity to local anesthetics:
  - ↓ dose up to 30%.
  - Likely due to hormones.
  - Engorgement of epidural venous plexus.
    - ↓ cerebrospinal fluid (CSF) volume.
    - ↓ volume of epidural space.
    - ↑ epidural space pressure.
    - ↑ chance of intravascular injection.

ABG in pregnancy:

Pao_2 ↑ by 10

$Paco_2$ ↓ by 10

Hco_3 ↓

pH normal 2° to compensatory metabolic acidosis

Respiratory System

- ↑ O_2 consumption (20–50%) and cardiac output contributes to rapid desaturation.
- ↓ functional residual capacity (FRC; at term up to 20%) also contributes to rapid desaturation.
  - Normalizes 48 hr after delivery.
  - Mandatory preoxygenation before inducing general, use supplemental oxygen during/after procedures.
  - Closing volume exceeds FRC in 50% of women at term.

Lung volume changes:

↑ V_T + unchanged RR → ↑ MV

↑ CC

↓ RV → ↓ FRC

↓ ERV + ↑ IC → normal VC

- ↑ minute ventilation (up to 50%).
- ↓ $PaCO_2$:
  - To 28-32 mmHg.
  - Compensatory ↓ in bicarbonate.
- ↑ 2,3-DPG to offset hyperventilation.
- ↑ hemoglobin P50.
- Thoracic breathing > abdominal breathing.
- ↑ intrapulmonary shunt.
- Airway:
  - Capillary engorgement of mucosa ↑ risk of trauma/bleeding/obstruction.
  - Use smaller endotracheal tube (ETT).

Cardiovascular System

- ↑ plasma volume:
  - ↑ aldosterone production.
  - Up to 45% ↑.
  - Dilutional anemia.
- ↑ blood volume:
  - +1000 to 1500 mL, ~90 mL/kg.
  - Normalizes 1–2 weeks postdelivery.
- ↑ heart rate (HR) × ↑ stroke volume (SV) = ↑ cardiac output (CO).
  - Greatest ↑ during labor and after delivery.
  - Normalizes 2 weeks postdelivery.
- Systolic ejection flow murmur, ↑ S1 splitting, or presence of S3.
- Rightward shift of hemoglobin dissociation curve.
- ↓ systemic vascular resistance (SVR) (↓ diastolic blood pressure [DBP] and ↓ systolic blood pressure [SBP]) during second trimester.
- ↓ response to adrenergic agents and vasoconstrictors.

Aortocaval Compression in Supine Position

After week 28:

- Supine hypotension syndrome in 20% of women
  - Pallor.
  - Diaphoresis.
  - Nausea and vomiting.
  - Can ↓ uterine and placental blood flow.
- Preventable cause of fetal distress
  - Turn patient on left side or place a > 15-degree wedge.
  - Avoid Trendelenburg positioning and systemic hypotension.

Renal System

A normal Cr value is abnormal in pregnancy.

- Renal vasodilatation, kidney enlargement.
- ↑ aldosterone + ↑ renin = ↑ Na retention.
- ↑ renal plasma flow and glomerular filtration rate (GFR) up to 50% during first trimester, normalizes in third trimester.
- ↓ serum creatinine and blood urea nitrogen (BUN).
- Mild glycosuria (1–10 g/d) and/or proteinuria (< 300 mg/d).

Gastrointestinal System

- Gastroesophageal reflux disease (GERD) and esophagitis:
  - Upward and anterior displacement of stomach.
  - ↑ progesterone causes ↓ GE sphincter tone.
  - Placental gastrin causes ↑ gastric acid (gastric pH < 2.5).
  - Gastric emptying—probably ↓ during labor.
  - Full stomach: > 60% of parturients have a gastric volume > 25 mL.
- ↑ risk of aspiration:
  - Opioids and anticholinergics ↓ lower esophageal sphincter pressure.
  - ↑ nausea and vomiting.
- Hepatic effects—overall unchanged:
  - ↑ alkaline phosphatase due to placental secretion.
  - Minor ↑ transaminases and LDH in third trimester.
  - Twenty-five to thirty percent ↓ in pseudocholinesterase activity—rarely prolongs succinylcholine.
  - ↑ gallstones secondary to inhibition of cholecystokinin release by ↑ progesterone levels.

Hematological System

- Hypercoagulable state:
  - ↑ fibrinogen.
  - ↑ factors VII, VIII, IX, X, XII.
- Leukocytosis.
- ↓ platelet count.
- Prevent deficiency anemia by supplementing iron and folate.

Metabolic Effects

- ↑ insulin levels, ↓ blood glucose.
- ↓ amino acids.
- ↑ free fatty acids/ketones/triglycerides.
- ↑ thyroid-binding globulin/triiodothyronine (T_3)/thyroxine (T_4), but patient remains euthyroid because free levels remain normal.

Musculoskeletal System

↑ relaxin:

- Softens cervix, inhibits contractions, relaxes pubic symphysis.
- Lax spine ligaments, which may contribute to patient's developing ↑ back pain.

UTEROPLACENTAL CIRCULATION

Uteroplacental insufficiency → intrauterine growth restriction (IUGR).

Uterine Blood Flow

- Uterus gets up to 10% of CO at term; of this:
  - 80% goes to placenta.
  - 20% goes to myometrium.

- Factors that ↓ uterine blood flow:
  - Hypocapnea: $PaCO_2$ < 20 mmHg.
  - Systemic hypotension (eg, aortocaval compression, hypovolemia, postregional sympathectomy).
  - Vasoconstriction (stress catacholamines).
  - Contractions (↑ venous pressure).
- See Table 10-1 for anesthetic effects on uteroplacental blood flow.

Drugs that do NOT cross placenta (secondary to ↓ lipid solubility/↑ weight/↑ ionization):

Heparin

Insulin

Glycopyrrolate

Nondepolarizing neuromuscular blockers (too large)

Succinylcholine (too ionized)

Placental Function and Anatomy

- Intervillous spaces contain fetal villi.
- Spiral branches of the uterine artery drain into uterine veins
- Umbilical cord: Two arteries, one vein.
- Five mechanisms of placental exchange:
  1. Diffusion.
  2. Bulk flow.
  3. Active transport.
  4. Pinocytosis.
  5. Breaks in placental membrane (possible route of Rh sensitization).
- Gas exchange:
  - PaO_2 of fetal blood from placenta = 40 mmHg.
  - Fetal O_2 dissociation curve is shifted to the left.
  - Maternal hyperventilation ↑ gradient across the placenta, facilitating CO_2 diffusion.
- Placental transfer of anesthetic agents:
  - Most inhalational agents freely cross and generally produce little fetal depression at < 1.0 MAC.
  - Neonates display ↑ sensitivity to respiratory depressant effects of morphine.

TABLE 10-1. Anesthetic Effects on Uteroplacental Blood Flow

Agent	Effect on Uteroplacental Blood Flow
Barbiturates	Small ↓ (↓ maternal BP)
Propofol	Small ↓ (↓ maternal BP)
Ketamine	< 1.5 mg/kg ↔, > 2 mg/kg may cause uterine hypertonus and resultant ↓
Midazolam	↓ only if used as an induction agent
Etomidate	Likely ↔
Volatile agents	↔ at < 1.0 MAC, ↓ at > 1.0 MAC
Local anesthetics	↓ at high intravascular doses—uterine vasoconstriction
Dilute epinephrine	↔

- Muscle relaxants are highly ionized, impeding placental transfer—minimal fetal effects.
- The more protein bound the agent, the lower the fetal blood levels.

Basic Fetal Physiology

- Two cardiac shunts (see Figure 10-1):
  - Foramen ovale
  - Ductus arteriosus
- Extrauterine life possible after 24–25 weeks' gestation.
- Surfactant:
  - Production at 30 weeks.
  - Sufficient amount at 34 weeks.
  - Accelerated by glucocorticoid administration.

Stage 1 involves T10–L1 (visceral pain). Stage 2 involves S2–S4 (visceral and somatic pain via pudendal nerve).

STAGES OF LABOR

First Stage

- From onset to complete cervical dilation.
- Two phases:
  - **Latent phase:** 2–4 cm slow contraction interval.
  - **Active phase:** Contractions 3–5 min apart.

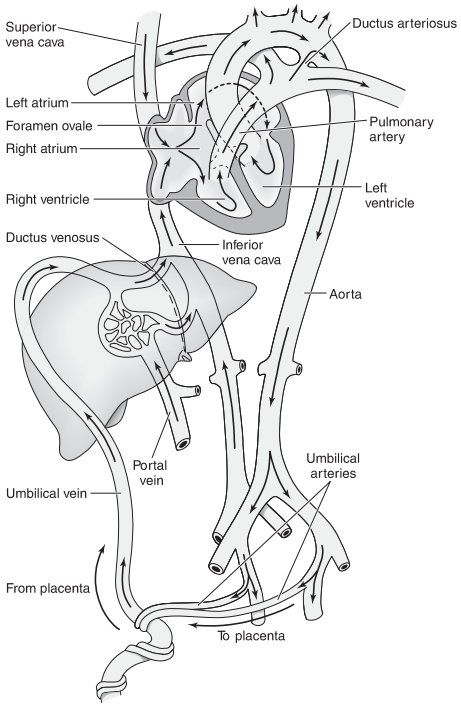

FIGURE 10-1. Fetal circulation with flow patterns through foramen ovale and ductus arteriosus.

(Reproduced, with permission, from Ganong WF. *Review of Medical Physiology*, 20th ed. New York: McGraw-Hill, 2001.)

- Duration: 8–12 hr (nulliparous), 5–8 hr (multiparous).
- Pain: Visceral pain from cervical and uterine plexuses:
  - T11–12 during latent phase.
  - T10–L1 during active phase.

Second Stage

- From complete cervical dilation to fetal descent and fetal delivery.
- Duration: 15–120 min.
- Pain: Perineal from stretching and compression of perineal structures:
  - T10–S4 (pudendal nerve S2–S4).
  - Not easily covered by labor epidural/combined spinal epidural (CSE).
- See Table 10-2 for effects of anesthetic agents on labor progression.

Third Stage

- From delivery of baby to delivery of placenta.
- Duration: 15–30 min.
- Greatest cardiac strain occurs after delivery, as CO can rise up to 80% above prelabor values.

ANESTHETIC MANAGEMENT

Nonpharmacologic Approaches

- Bradley, Dick-Read, Lamaze, Duola, LeBoyer techniques.
- Hypnosis.
- Transcutaneous electrical nerve stimulation (TENS).
- Biofeedback.
- Acupucture.

Pharmacologic Parenteral Approaches (Table 10-3)

Used in early stages of labor:

- Can cause fetal depression–respiratory depression, acidosis, abnormal neuro exam.
- Loss of beat-to-beat variability in fetal heart rate (FHR) tracing.

TABLE 10-2. Effect of Anesthetic Agents on Labor

Agent	Effect
Volatile agents	↓ uterine activity at equipotent doses
Opioids	↓ progression of labor
Ketamine	↔ effect
Regional	↔ effect when low concentrations of local and opioid used for CSE or epidural *(but there is a 10-fold ↑ in C-section rate in patients with severe pain and no regional anesthesia!)*

TABLE 10-3. Parenteral Agents/Doses/Effects

Parenteral Agent	Dose	Effects/Considerations
Meperidine	10–25 mg IV, 25–50 mg IM, up to 100 mg	↓ respiration (in 10–20 min if IV, in 1–3 hr if IM), given when delivery not expected for at least 4 hr.
Fentanyl	25–100 μg/hr	Onset 3–10 min, lasts 60 min initially, ↑ duration with repeat doses, respiratory ↓ outlasts analgesia, low doses associated with little or no fetal depression, no effect on Apgars.
Morphine	Not generally used	Causes respiratory depression in fetus.
Butorphanol	1–2 mg IV/IM	Little or no respiratory depression, but ↑ sedation.
Nalbuphine	10–20 mg IV/IM	Little or no respiratory depression, but ↑ sedation.
Promethazine	25–50 mg IM	↓ anxiety, ↓ nausea, ↓ opioid requirements.
Hydroxyzine	50–100 mg IM	↓ anxiety, ↓ nausea, ↓ opioid requirements.
Nonsteroidal anti-inflammatory drugs (NSAIDs)	Not recommended	↓ uterine contractions and promotes fetal patent ductus arteriosus closure.
Benzodiazepines	Not generally used	Prolonged neonatal depression, undesirable amnesia.
Ketamine	10–15 mg IV	Good analgesia, loss of consciousness in 2–5 min, doses > 1 mg/kg ↓ APGAR scores and ↑ hypertonic uterine contractions, may have psychomimetic effects.

- ↓ fetal movements.
- Premature fetus has ↑ sensitivity.
- Maternal: Respiratory depression, nausea, slow gastric emptying.

Regional Anesthesia

Spinal or Epidural Opioids Alone (Without Local Anesthetic)

- Can cause respiratory depression, nausea, pruritus, sedation.
- Useful for high-risk patients who cannot tolerate sympathectomy (eg, hypovolemia, cardiac disease).
- Morphine epidural dose > 5 mg not recommended because of ↑ risk of delayed respiratory depression; 30- to 60-min onset, 12- to 24-hr analgesia.
- Meperidine 50–100 mg provides good but brief (1–3 hr) analgesia.
- Fentanyl + morphine combo (eg, 2.5 mg morphine + 25–50 μg fentanyl) has faster onset, fewer side effects, prolonged analgesia.

Spinal, Epidural, CSE: Opioid/Local Anesthetic Combinations

- Continuous epidural is the most common because the catheter can be used for C-section.

VBAC is not a contraindication to neuraxial labor analgesia.

- Common epidural infusion: Bupivacaine or ropivacaine 0.625–0.125% + fentanyl 2–3 μg/mL or sufentanil 0.3–0.5 μg/mL (+ low concentration epinephrine is an option, too).
- Resuscitation equipment and medications must be available.
- BP and HR monitoring.
- Not contraindicated for vaginal birth after cesarean (VBAC): Changes in uterine tone and contraction pattern are more reliable signs of uterine rupture (rather than pain, which is inconsistent).
- For CSE, use needle-through-needle technique with 24- to 27-gauge spinal needle (smaller needle, less postdural puncture headache).

Contraindications

See Table 10-4.

Considerations When Placing Spinal/Epidural/CSE

- Dilute local + opioid infusions have little if any effect on labor progression.
- Earlier placement = easier positioning, better patient cooperation.
- Good to have in case of emergent C-section.
- To be initiated when patient requests it and the obstetrician approves.
- Conservative criteria: No fetal distress, contractions every 3–4 min, dilation 3–4 cm, engagement of fetal head.
- Can be done lateral decubitus or sitting, L3–L4 or L4–L5.
- Wet tap occurrence 0.25–9%. If this occurs, thread catheter subarachnoid or remove needle and try at higher level.
- Loss of resistance (LOR) to saline or air; excessive injection of air (> 2–3 mL) associated with patchy blocks and headache.
- Multiorificed catheter advanced 4–7 cm.
  - ↓ incidence of unilateral block.
  - ↓ false negative aspiration of CSF if intrathecal.

Activation of an Epidural

- 500–1000 mL Lactated Ringer's (LR) bolus (avoid glucose-containing solutions—can cause ↑ fetal insulin secretion and newborn hypoglycemia).
  - 3-mL test dose 1.5% lidocaine + 1:200,000 epinephrine may be used to test the epidural placement (controversial—some clinicians use only local anesthetic because of less toxicity and test doses are unreliable in patients in labor).
  - Intrathecal injections (sensory block 2–3 min, motor block in 3–5 min).

TABLE 10-4. Contraindications for Regional Anesthesia: Absolute vs. Relative

Absolute	Relative
Infection over site	Neurological disease
Coagulopathy	Back disorder
Thrombocytopenia	Some forms of cardiac disease
Marked hypovolemia	
True local anesthetic allergies	
Patient refusal/refusal to cooperate	
Anticoagulation	

- Intravascular injection (↑ HR 20–30 bpm within 1 min, perioral numbness, tinnitus).
- Catheters can erode into intrathecal or intravascular space over time.
- Give incremental doses of local + opioid mixture, waiting 1–2 min between doses. Total of 10 mL, 5 mL per incremental dose (maximum).
- Monitor BP for 15 minutes postepidural; give more fluids/ephedrine/phenylephrine if hypotensive.
- Can be activated in supine or left uterine displacement position (depending on patient and fetal status).

Other Regional Options (Less Frequently Used)

- Paracervical block—during stage 1, provides uterus and cervical anesthesia, risk of fetal bradycardia from uterine artery constriction.
- Caudal—during stage 1, provides perineal anesthesia, risks of subarachnoid/IV/fetal injection.
- Pudendal nerve block—during stage 2, provides perineal anesthesia.

Complications of Regional Anesthesia

- Hypotension: 20–30% ↓ BP or BP < 100 (left uterine displacement, ephedrine or phenylephrine).
- Intravascular injection: Seizure (intubate), cardiovascular collapse (initiate advanced cardiac life support).
- Intralipid 20% should always be immediately available wherever blocks are performed, for management of cardiac toxicity.
- Intrathecal injection and/or high spinal (intubate, circulatory support).
- Postdural puncture headache: Bed rest, hydration, analgesics, IV caffeine 500 mg, epidural saline injection, blood patch.
- Maternal fever: May result from shivering or inhibition of sweating. Should trigger further workup to rule out causes such as chorioamnionitis and neonatal sepsis.

Other Common Parenteral Drugs

Vasopressors

- Uterus has both α and β receptors:
  - α_1 uterine contraction
  - β_2 uterine relaxation
- Phenylephrine and ephedrine are both acceptable means of treating hypotension by ↑ arterial BP.

Drugs that ↓ uterine tone:

β_2 agonists (terbutaline, ritodrine)

Methylxanthines (indomethacin)

Nicardipine

Magnesium

Volatile agents

Nitroprusside

Ethanol

Drugs that ↓ Uterine Tone

- **β_2 agonists (terbutaline, ritodrine):**
  - Side effects: Bronchodilation, pulmonary edema, HPV inhibition, hypotension, arrhythmias, ↑ HR, ↑ glucose, ↓ Ca/K.
  - **Complications:** ↑ HR/CO/O_2 demand → MI; cerebral vasodilation.
- **Methylxanthines (indomethacin):**
  - Side effects: Nausea, GERD, PDA closure.
- **Nicardipine:**
  - Side effects: Hypotension, prolonged muscle relaxation, conduction defects.

- **Magnesium:**
  - Use: Premature labor, anticonvulsant, ↓ tone but not tocolytic.
  - Dose: 4 g over 20 min then 1–2 g/hr infusion.
  - Causes muscle relaxation by affecting calcium uptake and binding, ↓ the release of ACh and altering the sensitivity of the neuromuscular junction to ACh.
  - Side effects: Sedation, hypotension, heart block, muscle weakness, sedation, ↑ K, ↑ sensitivity to succinylcholine.
  - Calcium chloride ($CaCl_2$) counteracts the adverse effects of magnesium.

Drugs that ↑ uterine tone:

Oxytocin

Prostaglandins (hemabate)

Ergots (methergine)

Ketamine

Amides

Drugs that ↑ Uterine Tone

Oxytocin

- $T_{½}$: 3–5 min.
- Side effect: Hypotension with reflex tachycardia.
- Complications: Fetal distress, uterine tetany, maternal water intoxication.

Prostaglandins (Carboprost/Hemabate)

- Synthetic prostaglandin F2.
- Dose: 0.25 mg IM every 15–90 min, maximum 2 mg.
- Use: Stimulates uterine contractions, refractory postpartum hemorrhage.
- Side effects: Nausea, diarrhea, bronchospasm, V/Q mismatch → hypoxia.
- Contraindications: Asthma.

Ergots (Methergine)

- Dose: 0.2 mg IM.
- Faster onset than hemabate.
- Constricts smooth muscle.
- Use: Uterine atony.
- Side effects: HTN, bronchospasm.
- Contraindications: Preeclampia, asthma.

Magnesium Toxicity

Therapeutic	*4–6 mEq/L*
ECG changes (↑ PR, AV/SA block)	*5–10 mEq/L*
Loss of deep tendon reflexes	*10 mEq/L*
Respiratory paralysis	*15 mEq/L*
Cardiac arrest	*20 mEq/L*

Magnesium

- Treats premature labor.
- Anticonvulsant.
- Dose: 4 g over 20 min then 2 g/hr infusion.
- Causes muscle relaxation by affecting calcium uptake and binding, ↓ the release of acetylcholine and altering the sensitivity of the neuromuscular junction to acetylcholine.
- Side effects: Hypotension, heart block, muscle weakness, sedation.
- Calcium chloride ($CaCl_2$) counteracts the adverse effects of magnesium.

TABLE 10-5. General vs. Regional Anesthesia for C-Section Deliveries

Regional		General	
The Good	**The Bad**	**The Good**	**The Bad**
↓ fetal exposure to agents ↓ maternal aspiration risk Mother awake for birth Spinal opioids can be given for postop pain	If a complication from regional occurs, potentially difficult airway in an emergent situation → general anesthesia risks	Rapid and reliable Control of airway Potentially less hypotension	Chance of "cannot intubate, cannot ventilate" Risk of aspiration Fetal depression ↑ uterine relaxation/atony risk → bleeding

C-Section

- C-section rate 15–25%.
- Eighty to ninety percent under regional anesthesia.
- General anesthesia ↑ maternal mortality mostly due to airway complications (see Table 10-5).

Indications

See Table 10-6.

Regional Anesthesia for C-Section

- Need a T_4 level.
- Prehydration helps prevent hypotension (1000–1500 mL LR or normal saline (NS) bolus, 250–500 mL albumin).
- Positioning: Supine with wedge for left uterine displacement.
- Supplemental oxygen; keep BP > 100 with fluids, ephedrine, and phenylephrine.
- See Table 10-7 for medications, doses, and additives/fixes for regional anesthesia.

TABLE 10-6. Indications for C-Section

Labor Unsafe	Dystocia	Emergency
↑ risk of uterine rupture Previous classical incision Previous extensive myomectomy or reconstruction ↑ risk of hemorrhage Central/partial placenta previa Abruption of placenta Vaginal reconstruction	Fetopelvic disproportion Transverse/oblique lie Breech Dysfunctional uterine activity	Fetal distress Cord prolapse Maternal hemorrhage Chorioamnionitis Herpes with ruptured membranes Impending maternal death

TABLE 10-7. Regional Anesthesia for C-Sections

Type of Anesthesia	Medication and dose	Additives/Fixes
Spinal: Hyperbaric single shot or continous post-wet tap	Tetracaine 7–10 mg Lidocaine 50–60 mg Bupivacaine 10–15 mg	Add epinephrine 0.1 mg to prolong block if desired. Add fentanyl 5–20 g to enhance quality of block. Add morphine 0.2–0.3 mg for up to 24 hr postop analgesia (but ↑ respiratory depression).
Epidural: Load in 5-mL increments	Lidocaine 2% 15–25 mL Chloroprocaine 3% 15–25 mL (Bupivacaine can also be used, but less common)	± 1:200,000 epinephrine added to lidocaine, nothing to chloroprocaine; Add 1 mEq bicarb to lidocaine to advance onset time. Add fentanyl 50–100 g/sufenta 10–20 g to ↑ quality/intensity of block. 5 mg morphine good for 6–24 hr of postop pain relief.
Combined spinal epidural	Allows for rapid dense blockade and option to extend the block—mix and match with above options for spinal and epidural.	
Patchy block?	Ketamine 10–20 mg IV or 30% nitrous inhalation. If procedure still not tolerated, convert to general anesthesia	

General Anesthesia Considerations

- Indications:
  - Fetal distress during second stage.
  - Tetanic uterine contractions.
  - Breech extraction.
  - Version and extraction.
  - Retained placenta.
  - Uterine inversion.
  - Noncooperative patient.
- Left uterine displacement positioning.
- Important to preoxygenate for 3–5 min.
  - Aspiration occurs in 1:400 to 1:500 obstetric patients vs. 1:2000 nonobstetric patients.
  - Prophylaxis with sodium citrate 30 mL + IV metoclopramide 10 mg; can give glycopyrrolate 0.1 mg to ↓ secretions.
  - Preoxygenation, followed by rapid sequence induction and intubation; use orogastric tube to empty stomach.
  - Failed intubation 1:300 for obstetric patients due to airway edema vs. 1:2000 for nonobstetric patients.
  - Short-handled laryngoscope.
  - Difficult airway equipment (ie, glidescope, use of laryngeal mask airway) and jet ventilator available.
  - Review difficult airway algorithm: If can't intubate, can't ventilate → surgical airway.
  - Life of mother comes before the fetus—fatality rate with general anesthesia 16.7 times greater than that with regional, mostly due to airway complications.

- Extubate only when awake.
- Inhaled agents: Any may be used.
  - 1–2 MAC + 100% oxygen to start.
  - ↓ 0.5 MAC once fetus and placenta are delivered; supplement with propofol and opioid (helps prevent uterine atony).
- Muscle relaxants: Use short-acting nondepolarizing.
- Pregnancy-related morbidity and mortality.

General Considerations for Pregnant Patients

- History:
  - Age.
  - Parity.
  - Duration of pregnancy and complicating factors.
  - Maternal health history.
  - BP measurement.
  - Airway assessment.
  - Examination of back (if pertinent).
- IV access:
  - 18-gauge or larger: Enables rapid fluid resuscitation or transfusion.
  - IV fluids to prevent dehydration.
- Type and screen.
- NPO status/aspiration prevention:
  - For uncomplicated patients, small amounts of clears allowed (such as ice chips).
  - High risk patients should be kept NPO.
  - Minimum 6-hr fast for elective C-sections.
  - 15–30 mL sodium citrate to ↑ gastric pH > 2.5.
  - H_2 blocker and metoclopramide to high-risk patients and patients receiving general anesthesia.
- Uterine displacement with > 15-degree wedge under right hip.
- Tocodynamometer and fetal HR monitor.

FETAL MONITORING

Fetal Heart Rate (FHR) Monitoring

- Baseline FHR ranges from 120 to 160 bpm.
- Persistent tachycardia:
  - Chronic fetal distress.
  - Maternal fever.
  - Infection.
  - Drug effect—ritodrine, terbutaline, atropine, ephedrine.
- Baseline variability:
  - Reflects sympathetic and parasympathetic adjustments to various stimuli.
  - Causes of ↓ variability:
    - Fetal asphyxia.
    - Drug effect—benzodiazepines, opioids, volatiles, anticholinergics, atropine.
    - Anemia.
    - Prematurity.
  - Causes of ↑ variability: Ephedrine.
- Accelerations: reflect normal fetal oxygenation and considered reassuring.
- Prolonged decelerations are > 2 min but < 10 min.

FHR:

Accelerations 2° to fetal movement.

Early decelerations 2° to head compression.

Late decelerations 2° to uteroplacental insufficiency.

Variable decelerations 2° to transient umbilical cord compression.

Decelerations (Figure 10-2)

- **Early:** U-shaped, begins with onset of contraction; low point coincides with peak of contraction, returns to baseline when uterus relaxes. Fetal head compression → vagal tone.
- **Late:** Begins 20–30 sec or more after onset of contraction, low point occurs well after peak of contraction. Myocardial ischemia from uteroplacental insufficiency. Intervention: ↑ fetal oxygenation, correct maternal hypotension, alleviate aortocaval compression, ↓ uterine activity (eg, break tetanic contractions).
- **Variable:** Variable in shape and onset, from umbilical cord compression, > 15 bpm from baseline and < 100 bpm.

Fetal Pulse Oximetry

- Newer technique; not currently endorsed by American College of Obstetricians and Gynecologists (ACOG) because of cost and lack of outcome improvement.

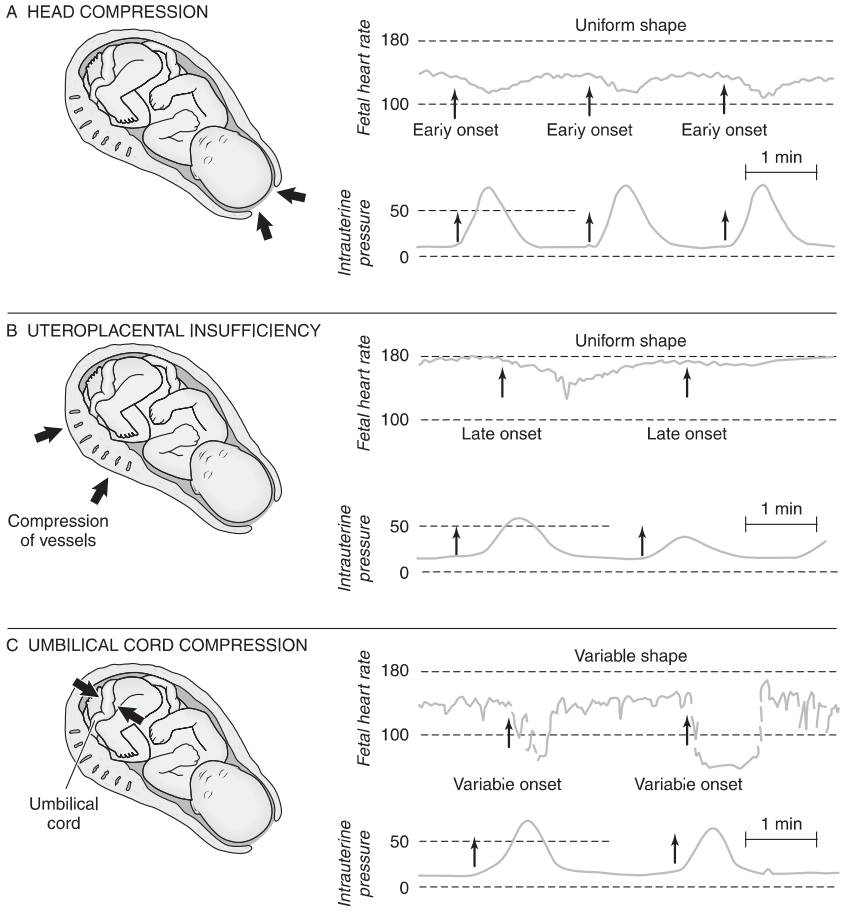

FIGURE 10-2. Classification and mechanism of fetal heart rate patterns.

(Reproduced, with permission, from Danforth DN, Scott JR. *Obstetrics and Gynecology*, 5th ed. Lippincott, 1986.)

- Used as adjunct to FHR monitoring if tracing is unreliable.
- O_2 saturation: 30–70% is normal.

Biochemical Monitoring—Scalp pH

Biochemical monitoring—Scalp pH > 7.25 is considered normal.

- pH 7.25 is lower limit of normal.
- < 7.20 indicates acidosis, 7.20–7.24 is preacidotic.
- Correlates with APGAR scores 1–2 min after birth:
  - Ninety-two percent with pH > 7.25 scored ≥ 7.
  - Eighty percent with pH < 7.16 scored ≤ 6.
- False positives and false negatives do occur.

Signs of Fetal Distress

- Nonreassuring fetal HR pattern:
  - Repetitive late decelerations.
  - ↓ variability and late decelerations.
- Fetal scalp pH < 7.2.
- Meconium.
- Oligohydramnios.
- IUGR.

COMPLICATIONS IN PREGNANCY

Dysfunctional Labor

- Prolonged latent phase:
  - > 20 hr in nulliparous.
  - > 14 hr in multiparous.
- Cervix ≤ 4 cm but completely effaced.
- Arrest of dilatation: Cervix stops dilating after 2 hr of active labor.
  - < 1.2 cm/hr in nulliparous.
  - < 1.5 cm/hr in multiparous.
- Disorder of descent:
  - < 1 cm/hr in nulliparous.
  - < 2 cm/hr in multiparous.
- Arrest of descent: Failure of fetal head to descend 1 cm in station after adequate pushing.

Preterm Labor/Delivery

- Eight to ten percent of all births.
- Eighty percent of early neonatal deaths.
- Preterm problems:
  - Respiratory distress syndrome.
  - ICH (poorly calcified cranium).
  - Hypoglycemia.
  - Hypocalcemia.
  - Hyperbilirubinemia.
- Fetal lung maturity:
  - Delaying delivery 24–48 hr may be beneficial if glucocorticoids (betamethasone) are administered.
  - Surfactant usually adequate after week 35.

Lecithin/sphingomyelin (L/S) ratio > 2.0 in 35+ weeks indicates that respiratory distress is unlikely.

- Tocolytics:
  - β_2 agonists: Terbutaline and ritodrine.
  - Methylxanthines (indomethacin).
  - Calcium channel blockers (nicardipine).
  - Magnesium.
  - Volatile agents.
  - Nitroprusside.
  - Ethanol.
- If nonemergent, delay anesthesia 3 hr after tocolysis to allow β-mimetic effects to subside—less hypotension.

Umbilical Cord Prolapse

- Occurs in 0.2–0.6% of pregnancies.
- Sudden fetal bradycardia and profound decelerations.
- Push presenting part back and place patient in Trendelenburg position.
- Immediate C-section.

Breech

- Occurs 3–4% of the time.
- Up to 10% of cord prolapse.
- Cephalic version attempted at 36–38 weeks.
  - Successful in 75% of patients.
  - Complications: Cord compression, placental abruption → C-section.
- Fetal head entrapment during vaginal or C-section delivery:
  - Nitroglycerin 50–100 μg IV can be tried.
  - Induce general anesthesia and administer volatile agent to relax the uterus.

Abnormal Vertex Presentation

- Occiput posterior.
- Face presentation.
- Shoulder dystocia:
  - Occurs in 0.2–2% of deliveries.
  - Fetal macrosomia.
  - If epidural not in place → general anesthesia.

Multiple Gestations

- Patients are prone to hypotension after regional.
- Mandatory IV fluid loading.
- Successive babies are more asphyxiated than the first.

Chorioamnionitis

- Regional anesthetic low risk.
- Avoid regional anesthesia if signs of impending sepsis are present:
  - High fever.
  - Mental status changes.
  - Tachypnea.

- Borderline hypotension.
- Thrombocytopenia/coagulopathy.

Preeclampsia and Eclampsia

- **Preeclampsia:**
- HTN (SBP > 140 mmHg or DBP > 90 mmHg or increase in baseline BP by 30 mmHg systolic or 15 mmHg diastolic) and proteinuria (> 3 g/24 hr) occurring after 20 weeks.
- Imbalance between placental thromboxane and prostacyclin—seven times more thromboxane than prostacyclin.
- Placental ischemia: Release of uterine renin.
- Proteinuria: Release of thromboplastin with subsequent deposition of fibrin in constricted glomerular vessels.
- Systemic vasoconstriction: Widespread arteriolar vasoconstriction → HTN, tissue hypoxia, endothelial damage.
- ↑ platelet aggregation:
  - Coagulopathy, occasional disseminated intravascular coagulation (DIC).
  - ↑ prothrombin time (PT), partial thromboplastin time (PTT): Indicate consumption of procoagulant.
- Endothelial cell injury:
  - ↓ placental perfusion and release of lipid peroxidases.
  - ↑ peripheral resistance.
- **Severe preeclampsia:**
  - Severe HTN (SBP > 160 mmHg or DBP > 110 mmHg) and severe proteinuria (> 5 g/24 hr).
  - Evidence of severe end-organ damage secondary to widespread vasospasm:
    - Refractory oliguria (< 400 mL/24 hr).
    - Cerebral or visual disturbances.
    - Pulmonary edema.
    - Epigastric pain/impaired liver function.
    - IUGR.
  - Complications:
    - Heart failure (may need PA catheter for fluid manage¬ment).
    - Periportal necrosis, rarely liver rupture and hemorrhage.
    - Reduced uric acid clearance.
    - Respiratory failure.
    - Ventilation/perfusion (V/Q) mismatch.
    - Abruption and rupture of fetal cotyledonary vessels
- **HELLP syndrome:**
  - **H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelets.
  - Form of severe preeclampsia.
  - BP elevations and proteinuria may be comparatively mild.
- **Eclampsia:** Preeclampsia + seizures.
  - Cerebral hemorrhage and edema account for ~50% of deaths.
  - Petechial hemorrhages often seen after convulsions.
  - Volume-depleted state: Mean plasma volume can be up to 40% below normal.
  - Definitive treatment is delivery of fetus and placenta.

Preeclampsia: *HTN (SBP > 140 mmHg or DBP > 90 mmHg or ↑ in baseline BP by 30 mmHg systolic or 15 mmHg diastolic) and proteinuria (> 3 g/24 hr) occurring after 20 weeks.*

Severe preeclampsia: *Severe HTN (SBP > 160 mmHg or DBP > 110 mmHg) and severe proteinuria (> 5 g/24 hr).*

Management

- **Anticonvulsant therapy:** Magnesium sulfate: See Magnesium under previous section titled Other Common Parenteral Drugs.
- **Antihypertensive therapy:**
  - Hydralazine: ↑ uteroplacental and renal blood flows.
  - Nitroprusside: For hypertensive emergency, usually tapered off in favor of longer-acting agent.
  - Nitroglycerin.
  - Labetalol.
  - Avoid ergot alkaloids (methergine) and ketamine.
- **Coagulopathy:**
  - Fresh whole blood, fresh frozen plasma (FFP), cryoprecipitate.
  - Regional anesthesia contraindicated in coagulopathic patients secondary to ↑ epidural hematoma risk.
- **Epidural analgesia:**
  - Not contraindicated in patients with adequate coagulation.
  - ↑ placental perfusion up to 75%.

Maternal Hemorrhage

Anesthetic considerations for all maternal hemorrhage situations:

- Two large-bore IVs (16- or 14-gauge preferable), +/– central line, +/– invasive monitoring.
- Blood available, may need massive transfusion protocol, may need FFP/cryoprecipitate/platelets/factors.
- Patient may need surgical intervention to control bleeding.
- General anesthesia utilized in uncontrolled hemorrhage/severe coagulopathy.
- Regional has been used in controlled/planned situations with success; judge on a case-by-case basis.

Placenta Previa

- Painless vaginal bleeding.
- Types:
  - Marginal placenta—close to internal os.
  - Partial placental previa—partially covers internal os.
  - Complete/central previa—covers internal os.
- Management:
  - Usually occurs after the seventh month.
  - < 37weeks: Bed rest and observation.
  - > 37weeks: C-section.
- Painless bleeding is placenta previa until proved otherwise; confirm by ultrasound.

Placenta Accreta

- Placenta adherent to internal surface of uterus.
- After one C-section: Occurs in 20–25% of patients with previa.

- After ≥ 4 C-sections: Occurs in 67% of patients with previa.
- **Placenta percreta:** Placenta penetrates myometrium and surrounding tissues.
- **Placenta increta:** Placenta invades muscle.

Placental Abruption

- Placental separation from the uterus.
- 0.2–2.4% of pregnancies in final 10 weeks of gestation.
- Uterine tenderness and hypertonus.
- Vaginal bleeding of dark, clotted blood.
- Common cause of fetal demise, perinatal mortality rate is > 50%.
- Risk factors:
  - HTN.
  - Trauma.
  - Short umbilical cord.
  - Multiparity.
  - Prolonged premature rupture of membranes (PROM).
  - Alcohol abuse.
  - Cocaine.
  - Abnormal uterus.
- Estimated blood loss (EBL) may be concealed in uterus—may need massive transfusion.
- Can cause coagulopathy → DIC, crash C-section.
- High maternal mortality: 1.8–11%.

Uterine Rupture

- Frank hemorrhage.
- Fetal distress.
- Hypotension.
- Continuous abdominal pain even with working epidural.
- ↓ uterine tone.
- Treat with immediate laparotomy under general anesthesia.

Amniotic Fluid Embolism

- Manifestations:
  - Sudden respiratory distress.
  - Circulatory collapse.
  - Uterine atony.
  - DIC.
- Management:
  - ABCs.
  - Fluids, inotropes.
  - Oxytocin, methergine, hemabate.
  - Platelets and coagulation factors.
  - Emergency cesarean section.

Consider possibility of pregnancy in all females of reproductive age.

Alteration in Physiologic Condition

- See previous sections. Treat patient like any other pregnant patient undergoing general anesthesia.
- Choose drugs with a long history of safety.
- FHR may be monitored if feasible, especially after 20 weeks' gestation (if it does not encroach on surgical field).

Teratogenicity

N_2O exposure associated with megaloblastic anemia and peripheral neuropathies

- Studies have failed to correlate anesthetic exposure with congenital defects in humans.
- However, type of condition that necessitated the surgery was correlated with ↑ fetal death (eg, pelvic procedures, obstetric procedures such as cerclage).
- IV agents such as thiopental, morphine, meperidine, and local anesthetics have not been shown to be teratogens.
- Benzodiazepines have been implicated in cleft palate anomalies.
- Nitrous oxide (N_2O) has teratogenic effect with longer exposure.
  - Inhibits methionine synthetase, which may cause myelination abnormalities.
  - Inhibits thymidine synthetase, which could affect DNA synthesis.
  - Single exposure to N_2O is not likely teratogenic.

Effects on Uteroplacental Blood Flow

- ↓ secondary to systemic hypotension.
- ↓ secondary to aortocaval compression.
- ↓ secondary to hemorrhage.
- ↓ secondary to ↑ uterine activity.
- ↓ secondary to pressors (epinephrine, norepinephrine).

Postpartum Patient Considerations

- Anemia.
- ↑ risk for aspiration still applies—treat them like they are still pregnant:
  - 8-hr fast.
  - H_2 blocker.
  - Sodium citrate.
  - Metoclopramide.
  - Rapid-sequence induction.
  - Extubate only when awake.
- ↓ plasma cholinesterase: Some prolongation of succinylcholine and mivacurium, sometimes vecuronium.
- Avoid high concentration of volatile agent: Theoretical risk of hemorrhage.
- IV opioids have little, if any, effect on neonates.

CHAPTER 11

Cardiac Anesthesia

General Evaluation

Preoperative Goals

- Determine severity of the lesion.
- Determine residual ventricular function.
- Secondary effects on pulmonary, renal, hepatic function.
- Rule out concomitant coronary artery disease (CAD) in older patients and patients with risk factors.
- Rule out myocardial ischemia in severe aortic stenosis (AS) or aortic regurgitation (AR) (without CAD).

History

- The New York Heart Association (NYHA) Functional Classification of Heart Disease is useful for grading the severity of heart failure, comparing patients and estimating prognosis (Table 11-1).
- Ask about exercise tolerance, fatigability, pedal edema, dyspnea, orthopnea, paroxsysmal nocturnal dyspnea, chest pain, neurological symptoms, and thromboembolic phenomenon.
- Prior procedures, valvotomy, valve replacement.
- Medications: Evaluate efficacy and exclude side effects. Common medications: Digoxin, diuretics, vasodilators, angiotensin-converting enzyme (ACE) inhibitors, antiarrhythmics, anticoagulants.
  - **Digoxin:** Controls ventricular rate in atrial fibrillation (AF). Ventricular rate: < 80–90/min at rest, < 120/min with stress/exercise. Preop vasodilator: ↓ preload, afterload, or both; if excessive, worsens exercise tolerance → postural hypotension. Signs of toxicity:
    - **Cardiac:** Arrhythmias; secondary to enhanced conduction and ↓ conductivity in the specialized cells of the atria, ventricles, sinoatrial (SA) and atrioventricular (AV) nodes.
    - **Gastrointestinal:** Nausea/vomiting.
    - **Central nervous system (CNS):** Confusion.
    - **Visual:** Altered color perception/scotomas.

TABLE 11-1. New York Heart Association (NYHA) Functional Classification

NYHA Class	Symptoms
I	No symptoms and no limitation in ordinary physical activity (eg, shortness of breath when walking, climbing stairs, etc.).
II	Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.
III	Marked limitation in activity due to symptoms, even during less-than-ordinary activity (eg, walking short distances [20–100 m]). Comfortable only at rest.
IV	Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients.

(Reproduced, with permission, from The Criteria Committee of the New York Heart Association. *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels,* 9th ed. Boston: Little, Brown & Co., 1994: 253–256.)

Physical Examination

- Most important: Search for signs of congestive heart failure (CHF).
  - Left-sided failure: S3 gallop/pulmonary rales.
  - Right-sided failure: Jugular venous distention, hepatojugular reflux, hepatosplenomegaly, pedal edema.
- Neurological deficits due to embolic phenomenon.

Laboratory Evaluation

- Liver function tests (LFTs): Severe/chronic right-sided failure → passive hepatic congestion → hepatic dysfunction.
- Arterial blood gas (ABG): Pulmonary symptoms.
- Coagulation studies: Prothrombin time (PT)/partial thromboplastin time (PTT)—indicate degree of anticoagulation reversal.

Electrocardiogram (ECG)

- Usually nonspecific: T wave/ST segment changes, arrhythmias, conduction abnormalities, QRS deviation reflecting ventricular hypertrophy.
- Digoxin toxicity: Prolonged P-R interval, arrhythmias.
- In order of decreasing frequency: Ventricular ectopy, paroxysmal atrial tachycardia (PAT) with 2:1 AV block, AV block, marked sinus bradycardia, low atrial or AV junctional rhythms, AV dissociation.

Chest X-ray (CXR)

- Cardiac size.
- Pulmonary vascular congestion.
- Specific cardiac chamber enlargement.

Special Studies

- Echocardiography.
- Radionuclide angiography.
- Cardiac catheterization.

Premedication

- Normal/near normal ventricular function: Standard doses.
- Poor ventricular function: Doses reduced in proportion to the severity of ventricular impairment.
- Usual medications on morning of surgery.
- Supplemental O_2 in patients with pulmonary HTN/pulmonary disease.

Antibiotic Prophylaxis

- The risk of infective endocarditis in patients with valvular heart disease following bacteremic events—including dental, oropharyngeal, nasopharyngeal, gastrointestinal, or genitourinary surgery or any incision and drainage—is well established.
- Antibiotic prophylaxis with dental procedures is recommended only for patients with cardiac conditions associated with the highest risk of adverse outcomes from endocarditis, including:
  - Prosthetic cardiac valve
  - Previous endocarditis

- Congenital heart disease only in the following categories:
  - Unrepaired cyanotic congenital heart disease, including those with palliative shunts and conduits.
  - Completely repaired congenital heart disease with prosthetic material or device, whether placed by surgery or catheter intervention, during the first 6 months after the procedure.
  - Repaired congenital heart disease with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization).
  - Cardiac transplantation recipients with cardiac valvular disease.

Anticoagulation Management

- Most patients can safely have their warfarin stopped 3 days prior to surgery and restarted 2–3 days postoperatively.
- The risk of thromboembolic complications ↑ with:
  - Prior history of embolism.
  - Presence of thrombus.
  - Atrial fibrillation.
  - Prosthetic mechanical valve:
    - Highest: Caged ball mechanical (Starr Edwards) in mitral/tricuspid location.
    - Intermediate: Tilting disc (St. Jude) valve.
    - Lowest: Bioprosthesis (porcine or bovine tissue valves).
- If the thromboembolism risk is high:
  - Anticoagulation can be stopped the day before surgery and reversed with vitamin K or fresh frozen plasma (FFP).
  - IV heparin therapy can then be instituted 12–24 hours postoperatively once surgical hemostasis is deemed to be adequate.

MS on physical exam:

Rumbling diastolic murmur.

Specific Valvular Disorders

Mitral Stenosis (MS)

Preoperative Considerations

- Almost always a delayed complication of rheumatic fever.
- Two-thirds of cases occur in females.
- Progressive fusion and calcification of valve leaflets → stenosis.
- Process begins minimum of 2 years from acute disease and is symptomatic after 20–30 years.
- Isolated MS < 50%.
- MS + mitral regurgitation (MR) > 50%.
- Aortic valve involved in 25%.

Pathophysiology

- Rheumatic process causes valve leaflets to thicken, calcify, and become funnel shaped.
- Transvalvular pressure gradient depends on:
  - Cardiac output (CO): ↑ CO → ↑ flows → ↑ gradient.
  - Heart rate (HR): ↑ HR → ↓ diastolic time → ↑ flows → ↑ gradient.
  - Atrial kick: Loss of normal atrial kick → ↑ flows → ↑ gradient.
- A dilated left atrium (LA) promotes supraventricular tachycardia (SVT), particularly atrial fibrillation.

- Blood flow stasis promotes thrombi formation in the LA appendage.
- Acute ↑ LA pressure is transmitted back to pulmonary capillaries → pulmonary edema.
- Chronic elevations in pulmonary capillary pressure are partially compensated by ↑ in pulmonary lymph flow and eventually result in pulmonary vascular changes → irreversible ↑ in pulmonary vascular resistance (PVR) → pulmonary HTN.
- Chronic dyspnea secondary ↓ lung compliance from ↑ work of breathing.
- ↑ right ventricular (RV) afterload → RV failure → marked dilatation of the RV → tricuspid regurgitation (TR) and pulmonary regurgitation (PR).

MS is associated with AF and emboli.

Common Complications

- Embolic events:
  - Due to dislodgement of clots from LA.
  - Common with MS and AF.
  - Cerebral vascular accidents—most common embolic event.
  - Pulmonary emboli.
- Hemoptysis: Rupture of pulmonary-bronchial venous communications.
- Chest pain: 10–15%.
  - Emboli to the coronary circulation.
  - Acute RV pressure overload.
- Hoarseness: Compression of left recurrent laryngeal nerve by the enlarged LA.
- Impaired LV function in 25%:
  - Residual damage from rheumatic myocarditis.
  - Coexistent HTN.
  - Coexistent ischemic heart disease.

Mitral Valve Area

- 4–6 cm^2: Normal.
- 1.5–2 cm^2: Asymptomatic/mild symptoms with exertion.
- 1–1.5 cm^2: Symptomatic with mild to moderate exertion.
- < 1 cm^2: Critical MS; dyspnea with minimal exertion with transvalvular gradient of 20 mmHg at rest; CO, although normal at rest, will fail to ↑ appropriately during exertion due to ↓ ventricular preload.

Treatment

- Time from onset of symptoms to incapacitation averages 5–10 years, death in 2–5 years.
- Medical management: Primarily supportive.
  - Limitation of physical activity.
  - Sodium restriction.
  - Diuretics.
  - Digoxin: AF with fast ventricular response.
  - β blocker to control HR in patients with mild to moderate symptoms.
  - Anticoagulants: History of emboli, > 40 years, large LA with chronic AF. MS is associated with AF and emboli.
- Surgical management:
  - MV replacement.
  - Percutaneous valvuloplasty:
    - Improves symptoms temporarily.
    - Pregnant patients or poor surgical candidates.

MS anesthetic goals: Avoid ↑ HR, ↓ SVR, and ↑ PVR.

Anesthetic Management

- Hemodynamic goals:
  - Rate: Avoid ↑ HR (can ↓ LV filling time) and ↓ HR (can ↓ CO); avoid pancuronium and ketamine; deepen anesthesia or give β blockers for tachycardia.
  - Rhythm: NSR; diltiazem or digoxin for AF.
  - Preload: Maintain; avoid hypovolemia and fluid overload.
  - Afterload: Maintain.
  - Contractility: Do not depress.
  - PVR: Avoid factors that ↑ PVR (hypoxia, hypercarbia, acidosis); avoid nitrous oxide.
- Choice of agents:
  - Epidural preferable to spinal due to more gradual onset of sympathetic blockade.
  - Volatile anesthetics can cause undesirable vasodilatation and junctional rhythms. Halothane is most suitable (↓ HR and least vasodilating).
- Monitoring:
  - Full hemodynamic monitoring in major surgical procedures (with large fluid shifts): intra-arterial pressure and pulmonary artery pressure monitoring.
  - ECG: Notched p wave.
  - Pulmonary capillary wedge pressure (PCWP) reflects the transvalvular pressure gradient (not the LV end-diastolic pressure [LVEDP]).
  - PCWP waveform: Prominent "a" waves, ↓ "y" descent.
- Intraoperative tachycardia:
  - Deepen anesthesia with opioid (not meperidine).
  - β blockers: Esmolol/metoprolol.
  - AF: Diltiazem or digoxin (ventricular rate control).
  - Verapamil may cause vasodilatation.
  - Cardioversion if hemodynamic deterioration.
  - Vasopressor: Phenylephrine preferred over ephedrine; phenylephrine lacks β-agonist activity.

Mitral Regurgitation (MR)

Preoperative Consideration

- **Acute MR:**
  - MI (papillary muscle dysfunction or rupture of chordae tendinae).
  - Infective endocarditis.
  - Chest trauma.
- **Chronic MR:**
  - Rheumatic fever.
  - Congenital anomalies of valve apparatus.
  - Dilatation, destruction, calcification of mitral annulus.

Pathophysiology

- Principal derangement: Reduction in forward SV due to backward flow of blood into LA during systole.
- Compensatory mechanism: LV dilates → ↑ LV end-diastolic volume (LVEDV); maintains normal CO, even as ejection fraction (EF) ↓.
- Eventually, "eccentric" ventricular hypertrophy → progressive impairment of contractility → ↓ EF.
- Severe MR: Regurgitant volume > forward stroke volume (SV).

- Regurgitant volume depends on:
  - Size of MV orifice.
  - HR (systolic time), (↑ HR → ↓ regurgitant volume).
  - LV–LA pressure gradient during systole:
  - SVR (↓ SVR → ↓ regurgitant volume).
  - LA compliance (↑ LA pressure → ↓ regurgitant volume).
- Clinical manifestations depend on atrial compliance:
  - Acute MR: Normal/↓ LA compliance → pulmonary vascular congestion and pulmonary edema.
  - Chronic MR: Large dilated LA/↑ LA compliance → ↓ CO.
  - Most patients exhibit symptoms of both.
- Regurgitant fraction and symptoms:
  - < 30%: Mild.
  - 30–60%: Moderate.
  - > 60%: Severe.

MR on physical exam: Holosystolic murmur at left sternal border.

Treatment

- Medical:
  - Digoxin, diuretics, vasodilators (ACE inhibitors).
  - Afterload reduction ↓ SVR → ↑ forward SV → ↓ regurgitant volume.
- Surgical:
  - Mitral valve replacement (MVR): For moderate to severe symptoms.

Anesthetic Management

- Hemodynamic goals:
  - Rate: normal—mild ↑ HR 80–100 bpm (↓ HR → ↑ regurgitant volume/↓ CO/↑ LVEDV).
  - Rhythm: Atrial fibrillation common.
  - Preload: Maintain; avoid excessive fluids (→ ↑ regurgitation volume).
  - PVR: Avoid factors that ↑ PVR (hypoxia, hypercarbia, acidosis); avoid nitrous oxide.
- Choice of agents:
  - Good ventricular function:
    - Tolerates most anesthetics.
    - Tolerates spinal and epidural.
  - Moderate to severe ventricular impairment:
    - Opioid-based anesthetic.
    - Use pancronium (causes tachycardia).
    - Avoid volatile anesthetics (cause myocardial depression).
- Monitoring:
  - Based on severity of ventricular dysfunction and procedure.
  - PCWP waveform: Large "v" wave, rapid "y" descent.
  - Color-flow Doppler transesophageal echocardiogram (TEE): Blood flow reverses in the pulmonary veins during systole in severe MR.

MR anesthetic goals: ↑ HR and ↓ SVR. Keep faster/fuller/vasodilated.

Aortic Stenosis (AS)

The most common cardiac valve lesion in the United States.

Causes

- **Congenital AS:**
  - Single most common congenital heart lesion.
  - Bicuspid aortic valves:

- Affect 1–2% of population.
- Affects mostly males.
- Present at birth (without problems).
- Cause problems in fifth to sixth decades.

- Abnormal valve cusp → turbulent flow → endothelial damage → inflammation and fibrosis → stenosis, infection, regurgitation.
- Associated with vascular abnormality → medial degeneration → dilatation, aneurysm, dissection.

- **Acquired AS:**
  - ↑ incidence with ↑ age.
  - Results from active inflammatory process similar to atherosclerotic vascular disease.
  - May be prevented by HMG-CoA reductase inhibitors.

AS on physical exam: Systolic ejection murmur.

Pathophysiology

- AV obstruction (slowly over years) → ↑ LV pressure → concentric hypertrophy (↑ wall thickness); maintains LV output for many years without dilatation or symptoms.
- Severe AS: Systolic gradient > 50 mmHg, aortic valve area < 0.8 cm^2.
- Maintainance of sinus rhythm is fundamental:
  - Ventricle: Hypertrophy → ↓ compliance → ↑ LVEDP.
  - ↑ dependence on atrial contraction for ventricular filling.
  - Loss of sinus rhythm → precipitous onset of symptoms.

Clinical Presentation

- Usually asymptomatic, clinically compensated for several decades.
- Classic triad:
  - Angina.
  - Dyspnea.
  - Syncope.
- Causes of MI in patients with AS without CAD:
  - ↓ myocardial oxygen supply:
    - ↓ aortic pressure.
    - ↑ LVEDP.
    - ↓ diastolic time.
  - ↑ myocardial oxygen demand:
    - ↑ LV mass.
    - ↑ LV systolic pressure.
    - ↑ LV ejection time.
- Causes of dyspnea:
  - LV dilatation and decompensated ventricles.
  - ↓ CO.
  - ↓ SV.
  - ↓ LV–aortic pressure gradient.
  - ↑ atrial and pulmonary pressures (diastolic failure).
- Causes of sudden pulmonary edema in patients with preserved LV systolic function: LV hypertrophy → ↑ muscle mass and ↑ muscle stiffness → ↑ chamber size → diastolic stiffness → pulmonary edema.

Prognosis and Treatment

- Angina: 50% 5–year survival rate.
- Syncope: 50% 3–year survival rate.
- Dyspnea: 50% 2–year survival rate.

- Patients with severely ↓ EF and AS: 2% operative mortality.
- Aortic valve replacement (AVR): For moderate to severe symptoms.
- Percutateous valvuloplasty: For poor surgical candidates; improves symptoms temporarily.

Anesthetic Management

AS anesthetic goals: Maintain NSR and normal HR and avoid ↓ SVR.

- Hemodynamic goals:
  - Rate: Avoid ↑/↓ HR (to assist with LV diastolic filling).
  - Rhythm: Maintain NSR (to assist with atrial kick).
  - Preload: Maintain hydration (to facilitate LV filling).
  - Afterload: Maintain DBP (for CPP) and SVR (for coronary perfusion), avoid LV ischemia.
  - Contractility: Avoid ↓ contractility.
- Choice of agent: Neuraxial anesthesia is poorly tolerated.

Aortic Regurgitation (AR)

Causes

- Acute: Commonly due to endocarditis, trauma, or dissection.
- Chronic:
  - Develops slowly.
  - Commonly due to abnormalities of valve (ie, bicuspid valve) or aortic root.

AR on physical exam: Decrescendo diastolic murmur.

Pathophysiology

- Acute:
  - Lack of compensation (LV dilation or hypertrophy).
  - Rapid ↓ SV and ↑ LVEDP → pulmonary edema and hypotension.
- Chronic:
  - Volume overload of LV.
  - SV reduced secondary to regurgitant flow.
  - Regurgitant fraction is dependent on HR and diastolic pressure gradient across AV.
  - Progressive dilation of LV with chronic regurgitation → initial ↑ LV compliance → normal SV.
  - Eventual deterioration of LV function → ↓ SV and ↑ LVEDP.

Anesthetic Management

AR anesthetic goals: Mildly ↑ HR and ↓ SVR.

- Hemodynamic goals:
  - Rate: Mild ↑ HR between 80 and 100 bpm (→ ↓ regurgitant fraction); pancuronium is useful choice.
  - Rhythm: Atrial fibrillation common (but atrial kick not as important as in AS).
  - Preload: Maintain (to maintain SV).
  - Afterload: ↓ afterload (vasodilation → ↓ regurgitant fraction).
  - Contractility: Maintain (for SV).
- Anesthetic techniques:
  - Spinal and epidural anesthesia tolerated.
  - Opioid-based anesthetic more tolerated, especially with depressed LV function.

HOCM anesthetic goals: Avoid ↑ HR and HTN.

Hypertrophic Obstructive Cardiomyopathy (HOCM)

- Other terms: Idiopathic hypertrophic subaortic stenosis (IHSS), muscular subaortic stenosis.
- Dynamic outflow obstruction: Patients with ventricular hypertrophy without an obvious cause such as HTN/AS.
- Most common genetic cardiac disease.
- Incidence: 1 in 500.
- Most common cause of sudden cardiac death in children and adolescents.
- Systolic anterior motion (SAM) of MV anterior leaflet results in LV outflow tract obstruction.
- Clinically, usually a benign course but with a risk of LV outflow obstruction.

Anesthetic Management

- ↑ preload: Fluids; avoid hypovolemia and nitroglycerin.
- ↑ afterload: Phenylephrine; avoid nitroprusside, isoflurane, morphine, calcium channel blockers.
- ↓ contractility: β blockers; avoid calcium channel blockers, digoxin, isoproterenol.
- Monitoring is based on severity of LV outflow obstruction and surgical procedure.

CONGENITAL HEART DISEASE

Three main classifications:

1. Obstructive lesions.
2. Left-to-right shunts.
3. Right-to-left shunts.

Anesthetic goals in left-to-right shunt: ↓ SVR and ↑ PVR to minimize shunt.

Obstructive Lesions

- Congenital aortic stenosis.
- Coarctation of the aorta.
- Pulmonic stenosis.

Induction in left-to-right shunt: IV induction is slow. Inhalation induction is rapid if ↓ CO but unchanged if CO normal.

Predominantly Left-to-Right Shunts (Simple Shunts)

Anesthetic Management

- Induction:
  - IV induction is slow.
  - Inhalation induction is rapid if ↓ CO but unchanged if CO normal.
- Maintenance: Goal to ↓ shunt by:
  - ↓ SVR
  - PPV → ↑ PVR
- Complications:
  - Pulmonary HTN
  - Heart failure
  - Eisenmenger's syndrome

Atrial Septal Defects (ASDs)

- Types: Ostium secundum (most common), ostium primum, sinus venosus.
- Most children are minimally symptomatic.
- May have recurrent pulmonary infections.

Ventricular Septal Defects (VSDs)

- Most common congenital heart disease.
- Functional abnormality of VSD depends on size of the defect, PVR, presence/absence of other abnormalities.
- Small VSDs:
  - Usually close during childhood.
  - Treated medically and followed by ECG and echocardiogram.
- Large VSDs:
  - Vary directly with SVR and indirectly with PVR.
  - Recurrent pulmonary infections and CHF.
  - Treatment: Surgical treatment before pulmonary vascular disease and Eisenmenger's physiology develop.

AV Septal (Endocardial Cushion) Defects

- Common lesion in patients with Down's syndrome.
- Contiguous ASD and VSD with very abnormal AV valves → large shunts at both levels.
- MR and TR → ↑ volume load on the ventricles → pulmonary HTN → Eisenmenger's syndrome.

Patent Ductus Arteriosus (PDA)

- Persistence of communication between the main pulmonary artery and the aorta → left-to-right shunts.
- Responsible for cardiopulmonary deterioration in premature infants.
- PDA may need to remain patent in certain cases of right-to-left shunt (pulmonary atresia, tetralogy of Fallot, coarctation) and liver transplant/failure.
- Place arterial line or pulse oximetry in right upper extremity.
- Factors that close PDA:
  - ↑ Pa_{O_2}
  - Prostacyclin
  - Indomethacin
- Factors that maintain PDA:
  - Hypoxia
  - Hypercarbia
  - Acidemia
  - Prostaglandin E

Partial Anomalous Venous Return

- One or more pulmonary veins, usually from the right lung, drains into the right side of the heart.
- Anomalous entry sites: RA, superior vena cava (SVC), inferior vena cava (IVC), coronary sinus.

Factors that close PDA:

↑ Pa_{O_2}

Prostacyclin

Indomethacin

Factors that maintain PDA:

Hypoxia

Hypercarbia

Acidemia

Prostaglandin E

- Results in variable left-to-right shunting.
- TEE: Large coronary sinus is a sign of anomalous drainage.
- Complicates management of cardioplegia.

Predominantly Right-to-Left Shunts (Complex Shunts)

- Both ventricular outflow obstruction and shunting.
- Mild shunting: Depends on ratio of SVR to PVR.
- Severe shunting: Direction and magnitude of the shunt is fixed.
- Cyanosis is not relieved with ↑ Fio_2.
- Survival depends on another shunt in the opposite direction.
- Lesions with ↓ pulmonary blood flow:
  - Tetralogy of Fallot.
  - Pulmonary atresia.
  - Tricuspid atresia.
- Lesions with ↑ pulmonary blood flow:
  - Transposition of the great vessels.
  - Truncus arteriosus.
  - Single ventricle.
  - Double-outlet RV.
  - Total anomalous pulmonary venous return.
  - Hypoplastic left heart.

Induction in right-to-left shunts:
IV induction is rapid.
Inhalation induction is slow.

Anesthetic Management

- Induction:
  - IV induction is rapid.
  - Inhalation induction is slow.
- ↑ SVR.
- Avoid crying.
- Avoid hypoxia/catecholamines.

Tetralogy of Fallot (TOF)

Physiology

- Four components:
  1. RV obstruction
  2. RV hypertrophy
  3. VSD
  4. Overriding aorta
- RV obstruction:
  - ↑ by sympathetic tone; causes hypercyanotic spells in very young patients.
  - RV obstruction + VSD: Unoxygenated RV blood + oxygenated LV blood → aorta.
- Right-to-left shunting has two components: Fixed component → RV obstruction; variable component → SVR and PVR.

Hemodynamic goal in right-to-left shunts: ↑ SVR.

Treatment

- Medical: IV prostaglandin E_1 (PGE_1) to prevent ductus arteriosus (DA) closure.

- Surgical:
  - Palliative: Modified Blalock-Taussig shunt (left subclavian artery–pulmonary artery) to ↑ pulmonary blood flow.
  - Complete correction: Closure of VSD, removal of obstructing infundibular muscle, pulmonic valvotomy, valvoplasty.
- Anesthetic considerations:
  - Maintain intravascular volume.
  - Maintain SVR.
  - Avoid ↑ in PVR, acidosis, ↑ airway pressures.
  - Avoid histamine-releasing muscle relaxants.
  - Ketamine: Good option, maintains/↑ SVR.
- Management of hypercyanotic spells:
  - IV fluids.
  - Phenylephrine: ↑ SVR, ↓ right-to-left shunt, ↑ pulmonary blood flow.
  - Propranolol: Relieves infundibular spasm, ↑ pulmonary blood flow.
  - Sodium bicarbonate: Treats acidosis.

Tricuspid Atresia

Physiology

- Blood flows out of RA via ASD or patent foramen ovale (PFO).
- Blood flows from LV to pulmonary circulation through VSD or PDA.
- Cyanosis is seen at birth; severity is dependent on the pulmonary blood flow.

Treatment

- PGE_1 infusion keeps DA open.
- Percutaneous Rashkind balloon septostomy (RA to LA).
- Modified Blalock-Taussig shunt: Severe cyanosis.
- Modified Fontan procedure: RA to right PA.
- Bidirectional Glenn shunt: SVC to main PA.
- Heart transplantation: Failed Fontan procedure.
- Anesthetic considerations:
  - After corrective procedure, blood from the systemic veins → LA.
  - ↑ systemic venous pressure: ↑ VR.
  - ↓ PVR: Avoid hypoxia, hypercarbia.
  - ↓ LA pressure.

Transposition of the Great Arteries

Physiology

- Pulmonary and systemic VR flow normally back to the RA and LA, respectively.
- Aorta arises from the RV, and PA from the LA, respectively.
- Deoxygenated blood—systemic circulation; oxygenated blood—pulmonary circulation.
- Survival requires mixing of systemic and pulmonary circulations through a PFO, PDA, or VSD.

Treatment

- **Medical:** PGE_1 infusion.
- **Surgical:**
  - Palliative: Rashkind septostomy.
  - Corrective: Arterial switch procedure.
    - Aorta reanastamosed to LV.
    - PA reanastamosed to RV.
    - Coronary arteries reimplanted into old PA root.
    - VSD closed.
    - Atrial switch (Senning procedure): Intra-arterial baffle created from the atrial wall and blood from pulmonary veins → ASD → RV → systemic circulation.

Truncus Arteriosus

Physiology

- Single arterial trunk supplies both systemic and pulmonary circulations.
- VSD present.
- After birth, ↓ PVR → ↑ pulmonary blood flow → heart failure.
  - If untreated, ↑ PVR → Eisenmenger's syndrome.

Treatment

- Rastelli repair: Closes the VSD, separates PA from the truncus, connects RV to PA with a conduit.
- Anesthetic considerations: Prevent right-to-left shunt.

Hypoplastic Left Heart Syndrome

Physiology

- Marked underdevelopment of the LV.
- RV is the major pumping chamber for both systemic and pulmonary circulations.
- Blood from RV → PA through PDA → aorta.
- Almost all the blood in the aorta comes from the PDA.
- Associated with other major noncardiac congenital anomalies.

Treatment

- Norwood procedure.
- Cardiac transplantation.

CARDIAC TRANSPLANTATION

Preoperative Considerations

- Most common indications: Ischemic and dilated cardiomyopathies.
- Transplantation criteria:
  - Absence of extensive end-organ damage.
  - Absence of other major systemic illnesses.
  - Reversible renal and hepatic dysfunction (common due to chronic hypoperfusion and venous congestion).
  - PVR normal or at least responsive to oxygen/vasodilators.

- Contraindications: Significant atherosclerotic disease, renal/hepatic diseases (immunosupressants can affect renal and hepatic function), irreversible pulmonary HTN.
- NYHA Class IV patients are often on maximal medical therapy with an EF < 20%, and some may have left ventricular assist devices (LVADs).

Preoperative Management

- NPO status should be reviewed, and rapid-sequence induction should be considered.
- Immmunosupressants are begun preoperatively.
- PA catheterization should be considered in the preoperative period.

Intraoperative Management

- Induction can be challenging as patients have poor cardiac reserve and ongoing hemodynamic instability.
- Slow induction utilized with etomidate, ketamine, and opioids.
- TEE examination evaluates the native and donor hearts.
- After sternotomy, cardiopulmonary bypass (CPB) is initiated with bicaval and aortic cannulation.
- The PA catheter is pulled back into the sheath and the native heart is excised, leaving the posterior atrial remnants in place.
- The donor atria are anastamosed followed by the anastamosis of the aorta and pulmonary arteries.
- Intracardiac air is evacuated and methylprednisolone is given prior to the release of the aortic cross-clamp.
- Inotropic support is initiated before separation from CPB: Isoproteronol is used often for direct β_1 agonist activity as the donor heart is denervated and will not respond to indirect agents.
- Epicardial pacing may be necessary after separation from bypass.
- Treatment of preexisting pulmonary HTN with acute right ventricular failure: Inhaled NO, prostaglandin E_1, or a right ventricular assist device can $\downarrow$ pulmonary vascular resistance.

Postoperative Considerations

- Patients are kept intubated and slowly weaned for extubation.
- Postoperative complications: Acute graft rejection, bleeding, renal dysfunction, infection.

Patient with a Transplanted Heart

Characteristics of the Transplanted Heart

- Totally denervated.
- No direct autonomic influence.
- Normal response to circulating catecholamines.
- Postoperative course:
  - Acute rejection.
  - Renal/hepatic dysfunction.
  - Infection.
- Normal cardiac impulse formation and conduction.
- High resting HR: 100–120 bpm, due to absence of vagal influences.
- Response to circulating catecholamines: Normal or enhanced due to denervation sensitivity $\rightarrow \uparrow$ receptor density.

Cardiac transplant patients have a resting tachycardia with two p waves.

- Partial reinnervation may occur in some patients after some time.
- Cardiac output:
  - Low-normal.
  - ↑ relatively slowly in response to exercise.
  - Response is dependant on an ↑ in circulating catecholamines.
- Preload dependent.
- Coronary autoregulation is preserved.

Preoperative Considerations

- Present early in the postop period for mediastinal exploration or retransplantation.
- Present later for incision and drainage of infections, orthopedic surgery, or unrelated procedures.
- Evaluate the functional status of the transplanted organ.
- Detect complications of immunosuppression.
- Rejection:
  - Highest incidence within first 3 months.
  - Rejection rates: One per patient-year.
  - Early signs of rejection: Arrhythmias (first 6 months), ↓ exercise tolerance.
- Monitoring:
  - Endomyocardial biopsy: Most reliable technique.
  - Periodic echocardiogram commonly used.
- Coronary atherosclerosis:
  - Very common and serious.
  - Myocardial ischemia/infarction: Silent; due to denervation.
  - Periodic evaluations include angiography.
- Immunosuppressive therapy:
  - Cyclosporine, azathioprine, prednisone.
  - Side effects: Nephrotoxicity, hepatotoxicity, bone marrow suppression, opportunistic infections, osteoporosis.
  - Stress doses of corticosteroids for major surgery.
- HTN and fluid retention: Diuretic and ACE inhibitor.

Anesthetic Management

- All anesthetic techniques including RA used.
- Maintain normal or high cardiac preload.
- Sensitive to rapid vasodilatation (absence of reflex ↑ in HR).
- Indirect vasopressors (eg, ephedrine and dopamine) are less effective due to absence of catecholamine stores in myocardial neurons.
- Heart rate:
  - To ↑ HR, use isoproterenol, dilute epinephrine (10 μg/mL).
  - Opioids and cholinesterase inhibitors do not cause bradycardia.
  - Anticholinergics, pancuronium, meperidine do not cause tachycardia.
  - Anticholinergic is still given during reversal to block the muscarinic side effects of acetylcholine.
  - ECG: Two sets of P waves—recipient's own SA node + donor's SA node.
- Major operations: Direct arterial, central venous, PA pressure monitoring required; strict asepsis during line placement.

Cardiac Tamponade

Causes

- Hemorrhagic pericarditis caused by:
  - Aortic dissection.
  - Ventricular free wall rupture after MI.
  - Anticoagulant-induced hemopericardium.
  - Trauma (stab wounds, central venous catheter).
- Uremic pericarditis:
  - Neoplastic pericarditis (mesothelioma, lymphoma).
  - Serous pericarditis (rheumatoid disorders, irradiation, viral infection).

Pathophysiology

- Primary determinants of physiological impairment:
  - Rapidity of fluid accumulation.
  - Pericardial sac compliance.
- Acute accumulation of small volume → noncompliant compartment → rapid cardiovascular collapse.
- Slow accumulation of large volume → compliant compartment → well tolerated.
- Accumulation of fluid → cardiac chamber collapse → limited SV.
- Compensation through vasoconstriction to maintain venous return and tachycardia to maintain CO.
- Clinical manifestations: Dyspnea, orthopnea, tachycardia, paradoxical pulse, hypotension.
- RA, RV, LV diastolic collapse on TEE.

Anesthetic Considerations

- Avoid vasodilators and myocardial depressants.
- Ketamine is good option.

Tamponade management:

Full, tight, fast.

Constrictive Pericarditis

Causes

- Idiopathic.
- Viral.
- Radiation therapy.
- Post cardiac surgery.
- Post MI → Dressler's syndrome.
- Connective tissue disorders.
- Renal failure.

Pathophysiology

- Diastolic filling of the heart is restricted.
- Pericardial calcification, pericardial effusion.
- Myocardial involvement: Myocardial atrophy → systolic dysfunction.
- Coronary involvement: Scar-induced compression of coronary arteries.

Symptoms/Signs

- ↓ cardiac output: Fatigue, malaise.
- ↓ VR to the left heart: Dyspnea, cough.
- ↓ VR to the right heart: Enlarged liver, ascites.
- Equalization of diastolic intracavitary pressures.
- Pronounced ↓ in pressure in early diastole: Pronounced y descent on central venous pressure (CVP): "Square root sign."

Primary Cardiac Tumors

Primary cardiac tumors are rare: Benign are more common than malignant. Myxomas are the most common benign tumors; 85% of these occur solitarily in the LA.

Clinical Manifestations

- Nonspecific systemic features:
  - Mediated by interleukin-6 (IL-6) synthesis by tumor tissue.
  - Fever, malaise, cachexia, arthralgia, rash, behavioral changes.
- Thromboembolic phenomenon:
  - Due to the intracavitary location and the friable nature.
  - Left-sided tumor → systemic emboli.
  - Right-sided tumor → pulmonary emboli.
- Local cardiac effects depend on specific location.
  - Myocardial tumors: Conduction disturbances.
  - LA myxoma: Most common tumor; mimics MS; dyspnea on exertion, orthopnea, cough, hemoptysis.

ANESTHETIC MANAGEMENT FOR CARDIAC SURGERY

Preoperative Evaluation

- Surgical procedure: Indication for surgery and the determination of the pathophysiologic implications of the underlying disease process.
- Risk factors for cardiovascular disease and their implications for comorbid disease (eg, cigarette use, HTN, diabetes).
- Functional status according to NYHA classification.
- Specialized investigative data:
  - Ischemia:
    - Exercise stress tests.
    - Myocardial perfusion imaging (rest, exercise, pharmacologic).
    - Angiography.
  - Myocardial function/valve function:
    - Hemodynamics: CO, pressure gradients, valve areas.
    - Ventriculography.
    - Echocardiography.

- Systemic disease:
  - Cerebrovascular disease (duplex studies): There are as yet no clear data to indicate if outcome in patients with significant cerebrovascular and coronary disease is optimized by performing combined carotid endarterectomy and coronary artery bypass graft (CABG) or sequential surgeries in either order.
  - Atherosclerotic aortic disease.
  - Renal impairment.
  - COPD.

Anesthetic Agents

- Hemodynamic dictates of the patient's underlying cardiac condition include:
  - HR control.
  - Coronary perfusion pressure requirements.
  - Myocardial oxygen supply/demand determinants.
  - Effects on LV function.
- Balanced anesthetic technique most frequently used.
- Opioids: Core component.
  - Stable hemodynamics.
  - Vagotonic-induced bradycardia.
- Low/moderate doses of volatile anesthetics:
  - Lack of consciousness, amnesia.
  - Induce preconditioning: Potentially extremely important phenomenon in patients either certain (CPB with aortic cross-clamping) or likely (off-pump coronary artery bypass [OPCAB], manipulation-associated hypotension and ↓ coronary perfusion pressure, occlusion of coronary vessels for distal anastomosis) to be subjected to myocardial (and perhaps neurologic) ischemic insults.
- Benzodiazepines: Central component. Midazolam—minimal effects on coronary blood flow autoregulation.
- Avoid N_2O:
  - Undesirable effect on pulmonary vasculature and myocardial function.
  - Ability to ↑ gaseous bubble size.
- Propofol: Not a primary induction agent; → hypotension. ↓ in SVR, mild myocardial depression.

Regional Anesthetic Techniques

- Intrathecal and epidural anesthetic techniques have desirable effects on:
  - Stress response.
  - Hemodynamics.
  - Coronary perfusion pressure.
  - Myocardial blood flow redistribution.
  - Potential early extubation.
  - Certain patients with compromised pulmonary function (COPD).
- Disadvantages:
  - Risk of hematoma formation in the neuraxis with anticoagulation.
  - Specific side effects: Hypotension, pruritis, etc.

CPB Circuitry

The CPB pump has several features (see Figure 11-1):

- Arterial cannula: In ascending aorta.
- Venous cannula:
  - One or two cannulas in RA or RA and SVC.
  - Femoral venous cannulation: Temporizing to decompress the heart during repeat high-risk surgery.
- Reservoir:
  - Receives blood from cannulas in RA or SVC/IVC.
  - Flows to reservoir via gravity.
  - Air embolism can occur if fluid levels run low.
- Oxygenator:
  - Receives blood from reservoir.
  - Blood equilibrates with oxygen-containing gas mixture.
- Heat exchanger:
  - Receives blood from oxygenator.
  - Heat transfer via conduction.
  - Filter to catch bubble that may arise from rewarming as gas solubility ↓ as temperature rises.
- Pump:
  - Roller pump:
    - Produces flow by compressing large bore tubing.
    - Continuous pulsatile flow.
    - Causes RBC trauma.
  - Centrifugal pump:
    - Cones spin and propel blood from central inlet to periphery.
    - Flow is pressure sensitive.
    - Less traumatic to RBCs.
  - Pulsatile flow: Possibly improves tissue perfusion, attenuates response of stress hormones, enhances O_2 extraction.
- Arterial filter: Prevents systemic embolism from thrombi/fat/calcium/tissue debris.

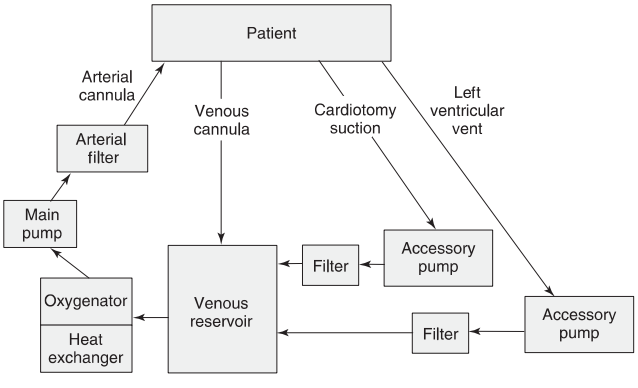

FIGURE 11-1. The basic design of cardiopulmonary bypass machine.

(Reproduced, with permission, from Morgan GE et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006: 492.)

- Cardiotomy suction:
  - Aspirates blood from surgical field and returns it to reservoir.
  - Excessive pressure can damage RBCs.
- Left ventricular vent:
  - Catheter inserted into LV via right superior pulmonary vein and LA to decompress blood accumulation in LV from residual pulmonary flow (via bronchial arteries, thebesian veins, and pericardial collateral flow).
  - Distention of the LV is a risk during CPB with aortic cross-clamping.
  - LV distention → subendocardial myocardial ischemia.
- Monitors and safety devices:
  - In-line filters and bubble traps.
  - In-line blood gas monitors.
  - Pressure and flow measurement mechanisms.
  - Oxygen and gas flow measurement devices.
  - Temperature monitoring.
  - Low-level sensor detectors on the venous reservoir.

CPB Complications

- ↑ aortic pressure secondary to occlusion, small or kinked cannula, improper cannula positioning (in innominate), or aortic dissection.
- ↓ venous return secondary to occlusion, bleeding, or aortic dissection.
- Hypotension secondary to pump malfunction, ↓ venous return, ↓ peripheral resistance (from ↓ Hct or vasodilation).
- HTN secondary to excessive flow or inadequate anesthesia.

Anticoagulation for CPB

- Anticoagulation necessary during CPB to prevent clot formation in pump and DIC.
- Heparin remains the only drug available for anticoagulation of patients for CPB.
- Negatively charged mucopolysaccharide that has no anticoagulant properties by itself.
- Mechanism of action: Heparin binds to antithrombin III (AT–III) and potentiates its effect. AT–III binds to thrombin and the complex is rapidly removed from the circulation. AT–III also binds other factors in the coagulation cascade, such as factors Xa, IXa, XIa, and XIIa, but the potentiating effect of heparin on these factors is much less potent (10– to 100–fold) than that on the AT–III/thrombin reaction.
- Efficacy is inversely related to molecular weight of heparin.
- Humans differ from one another in the levels of AT–III and other coagulation factors present in their serum. Also, the levels of AT-III in the same patient may change over time, in the presence of disease, and in the presence of medications, including heparin. As AT–III is produced by the liver, ↓ AT–III levels in the presence liver disease.
- If heparin fails to achieve adequate anticoagulation, AT-III levels may be supplemented by FFP, AT-III concentrate, or recombinant AT-III.
- Administration:
  - Dose: Heparin 300–400 IU/kg pre-CPB.
  - Effective within one circulation time.
  - Side effects: Uncommon. Allergic reactions, histamine release, calcium chelation.

ACT of 400–450 is acceptable to initiate CPB.

- **Activated clotting time (ACT):**
  - Relationship between heparin dose and ACT is linear (up to 600s). ACT values of 400–450s are acceptable initiate CPB.
  - ACT uses diatomaceous earth (celite) or aluminum silicate (Kaolin) to activate the intrinsic cascade. Celite-based ACT is prolonged by aprotinin but not kaolin–based ACT.

Antifibrinolytics

- Lysine analogues (ε-aminocaproic acid, tranexamic acid):
  - Mechanism of action: Binds to lysine-binding sites on plasminogen and fibrinogen, inhibiting plasminogen activator and plasmin release.
  - Beneficial effects during CPB: Inhibits fibrinolysis, ↓ mediastinal bleeding, ↓ transfusion requirements.
- Aprotinin:
  - Nonspecific protease inhibitor.
  - Inhibitory action on intrinsic coagulation cascade, complement activation, fibrinolysis, bradykinin, kallikrein formation.
  - May induce antibody formation, immunoglobulin G (IgG) antibody.
  - May inhibit endothelial nitric oxide synthase in the coronary circulation—harmful.
  - May ↓ stroke rate—beneficial.
  - Taken off market in 2007 after concerns that it ↑ risk of death due to thrombosis when used to prevent bleeding in heart surgery.

Pathophysiology of CPB

- CPB elicits a rapid and profound inflammatory response that is highly complex and involves several interactive and cellular and humoral pathways.
- The spectrum of this inflammatory response ranges from less severe systemic inflammatory response syndrome (SIRS) to severe multiple organ dysfunction syndrome (MODS).
- Several host (eg, diabetes mellitus), environmental (eg, nosocomial infections), and intraoperative (eg, gut translocation) factors as well as CPB itself may predispose patient to infection.
- Several preoperative factors and patient-related conditions (eg, active ischemia, impaired ventricular function, diabetes) may modulate the host inflammatory response while CPB itself evokes a profound, multifaceted, complex inflammatory host response.
- The inflammatory response to CPB is initiated immediately when the patient's blood is exposed to the CPB tubing and reservoir. Ischemia—reperfusion injury during CPB also contributes.
- Specific pathways involved in response to CPB:
  - Cellular immune activation with adhesion, margination, and translocation of granulocytes and macrophage activation. This is the central cellular response.
  - The complement pathway, specifically the alternative pathway.
  - The cytokine system with both proinflammatory and anti-inflammatory cytokine activation resulting in immune modulation.
  - Coagulation and fibrinolytic system activation.
  - Upregulation of inducible NOS.

- Activation of oxidant stress pathway resulting in the production of reactive oxygen species (ROS), including super oxide and hydroxyl radicals and peroxynitrite.
- Several commonly used anesthetics, including propofol, fentanyl, and midazolam, modulate one or more of the previously mentioned pathways.
- Pulsatile flow, membrane oxygenators, heparin-bonded circuits, and moderate hypothermia exert a favorable influence.

Myocardial Protection

- Broadly, refers to all interventions undertaken in the preoperative, intraoperative, and postoperative periods that optimize myocardial oxygen supply and demand.
- Narrowly, refers only to the interventions undertaken during CPB and specifically during the period of aortic cross-clamping.
- **Techniques:**
  - Systemic hypothermia.
  - Topical hypothermia (risk of phrenic nerve injury).
  - Cardioplegia.
  - Blood cardioplegia.
- **Goals:**
  - Minimize oxygen consumption.
  - Minimize consequences of oxygen deprivation during aortic cross-clamping.
- **Aortic cross-clamping:**
  - **Consequences:**
    - Ischemia during aortic cross-clamping.
    - Reperfusion injury at the end of aortic cross-clamping.
  - **Consequences of ischemia:**
    - Rapid depletion of adenosine triphosphate (ATP).
    - ↑ anaerobic glycolysis.
    - Accumulation of lactic acid.
    - Inability to maintain cellular ionic gradients.
    - Intracellular accumulation of osmotically active ions.
    - Cell swelling.
    - Eventual cell death.
  - **Consequences of reperfusion injury:**
    - Additional cellular injury.
    - Production of ROS.
    - Leukocyte activation.
    - Accumulation of intracellular calcium.
    - Manifestations: Dysrhythmias, stunned myocardium.
- **Cardioplegia:**
  - Antegrade: Follows normal cardiac circulation.
  - Retrograde: Delivered via coronary sinus.
    - Technically more difficult.
    - Delivered with pressures < 40 mmHg.
    - Preferred technique in aortic incompetence and severe coronary obstruction.
  - Through saphenous vein grafts on completion of distal anastamoses.
  - Duration, quantity, and frequency of cardioplegia administration varies from surgeon to surgeon.

Oxygen consumption (Vo_2) ↓ approximately 5–7% for every 1°C decrease.

- **Blood cardioplegia:**
  - Used in 80–85% of cardiac surgery procedures in the United States.
  - Retrograde approach is used alone or in combination in 45% of cases.
  - In patients with EF < 40%, blood cardioplegia is superior to crystalloid cardoiplegia as determined by enzyme leak, rhythm, and conduction abnormalities.

Management of CPB

- Temperature.
- Blood pressure, flow, flow patterns.
- Hematocrit and rheologic issues.
- Acid-base management.
- Glycemic control.

Temperature

- Most centers perform cardiac surgery with CPB using hypothermia at systemic temperatures ranging from 27°C to 32°C, with myocardial temperatures of 12°C to 15°C.
- The metabolic rate and temperature are directly but not linearly related.
- Oxygen consumption (Vo_2) ↓ approximately 5–7% for every 1°C decrease.
  - A 10°C decrement in temperature from 37°C (termed Q10) will cause a two- to threefold ↓ in oxygen consumption.
  - Hypothermia with lower Vo_2 allows lower flow rates on CPB, less myocardial collateral flow, and less myocardial rewarming.
- Temperature gradient between venous outflow blood and arterial inflow:
  - ↓ temperature → ↑ solubility of gases *and* ↑ temperature → ↓ solubility of gases.
  - ↑ temperature gradient → ↑ the rate at which gases come out of solution → ↑ bubble formation → thromboembolism.
  - Gradients should not exceed 10°C.
- During rewarming on CPB, abnormal pressure, flow, and flow patterns are present. ↑ in temperature → ↑ Vo_2 especially in well-perfused areas like the brain. Warming inflow blood to higher than 37°C may have deleterious neurologic complications (eg, stroke).

Pressure Flow and Flow Patterns

- Mean arterial pressure (MAP):
  - Most centers maintain MAP between 40 and 60 mmHg during CPB using nonpulsatile flow rates of 50–60 mL/kg.
  - Low MAP: 40–60 mmHg.
  - High MAP: > 80 mmHg.
- At any particular temperature, MAP and flow rates influence organ perfusion, including myocardial and cerebral perfusion.
- ↑ MAP → ↑ perfusion pressure → ↑ collateral flow → effects.
  - ↑ myocardial rewarming (harmful).
  - Obscuring surgical field (harmful).
  - ↑ blood flow distal to obstructed lesions (beneficial).
  - Higher MAP (80–100 mm Hg) significantly improves neurologic outcome: Used in patients with documented cerebrovascular disease.

- Pulsatile blood flow improves capillary perfusion, which improves tissue perfusion. Renal blood flow and urinary output seem better preserved.

Hematocrit and Rheologic Issues

- **Effect on oncotic pressure:** Initiation of CPB—use of crystalloid priming solutions → abrupt ↓ in hematocrit and oncotic pressures, which alters microvascular Starling's forces → efflux of fluid from the intravascular to extravascular compartments → generalized tissue swelling.
- **Effect on blood pressure:**
  - Initiation of CPB → ↓ in hematocrit → ↓ in viscosity → hypotension.
  - CPB progresses → progressive hypothermia, which ↑ viscosity → ↑ MAP.
- **Effect on oxygen-carrying capacity:** Large changes in hematocrit have only a modest influence on oxygen-carrying capacity. Hematocrit (Hct) 20% ↓ O_2 carrying capacity to only 90% of maximal. Hct values of 22–25% are usually well tolerated.

Alpha stat pH has an uncorrected P_{CO_2} and is associated with better neurologic outcomes.

Acid-Base Management

- pH of neutrality of water, the pKa of buffer systems and the pH of blood are all inversely related to temperature.
- Hypothermia ↑ the pKa of the imidazole buffer system.
  - **Alpha stat:** No CO_2 is added.
  - **pH stat:** CO_2 added to keep pH 7.4.
- Alpha stat is associated with better preservation of cerebral autoregulation, metabolic coupling, and enzyme activity.
- Alpha stat → lower P_{CO_2}. Cerebral blood flow (CBF) is actually ↓, which ↓ cerebral emboli → improved neurologic outcomes.
- However, may impede brain cooling, which is important with deep hypothermic circulatory arrest. Use pH stat during cooling, add CO_2 to improve CBF, then revert back to alpha stat approach.

Glycemic Control

- Hyperglycemia exacerbates neurologic injury.
- Aggressive glucose homeostasis is associated with improved outcome in ICU patients.

Separation from CPB

Checklist before separation from CPB.

- Cardiac:
  - Surgical: Bleeding, valve function (TEE), intracardiac air (TEE), aorta (TEE, confirm no dissection).
  - Rate, rhythm (ECG).
  - Ischemia (ECG).
  - Myocardial function (visual observation, TEE, cardiac output/filling pressure).
- Temperature:
  - Temperature in high blood flow region > 37°C.
  - Temperature in low blood flow region > 35°C.

- Inflow/outflow temperature.
- Duration of rewarming.
- Hematocrit.
- Electrolytes, acid-base status.
- Ventilation, oxygenation (ability to ventilate both lungs, especially the left lung and its lower lobe).
- Inotropic support:
  - Low doses of selected inotropic agent are started to augment inotropy, chronotropy, and lusitropy during separation and after CPB.
  - The threshold for, the type of, and the magnitude of inotropic support depend on the magnitude of the myocardial insult.
  - Inotropic support also ↓ the dependency of cardiac output on preload and thus on fluid administration.
  - Importantly, inotropic support should not be started during the period of maximum, obligatory, oxygen debt that occurs immediately after removal of aortic cross-clamp.
- Rate and rhythm disturbances:
  - Bradycardia +/– varying degrees of heart block: Common after aortic cross-clamping, especially with surgery adjacent to the bundle of His, aortic valve surgery, and direct tissue injury. Treated with combination of pacing.
  - Ventricular dysrhythmias: Ventricular fibrillation most common.
    - Usually treated with lidocaine loading and electrical defibrillation.
    - Amiodarone for intractable dysrhythmias.
  - Atrial fibrillation: Common dysrhythmia, develops 2–4 days postoperatively.

1 mg protamine to neutralize 100 IU heparin.

Heparin Reversal

- Protamine is the only compound currently available to reverse heparin.
- Polycationic compound with arginine residues derived from fish that forms an ionic bond with the anionic residues of heparin.
- Binds only the "free" heparin in the circulation.
- Does not affect/reverse heparin bound to AT-III.
- Heparin rebound: Protamine has shorter half-life than heparin. Heparin bound to tissues reequilibrates into the circulation → post-CPB bleeding.
- Dosing:
  - Fixed-dose regimen: 1 mg protamine/100 IU heparin.
  - Heparin rebound: 0.3 mg protamine/100 IU heparin.
- Fixed-dose regimen does not take into account:
  - Hepatic and renal clearance of heparin.
  - Heparin–AT–III complex.
  - Dose-dependent side effects of protamine.
  - Contribution of excess protamine to post-CPB bleeding.
  - Hence, a minimal dose of protamine to reverse heparin, via a combination of ACT and heparin-protamine titration system, is used.
- Protamine reactions:
  - Systemic hypotension: Most common; protamine administered too rapidly → histamine release → hypotension → ↓ coronary perfusion pressure/MI/flushing.
  - IgE mediated: Dose-dependent allergic reactions are seen in patients with previous exposure who are sensitized to protamine.
  - Type III reactions: Protamine-heparin complex → release of thromboxane A_2 from platelets and macrophages → pulmonary HTN → right heart failure. Effect is attenuated by pretreatment with cyclooygenase (COX) inhibitors.

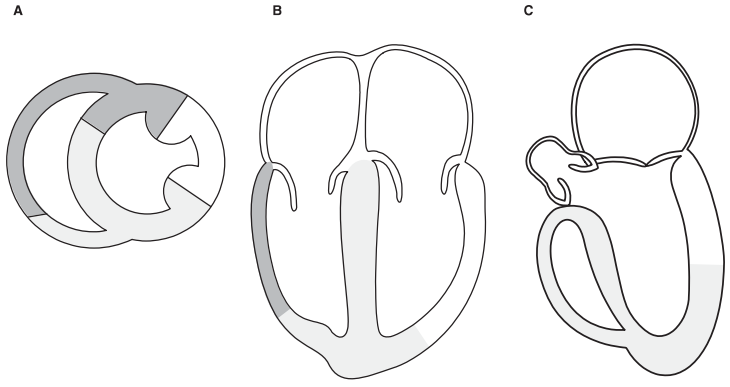

FIGURE 11-2. Coronary artery supply of the left and right ventricles in three views: The short-axis view (A), the four-chamber view (B), and the three-chamber view (C). Dark gray, RCA; light gray, LAD; white, CX.

(Reproduced, with permission, from Morgan GE et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006: 505.)

- Contributes to post-CPB bleeding:
  - Causes sequestration of platelets in the pulmonary circulation → egress of large platelets from the circulation.
  - Induces platelet shrinkage.

Common Problems After Cardiopulmonary Bypass

Hemodilution

Reasons for hemodilution:

- Crystalloids used to prime the CPB circuit → hemodilution and ↓ in concentration of platelets and coagulation factors at the initiation of CPB.
- Crystalloid cardioplegia → hemodilution at the initiation of CPB.
- Crystalloids to the pump to maintain safe reservoir volumes and safe target flows to the patient.
- Cell savage techniques → hemodilution and loss of coagulation factors and platelets.

Heparin, Protamine, Hypothermia, and Coagulopathy

Reasons for bleeding after CPB:

- Heparin rebound.
- Heparin-induced inhibition of platelet function.
- Protamine inhibition of thrombin at high concentration.
- Protamine-induced ↑ egress of large platelets from the circulation.
- Heparin + protamine → complement release → acute ↓ in platelets.
- Hypothermia → inhibitory effects on platelet function.

Myocardial Ischemia

- ECG changes based on coronary distribution (see Figure 11-2).
- Incidence: 60% of patients undergoing revascularization.

- Commonly seen with:
  - Aortic cross-clamping.
  - Anoxic cardiac arrest.
  - OPCAB grafting.
  - Minimally invasive procedures.
  - Open heart procedures (eg, valve replacement).
- Possible causes:
  - Vessel spasm (native coronary arteries, internal mammary artery).
  - Emboli (air, particulate).
  - Technical graft anastamotic issues.
  - Kink/clotting of grafts, native vessels.
  - Nongraftable vessels.
- Most common changes: ST and T wave changes, usually resolve; not associated with ↑ morbidity and mortality.
- May progress to Q-wave changes or high levels of enzyme leak → adverse outcomes.
- Regional changes are signs of local problem:
  - Kinked graft.
  - Anastamotic issue.
  - Embolic event.
  - Inadequate myocardial protection distal to a critical lesion.
- Generalized changes are signs of difficulties with myocardial protection.

Treatment

- Treat the cause, if found.
- Surgical: Redo an anastamosis.
- Medical: Treat arterial conduit spasm with a calcium channel blocker.
- Pharmacological: Optimize determinants of myocardial oxygen supply and demand.
- IABP counterpulsation.

Myocardial Dysfunction (Left/Right, Systolic/Diastolic Dysfunction)

Etiology

- Exacerbation of preexisting dysfunction and relative intolerance of anoxic arrest and aortic cross-clamping.
- Inadequate myocardial protection:
  - Role of underlying cardiac anatomy.
  - Route of cardioplegia administration.
  - Type of cardioplegia.
- Ischemia/infarction:
  - Vessel spasm (native coronary arteries, internal mammary artery).
  - Emboli (air, particulate).
  - Technical graft anastamotic issues.
  - Kink/clotting of grafts, native vessels.
  - Nongraftable vessels.
- Reperfusion injury.
- Unmasked ventricular dysfunction: Mitral valve replacement/repair with MR.
- Uncorrected lesions:
  - Hypertrophic cardiomyopathy
  - Valve gradients
  - Shunts

Management of LV Failure

- Identify the treatment course (if possible).
- Optimize forward flow:
  - Optimize heart rate, rhythm +/– pacing.
  - Manipulate loading conditions.
  - Manipulate inotropic conditions.
  - Manipulate lusitropic conditions.
  - Medications: Nitroglycerin, sodium nitroprusside, epinephrine, dobutamine.
- Treat acidosis.
- If RV failure:
  - Optimize specific afterload determinants: P_{O_2}, P_{CO_2}, pH, airway/intrathoracic pressures.
  - Nitrates, inhaled NO.
- Mechanical circulatory support systems.

Causes of RV Failure

- RV ischemia.
- Pulmonary HTN and elevated PVR.
  - **Pulmonary arterial HTN:**
    - Primary pulmonary hypertension: Sporadic, familial.
    - Related to collagen vascular disease, congenital systemic-to-pulmonary shunts, portal HTN, HIV infection, drugs/toxins, persistent pulmonary HTN of the newborn.
  - **Pulmonary venous HTN:** Pulmonary HTN associated with the disorders of the respiratory system and /or hypoxemia:
    - COPD.
    - Interstitial lung disease.
    - Sleep-disordered breathing.
    - Alveolar hypoventilation disorders.
    - Pulmonary HTN caused by chronic thrombotic and/or embolic disease.
- Altered interventricular dependence (eg, after placement of LV assist device).

Management of RV Failure

- Optimize preload (CVP 12–15 mmHg).
- Inotropic support.
- Afterload reduction:
  - Nonspecific: Optimize blood gases, pH, airway pressures.
  - Specific: Phosphodiesterase inhibitors, vasodilators, NO.
- Mechanical assist device.

Diastolic Heart Failure

- The inciting event in heart failure may be:
  - Cardiac (MI, valve disease).
  - Extracardiac (chronic ventricular overload).
- Progressive heart failure results from changes in multiple interrelated mechanisms involving hemodynamic, nonhemodynamic, neurohumoral, energetic and genetic factors.
- **Diastolic function:** Ability of the heart to fill.
- **Diastolic dysfunction:** Abnormal index of diastolic function.

- **Diastolic failure:** Associated clinical syndrome.
- Diastolic dysfunction and even diastolic failure are frequent perioperative developments after anoxic cardiac arrest.
- Diastolic dysfunction can occur with or without systolic dysfunction.

TREATMENT

- Alleviating symptoms: Pulmonary congestion with nitrates.
- Treating the cause: Ischemia.
- Manipulating other activated mechanisms: Renin-angiotensin system: ACE inhibitors, angiotensin receptor blockers, aldosterone antagonists.
- Calcium channel blockers: Contraindicated in systolic failure.
- β blockers: May be used acutely to ↓ HR, ↑ diastolic interval and ventricular filling (contraindicated in acute systolic failure).
- Natriuretic peptide analogues: Nesiritide.
- Arginine vasopressin antagonists.
- IABP counterpulsation: Used commonly.
- Mechanical circulatory support: LV assist devices (LVADs) and RV assist devices (RVADs).

Intra-aortic Balloon Counterpulsation

- Alternating inflation during diastole and deflation during systole of a helium-filled balloon positioned in the proximal descending aorta to left subclavian take-off.
- **Introduction site:**
  - Femoral artery: Most frequent.
  - Proximal descending aorta: Intraoperative placement when severe peripheral vascular disease precludes femoral approach.
- **Major indications:**
  - Myocardial ischemia, intractable to maximal medical therapy; used as a stabilizing measure before definitive intervention.
  - LV dysfunction inadequately managed with inotropic therapy, usually seen intraoperatively post CPB.
- **Beneficial hemodynamic effects:**
  - ↑ diastolic BP → ↑ coronary perfusion.
  - ↓ LV afterload → ↓ myocardial oxygen consumption.
- **Efficacy depends on:**
  - Proper placement in the aorta: Close to the heart but distal to the great vessels.
  - Proper timing of inflation and deflation: Inflated in synchrony with the dicrotic notch on an arterial blood pressure.
- **Contraindications:**
  - Aortic incompetence.
  - Arterial dissection.
  - Peripheral vascular disease.
- **Complications:**
  - Thromboembolic phenomenon.
  - Distal limb ischemia (thromboembolism and mechanical obstruction to the femoral artery).
  - Thrombocytopenia.
  - Gas emboli (rupture of balloon).
  - Infection.

IABP hemodynamic effects: ↑ coronary perfusion and ↓ LV afterload.

Dysrhythmias

Atrial Fibrillation (AF)

- Most frequent postoperative arrhythmia after cardiac surgery; may occur intraoperatively.
- Incidence: 15–40%.
- Usually 2–4 days postoperatively.
- May develop for the first time after surgery/represent exacerbation of preoperative dysrhythmia.
- Nonsurgical risk factors: Preexisting AF and age.
- Surgical risk factors: Combined valve/coronary surgery, duration of aortic cross-clamping, bicaval cannulation, pulmonary vein venting.
- Associated with prolonged hospital stay, ↑ incidence of cerebrovascular accidents (CVAs), ventricular fibrillation/ventricular tachycardia.
- Not independently associated with ↑ in mortality.
- Intraoperative preventive measure: Optimize electrolyte balance including magnesium homeostasis.
- Goals of treatment: (1) control of ventricular response rate, (2) convert to sinus rhythm.
- β blockers, magnesium: Modify ventricular response rate and promote conversion to sinus rhythm.
- Other treatment modalities: Electrical cardioversion, pacing.
- Agents with negative inotropy may be better avoided in this specific setting.
- Cox maze procedure: Involves multiple surgical incisions in the atria by epicardial radiofrequency ablation—chaotic atrial conduction is interrupted. It is an effective surgical approach in chronic AF with mitral valve disease and makes 78% of patients AF free at 3 years.

Ventricular Tachycardia/Fibrillation (VT/VF)

- Nonsustained ventricular tachycardia: Common after cardiac surgery.
- Incidence: 50%; does not adversely affect long-term outcome.
- Sustained VF/VT:
  - Most likely if ventricular function is depressed.
  - In association with ischemia.
  - After aortic valve replacement, especially with ventricular dilatation.
- Long-term management: Depends on electrophysiology studies.
- Intraoperatively:
  - Treat ischemia.
  - Optimize electrolyte balance.
  - Cardioversion.
  - Pharmacologic therapy initiated when appropriate: Bretylium, amiodarone more effective than lidocaine.

New and Emerging Approaches to Cardiac Surgery

Off-Pump Coronary Artery Bypass Grafting (OPCAB)

- **Advantages:**
  - ↓ cerebral emboli and stroke rates: Reduced aortic manipulation.
  - ↓ bleeding and blood product use: Avoidance of CPB.
  - ↓ time to extubation.
  - ↓ length of stay.
  - ↓ overall cost.

- **Anesthetic implications:** Profound hemodynamic fluctuations:
  - ↓ preload → ↓ CO.
  - Pericardial sling sutures in the posterior pericardium → constriction of IVC and/or pulmonary veins or both; ↓ venous return (VR) to right and/or left side of the heart.
  - Lifting of heart during conduit anastamoses on the posterior surface → ↓ VR.
  - To overcome the hypotension, augment preload:
    - Steep Trendelenburg position.
    - Crystalloid/colloid.
    - Monitor CVP and PA pressures, optimize loading conditions.
  - TEE: Direct visualization of the adequacy of preload, detects new-onset valvular regurgitation or diastolic dysfunction.

Minimally Invasive Direct Coronary Artery Bypass Surgery (MIDCAB)

- Performed through left thoracotomy.
- **Advantages:**
  - Avoidance of CPB.
  - Less risk of stroke or neuropsychiatric deficits.
  - Shorter hospitalization, ICU length of stay.
  - Cost saving.
  - ↓ transfusion requirements.
- **Disadvantages:**
  - Technically more demanding.
  - Multivessel disease contraindicated.
  - Limited to patients with amenable cardiac anatomy.
  - May not be a reproducible procedure.
  - Hemodynamic instability and arrhythmias.
  - Unsafe in unstable patients.
  - Steep learning curve.

Port Access Coronary Artery Bypass Surgery

- Cardiac surgery with CPB and cardiac arrest but without a median sternotomy.
  - Venous drainage: Cannula in femoral vein advanced into IVC and RA.
  - Cardiac venting: Large-bore catheter in PA through right internal jugular (IJ).
  - Coronary sinus catheter: Through right IJ.
  - Arterial catheter: Special endarterial cannula through femoral artery cutdown, allows placement of end aortic clamp in the ascending aorta.
  - TEE facilitates correct placement.
  - Heart isolated by inflation of balloon in the aortic lumen.
  - Cardioplegia through port emptying in the ascending aorta proximal to the balloon.
  - Left minithoracotomy → exposure of the heart for surgical anastamoses.
- Reported results have varied range, from comparable to CPB → significant complications related to femoral artery dissection.
- Other disadvantages: Expensive, steep learning curve.

Robotics

- **Advantages:**
  - Allows precise movements in small places ↑ potential for port-access surgery.
  - Potential for remote surgery—telesurgery.
- **Anesthetic implications:**
  - Similar to other port-access cardiac surgery procedures.
  - Double-lumen tube, external defibrillator pads, TEE, lateral position.
  - Maintaining the ability to resuscitate the patient in spite of the patient's lateral position and use of the robot (bed locked).

Anesthesia for Cardioversion (CV)

- **Indications for elective CV:**
  - AF/atrial flutter.
  - AV nodal reentry.
  - Reciprocating tachycardias from preexcitation syndromes.
- Not effective against:
  - Enhanced activity: Multifocal atrial tachycardia (MAT).
  - Triggered activity: Digitalis toxicity.
- **Indications for emergency CV:** Tachyarrhythmia associated with significant hypotension/CHF/angina.
- **Specific indications for CV in patients with AF:**
  - Symptomatic fibrillation of < 12 months' duration.
  - History of embolism.
  - Recent onset.
  - No response to medications.
- **High recurrence rate associated with:**
  - Long-standing fibrillation.
  - COPD.
  - CHF.
  - Mitral requrgitation.
- **Preoperative considerations:**
  - NPO for 6–8 hr.
  - Twelve-lead ECG prior to the procedure to confirm presence of the arrhythmia.
  - Correct metabolic, electrolyte, and acid-base abnormalities.
  - Continue the antiarrhythmics (digitalis, quinidine, etc.) started 1–2 days prior.
  - Continue anticoagulation (warfarin) started 1–2 weeks prior.
  - TEE: Performed prior to CV to rule out an LA blood clot, usually in the LA appendage.
- **Monitoring**
  - ECG, BP, pulse oximetry.
  - Reliable venous access.
  - Functional bag-mask device capable of delivering 100% oxygen.
  - Oxygen source from wall outlet or full tank.
  - Airway kit with oral/nasal airways, laryngoscopes, and endotracheal tubes.
  - Functional suction apparatus.
  - Anesthetic drug kit that includes at least one sedative-hypnotic as well as succinylcholine.
  - Crash cart that includes all necessary drugs and equipment for cardiopulmonary resuscitation.

Anesthetic Technique

- Very brief (1–2 min) amnesia or light general anesthetic is required.
- Preoxygenation with 60–100% oxygen for 3–5 min.
- Short-acting barbiturate (methohexital), propofol, etomidate, or a benzodiazepine (eg, midazolam, diazepam) is used.
- Shock is delivered when the patient is unable to respond verbally or has lost eyelid reflex.
- Transient airway obstruction or apnea may be observed.

Cardioversion

- Mechanism of action:
  - Simultaneously depolarizes the entire myocardium.
  - Prolongs the refractory period.
- Synchronization:
  - Indications: All tachyarrhythmias except VF.
  - Shock is delivered during the QRS complex.
  - Shock occurs in the ST segment/T wave → a more serious arrhythmia (eg, VF).
- Placement of electrodes:
  - Anterolateral: One R second intercostal space (ICS) next to the sternum. Second L fifth ICS medial collateral ligament (MCL).
  - Anteroposterior: One anteriorly, LV apex in fifth ICS. Second posteriorly, L infrascapular region.
- Energy levels:
  - Supraventricular tachycardia (except AF): 25–50 J.
  - AF: 50–100 J (minimum).
  - Hemodynamically stable VT: 25–50 J.
  - VF and hemodynamically unstable VT: 200–400 J.
  - If the first shock is ineffective, higher energy level is required.
  - If ventricular arrhythmia develops after initial shock, lidocaine is given prior to next shock.

Complications

- Transient myocardial depression.
- Postshock arrhythmias:
  - Due to inadequate synchronization.
  - Most transient and resolve spontaneously.
- Arterial embolism may cause delayed awakening

Recovery

- Similar to general anesthetic.
- Monitor for recurrence of arrhythmias.
- Monitor for signs of cerebral embolism.

CHAPTER 12

Thoracic Anesthesia

PHYSIOLOGICAL CHANGES DURING THORACIC SURGERY

Thoracic surgery presents specific physiological situations for the anesthesiologist.

- Lateral decubitus position provides optimal access for the majority of operations (lungs, pleura, esophagus, great vessels).
- Alters the normal pulmonary ventilation/perfusion dynamics (see Figure 12-1).
- ↓ in functional residual capacity (FRC) with general anesthesia (GA), moves the upper lung to a more favorable position on the compliance curve. Lower lung moves to a less compliant position (see Figure 12-1).
- This ↓ in FRC during GA is due to numerous factors, such as:
  - Cephalad shift of diaphragm due to loss of muscle tone.
  - Exertion of cephalad pressure from abdominal content.
  - Change in intrathoracic volume secondary to ↑ lung blood volume.
  - Changes in chest wall shape.
  - Surgical positions (ie, Trendelenberg) and surgical manipulation.
- Controlled positive pressure ventilation favors the upper lung because it is more compliant due to the compression on the dependent lung/hemidiaphragm from mediastinal and abdominal structures. This is further accentuated by opening the nondependent chest for the surgical procedure.
- Opening of the chest wall disrupts normal lung physiological parameters.
  - Normally, lungs are kept expanded by negative pleural pressure.
  - After chest wall is opened, negative pleural pressure is lost, as is the elastic recoil, so lung collapses.
  - Paradoxical mediastinal shift.
- One-lung ventilation:
  - Large right-to-left intrapulmonary shunt is created.
  - Mixture of unoxygenated blood from upper lung with oxygenated blood from lower lung widens A-a gradient, which can result in hypoxemia.

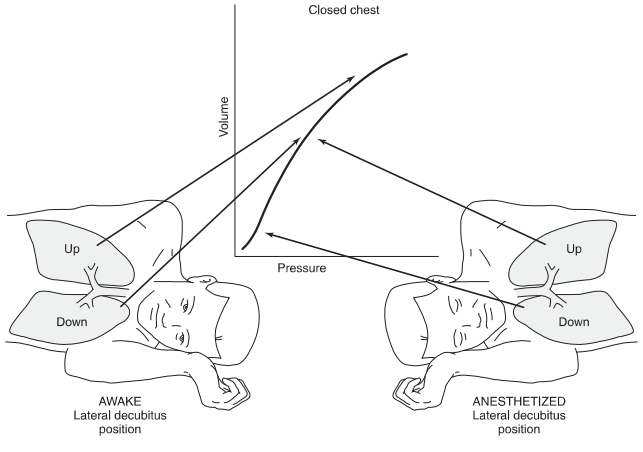

FIGURE 12-1. The effects of anesthesia on lung compliance in lateral decubitus position.

(Reproduced, with permission, from Morgan GE et al. *Clinical Anethesiology*, 4th ed. New York: McGraw-Hill, 2006: 587.)

- Blood flow to upper lung is ↓ by hypoxic pulmonary vasocontriction (HPV), which is a physiological mechanism causing pulmonary vasoconstriction in response to regional ↓ in alveolar PO_2/hypoxia. The purpose of this response is to divert blood from underventilated regions of the lung to maintain ventilation/perfusion (V/Q) relationship.
- Factors affecting HPV:
  - Triggers: ↓ PaO_2, ↑ $PaCO_2$.
  - Inhibitors: inhalationals (volatiles, N_2O, NO), vasodilators (nitroprusside, nitroglycerin, β agonists, dobutamine), hypocapnea, ↑ PA pressure, ↑ PEEP.

Drugs that negatively affect HPV: Volatiles, N_2O, NO, nitroprusside, nitroglycerin, β agonists, dobutamine.

ONE-LUNG VENTILATION (OLV)

Techniques for One-Lung Ventilation

Double-Lumen Tubes (DLTs)

Absolute indications for a DLT:

- *Avoid contamination (abscess, hemorrhage).*
- *Differential ventilation: Bronchopulmonary fistula, tracheobronchial disruption, large cyst/bullae.*
- *BAL.*

- Indications:
  - Absolute:
    - Avoid contamination (abscess, hemorrhage).
    - Differential ventilation: Bronchopleural fistula, tracheobronchial disruption, large cyst/bullae.
    - Bronchoalveolar lavage (BAL).
  - Relative:
    - High-priority surgical exposure: Thoracic aneurysm surgery, upper lobectomy, pneumonectomy.
    - Low-priority surgical exposure: Esophageal resection, middle/lower lobectomy.
- Contraindications: Bronchial or carinal obstructions.
- Advantages: Ease of placement, ability to ventilate/suction either lung.
- Disadvantages: Should not be used for prolonged ventilations, complicated placement in difficult airways.
- Complications:
  - Hypoxemia due to misplacement.
  - Traumatic laryngitis.
  - Tracheobronchial rupture from over inflation of cuff.
  - Suturing of tube to bronchus during surgery
- Complications of double-lumen tubes:
  - Hypoxemia due to misplacement.
  - Traumatic laryngitis.
  - Tracheobronchial rupture from over inflation of cuff.
  - Suturing of tube to bronchus during surgery.
- Placement of DLT (Figure 12-2):
  - Either Macintosh or Miller blade can be used.
  - DLT is passed through the vocal cords with the distal curvature pointed anteriorly and is rotated 90 degrees, toward side of the bronchus to be intubated.
  - Advance until resistance is felt. Average depth of insertion is 29 cm.
  - Confirm with flexible fiber-optic bronchoscope.
  - If difficulty arises, placement of smaller tube should be attempted.
  - Troubleshooting of malpositioned DLT:
    - Check tube depth insertion.
    - Make sure tube did not enter contralateral side.
    - Right double-lumen tubes can occlude orifice of right upper lobe.

Alternatives to DLT

- Endobronchial insertion of single-lumen ETT.
- Bronchial blocker (BB) placed through or alongside single-lumen ETT (Figure 12-3).
  - Advantages: Used in pediatric patients or patients with difficult airway in which DLT is not possible; tube does not need to be exchanged at end of case if prolonged ventilation expected.
  - Disadvantages: Inability to suction isolated lung; inability to ventilate isolated lung; loss of seal or balloon slippage can occur.
- Univent—single-lumen ETT with built-in BB.
  - Advantages: Use in difficult airways, continuous ventilation during BB placement, CPAP through BB lumen.
  - Disadvantages: Inability to ventilate while maintaining isolation, slow lung inflation/deflation, occlusion of BB lumen.

Anatomic Considerations (Figure 12-4)

- Adult trachea is 11–13 cm long.
- Wider right mainstem bronchus diverges away from trachea at 25 degrees.
- Left bronchus diverges at 45 degrees.
- Orifice of right upper lobe bronchus is 1.5 cm vs. left upper lobe, which is about 5 cm, making it preferable to place left-sided DLT.

OLV Ventilator Settings

FiO_2 100%.
V_T ~10 cc/kg.
RR ↑ 20-30% to make up for ↓ V_T.

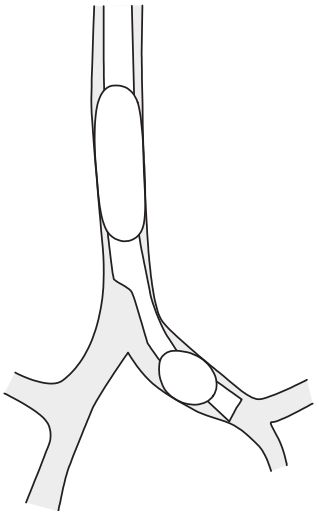

FIGURE 12-2. Optimal placement of left double-lumen tube.

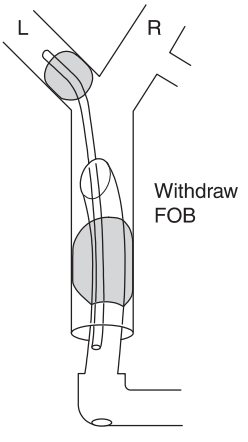

FIGURE 12-3. Proper placement of bronchial blocker in left mainstem.

(Reproduced, with permission, from Longnecker DE et al (eds). *Anesthesiology.* New York: McGraw-Hill, 2008: 1233.)

Hypoxemia Management on OLV

1. Ensure FiO_2 100%.
2. Check position of DLT.
3. CPAP 5–10 cmH_2O to collapsed lung.
4. PEEP 5–10 cmH_2O to ventilated lung.
5. CPAP 10–15 cmH_2O to collapsed lung.
6. PEEP 10–15 cmH_2O to ventilated lung.
7. Intermittent two lung ventilation.
8. Surgical occlusion of pulmonary artery.

ANESTHESIA FOR LUNG RESECTION

Predominantly performed for the diagnosis and treatment of pulmonary tumors, infections, and bronchiectasis.

Tumors

Malignant vs. benign. Can occur at the periphery or centrally.

- Benign pulmonary tumors include hamartomas, bronchial adenomas (carcinoids, cylindromas, and mucoepidermoid adenomas).
- Carcinoids may secrete multiple hormones such as adrenocorticotropic hormone (ACTH) and vasopressin.
- Malignant pulmonary tumors are divided into small cell and non–small cell carcinomas.
- Non–small cell include squamous cell, adenocarcinoma, and large cell carcinomas.
- All types most commonly associated with smokers.

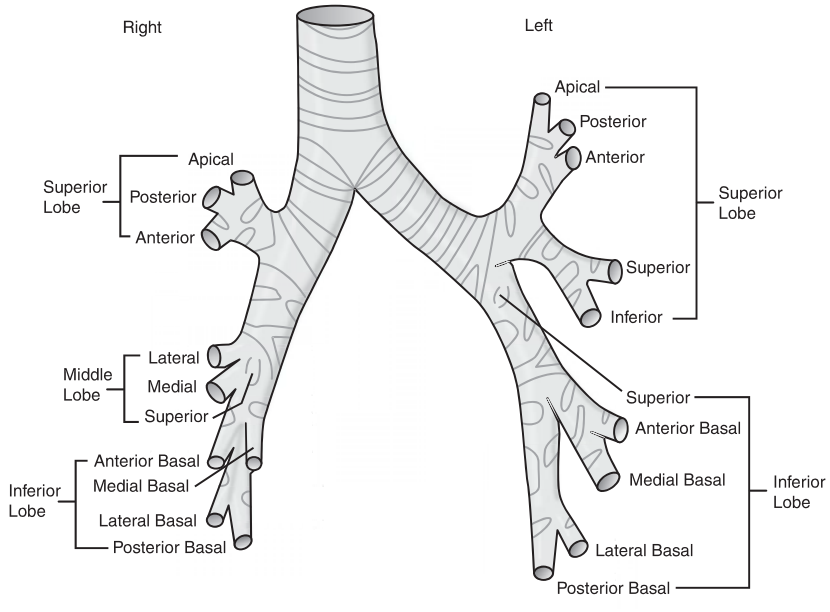

FIGURE 12-4. Anatomy of the tracheobronchial tree.

Symptoms

- Cough, hemoptysis, dyspnea, wheezing, weight loss, fever, productive sputum.
- Pleuritic chest pain can suggest pleural extension.
- Involvement of mediastinal structures is suggested by hoarseness from compression of recurrent laryngeal nerve.
- Horner's syndrome is caused by involvement of the sympathetic chain.
- Elevated hemidiaphragm is caused by compression of the phrenic nerve.
- Dysphagia is caused by compression of the esophagus or superior vena cava syndrome.
- Paraneoplastic syndromes: Cushing's syndrome, hyponatremia, hypercalcemia, cerebellar degeneration, peripheral neuropathy, polymyositis, migratory thrombophlebitis, and nonbacterial carditis.

Treatment

- Surgery is the treatment of choice for the curative treatment of lung cancer, especially in non–small cell carcinomas.
- Small cell carcinomas are treated with chemotherapy and radiation since a diagnosis is usually made after metastases.

Infections

- Can present as solitary nodule or cavitary lesion (necrotizing penumonitis).
- Lung resection is indicated for cavitary lesions that are refractory to antibiotic treatment, are associated with refractory empyema, or result in massive hemoptysis.

Bronchiectasis

- Permanent dilation of bronchi, which is end result of severe or recurrent inflammation and obstruction of bronchi.
- Bronchial muscle and elastic tissue is replaced by fibrous tissue, which can cause hemoptysis.
- Pulmonary resection is usually indicated for massive hemoptysis.

Preoperative Assessment

CXR and ECG are beneficial.
ABG and spirometry are helpful depending on the disease severity.

Assessment of patient's ability to tolerate procedure:

1. PFTs show preop $FEV_1 > 2$l and normal P_{CO_2} on RA indicate procedure will be tolerated. If not, then . . .
2. V/Q scan shows postop $FEV_1 > 800$ cc.

   [Predicted postop FEV_1 = preop FEV_1% x (1 – % lung removed / 100)]

   If not, then…
3. Split lung function test shows PAP < 40 mmHg with normal MAP and normal ABG on RA. If not, then procedure will not be tolerated.

Preoperative Management

- Little sedative premedication for patients with moderate to severe pulmonary compromise.

- Consider anticholinergics to reduce copious secretions.
- Monitors/access: Arterial line, large-bore IV, consider central line.

Maintenance

- Halogenated agents and opioids.
- N_2O is generally avoided as it can decrease FiO_2.
- Neuromuscular blockade will facilitate surgery.
  - Restrict IV fluids to only maintenance requirements and blood loss replacement. This prevents gravity-induced transudation of fluid into the dependent lung causing intrapulmonary shunting and nondependent lung reexpansion edema.

Postoperative Management

- Extubate early to ↓ risk of barotraumas and pneumonia.
- Change double-lumen to single-lumen if expected prolonged intubation.
- Supplemental oxygen, incentive spirometry, semiupright position.
- Thoracic epidurals provide excellent pain relief and reduce pulmonary complications.
- Intrapleural catheters and intercostal blocks can also be used.
- Complications: Atelectasis, pneumonia, bronchospasm, bronchopleural fistula, reexpansion pulmonary edema, transudation of fluid into dependent lung, hemorrhage, herniation of lobe causing torsion (→ pulmonary vein obstruction → infarction), cardiac herniation into operative hemithorax, RV failure, phrenic/vagus/left recurrent laryngeal nerve damage, spinal cord damage.

ANESTHESIA FOR TRACHEAL RESECTION

- Indications: Tracheal stenosis, tumors, and congenital abnormalities.
- Compromise of tracheal lumen results in progressive dyspnea, wheezing, and/or stridor.
- Flow volume loops can assist in the location and severity of the obstruction (see Figure 12-5).

Anesthetic Considerations

- Administer little or no premedication since most patients have some level of airway obstruction.
- Left radial artery cannulation for pressure monitoring is preferable because of potential of innominate artery compression.
- Inhalation induction is performed for patients with severe obstruction so that spontaneous ventilation is maintained.
- Avoid neuromuscular blockade due to its potential complete airway obstruction.
- Surgeon may perform direct bronchoscopy to evaluate lesion.
- Use smallest tube possible to allow easy passage through lesion.
- Jet ventilation can be used as an alternative.

ANESTHESIA FOR ESOPHAGEAL SURGERY

- Indications: Tumors, gastroesophageal reflux, motility disorders.
- Procedures: Simple endoscopy, esophageal dilation, cervical esophagomyotomy, open or distal esophagomyotomy, blunt esophagectomy.

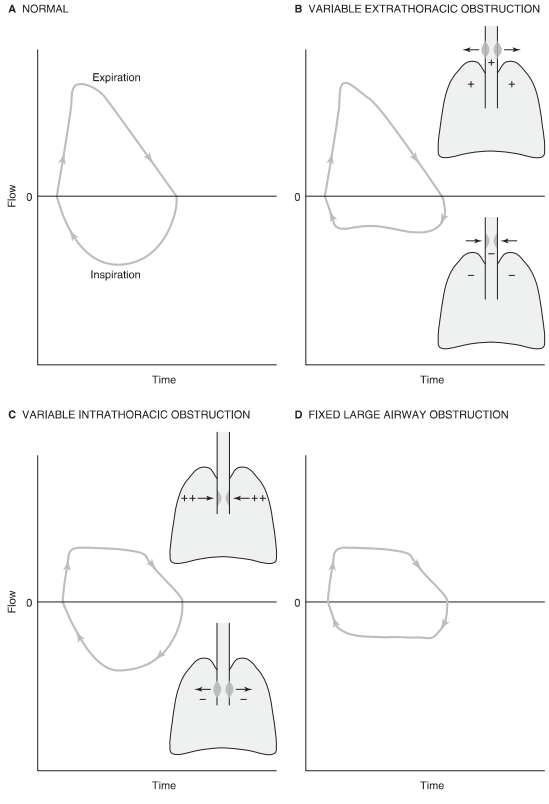

FIGURE 12-5. Flow volume loops.

(Reproduced, with permission, from Morgan GE et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006: 604.)

Anesthetic Considerations

- Major concern is risk of pulmonary aspiration secondary to underlying disease (ie, obstruction, altered motility, or reflux disease).
- Preoperative administration of an H_2 blocker, metoclopramide, or proton pump inhibitor.
- An awake nasogastric tube may help in preventing aspiration.
- An upright position with rapid-sequence intubation should be used, or consider an awake fiber-optic technique.
- Depending on the procedure, a double-lumen tube should be used.
- Monitoring of arterial pressure and central venous access is indicated.
- Interoperative complications: Surgical dissection, especially during retraction and the freeing of the esophagus from the posterior mediastinum, can induce vagal stimulation, interfere with cardiac filling, and cause hypotension.
- Postoperative complications: Damage to the phrenic, vagus, and left recurrent laryngeal nerves secondary to surgical manipulation.

ANESTHESIA FOR THORASCOPIC SURGERY

- Surgical technique used for majority of procedures (lung biopsies, segmental and lobar resections, pleurodesis, and esophageal procedures).
- One-lung ventilation is mandatory.
- Anesthetic management similar to pneumonectomy and other thoracotomy procedures.

DIAGNOSTIC THORACIC PROCEDURES

Rigid Bronchoscopy

- For tracheal dilation, requires general anesthesia.
- Usually short in duration.
- Airway must be shared with surgeon.
- Intravenous induction maintained with potent halogenated anesthetic, 100% oxygen, and a short-acting neuromuscular blocker is ideal.
- Total IV anesthesia can be used alternatively.
- Common techniques employed:
  - Apneic oxygenation with small catheter alongside bronchoscope.
  - Conventional ventilation through the side arm of a ventilating bronchoscope.
  - High-frequency ventilation through an injector-type bronchoscope (16- to 18-gauge cannula in the proximal end of the bronchoscope).

Mediastinoscopy

- Provides access to the mediastinal lymph nodes.
- General tracheal anesthesia with muscle relaxation is the common practice.
- Adequate venous access is necessary due to risk of innominate artery damage and ↑ risk of bleeding (see Figure 12-6).

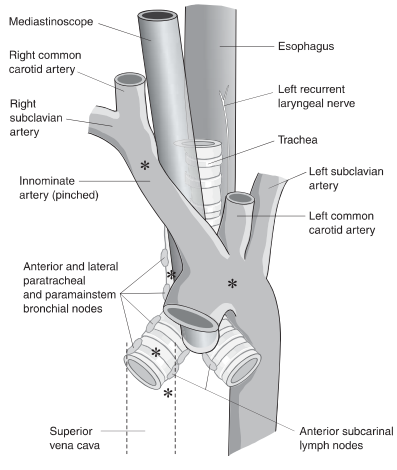

FIGURE 12-6. Placement of mediastinoscope in anterior mediastinum.

The mediastinoscope is in close proximity to the innominate artery. (Reproduced, with permission, from Longnecker DE et al (eds). *Anesthesiology.* New York: McGraw-Hill, 2008: 1253.)

Oxygen saturation monitoring in right hand can reflect innominate artery compression during mediastinoscopy.

- Oxygen saturation should be measured on right upper extremity to monitor for innominate artery compression.
- Complications: Reflex bradycardia, hemorrhage, cerebral ischemia, pneumothorax, air embolism, recurrent laryngeal or phrenic nerve injury.

Bronchoalveolar Lavage

- Used for patients with pulmonary alveolar proteinosis.
- Patients unable to clear copious amounts of surfactant.
- Procedure indicated for severe hypoxemia or worsening dyspnea.
- Unilateral lavage is performed under general anesthesia with a double-lumen tube.
- At the completion of the procedure, the double-lumen tube is replaced with single lumen.

CHAPTER 13

Anesthesia for Vascular Surgery

GENERAL PREOPERATIVE CONSIDERATIONS

Coexisting Diseases

- **Cardiac:** A major source of morbidity and mortality as many patients have coronary artery disease (CAD). Risk factors: congestive heart failure (CHF), myocardial infarction (MI), hypertension (HTN), angina, arrhythmias, valvular heart disease.
- **Pulmonary:** Many patients may have a smoking history and may have chronic obstructive pulmonary disease (COPD) or underlying malignancy.
- **Renal:** HTN, diabetes, arteriosclerosis, and volume depletion may → renal insufficiency.
- **Central nervous system (CNS):** Vascular disease is a "whole-body" phenomenon. Presence of carotid bruits, history of cerebrovascular accident (CVA) or transient ischemic attacks (TIAs), amaurosis fugax, or syncope may require further evaluation.
- **Endocrine:** Diabetes and its end-organ complications are a concern. Tight glucose control should be achieved.
- **Hematologic:** Many patients are on anticoagulants. Ascertain a thorough history of type, dose, and last time taken. Coagulation profile (prothrombin time [PT]/partial thromboplastin time [PTT]/International Normalized Ratio [INR]/platelets) should be available. It may be necessary to continue clopidogrel in patients with drug-eluding cardiac stents. Check with the cardiologist if clopidogrel can be stopped safely.

Amaurosis fugax can be divided into five groups of etiology:

1. *Embolic*
2. *Hypoperfusion*
3. *Angiospasm*
4. *Neurologic*
5. *Idiopathic*

Preoperative Medications

See Table 13-1.

Preoperative Testing

- Based on individual patients' comorbidities, risk of surgery, and exercise tolerance.
- Further testing may not be necessary based on new *ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery* (Figure 13-1).
- **High risk** (reported cardiac risk often > 5%):
  - Aortic and other major vascular surgery.
  - Peripheral vascular surgery.
- **Intermediate risk** (reported cardiac risk generally < 5%): Carotid endarterectomy.

CAROTID ARTERY SURGERY

Preoperative Considerations

INDICATIONS

- TIAs associated with severe ipsilateral carotid stenosis (> 70% occlusion).
- Severe stenosis in patients with minor stroke.
- Moderate occlusion (30–70%) in patients with ipsilateral symptoms.

TABLE 13-1. Management of Preoperative Drug Therapy

Medication	Side Effect or Potential Concern in the Perioperative Period	Recommendation for Perioperative Use
Aspirin	Platelet inhibition may ↑ bleeding. ↓ GFR.	Continue until day of surgery. Monitor fluid and urine status.
Clopidogrel	Platelet inhibition may ↑ bleeding. Rare thrombotic thrombocytopenic purpura.	Hold for 7 days before surgery except for CEA and severe CAD. Consider blood crossmatch. Avoid neuraxial anesthesia if not held for at least 7 days.
HMG-CoA reductase inhibitors	LFT abnormalities. Rhabdomyolysis.	Assess LFTs and continue through morning of surgery. Check CPK if myalgias.
β blockers	Bronchospasm. Hypotension. Bradycardia. Heart block.	Continue through perioperative period.
ACE inhibitors	Induction hypotension. Cough.	Continue through perioperative period. Consider one-half dose on day of surgery.
Diuretics	Hypovolemia. Electrolyte abnormalities.	Continue through morning of surgery. Monitor fluid and urine status.
Calcium channel blockers	Perioperative hypotension, especially with amlodipine.	Continue through perioperative period (consider withholding amlodipine on the morning of surgery).
Oral hypoglycemics	Hypoglycemia preoperatively and intraoperatively. Lactic acidosis with metformin.	When feasible, switch over to insulin preoperatively. Monitor glucose status perioperatively.

ACE, angiotensin-converting enzyme; CAD, coronary artery disease; CEA, carotid endarterectomy; CPK, creatine phosphokinase; GFR, glomerular filtration rate; HMG-CoA, 3-hydroxy-3-methyl-glutaryl-CoA; LFT, liver function test.

(Adapted, with permission, from Barash PG. *Clinical Anesthesia,* 5th ed. Lippincott Williams & Wilkins, 2006: 936.)

Patient Characteristics

- Generalized atherosclerosis may be present, particularly in coronary arteries.
- Coexisting HTN and diabetes may ↑ morbidity when uncontrolled.
- Preexisting neurologic deficits should be documented.

Major cause of mortality in CEA patients is myocardial ischemia (1–2%).

Monitoring

- Arterial catheterization is used for close regulation of blood pressure.
- **Electrocardiogram (ECG):** Monitor for ischemia in leads II and V5; utilize continuous ST segment analysis if available.
- Pulmonary artery (PA) catheterization is usually not necessary.

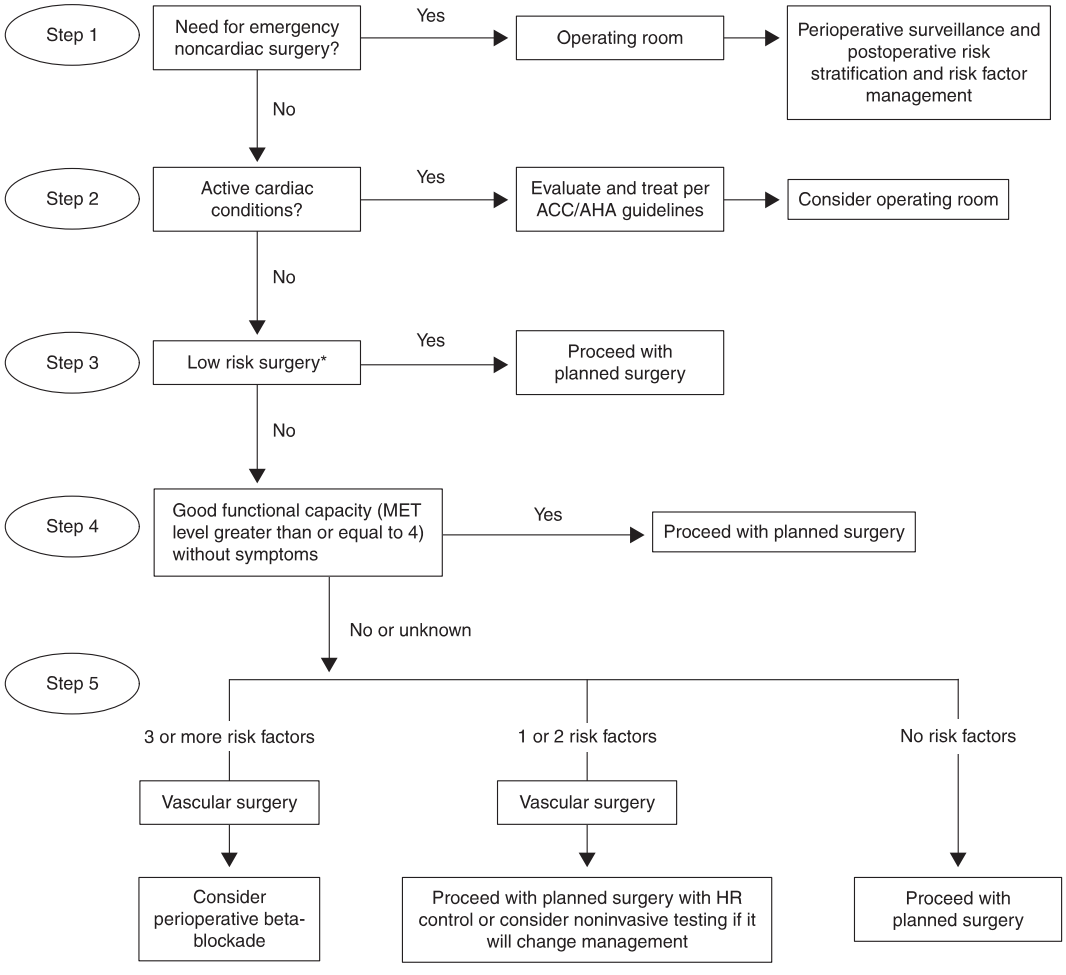

FIGURE 13-1. ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery.

(Reproduced, with permission, from Fleisher L et al. ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Cardiology/American Heart Association Task Force on Practice Guidelines. Care for Noncardiac Surgery: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation* 2007; 116: e418–e500.)

A patient with history of severe anxiety or an elderly patient with poor baseline mental status is a poor candidate for awake CEAs.

- **Cerebral function evaluation:** A temporary shunt may be placed if evaluation shows the development of cerebral ischemia; shunts carry a risk of thromboembolic complications.
  - **Regional anesthesia:** Patient cooperation is necessary to evaluate speech and motor function.
  - **General anesthesia:** Electroencephalogram (EEG), somatosensory evoked potentials (SSEPs), distal stump pressure, transcranial Doppler measurement of middle cerebral artery (MCA) flow velocity, cerebral oximetry.

Anesthetic Management

The use of general vs. regional anesthesia should be a decision made based on the experience of the anesthesiologist and surgeon. Many studies show no difference in outcome between the two techniques.

- **Regional anesthesia:** Superficial and deep cervical plexus blockade of C2–C4 performed in a cooperative patient allow for adequate surgical anesthesia.
  - **Advantages:** Intraoperative neurologic examination (assessment of level of consciousness, speech, and contralateral handgrip) can be performed and may better assess cerebral perfusion during cross-clamping.
  - **Disadvantages:**
    - Airway not secured and may be difficult to secure once surgery starts.
    - Change in mental status may cause combative patient and allow for poor surgical field.
    - No potential pharmacologic cerebral protection.
- **General anesthesia:**
  - **Induction:** Goal of induction is to maintain hemodynamic stability while maximizing cerebral protection.
    - Keep mean arterial pressure at or slightly above patient's baseline.
    - Avoid tachycardia.
  - Thiopental, propofol, and etomidate ↓ cerebral metabolic rate ($CMRO_2$) more than cerebral blood flow (CBF).
  - Opioids can be used to avoid hypertensive response to endotracheal intubation.

Temporary shunt carries a thromboembolic event risk of 0.7%.

MAINTENANCE

- Isoflurane is volatile of choice due to its greatest ↓ in $CMRO_2$, but desflurane has gained popularity due to its quick awakening.
- **HTN:** Treat with a vasodilator such as nitroglycerin or nitroprusside.
- **Hypotension:** Treat with a titratable direct-acting α agonist such as phenylephrine.
- **β blockers:** Use with caution, as profound bradycardia may be encountered with manipulation of carotid baroreceptors.
- **Maintain normocapnia:** Hypercapnia can cause cerebral steal syndrome, and hypocapnia can ↓ cerebral perfusion.
- **Carotid cross-clamping:**
  - Heparin 50–100 units/kg given before cross-clamping.
  - Blood pressure kept elevated to ↑ CBF via circle of Willis.
  - Shunt: Placed if there are changes in EEG during general anesthesia or there are changes on neurologic exam during regional anesthesia; may ↑ potential for ↑ morbidity from thromboembolic events; shunts may impede access to carotid and ↑ cross-clamp time; patients may still develop EEG abnormalities despite placement of shunt, which requires adjustment of the shunt.
  - Traction on the carotid sinus may cause intense vagal stimulation, resulting in severe bradycardia and hypotension, and may require treatment with atropine (can be ↓ with local infiltration of field).
  - Protamine 50–150 mg is given slowly for reversal of heparin prior to skin closure.

Postoperative Management and Complications

- Smooth emergence from general anesthesia is ideal in maintaining hemodynamic stability.
- **Postoperative HTN**—more common.
  - **Causes:** Pain, hypoxemia, hypercarbia, surgical denervation of the ipsilateral carotid baroreceptor.

- **Treatment:** Short-acting agents such as nitroglycerin, labetatol, nicardipine.
- **Postoperative hypotension**—less common.
  - **Causes:** Removal of atheroma exposes baroreceptors to higher pressures, causing brain stem–mediated hypotension and bradycardia.
  - **Treatment:** Usually resolves in 12–24 hr; obtain ECG to ensure no cardiac etiology; IV fluids and vasopressors if neurologic status is compromised.
- **Respiratory insufficiency:**
  - **Causes:** Recurrent laryngeal nerve damage, hypoglossal nerve damage, impaired carotid body response to hypoxemia, neck hematoma.
- **Neurologic deficits:**
  - **Causes:** Thromboembolism (leading to cerebral hypoperfusion), regional cerebral hyperperfusion.

Ascending aorta = between aortic valve and innominate artery.

Aortic arch = b/w innominate artery and left subclavian artery.

Descending aorta = distal to the left subclavian artery.

AORTIC SURGERY

Preoperative Considerations

- Anesthetic management of the patient for aortic surgery can be challenging, especially in the emergent, nonoptimized case,
- Potential for large blood loss requiring aortic cross-clamp.

Aortic Cross-Clamp

- Effects of aortic cross-clamp:
  - ↑ left ventricular afterload.
  - ↓ distal organ perfusion.
  - Worsening of severe HTN, myocardial ischemia, and valvular regurgitation.
  - Renal failure secondary to hypoperfusion.
  - Paraplegia from compromised flow to the spinal cord via the artery of Adamkiewicz.
- Cross-clamp release causes hypotension as a result of:
  - Sudden ↓ in afterload.
  - Coagulopathy and ↑ bleeding.
  - Release of metabolites from ischemic lower body, causing diffuse vasodilatation.
  - Slow release of clamp, volume loading, briefly ↓ anesthetic depth, and intermittent dosing of vasopressors can alleviate the amount of hypotension.
- **Complications:**
  - Paraplegia and spinal cord ischemia caused by surgical damage to artery of Adamkiewicz, which is the major artery supplying lower thoracic and lumbar spinal cord.
  - Anterior spinal artery syndrome: Loss of motor function and sensation to pinprick, with intact proprioception and vibration.
  - Protection against spinal cord injury: Short cross-clamp time, higher perfusion pressures, shunts, partial cardiopulmonary bypass (CPB), steroids, hypothermia, mannitol, cerebrospinal fluid drainage via lumbar drain
  - **Renal failure:**
    - **Risk factors:** Preexisting renal disease with emergency cases, prolonged cross-clamp times, prolonged hypotension.
    - Greater incidence with suprarenal over infrarenal clamping.

- **Prevention:** Infusion of mannitol and fenoldopam, renal-dose dopamine may not be beneficial.

Indications for Surgery

Aortic Dissection

Tear in the tunica intima of the aorta results in a false lumen, allowing blood to flow into the media and potentially extend the length of the vessel.

Risk Factors

Connective tissue disorders (Marfan's syndrome and Ehlers-Danlos syndrome) as a result of medial cystic necrosis.

Complications

Occlusion of aortic lumen, extension proximally to root, rupture into the pericardium causing tamponade.

- **Classification** (see Figure 13-2):
  - **Stanford classification:**
    - Type A (DeBakey types I and II) involves the ascending aorta.
    - Type B (DeBakey type III) involves the descending aorta.
    - Type A dissections require surgery, while type B dissections can usually be managed medically.
  - **DeBakey classification:**
    - Type I involves the ascending aorta, aortic arch, and descending aorta.

Artery of Adamkiewicz

(orgin of)

T5–T8 = 15%

T9–T12 = 60%

L1–L2 = 25%

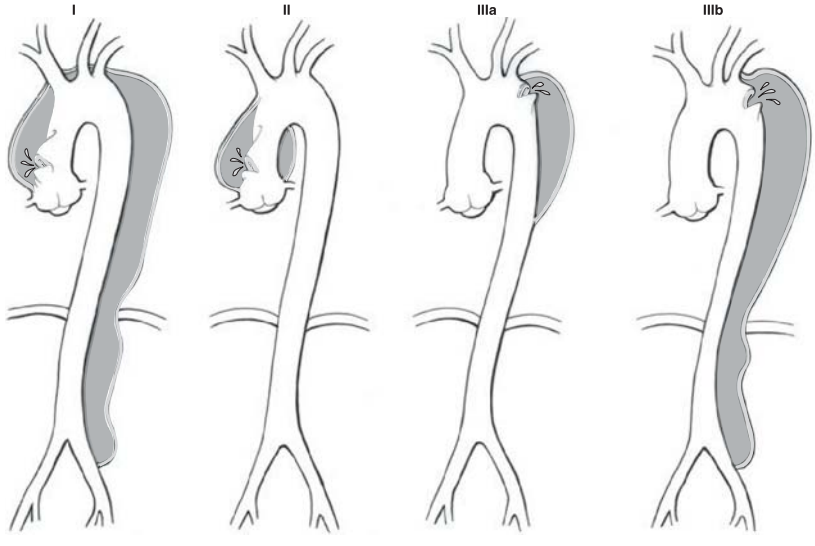

FIGURE 13-2. Classification of aortic dissections.

Stanford type A includes any involvement of ascending aorta, while type B is limited to the descending aorta. DeBakey types I and II can also be considered Stanford type A. DeBakey type III is the same as a Stanford type B. (Reproduced, with permission, from Cohn LH (ed). *Cardiac Surgery in the Adult,* 3rd ed. New York: McGraw-Hill, 2008: 1196.)

- Type II is confined to the ascending aorta.
- Type III is confined to the descending aorta distal to the left subclavian artery.

Aortic Aneurysms

- Aneurysms are defined as a focal dilatation with at least a 50% ↑ over normal arterial diameter.
- Most commonly involves the abdominal aorta.
- **Etiology:** Atherosclerosis (most common), rheumatoid arthritis, syphilis, trauma.

Complications

- Aortic regurgitation, tracheal compression and/or deviation, hoarseness, superior vena cava syndrome, rupture.
- Rupture manifests as acute severe back pain.
- Risk of rupture ↑ with increasing aneurysm size: > 6 cm correlates to 50% rupture within 1 year.

Treatment

Repair is usually performed in patients with > 4-cm aneurysms; many patients are candidates for endovascular repair.

Aortic Occlusive Disease

- Etiology: Atherosclerosis.
- Occurs at aortic bifurcation (Leriche's syndrome).

Treatment

Aortobifemoral bypass and/or endarterectomy.

Normal aortic size in adults is 2–3 cm in width.

Coarctation of the Aorta

Congenital heart defect classified in relation to the ductus arteriosus:

- **Preductal type** often diagnosed during infancy:
  - Diminished pulses in lower extremities.
  - Lower-body cyanosis.
  - Upper-body perfusion from aorta; lower-body perfusion from pulmonary artery.
- **Postductal type** often diagnosed in adulthood.
  - Severity of lesion and amount of collateral circulation determines severity of symptoms.
  - Upper-extremity HTN.

Monitoring

- Large IV access above the diaphragm should be established prior to induction.
- Arterial catheterization (preferably on the right) can be obtained preinduction if significant comorbidities or wide changes in hemodynamics are expected during induction; otherwise, postinduction arterial line placement is suitable.

- Central venous access with the insertion of a pulmonary artery catheter (PAC) can be obtained after induction. PAC detects left ventricle failure, particularly during suprarenal cross-clamping.
- Transesophageal echocardiography (TEE):
  - Highly sensitive and specific in the diagnosis of an acute aortic dissection.
  - Helpful particularly in type A dissections in the assessment of the aortic valve, tamponade, and left ventricular dysfunction.
  - Limited images of distal ascending aorta and proximal aortic arch because of interposition of trachea or bronchus between esophagus and aorta.

Anesthetic Management

Surgery on the Ascending Aorta

- Involves sternotomy and CPB.
- May need left radial artery, femoral or dorsalis pedis for arterial blood pressure monitoring, as the innominate artery may be clamped.
- CPB may be established via the femoral artery in cases involving dissections.
- Intraoperative course can be complicated by large volume shifts, blood loss, long cross-clamp times, and new or worsening aortic regurgitation, often requiring valve replacement.
- Nitroglycerin or nitroprusside is often used for precise blood pressure control.
- β blockers should be used with caution, as bradycardia can worsen aortic regurgitation.

Surgery on the Aortic Arch

- Involves sternotomy, CPB, and hypothermic circulatory arrest.
- Cooling to 15–18°C, steroids, mannitol, and thiopental can be used to achieve cerebral protection.
- Associated coagulopathies should be corrected during rewarming period.

Double-lumen ETT for descending aorta thoracic surgery is a relative, not absolute indication.

Surgery on the Descending Thoracic Aorta

- Involves left thoracotomy without CPB for open procedures.
- One-lung anesthesia using double-lumen tube or bronchial blocker can facilitate surgical exposure.
- Shunts and left atrial–femoral artery and femoral vein–femoral artery bypasses can reduce complications caused by cross-clamping.
- CSF drains may be required if shunts or bypasses are not used.
- Elective cases can benefit from a thoracic epidural for postoperative pain management but may be complicated with use of anticoagulation.
- Endovascular repair considered for those not candidates for open repair.
- Right radial arterial line, as clamping of left subclavian artery may be necessary.
- Cross-clamping causes HTN above clamp and hypotension below clamp.
- Vasodilator agents (nitroglycerin and nitroprusside) ↓ blood pressure in the acute setting.
- Correction of coagulopathy and dosing of calcium chloride may benefit those receiving massive transfusions.

Surgery on the Abdominal Aorta

- Involves anterior abdominal or anterolateral retroperitoneal approach.
- Endovascular repair considered for those not candidates for open repair.
- Combined epidural–general anesthesia may provide benefit to ↓ release of stress hormones and ↓ requirements for inhalation agents.
- More distal cross-clamping produces less effect on left ventricular afterload and hemodynamics.
- Fluid replacement is of concern due to large surgical exposure.

Partial tear or complete aortic transection is usually seen as a widened mediastinum on CXR.

Postoperative Management

- Most patients undergoing surgery to the proximal aorta should remain intubated and ventilated for the immediate postoperative period.
- Most patients undergoing surgery of the abdominal aorta can be extubated.
- Immediate postoperative goal is to maintain stable hemodynamic parameters and correct coagulopathies.

UPPER- AND LOWER-EXTREMITY REVASCULARIZATION

Preoperative Considerations

Indications

- Occlusive arterial disease from atherosclerosis and emboli.
- Aneurysms of peripheral vessels.
- Pseudoaneuryms.
- Vascular injuries.

Anesthetic Management

- Can be performed under regional or general anesthesia alone or as a combined regional-general technique.
- Neuraxial anesthesia: These patients may already be on antiplatelet medication or will require anticoagulation during surgery; timing of epidural placement and removal should be made accordingly.
- Vasopressor: Phenylephrine minimizes tachycardia.
- Arterial catheterization may be required for patients with co-morbidities and severe vascular disease.
- Maintain normothermia: Shivering can ↑ myocardial oxygen demand.
- Conservative fluid management, as many of these patients are prone to CHF.

Postoperative Management

- Patients are closely observed for graft occlusion and may need revision.
- Epidural catheters are typically left in place for postoperative analgesia.
- Extremes of blood pressure and heart rate should be avoided.

CHAPTER 14

Neurosurgical Anesthesia

Cerebral Blood Flow (CBF)

- Average: 50 mL/100 g/min.
- Gray matter receives ~80 mL/100 g/min.
- White matter receives ~20 mL/100 g/min.
- Average brain CBF: 750 mL/100g/min (average brain weighs 1.5 kg and consumes 15–20% of CO).
- Electroencephalogram (EEG) slowing: 20–25 mL/100 g/min.
- Flat EEG: 15–20 mL/100 g/min.
- Irreversible brain damage: < 10 mL/100 g/min.

Only hypothermia decreases the basal $CMRO_2$ of the brain.

Determinants of Cerebral Blood Flow

- **Cerebral metabolic rate (CMR):**
  - Greatest in gray matter of cerebral cortex.
  - Parallels cortical electrical activity.
  - Expressed as mL O_2/100 g brain tissue/min.
  - Changes in CMR directly coupled with CBF: ↑ (or ↓) in CMR result in ↑ (or ↓) in CBF, relationship uncoupled by volatiles.
  - CMR in young adults and elderly: ~3.5 mL O_2/100 g brain tissue/min.
  - CMR in children (6 years old) is higher ~ 5.2 mL O_2/100 g brain tissue/min.
- **$PaCO_2$:**
  - Carbon dioxide tension between 20 and 80 mmHg is directly proportional to CBF:
    - 40–80 mmHg: CBF doubled.
    - 20–40 mmHg: CBF halved.
  - Effect is transient as CBF returns to normal in 6–8 hr even if altered CO_2 levels are maintained.
  - CBF ↑ 1 mL/100 mg/min for 1 mmHg ↑ in $PaCO_2$ from 40 mmHg.
- **Cerebral steal:**
  - Occurs in hypoventilated patients with focal ischemia.
  - Hypoventilation → ↑ $PaCO_2$ → vasodilation. Blood vessels in ischemic areas are already maximally dilated. Blood flow is shunted away from ischemic areas to normal areas.
- **Reverse steal or Robin Hood effect:**
  - Occurs in hyperventilated patients with focal ischemia.
  - Hyperventilation → ↓ $PaCO_2$ → vasoconstriction. Blood vessels in ischemic areas remain more vasodilated due to lower pH. Blood flow is shunted away from normal areas to ischemic areas.
- **PaO_2:** CBF is ↑ when PaO_2 < 50 mmHg.
- **Cerebral perfusion pressure (CPP) and autoregulation:**
  - CPP = mean arterial pressure (MAP) – intracranial pressure (ICP) (or central venous pressure [CVP] if it is > ICP).
  - Normal CPP = 80–100 mmHg.
  - Normal ICP < 10 mmHg.

EEG Finding	CPP
EEG slowing	< 50 mmHg
Isoelectric EEG	25–40 mmHg
Irreversible brain damage	< 25 mmHg

- **Autoregulation** (see Figure 14-1):
  - CBF maintained constant between as MAP varies from 50–150 mmHg.
  - Below and above this range, CBF is dependent on CPP.
  - MAP < 60: Ischemia.
  - MAP > 160: Potential disruption of blood-brain barrier and cerebral edema.
  - Autoregulation is disrupted by ischemia, tumor masses, trauma, seizures, hypoxemia/hypercarbia.
  - Autoregulation curve is shifted right in hypertensive patients.
- **Temperature:**
  - CBF varies in a direct relationship with temperature.
  - For each 1°C change in temperature, CBF varies 5–7%.
  - Hypothermia ↓ both CMR and CBF.

At 20° C, the EEG is isoelectric.

Cerebrospinal Fluid (CSF)

- Protects the CNS against trauma.
- Primarily formed by transport of sodium, chloride, and bicarbonate ions.
- Produced mostly by ependymal cells in the choroid plexus.
- Absorbed into venous system of the brain by villi in the arachnoid membrane.
- Absorption directly proportional to ICP and inversely proportional to CVP.
- Rate of production: 0.3–0.4 mL/min.
- Total volume: 100–150 mL.
- Medications that ↓ CSF formation:
  - Furosemide: Blocks transports of sodium and chloride ions.
  - Acetazolamide: Blocks transport of bicarbonate ions.
  - Spironolactone.
  - Isoflurane.
  - Vasocontrictors.

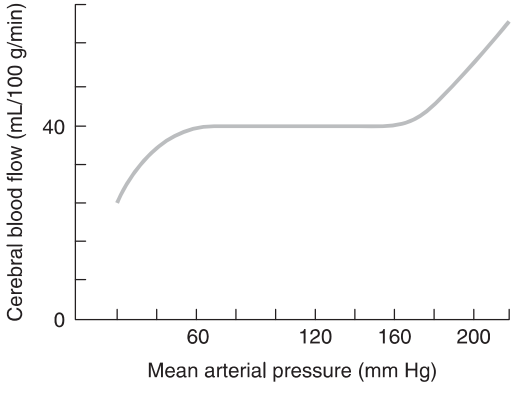

FIGURE 14-1. Autoregulation.

(Reproduced, with permission, from Morgan GE et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006: 616.)

Intracranial Pressure (ICP)

- Normal: < 10 mmHg.
- Major components that occupy space in the cranium:
  - Brain parenchyma: Neurons and glia.
    - 80% of intracranial volume.
    - Causes of ↑ ICP: Tumors, vasogenic or cytotoxic cerebral edema.
  - CSF:
    - Occupies 8% of intracranial volume.
    - Causes of ↑ ICP: Communicating or obstructive hydrocephalus.
  - Blood:
    - Occupies 12% of intracranial volume.
    - Cerebral blood volume: 3–7 mL/100 g brain weight.
    - Causes of ↑ ICP: Cerebral hemorrhage or vasodilation of vessels.
    - Because the skull cannot expand, its volume is fixed; any ↑ in parenchyma, CSF, or blood is accompanied by an ↑ in ICP.

Symptoms of ↑ ICP

Headache, vomiting, papilledema, drowsiness, loss of consciousness, decerebrate posturing, oculomotor nerve palsy, abnormal brain stem reflexes, abnormal respiratory patterns, Cushing reflex (hypertension [HTN] and bradycardia).

Complications of ↑ ICP

- ↓ CPP.
- Brain herniation (for types, see Figure 14-2).

CBV ↑ 0.05 mL/100 grams of brain per 1 mmHg increase in $Paco_2$.

Treatment of ↑ ICP

- ↓ blood volume:
  - Head elevation: ↑ venous return.
  - Hyperventilation: $PaCO_2$ of 25–30 mmHg reduces ICP for 24–36 hr without affecting acid-base status and cerebral O_2 delivery.
  - Mild fluid restriction (one-third to one-half of maintenance requirements).
- ↓ CSF volume: Drain CSF—extraventricular drain (EVD) or lumbar drain.

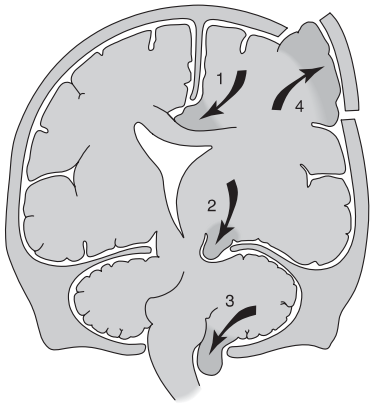

FIGURE 14-2. Different types of brain herniation: (1) cingulated gyrus, (2) temporal lobe (uncal), (3) cerebellar, and (4) transcalvarial (postoperative or traumatic).

(Reproduced, with permission, from Fishman RA. Brain edema. N *Engl J Med* 1975; 293: 706; or Morgan GE et al. *Clinical Anesthesiology*, 4th ed. New York: McGraw-Hill, 2006: 619.)

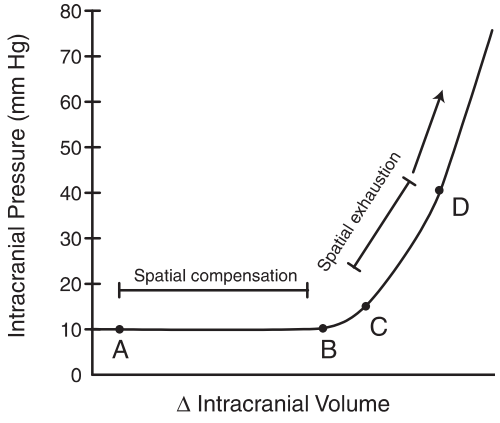

FIGURE 14-3. Initial compensatory mechanisms (from point A to B).

(1) Displacement of CSF from cranial vault to spinal compartment; (2) ↑ CSF absorption; (3) ↓ CSF production; (4) translocation of blood out of the intracranial vault. (Reproduced, with permission, from Sulek CA. Intracranial pressure. *Clinical Neuroanesthesia,* 2nd ed. Philadelphia: Churchill Livingstone, 1998: 73–123.)

- ↓ parenchymal volume:
  - Osmotic diuretics.
  - Steroids.
- ↓ CMR:
  - Barbiturates.
  - Moderate hypothermia (35°C)—↓ CMR and CBF.
  - Avoid positive end-expiratory pressure (PEEP) if possible—may ↓ venous effluent.
  - Avoid succinylcholine—has potential to ↑ ICP.
  - Anesthetic agents.
    - Barbiturates/propofol/benzodiazepine/etomidate—↓ CBF and ICP.
    - Narcotics/lidocaine/esmolol—blunt sympathetic and hypertensive response to laryngoscopy.
    - Inhalational agents—↑ CBF and ICP.
  - Avoid hypo-osmolar glucose containing fluids—can aggravate cerebral edema and ↑ ICP.

No further ↓ in CMR is observed once EEG is isoelectric.

Intracranial Elastance

$$\text{Elastance} = \text{Compliance}^{-1} = \Delta \text{ Pressure} / \Delta \text{ Volume}$$

- ↑ in volume are initially well compensated until the limit of spatial compensation is reached.
- After this point, further ↑ volume cause large ↑ ICP.
- In other words, elastance is ↑ (and compliance ↓) (see Figure 14-3).

Cerebral Edema

↑ in brain water content. Types:

- **Vasogenic edema:** Disruption of the blood-brain barrier (most common). Etiology: ↑ blood pressure (BP), mechanical trauma, inflammation, brain tumors, infarction.

TABLE 14-1. Effects of Anesthetic Agents on Cerebral Physiology

Agent	CMR	CBF	CSF Production	CSF Absorption	CBV	ICP
Volatile Agents						
Halothane	↓↓	↑↑↑	↓	↓	↑↑	↑↑
Enflurane	↓↓	↑↑	↑	↓	↑↑	↑↑
Isoflurane	↓↓↓	↑	±	↑	↑↑	↑
Desflurane	↓↓↓	↑	↑	↓	?	↑↑
Sevoflurane	↓↓↓	↑	?	?	?	↑↑
Nitrous oxide	↓	↑	±	±	±	↑
IV Agents						
Barbiturates	↓↓↓↓	↓↓↓	±	↑	↓↓	↓↓↓
Etomidate	↓↓↓	↓↓	±	↑	↓↓	↓↓
Propofol	↓↓↓	↓↓↓↓	?	?	↓↓	↓↓
Benzodiazepines	↓↓	?	±	↑	↓	↓
Ketamine	±	↑↑	±	↓	↑↑	↑↑
Opioids	±	±	±	↑	±	±
Lidocaine	↓↓	↓↓	?	?	↓↓	↓↓

↑, increase; ↓; decrease; ± = little or no change; ? = unknown; CMR, cerebral metabolic rate; CBF, cerebral blood flow; CSF, cerebrospinal fluid; CBV, cerebral blood volume; ICP, intracranial pressure.

Note: Enflurane can cause seizure patterns on EEG particularly combined with hypocapnea.

(Adapted, with permission, from Morgan, GE et al. *Clinical Anesthesiology,* 4th ed. New York: McGraw Hill, 2006: 161.)

- **Cytotoxic edema:** Failure of active Na^+ pump to maintain normal gradient, free water enters cells. Causes: metabolic insults such as hypoxemia or ischemia.
- **Interstitial edema:** Entry of CSF into brain interstitium as a result of obstructive hydrocephalus.
- **Water intoxication:** Intracellular movement of water from acute drop in serum osmolality.

EFFECTS OF ANESTHETIC AGENTS ON BRAIN PHYSIOLOGY

See Table 14-1.

Inhalational Agents

Volatile Anesthetics

- Uncouple CBF and CMR, impair cerebral autoregulation.
- Dose-dependent ↓ CMR: Isoflurane > desflurane/sevoflurane > halothane.
- Dose-dependent ↑ CBF: Halothane > enflurane > isoflurane/desflurane/sevoflurane.
  - Vasodilation and impairment of CBF autoregulation.
  - Blood flow returns to normal after continued administration (2–5 hr).
  - Effects can be abolished by hyperventilation.
  - Can contribute to cerebral steal—volatiles ↑ CBF away from ischemic areas.
- ICP alterations:
  - Isoflurane for patients with ↓ compliance.
  - Desflurane may ↑ ICP more than other agents.

Nitrous Oxide

Minimal effects on CBF, CMR, and ICP.

Intravenous Agents

- Most IV anesthetic agents ↓ CBF and CMR (except ketamine).
- All IV agents preserve autoregulation and CO_2 responsiveness.

Luxury perfusion = the combo of ↓ in neuronal metabolic demand and ↑ in CBF (cerebral blood flow).

Barbiturates

Major CNS actions:

- CMR depression (↓ CMR > ↓ CBF → metabolic supply > metabolic demand [luxury perfusion]).
  - Dose-dependent depression.
  - ↓ CMR evenly throughout brain.
- CBF reduction*
  - Dose-dependent depression.
  - Caused by vasoconstriction in nonischemic areas.
  - Robin Hood effect (reverse steal)—blood flow redistributed to ischemic areas.
- Hypnosis.
- Anticonvulsant.
- Facilitates CSF absorption—helps lower ICP.

Detrimental circulatory steal phenomenon is possible with volatile anesthetics in settings of focal ischemia.

Opioids

- Minimal effects on CBF, CMR, and ICP.
- All can ↓ CPP secondary to a hypotension.
- Alfentanil: Can activate seizure foci in patients with epilepsy.
- Morphine: Not ideal due to poor lipid solubility.
- Remifentanil: A rapidly metabolized opioid allows rapid neurologic assessment.
- Naloxone: Can cause severe HTN.

Normeperidine (metabolite of meperidine) can induce seizures in patients with renal failure.

Etomidate

- ↓ CMR, CBF, and ICP.
- ↓ CSF production and enhances absorption.
- Effect on CMR is more pronounced in cerebral cortex than the brain stem.
- Long-term use can cause adrenal suppression.
- Induction doses are associated with myoclonic movements.
- Can activate seizure foci in patients with epilepsy.

Propofol

- ↓ CMR, CBF, and ICP.
- ↓ CBF > ↓ CMR, luxury perfusion.
- Associated with hypotension and cardiac depression, which can compromise CPP.
- Anticonvulsant activity.

Benzodiazepines

- ↓ CMR, ↓ CBF but effect is less pronounced than barbiturates, etomidate, and propofol.
- Anticonvulsant activity.
- Flumazenil: Benzodiazepine antagonist; ↑ CMR, ↑ CBF, ↑ ICP; use with caution.

Ketamine

- ↑ CBF, ↑ CBV, ↑ CSF → ↑↑ ICP.
- ↑ CBF by 50–60%.
- Only IV anesthetic that is a cerebral vasodilator.
- Total CMR unchanged: Selectively activates some areas, while depressing others. Global CMR remains unchanged.
- ↓ CSF absorption.
- Can cause seizure activity in limbic and thalamic areas.

Dexmedetomidine

- Adrenergic α_2 selective agonist with analgesic and sedative effects.
- ↓ CBF via cerebral vasoconstriction.

Lidocaine

- ↓ CMR, ↓ CBF, ↓ ICP but its effect is less compared to other IV agents.
- ↓ CBF by increasing cerebral vascular resistance.
- Blunts laryngeal reflex, useful adjunct for intubation and extubation.
- Toxicity and seizure risk.

NEUROMONITORING

Refer to Chapter 5 for details.

Electroencephalogram (EEG)

- Monitors cerebral function and adequacy of CPP during general anesthesia (see Table 14-2).
- **Uses:** CEA, cerebral aneurysm clipping, cerebral arteriovenous malformation (AVM) surgery, deliberate hypotension, assessment of anesthetic depth.
- **Interpretation:**
  - Activation (high frequency, low voltage): Light anesthesia or surgical stimulation.
  - Depression (low-frequency, high-voltage activity): Deep anesthesia and ischemia.
- Most anesthetic agents produce biphasic pattern on EEG—activation followed by dose-dependent depression. Opioids are an exception to this rule; they exhibit a monophasic pattern with dose-dependent depression (see Table 14-3).

SSEP signals can not detect anterior cord ischemia.

Evoked Potentials

Somatosensory Evoked Potentials (SSEPs)

Reflect ability of neural pathway to conduct a signal from periphery to cerebral cortex.

Types

- **Somatosensory:** Tests sensory cortex and integrity of dorsal columns of the spinal cord. Uses: spinal tumor resection, instrumentation of the spine, CEA, abdominal aortic aneurysm (AAA).
- **Auditory (BAEP):** Tests integrity of CN VIII and auditory pathways. Use: posterior fossa surgery.
- **Visual (VEP):** Tests optic nerve function and upper brain stem function. Use: resection of large pituitary tumors.

Interpretation

- Latency: Time from stimulation to specific peak.
  - Short latency: Arises from brain stem, least affected by anesthetics.
  - Intermediate latency: Arises from cortex, affected by anesthetics.

TABLE 14-2. EEG Frequency Ranges

Delta rhythm (0–3 Hz)	Deep sleep, deep anesthesia, or pathologic states (eg, brain tumors, hypoxia, metabolic encephalopathy).
Theta rhythm (4–7 Hz)	Sleep and anesthesia in adults, hyperventilation in awake children and young adults.
Alpha rhythm (8–13 Hz)	Resting, awake adult with eyes closed; predominantly seen in occipital leads.
Beta rhythm (> 13 Hz)	Mental activity, light anesthesia.

(Reproduced, with permission, from Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 758.)

TABLE 14-3. EEG Changes with Anesthetic Drugs, Pao_2, $Paco_2$, Temperature

Increased Frequency
Barbiturates (low dose)
Benzodiazepines (low dose)
Etomidate (low dose)
Propofol (low dose)
Ketamine
N_2O (30–70%)
Inhalation agents (< 1 MAC)
Hypoxia (initially)
Hypercarbia (mild)
Seizures
Decreased Frequency/Increased Amplitude
Barbiturates (moderate dose)
Etomidate (moderate dose)
Propofol (moderate dose)
Opioids
Inhalation agents (> 1 MAC)
Hypoxia (mild)
Hypocarbia (moderate to extreme)
Hypothermia
Decreased Frequency/Decreased Amplitude
Barbiturates (high dose)
Hypoxia (mild)
Hypercarbia (severe)
Hypothermia (< 35°C)
Electrical Silence
Barbiturates (coma dose)
Etomidate (high dose)
Propofol (high dose)
Desflurane (2 MAC)
Isoflurane (2 MAC)
Sevoflurane (2 MAC)
Hypoxia (severe)
Hypothermia (< 15–20°C)
Brain death

(Reproduced, with permission, from Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 759.)

- Long latency: Arises from cortex, highly affected by anesthetics, not used for monitoring.
- Amplitude: Voltage measurement from the peak apex to baseline.
  - VEP: Most affected by anesthetics.
  - BAEP: Least affected by anesthetics.
  - ↓ amplitude of 50% or ↑ latency 10% indicates a disruption of the pathway.

The anesthetics that ↓ SSEP signals also ↓ MMEP signals but to a lesser degree.

Anesthetic Considerations

- **Inhalational:**
  - Volatiles: Dose-dependent ↓ amplitude and ↑ latency.
  - Nitrous oxide: ↓ amplitude but does not affect latency.
- **Intravenous:** Less effect on SSEPs with ↓ amplitude and ↑ latency at high doses (see Table 14-4).
- Other variables altering SSEPs:
  - Temperature: Hypothermia ↑ latency and ↓ amplitude.
  - Hypotension: ↓ amplitude.
  - Hypoxia: ↓ amplitude.
  - Hypocarbia: ↑ latency.
  - Isovolemic hemodilution: ↑ latency when Hct < 15%, ↓ amplitude when Hct < 7%.

Motor Evoked Potentials (MEPs)

- Transcranial stimulation of motor cortex elicits contralateral peripheral nerve signals, electromyographic signals, and limb movements.
- Tests integrity of dorsolateral and ventral spinal cords pathways.
- **Uses:** Intramedullary tumor resections, scoliosis correction, AAA.
- Anesthetic considerations: Extremely sensitive to volatile anesthetics, requires total intravenous anesthesia (TIVA).
- **Caveat:** Not all motor stimulations are MEPs (eg, facial nerve monitoring during head/neck surgery or direct stimulation of peripheral nerves). This type of monitoring measures the integrity of peripheral nerves only and is not affected by volatile anesthetics—it is sensitive only to neuromuscular blockade.

TABLE 14-4. Effects of Intravenous Anesthetics on SSEP Monitoring

Drug	Amplitude	Latency
Midazolam	↓	0
Diazepam	↓	↑
Thiopental	↑/0	↑
Etomidate	↑↑↑	↑
Propofol	0	↑
Ketamine	↑	
Opioids		↑
Volatiles	↓	↑
Nitrous oxide	↓	0

(Adapted, with permission, from Faust RJ et al. *Anesthesiology Review,* 3rd ed. Philadelphia: Churchill Livingstone, 2002: 65.)

Intracranial Pressure Monitoring

- Allows optimization of CPP, early detection and treatment of brain hemorrhage, edema, and herniation.
- **Techniques:** Ventriculostomy catheters, subdural-subarachnoid bolts or catheters, epidural transducers, intraparenchymal fiber-optic catheters.
- **Uses:** Head trauma, large brain tumors, ruptured intracranial aneurysms, cerebrovascular occlusive disease, hydrocephalus.

CONSIDERATIONS DURING NEUROSURGICAL CASES

Intracranial Hypertension

Occurs when ICP > 15 mmHg (normal < 10 mmHg).

Causes

Expanding tissue or fluid mass, interference with normal CSF absorption, ↑ CBF, conditions → cerebral edema.

Presentation

- Usually asymptomatic initially, but gradual ↑ in ICP lead to headache, nausea, vomiting, papilledema, altered consciousness, and focal neurologic deficits.
- Cushing's reflex: Abrupt ↑↑ arterial BP with reflex bradycardia.
- Complications: Cerebral ischemia and herniation.

With mannitol administration, serum osmolality goal is 300–315 mOsm/L.

Treatment

- Direct at the underlying cause.
- Correct metabolic disturbances.
- Osmotic agents and loop diuretics.
  - **Mannitol:**
    - 0.25–2.0 g/kg, action in 10–15 min and effective for 2 hr.
    - Reduces ICP by reducing volume of brain parenchyma.
    - Can cause transient cerebral vasodilation and ↑ ICP initially.
    - Must be given as an infusion to avoid ↑ ICP.
    - Effective when blood-brain barrier is intact (can cause ↑ ICP if not intact).
    - **Uses:** Usually limited to craniotomies and tumor resections.
    - **Complications:** Pulmonary edema in patients with borderline cardiac or renal function, hematoma expansion as volume of normal brain tissue around hematoma ↓, subdural hematoma in elderly with fragile bridging veins entering sagittal sinus, rebound edema.
  - **Furosemide:**
    - Requires up to 30 min to work.
    - Reduces ICP by systemic diuresis and ↓ CBV.
    - Also ↓ CSF production.
    - Resolves cerebral edema through improving cellular water transport.
    - Check serum K^+ levels, especially if used in combination with osmotic diuretics.
- Moderate hyperventilation to $PaCO_2$ of 25–30 mmHg.
- Corticosteroids: Promote repair of blood-brain barrier if intracranial hemorrhage results from vasogenic edema secondary to tumors.

Intracranial Masses and Anesthesia

- Types of intracranial masses:
  - Congenital.
  - Neoplastic (benign, malignant).
  - Infectious (cysts, abscesses).
  - Vascular (hemangiomas, AVMs).
- Types of primary tumors:
  - Glial cell tumors (astrocytoma, oligodendroglioma, glioblastoma).
  - Ependymal cells (ependymoma).
  - Supporting tissues (meningioma, schwannoma, choriodal papilloma).
  - Childhood tumors (medulloblastoma, neuroblastoma, chordoma).

Presentations of Intracranial Masses

- Headaches, seizures, general decline in cognition or specific neurologic functions, signs of intracranial HTN.
- Supratentorial masses (meningiomas, gliomas, metastatic lesions): Associated with ↑ ICP.
  - Seizures
  - Hemiplegia
  - Aphasia
- Infratentorial masses: Associated with mass effects on brain stem structures and ↑ ICP as result of obstructive hydrocephalus.
  - Cerebellar dysfunction (ataxia, nystagmus, dysarthria).
  - Brain stem compression (cranial nerve palsies, abnormal respiration, altered consciousness).

Preoperative Management

- Determine presence or absence of ↑ ICP: Computed tomography (CT)/ magnetic resonance imaging (MRI) signs of cerebral edema, enlarged cerebral ventricles, midline shift.
- Neurologic exam: Mental status, sensory or motor deficits.
- Review medications: Use of steroids, anticonvulsants, diuretics.
- Labs: Look for electrolyte abnormalities, hyperglycemia.

ICP may also be ↓ by minimizing IV fluids.

Premedication

- Not necessary in lethargic patients.
- Benzodiazepines for alert and anxious patients.
- Small doses of opioids for preinduction insertion of invasive monitoring devices in awake, conversant patients to alleviate discomfort.
- Avoid respiratory depressants in patients with ↑ ICP.
- Continue corticosteroids and anticonvulsant therapy until time of surgery.

Monitoring

- ASA standard monitors.
- Temperature probe.
- Arterial line: Continuous BP monitoring, arterial blood gases.
- Foley catheter: Guides fluid management in the face of blood loss and osmotic diuresis.

- Twitch monitor: Place on *unaffected* side in patients with hemiplegia (often resistant on the affected side).
- Possible central venous catheterization for patients with severe comorbid conditions.
- ICP monitoring: After ventriculostomy or subdural bolt placement by the neurosurgeon.

A cooperative patient may be asked to hyperventilate themselves prior to induction.

Induction and Intubation

- Goal: Smooth, controlled induction without ↑ ICP or ↓ CBF.
- Optimize intracranial compliance with osmotic diuresis, steroids, or removal of CSF prior to induction.
- Hyperventilate to lower ICP.
- Propofol (1.25–2.5 mg/kg) or thiopental (3–5 mg/kg) are commonly used as induction agents.
- Lidocaine (1.5 mg/kg) used ~90 sec before intubation to blunt laryngeal reflexes.
- Opioids (fentanyl: 5–10 μg/kg) blunts sympathetic response during intubation.
- Esmolol (0.5–1.0 mg/kg) controls tachycardia in hypertensive or cardiac patients.
- Combination of etomidate (6–8 mg) can be used in hemodynamically unstable patients.
- Nondepolarizing muscle relaxants: Usually given to prevent coughing or straining that could ↑ ICP. Succinylcholine has been shown to ↑ ICP and is not normally used in elective neurosurgery cases, but is still a muscle relaxant of choice in patients with risk of aspiration, difficult airway. Avoid succinylcholine in patients with chronic muscle weakness.

Positioning

- Frontal, temporal, parieto-occipital craniotomies are done in supine position with a slight head elevation (15–30 degrees) to facilitate venous and CSF drainage.
- Excessive flexion or rotation of the head can impede jugular venous drainage and ↑ ICP.
- Operating table is usually turned 90–180 degrees away from the anesthesiologist.
  - Ensure that breathing circuit connections are tight and endotracheal tube (ETT) is adequately secured.
  - Ensure that all monitors, IV and arterial lines are in good position prior to draping.

Vasodilators should be avoided due to ↑ in ICP as a result of ↑ CBV.

Maintenance of Anesthesia

- Balanced anesthetic with volatile agents, opioids, and muscle relaxant.
- TIVA is required with transcranial MEP monitoring.
- Maintain normocarbia: In cases of significantly ↑ ICP, hyperventilation may be necessary with a $PaCO_2$ between 30 and 35 mmHg.

- Avoid high PEEP or high mean airway pressures (high tidal volumes with low rates), which can potentially cause venous congestion and ↑ ICP.
- Avoid glucose-containing IV fluids (hyperglycemia has been implicated in ischemic brain injury).
- Crystalloids to replace maintenance fluids and colloids to restore intravascular volume deficits from blood loss.

Emergence and Extubation

- IV lidocaine (1.5 mg/kg) can be given 90 sec prior to extubation to minimize cough, straining, and HTN.
- Antihypertensive agents (labetalol, esmolol, nicardipine): Control HTN during emergence.
- Extubate patient only if complete systemic or brain homeostasis is achieved.
- Extubate only after patient is fully reversed from muscle paralysis, awake, and following commands (see Table 14-5 for causes of delayed awakening).
- Brief neurologic examination should be performed before transporting patient out of the OR with oxygen and monitors.

Complications of Posterior Fossa Surgery

Cardiovascular Instability

Causes

- ↓ venous return.
- Surgical stimulation
- Trigeminal nerve stimulation → bradycardia, HTN.
- Glossopharyngeal or vagal stimulation → bradycardia, hypotension, asystole.

Treatment

- Preoperative hydration, compression stockings, slow positional change.
- Notify surgeon to stop the stimulation (usually resolves the problem).
- Atropine, glycopyrrolate, or ephedrine is a rarely necessary next step.

TABLE 14-5. Causes of Delayed Awakening

Preoperative ↓ level of consciousness
Residual anesthesia
Metabolic or electrolyte disturbance
Residual hypothermia
Pneumocephalus
Intraoperative complications:
Seizures
Cerebral edema
Hematoma
Pneumocephalus
Vessel occlusion/ischemia

(Reproduced, with permission, from Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 772.)

Obstructive Hydrocephalus

- Obstruction of CSF flow at the level of fourth ventricle or cerebral aqueduct.
- Can markedly ↑ ICP.
- Ventriculostomy usually placed prior to induction of general anesthesia.

Brain Stem Injury

Patients with cerebral atrophy are more at risk for pneumocephalus.

- Results from direct surgical trauma, retraction, or ischemia.
- Vital respiratory, circulatory centers, cranial nerves and nuclei can be affected.
- Signs include acute changes in blood pressure, HR, and heart rhythm.
- Communication with the surgeon is of utmost importance.
- Facial nerve monitoring is occasionally done but requires absence of muscle relaxant.

Pneumocephalus

- Air readily enters the subarachnoid space as CSF is lost during surgery.
- Can cause delayed awakening.
- Expansion of pneumocephalus can compress brain parenchyma after dural closure.
- Nitrous oxide not used in sitting craniotomies, risk of venous air embolism.

Unusual Positioning

- Surgery performed in modified lateral, prone position, or sitting position.
- Head is above the level of the heart.
- Protect pressure points in elbows, heels, forehead, and ischial spines.
- Excessive neck flexion:
  - Jugular venous obstruction → swelling of upper airway.
  - Quadriplegia from compression of cervical spinal cord, especially in patients with existing cervical spinal stenosis.

Venous Air Embolism

Highest incidence (40–45%) in patients operated in the sitting position but can occur whenever the surgical incision is above the level of the heart.

Pathophysiology

- Subatmospheric pressure in open vein → entrapment of air in right heart and pulmonary vasculature → mechanical obstruction of small arterioles/arteries and release of vasoactive agents → pulmonary vasoconstriction → V/Q mismatch → ↑ pulmonary vascular resistance → ↑ right ventricular (RV) afterload → ↑ CVP as RV fails → ↓ CO → hypotension.
- Air can directly enter arterial circulation via right-to-left shunts (patent foramen ovale) leading to paradoxical air embolism → stroke or MI.
- Monitoring techniques for venous air embolism:
  - TEE (most sensitive).
  - Precordial Doppler.
  - PA catheter.
  - $ETCO_2$.
  - Mass spectrometry (ETN_2) (least sensitive).

Presentation

↓ O_2 saturation → ↓ $ETCO_2$ → hypotension.

Treatment

- Flood surgical field with saline or pack entry site if identified.
- Discontinue nitrous oxide if used—can expand entrapped air.
- Compression of internal jugular veins.
- Aspirate entrained air with central venous catheter—many consider central venous catheter as necessity for sitting craniotomy.
- ↑ CVP: ↑ IV volume infusion, compression of internal jugular veins, PEEP (controversial—could reverse pressure gradient between left and right atria and allow passage of air across patent foramen ovale).
- Vasopressors if hypotension ensues.
- Place patient in head-down position and close wound.
- CPR if circulatory arrest occurs.

Optimal recovery of venous air embolism is with multiorifice catheter positioned at junction of SVC and right atrium.

Stereotactic Surgery

- Usually performed through a burr hole, a three-dimensional reference grid is attached to head with pins placed in outer table of the skull. It allows localization of a discrete area of brain for biopsy or evaluation.
- Anesthesia is usually given under local anesthesia and sedation. (*Note*: sedation should be given with caution since stereotactic approach usually precludes full access to the airway.)
- Used as treatment for:
  - Involuntary movement disorders.
  - Intractable pain.
  - Epilepsy.
  - Diagnosis and treatment of deeply situated brain tumors.

Transsphenoidal Resection of Pituitary Gland

Pituitary adenomas: Most are nonfunctional but may still present with endocrine deficiency secondary to mass effect compression of normal tissue.

Surgical Approaches

- Transphenoidal:
  - Advantages: Eliminates frontal lobe retraction, reduces blood loss, ↓ incidence of diabetes insipidus.
  - Disadvantages: ↑ risk of CSF leakage and meningitis, inaccessibility of tumor, bleeding from cavernous sinuses and carotid.
- Bifrontal craniotomy:
  - Advantages: Direct visualization, access to larger tumors.
  - Disadvantages: Damage to olfactory nerves, frontal lobe vasculature, and optic nerves.

Anesthetic Considerations

- **Cushing's disease:** Hyperglycemia, HTN, CHF, ↓ K^+, ↑ intravascular volume, muscle weakness
- **Acromegaly:** Difficult airway, hyperglycemia, HTN, CHF, V/Q mismatch, muscle weakness.
- Oropharyngeal packing to prevent bleeding into esophagus.

- Use of epinephrine and lidocaine in nasal mucosa to reduce bleeding.
- Placement of spinal needle into lumbar subarachnoid space for draining CSF or injecting air to facilitate surgical exposure (air delineates brain stem from pituitary on fluoroscopy).

Complications

Carotid artery spasm or hemorrhage, venous hemorrhage, venous air embolism, diabetes insipidus.

Monitoring

- ASA standard monitors.
- Limited access to airway: Secure ETT firmly, ensure IVs are working, confirm all monitors working properly prior to prepping and draping
- Arterial line placement depending on degree of blood loss and condition of patient.

Postoperative Management

- Patient should be told to breathe through his/her mouth (nasal packing placed postoperatively).
- Diabetes insipidus: Treat with IV fluids and subcutaneous vasopressin.
- Postoperative adrenal insufficiency: Treat with corticosteroids (avoid giving dexamethasone intraoperatively so that pituitary function can be monitored postoperatively).

Anesthesia for Head Trauma

- Approximately 50% of deaths due to trauma are from head injuries.
- Trauma patients tend to be adolescents, young adults, or > 75 years of age.
- Patients often have associated intra-abdominal injuries, long-bone fractures that result in significant blood loss, systemic hypotension, and hypoxia.
- Severity of head injuries correlates with:
  - Extent of irreversible neuronal damage.
  - Extent of secondary insults:
    - Seizures, infection, sepsis.
    - Formation and expansion of intracerebral, epidural, or subdural hematoma.
  - Intracranial HTN and cerebral edema.
- Surgical and anesthesia management goal is to prevent or minimize secondary insult through prevention of hypoxia, hypercarbia, hypotension, anemia, and hyperglycemia.
- Classification of severe head injury is based on Glasgow Coma Scale (GCS) (see Table 14-6.)
- Severe head injury is determined as GCS score ≤ 8 for more than 6 hr and is associated with ~35% mortality.
- ↑ morbidity seen in patients who suffered > 5mm midline shift, lesion larger than 25 mL, or ventricular compression on CT scan due to edema.

Glasgow Coma Scale:

- *Eye opening: 4 eyes*
- *Verbal response: Jackson 5*
- *Motor response: 6-cylinder engine*

Types of Lesions

- Skull fractures: ↑ the risk of intracranial pathology. Basilar skull fractures → CSF rhinorrhea, pneumocephalus, cranial nerve palsies, hemotympanum, raccoon eyes (ecchymosis into periorbital tissues).
- Subdural and epidural hematomas.

Battle's sign = ecchymosis behind the ears.

TABLE 14-6. Glasgow Coma Scale

Modified Glasgow Coma Scale	
Eye Opening	
Spontaneously	4
To verbal command	3
To pain	2
None	1
Best Verbal Response	
Oriented, conversing	5
Disoriented, conversing	4
Inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
Best Motor Response	6
Obeys verbal commands	5
Localizes to pain	4
Flexion/withdrawal	3
Abnormal flexion (decorticate)	2
Extension (decerebrate)	1
No response (flaccid)	

Mild head injury = 13–15; moderate = 9–12; severe = ≤ 8.

(Adapted, with permission, from Teasdale G, Jennett B. Assessment of coma and impaired consciousness: A practical scale. *Lancet* 1974; 2: 81; or Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 782.)

- Brain contusion and/or concussions.
- Penetrating head injuries.
- Traumatic vascular occlusions and dissections.

Operative Management

Usually reserved for depressed skull fractures, evacuation of hematomas, debridement of penetrating injuries.

Preoperative Evaluation

- **ABCs:** Patency of **A**irway, adequacy of **B**reathing (oxygenation* and ventilation), and adequacy of **C**irculation (hemodynamic stability).
- Associated injuries.
- Neurologic examination for gross and focal deficits.
- Preexisting chronic illnesses.
- Circumstances of the injury: Time, duration of unconsciousness, associated alcohol or drug use.
- Assume **all** head trauma patients have cervical spine injury until proven otherwise.
- In-line stabilization should be used during airway manipulations.

*Majority of head trauma patients are hypoxemic from pulmonary contusions, fat emboli, or neurogenic pulmonary edema. Neurogenic pulmonary edema is secondary to intense sympathetic nervous system activity, which causes systemic and pulmonary hypertension. Treatment includes intubation, mechanical ventilation, 100% oxygen, suctioning, diuresis.

ICP > 60 mm Hg (sustained) → irreversible brain damage.

Intubation

- Indicated for: Inadequate ventilation, absence of gag reflex, persistent GCS ≤ 8.
- Assume all patients have full stomachs requiring a rapid sequence induction with muscle relaxation.
- Use of succinylcholine is controversial in closed head injuries secondary to its potential to cause ↑ ICP and hyperkalemia.
- Awake fiber-optic intubation or tracheostomy may be necessary if difficult intubation is anticipated.
- Blind nasal intubation is *contraindicated* in basilar skull fractures.

Hypotension

0**Etiology:** Related to other associated injuries such as intra-abdominal bleeding. Spinal cord injuries → sympathectomies (especially lesions to T1–4) → spinal shock (vasodilatation and hypotension).

Treatment

- Fluid resuscitation with colloid and blood for intravascular volume expansion.
- Temporary vasopressor usage (dopamine).
- Avoid glucose containing crystalloid solutions.

Intraoperative Management

- **Monitoring:** Arterial lines, CVP, or PA catheterization may be needed but not at the expense of delaying surgical intervention in rapidly deteriorating patient.
- **Maintenance:** Usually with various combinations of barbiturates, benzodiazepines, opioids, and sub-MAC concentrations of inhalational agents and muscle relaxants.
- Maintain normocarbia if ICP is normal.
- Intraoperative HTN: Surgical stimulation under light anesthesia.
  - ↑ ICP (+ bradycardia + HTN) = Cushing's triad.
  - Treatment: Deepen anesthesia, pain control, β-adrenergic blockade.
  - Maintain CPP between 70 and 110 mmHg.
- Disseminated intravascular coagulation (DIC).
  - Severe head injuries → release of brain thromboplastin.
  - Diagnosis: Coagulation studies.
  - Treatment: Fresh frozen plasma, cryoprecipitate.
- Acute respiratory distress syndrome:
  - May be associated with DIC.
  - Pulmonary aspiration and neurogenic pulmonary edema.
  - Treatment mechanical ventilation with PEEP (apply PEEP only after dura is opened or ICP is actively monitored).

Extubation

- Consider the severity of injury, patient's preoperative mental status, comorbid conditions, coexisting injuries.
- Patients with ↑ ICP need sedation, paralysis, hyperventilation; thus, should be left intubated.

Aneurysms > 7 mm are considered for surgical obliteration.

Anesthesia for Cerebral Aneurysms

- Typically occur at the bifurcation of large arteries at the base of the brain—majority occur in the anterior circle of Willis.
- Saccular aneurysms:
  - ~5% incidence.
  - Only a few have complications.
  - Rupture of saccular aneurysm—most common cause of SAH.
  - 10% acute mortality.
  - 25% of survivors die from delayed complications.
  - 50% of survivors suffer neurologic deficits.
- Emphasis on prevention of rebleeding from aneurysm.
- Treatment of aneurysms includes endovascular coiling or craniotomy with clipping.

Unruptured Aneurysms

- Most common presentations:
  - Headaches.
  - Third-nerve palsy.
  - Seizures.
  - Visual field defects.
  - Trigeminal neuralgia.
  - Cavernous sinus syndrome.
  - Hypothyroidism.
  - Pituitary dysfunction.

Diagnosis

- Angiography
- MRI angiography or helical CT angiography.

Ruptured Aneurysms

- Factors → rupture:
  - Large aneurysm size.
  - Weak aneurysm wall.
  - Previous rupture.
  - Large transmural pressure (TMP) gradient: TMP = MAP – ICP.
- Common presentations from major bleeding:
  - Subarachnoid hemorrhage: Sudden severe headache, nuchal rigidity, photophobia, nausea/vomiting, loss of consciousness.
  - Epidural hemorrhage or cerebral hemorrhage—less common.
  - Severe headache and/or nausea/vomiting without focal neurologic deficits.
  - Transient loss of consciousness from sudden ↑ in ICP and ↓ in CPP (persistent ↑ in ICP → death).

Triple H therapy: Hemodilution, hypertension, hypervolemia.

- Common presentations from minor bleeding:
  - Mild headache.
  - Vomiting.
  - Nuchal rigidity.
- Delayed complications (secondary to cerebral ischemia and infarction):
  - Rerupture within 48 hr, 30% incidence with 60% mortality.
  - Cerebral vasospasm within 3–12 days.
  - Intracranial HTN: Normalizes by one week, can cause intracerebral hemorrhage.
  - Hydrocephalus: 20% incidence.
  - Seizures.
- *Note:* Glucocorticoids do not reduce cerebral edema following rupture.

Anesthetic Considerations

- **Preoperative evaluation and management:**
  - Determine patient's surgical risk grade (see Table 14-7).
    - Grade 0-2 SAH: Normal ICP; CPP is not compromised and sedation can be given preoperatively.
    - Grade 3-5 SAH: ↑ ICP, CPP compromised, avoid sedation and hypercarbia.
  - Comorbid conditions, especially HTN, renal, cardiac, cerebrovascular disease.
  - Degree of vasospasm, if any.
  - ECG changes may be often seen with SAH, but they do not necessarily reflect cardiac disease. These include arrhythmias, ST changes, T-wave inversions.
- Blood should be available in the room.
- Intraoperative management: Smooth induction (and emergence) using lidocaine, β blockers, opioid to avoid large ↑ in transmural pressure (TMP) during laryngoscopy.
- BP management on induction:
  - Balance risk of elevated TMP = MAP – ICP vs. risk of ↓ CPP = MAP – ICP.
    - Grade 0–2 SAH: Normal ICP, therefore CPP will be preserved if we try to minimize TMP. BP can be maintained ± 20–25% baseline.

TABLE 14-7. Hunt-Hess Classification of Intracranial Aneurysms According to Surgical Risk

Grade	Characteristics
0	No bleed, incidental finding.
I	Asymptomatic or minimal headache and slight nuchal rigidity.
II	Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy.
III	Drowsiness, confusion, mild focal deficit.
IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity, vegetative disturbances.
V	Deep coma, decerebrate rigidity, moribund.

(Adapted, with permission, from Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneuryms. *J Neurosurg* 1968; 28: 14; or Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 777.)

    - Grade 3–5 SAH: ↑ ICP. CPP will be ↓ if we try to minimize TMP. BP should be maintained around the baseline BP.
  - Arterial line and CVP or PA pressure monitoring.
  - Foley catheter to guide fluid therapy.
  - Avoid calcium channel blockers (vasodilation → hypotension).
  - Osmotic diuretics (mannitol): ↓ ICP, facilitate surgical exposure, and ↓ tissue swelling. Mannitol is typically given on skin incision, such that its peak effect will occur on opening of dura. It should be given as a slow infusion to avoid transient ↑ in ICP. Also has a cerebral protective effect via free-radical scavenging in ischemic areas.
- Hyperventilation: Avoid in grade 0–2 SAH, as it may raise the TMP and cause rebleeding of aneurysm. Mild hyperventilation may be useful to ↓ ICP in grade 3–5 SAH but must be done gradually to avoid rupturing aneurysm.
- Controlled hypotension:
  - ↓ intraoperative blood loss.
  - ↓ TMP pressure on aneurysm ↓ risk of rupture.
- Temporary clips: Occlusive clips placed on afferent arteries of aneurysm for up to 20 min.
  - ↓ risks of systemic hypotension.
  - Requires normal to supra-systemic BP to maintain perfusion through collaterals during clipping.
- Mild hypothermia for brain protection.
- Remifentanil and desflurane commonly used to allow rapid wake-up and neurological exam.

Arteriovenous Malformation

AVM bleeding most common in age group: 30 year olds to 50 year olds.

- Congenital abnormalities can cause fistulas between arteries and veins.
- Parenchymal bleeding is more common than subarachnoid bleeding, as opposed to aneurysms where SAH is more common.
- Can present at any age but most commonly at ages 30–50.

Treatment

- Embolization and stereostatic radiosurgery.
- Surgical excision (be prepared for the extensive blood loss and have blood available in OR).
- Embolization can be performed prior to surgical excision to ↓ blood loss.

Anesthesia for Spine Surgery

Indications

- Symptomatic nerve root or spinal cord compression.
  - Protrusion of intervertebral disk.
  - Osteophytic bone (spondylosis) into the spinal canal.
- Disk herniation: Usually occurs at L4–L5 or C5–C6 levels in 30- to 50-year-olds.
- Correction of spinal deformities (scoliosis).
- Decompression of the spinal cord.
- Fusion of spine following spinal trauma.
- Resection of a tumor, abscess, or vascular malformation.

Preoperative Management

- Effect of spinal pathology/deformity on pulmonary function.
- Recognition of neurologic deficits.
- Airway assessment particularly limited neck range of motion and anatomic abnormalities.
- Preoperative level of pain.

Intraoperative Management

- **Concerns:**
  - Many spinal operations performed in the prone position.
  - Multilevel, fusion, instrumentation → potential for extensive blood loss and nerve injury.
  - Transthoracic approach → need for one-lung ventilation.
  - Possibility of visual loss following spine surgery.
- **Positioning:**
  - Majority in prone position. Be mindful to prevent abrasions or pressure necrosis on eyes, nose, ears, breasts, male genitalia.
  - Head to side or down.
  - Chest on parallel rolls to facilitate ventilation.
  - Abdominal compression prevention to avoid impedance of venous return.
  - Anterior approach for cervical spine → potential for injuries to trachea, esophagus, laryngeal nerves, carotid arteries, jugular veins, sympathetic chain.
- **Monitoring:**
  - Standard ASA monitors.
  - Arterial line and/or CVP prior to repositioning if and prone position.
  - Use of SSEPs or MEPs.

C3, 4, 5 "keeps the diaphragm alive."

Acute Spinal Cord Injury

Pathophysiological Effects

- **Respiratory:**
  - ↓ respiratory function with lesions at T6 or higher.
  - Sleep apnea (Ondine's curse): Lesions involving C2–C4.
  - Diaphragmatic paralysis.
  - Lesion at C4 or higher: Paralyzed diaphragm with ventilation impairment.
  - Lesion at C5: Partially paralyzed diaphragm with reduced ventilation.
  - Lesion at C6: Diaphragm intact.
  - Intercostal paralysis.
- **Gastrointestinal:** Aspiration risk secondary to ↑ secretions from paralyzed sympathetic nervous system, gastric dilation, and ineffective cough.
- **Cardiovascular:** Sympathetic discharge involving HTN and tachycardia immediately following injury followed by parasympathetic reflexes involving arrhythmias, bradycardia if cardioaccelerator fibers (T1–T4) affected.
- **Spinal shock:** Loss of vascular tone and vasopressor reflex with flaccid paralysis below level of lesion, lasts a few hours to several weeks.
- **Metabolic:**
  - Hyperkalemia: Succinylcholine administration within 48 hours of injury is considered safe. Serum potassium levels peak between 4 weeks

and 5 months and succinylcholine could precipitate ventricular fibrillation if administered due to proliferation of acetylcholine receptors and their super-sensitivity to depolarizing muscle relaxants.
    - Hypercalcemia: Reduced muscle activity causes a mobilization in calcium, starts 10 days after injury and peaks 10 weeks after injury; watch for arrhythmias.
- **Thermoregulation:** Lesion above C7 abolishes sweating and hyperthermia may ↑ oxygen demand.

May use succinycholine > 6 months after injury, but with caution.

Anesthestic Concerns

- Improved ventilation in spontaneously breathing quadriplegics in supine (vs. upright) position: ↓ end-expiratory volume and ↑ vital capacities secondary to greater excursion of diaphragm.
- Anticipate difficult intubation and consider awake fiber-optic intubation.
- Cardiovascular management: High lesions risk hypotension and spinal cord ischemia yet fluid overload risks pulmonary edema, consider PA catheter to guide fluid management in addition to arterial line and urine output.
- Sensory and evoked potentials during instrumentation of vertebral column.
- Methylprednisone: Improves outcome if started within 8 hours of injury; 30 mg/kg bolus followed by 5.4 mg/kg/mL × 24–48 hr.

Autonomic Hyperreflexia

- Acute sympathetic hyperreactivity in response to stimulation below T4–7 lesions.
- Prevalent in 66–85% of patients with spinal cord injury.
- Appears from 6 months to 2 years from time of injury.
- Triggers: Distention of hollow viscus (usually bladder distention), spasm or distension of other viscera, stimulation of skin.

Pathophysiology of Reflex

Lack of supraspinal inhibition of signals arising from afferent pathways originating in mucosa and muscle of hollow organs and ascending to spinothalamic tracts and dorsal columns → unopposed sympathetic output to stimulus → sympathetic stimulation below lesion and parasympathetic stimulation above lesion.

Presentation

- Paroxysmal HTN and compensatory bradycardia, dysrhythmias, vasoconstriction below and vasodilation above lesion.
- Symptoms: Sweating, flushing, nasal obstruction, headache, breathing difficulties, nausea, shivering, visual changes.
- Complications of untreated hypertensive crisis: Seizures, intracranial hemorrhage, MI.

Factors Affecting Severity of Response

- Lesion level:
    - T5 or above: Full syndrome.
    - Between T5–10: Mild BP elevation with some symptoms.
    - T10 or below: Minimal BP response and no sweating.
- Trigger level: The more caudad the stimulus, the greater the sympathetic response.

Regional anesthesia and general anesthesia are both effective in the prevention of hyperreflexia response.

Prevention and Treatment

- Topical anesthesia.
- Spinal anesthesia: For procedures in lower extremities and lower abdomen.
- Epidural anesthesia: Not reliable; often miss S2–S4 segments, which give the strongest stimuli (located furthest from lesion).
- GA: Reliable if good depth achieved.
- BP control:
  - Adrenergic α blocker: Not effective, act on circulating NE not NE release at nerve terminals, which is thought to be the mechanism.
  - Nitroprusside: For acute crisis.
  - Calcium channel blocker: For prophylaxis and acute crisis.

CHAPTER 15

Anesthesia for Trauma

TRAUMA STATISTICS

- Most common cause of death for ages 1–45.
- Overall mortality: Heart > cancer > cerebrovascular > trauma.
- One-third die within first 4 hours.
- Three-fourths die within 48 hours.
- Most common causes: motor vehicle accident (MVA), falls, poisoning, fires, drowning.

Trauma patients have an ↑ likelihood of drug abuse, intoxication, and to be carriers of hepatitis or HIV.

INITIAL ASSESSMENT

- Assumptions for all trauma patients:
  - Full stomach precautions.
  - Require in-line cervical stabilization.
  - Altered mental status secondary to head injury until proven otherwise.
  - Partial airway obstruction can rapidly progress to complete airway obstruction.
  - Hypotension secondary to hypovolemia until proven otherwise.
- Initial assessment (see Figure 15-1).
- Rapid overview: Stable, unstable, dead or dying.
- Primary survey: ABCs, neuro exam, quick exam of the undressed patient.
- Secondary survey: Systematic examination of body.

Airway

- In-line cervical stabilization *before* any airway manipulation (semirigid collar, bindings, backboard).
- Maintain patent airway by removing any secretions, debris, blood, or vomit.
- All patients should receive supplemental oxygen.
- Consider prophylactic intubation.

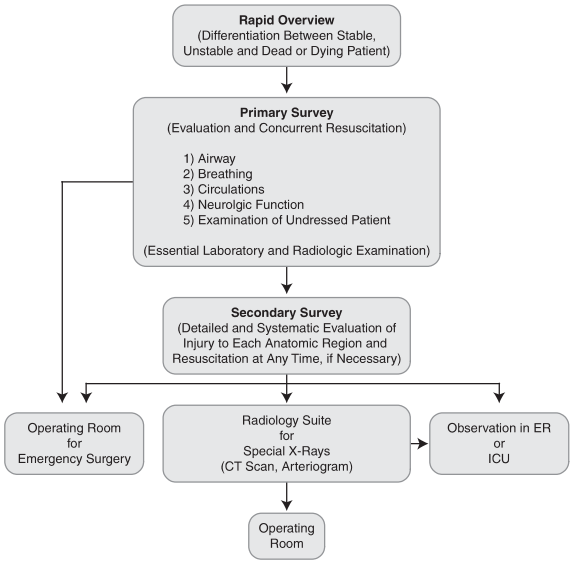

FIGURE 15-1. Clinical sequence for initial management of the major trauma patient. CT, computed tomography; ER, emergency room; ICU, intensive care unit.

(Reproduced, with permission, from Barash PG et al (eds.). *Clinical Anesthesia,* 6th ed. Lippncott Williams & Wilkins, 2009: 1263.)

- Confirm placement of endotracheal tube (ETT) in patients intubated in the field.
- Indications for intubation in trauma:
  - Apnea.
  - Obstruction.
  - Unconsciousness.
  - Severe head injury (Glasgow Coma Scale [GCS] < 9).
  - Maxillofacial trauma.
  - Penetrating neck injury with expanding hematoma.
  - Major chest injuries.
  - Aspiration prevention.
  - Impending respiratory failure.

Head Injury

- Requires deep anesthesia and muscle relaxants prior to induction to prevent hypertension (HTN), bucking, coughing (which cause ↑ intracranial, intraocular, and intravascular pressures).
- Preoxygenation.
- Avoid ketamine—can increase intracranial and systemic vascular pressures.
- Avoid nasotracheal intubation or orogastric (OG)/nasogastric (NG) tube in basilar skull fractures.

Cervical Spine Injury

- Factors increasing risk of cervical instability: Neck pain, severe distracting pain, neurological signs/symptoms, intoxication, loss of consciousness.
- In-line stabilization during direct laryngoscopy, nasal intubation or fiberoptic intubation.

Airway Obstruction

Primary survey:

A = *airway*

B = *breathing*

C = *circulation*

D = *disability*

E = *exposure*

Causes

- Displaced or lacerated pharyngeal soft tissues.
- Hematoma, bleeding, secretions, foreign bodies.
- Displaced bone or cartilage fragments.

Signs/Symptoms

Dyspnea, hoarseness, stridor, dysphonia, subcutaneous emphysema, hemoptysis.

Treatment

Chin lift, jaw thrust, clearing of oropharyngeal cavity, placement of oro/nasopharyngeal airway, bag ventilation, intubation, cricothyroidotomy.

Direct Airway Injuries

- **Maxillofacial:** Oropharyngeal hematoma or edema → airway compromise and obstruction.
- **Cervical airway:**
  - Blunt trauma:
    - Asymptomatic, hoarseness, muffled voice.

- Stridor, dysphagia, odynophagia.
- Cervical pain and tenderness.
- Ecchymosis, subcutaneous emphysema, flattening of Adam's apple.
- Penetrating trauma: Escape of air, hemoptysis, coughing.

- **Thoracic airway:**
  - Blunt trauma:
    - Occurs at membranous portion of trachea and mainstem bronchi.
    - Associated with pneumothorax, pneumomediastinum, pneumopericardium, subcutaneous emphysema, continuous air leak from chest tube.
  - Penetrating trauma: Occurs at any segment of intrathoracic airway.

Breathing

- Assess for cyanosis, accessory muscle use, flail chest, penetrating chest injuries, subcutaneous emphysema, tracheal shift, broken ribs, presence/absence, diminution of breath sounds
- Controlled ventilation with FiO_2 of 1.0 until oxygenation assessed by arterial blood gas (ABG).

Circulation

- Hypotension in trauma is due to hypovolemia until proven otherwise.
- Vital signs such as heart rate (HR), blood pressure (BP), urine output (UO), respiratory rate (RR) reflect the severity of hemorrhagic shock (see Table 15-1).
- Priorities: Stop bleeding and replace intravascular volume.

Resuscitation in pregnant women in shock is successful only after delivery of the fetus.

TABLE 15-1. Classification of Hemorrhagic Shock

	Class I	Class II	Class III	Class IV
EBL (mL)	< 750	750–1500	1500–2000	> 2000
EBL (% blood volume)	< 15	15–30	30–40	> 40
Pulse rate (per min)	< 100	> 100	> 120	> 140
BP	Normal	Normal	↓	↓
Pulse pressure	Normal	↓	↓	↓
RR (breaths/min)	14–20	20–30	30–40	> 35
UO (mL/hr)	> 30	20–30	5–15	Negligible
Mental status	Slightly anxious	Slightly anxious	Anxious and confused	Confused and lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

BP, blood pressure; EBL, estimated blood loss; RR, respiratory rate; UO, urine output.

(Adapted from The American College of Surgeons. Shock. In: The American College of Surgeons (ed) *Advanced Trauma Life Support.* 1990, p. 59–73.)

- Methods to stop bleeding:
  - Extremities: Pressure dressings and packs.
  - Chest trauma: Slows bleeding when lung is reinflated following chest tube drainage.
  - Abdominal: Bleeding tamponaded within cavity, pneumatic antishock garments. ↓ bleeding in abdomen and lower extremities and ↑ pulmonary vascular resistance (PVR) and ↑ perfusion of heart and brain (contraindicated for bleeding in thorax or head).
- Intravascular volume replacement:
  - Two large-bore IVs or surgical cutdown.
  - Warm crystalloid resuscitation before starting blood transfusion.
  - **Crystalloid:**
    - Lactated Ringer's (LR): Less likely to cause hyperchloremic acidosis, incompatible with packed red blood cells (PRBC) secondary to calcium, can aggravate cerebral edema in large doses.
    - Normal saline (NS): More likely to cause hyperchloremic acidosis.
    - Dextrose-containing solutions: Exacerbate ischemic brain damage.
    - Hypertonic NS (3% or 7.5%): Associated with less cerebral edema than LR, hypernatremia.
  - **Colloid:**
    - Albumin: Effective in restoring intravascular volume, expensive.
    - Dextran/hetastarch: Avoided due to coagulopathic potential.
  - PRBC: Uncrossed O negative or type specific until fully crossmatched blood available (usually after 45–60 min).
- Signs of adequate fluid replacement:
  - HR< 100.
  - UO > 0.5 mg/kg/hr.
  - Absence of metabolic acidosis.
  - Minimal effects of positive pressure ventilation (PPV) on systolic blood pressure (SBP).
- Causes of shock:
  - Hemorrhagic (most common).
  - Cardiac: Contusion, tamponade, valvular injury, preexisting coronary artery disease (CAD).
  - Pneumothorax, hemothorax.
  - Spinal cord injury.
  - Anaphylaxis.
  - Sepsis.

In hypovolemia:

- *Tachycardia may be absent secondary to ↑ vagal tone or chronic cocaine use.*
- *SBP may be maintained secondary to ↑ catacholamines and pain.*

Hemorrhage

Etiology

Hypovolemia.

Signs/Symptoms

- Tachycardia.
- Narrow pulse pressure.
- Cold, clammy skin.

Treatment

Initial crystalloid resuscitation → RBC transfusion if no response post-2L crystalloid over 15 min.

Myocardial Contusion

Etiology

↑ PVR and ↓ ventricular performance.

Signs/Symptoms

Dysrhythmia, tachycardia, hypotension.

Treatment

Fluids → vasodilators → inotropes.

Cardiac Tamponade

Etiology

↓ venous return.

Myocardial contusion can be diagnosed by ECG (possible signs of ischemia), elevated cardiac enzymes, or abnormal ECHO.

Signs/Symptoms

- Beck's triad (hypotension, distended neck veins, muffled heart sounds).
- Paradoxical pulse (> 10 mmHg ↓ BP with inspiration).
- Pulsus electrical alternans.
- Tachycardia.

Treatment

Pericardiocentesis → pericardial window → thoracotomy.

Pneumothorax or Hemothorax

Etiology

Lung collapse → mediastinal shift → inflow and outflow obstruction of the heart.

Signs/Symptoms

- Tachycardia.
- Hypotension.
- Distended neck veins.
- Absent breath sounds.
- Hyperresonance to percussion.
- Tracheal shift.
- Dyspnea.
- Subcutaneous emphysema.

Treatment

- Placement of 14-gauge catheter → chest tube placement.
- Maintain filling pressures.
- Avoid PPV, ketamine as induction agent of choice.

Spinal Cord Injury

Etiology

Sympathectomy → vasodilation.

Signs/Symptoms

- Hypotension without tachycardia.
- Cutaneous vasoconstriction.
- Narrow pulse pressure.

Treatment

Fluids → vasopressors → inotropes.

Venous distention in the lower extremities may be a sign of spinal cord injury.

Sepsis

Etiology

Intestinal perforation → peritoneal contamination.

Signs/Symptoms

Gradual onset, fever, tachycardia, wide pulse pressure.

Treatment

Fluids and antibiotics → inotropes.

HEAD TRAUMA

- Cause of 40% of deaths in trauma patients.
- Contributor to secondary brain injury: Hypotension, hypoxia → lactic acidosis, free radical generation, lipid peroxidation, prostaglandin synthesis, glutamate release, cell membrane breakdown → brain edema.

Complications

- Intracranial hemorrhage (ICH), herniation, seizures.
- Neurogenic pulmonary edema.
- Dysrhythmias, bradycardia, hypertension.
- Coagulopathy.

Diagnosis

- Glasgow Coma Scale (see Chapter 14, Table 14-6).
- Physical exam:
  - Compression of CN III nerve by uncus → dilated and sluggish pupil
  - Uncal herniation → dilated, fixed, "blown" pupil.
- Computed tomography (CT): Assess for midline shift, distorted ventricles and cisterns, effacement sulci, hematoma.

- Subdural: Concave.
- Epidural: Convex, "lenticular."

Anesthetic Management

- Goal is to avoid secondary brain injury.
- Maintain CPP > 60 mmHg.
- Maintain oxygen delivery with $SpO_2 > 95$.
- Maintain SBP > 60 mmHg: Impaired cerebral autoregulation means cerebral perfusion dependent on SBP.
- Reduction of intracranial pressure (ICP):
  - Mannitol: 0.25–0.5 g/kg every 4–6 hr, SE: hyponatremia, high serum osmolality, transient hypervolemia (caution in congestive heart failure [CHF]).
  - Lasix: Adjunct with mannitol.
  - Hyperventilation for short periods of time.
  - Elevation of head 30 degrees.
  - Steroids.
  - Cerebrospinal fluid (CSF) drainage.
  - Barbiturates.
- Avoid hyperthermia.
- Correct electrolyte abnormalities/coagulopathies.
- Maintain normovolemia: Use non-glucose-containing crystalloids or colloids.

NECK INJURY

Causes

- Trauma to C-spine, vessels, respiratory, and digestive tracts and CNS → hemorrhage, asphyxia, mediastinitis, paralysis, stroke, or death.
- Cervical vascular injury: Airway compromise or obstruction, brisk bleeding, expanding pulsatile hematoma, shock; requires immediate airway management and vascular control.
- Cervical arterial injury: ↓ or absent upper extremity/distal carotid pulses/carotid bruit/thrill, hemothorax, pneumothorax, air embolism, respiratory distress, stridor, dysphonia, hoarseness, cough, hemoptysis, cyanosis, air bubbling from wound, subcutaneous crepitus, laryngeal tenderness.
- Esophageal injury: Dysphagia, odynophagia, hematemesis, subcutaneous crepitus, prevertebral air on lateral cervical radiograph.

High thoracic lesions lead to bradycardia (T1–T4).

SPINE AND SPINAL CORD INJURY

Causes

MVA, falls, penetrating trauma with neurologic deficit.

Signs/Symptoms

- Flaccid areflexia, loss of rectal sphincter tone, diaphragmatic breathing, bradycardia in hypovolemic patients, pain and tenderness over vertebrae.
- **Spinal shock:**
  - **Cause:** Loss of sympathetic tone in vessels below level of lesion.
  - **Signs/symptoms:**
    - Hypotension.
    - Bradycardia.
    - Areflexia.

- Gastrointestinal atony.
- Lower-extremity venous distention.

- **Spinal cord injury:**
  - Incomplete: Intact sensory perception over sacral distribution and voluntary contraction of anus (sacral sparing); 50% of patients regain neurologic functions.
  - Complete: Unlikely to regain neurologic functions.

Diagnosis

Chest x-ray (CXR), computed tomography (CT).

Treatment

Steroids, immobilization, intubation/ventilation.

CHEST INJURY

Chest Wall Injury

- Rib fractures causing pneumothorax or hemothorax.
- Three or more fractured ribs or lower rib fractures have greater risk of hepatic and splenic injury.
- First rib fracture indicates trauma to major vessels.
- Scapular fractures indicate damage to heart and lungs.
- Sternal fractures common in elderly females in MVAs wearing seat belts.

Avoid N_2O in trauma patients as it may worsen a developing/undiagnosed pneumothorax.

Treatment

Thoracic epidural pain relief or opioids.

Flail Chest

Causes

Comminuted fractures of at least three adjacent ribs, rib fractures with associated costochondral separation, sternal fracture.

Signs/Symptoms

Paradoxical chest wall movement, splinting.

Diagnosis

Physical findings, CXR, ABG.

Treatment

- Better outcomes with adequate pain control over mechanical ventilation.
- Supplemental oxygen, continuous positive airway pressure (CPAP), airway humidification.
- Chest physiotherapy, incentive spirometry.
- Bronchodilators, airway suctioning.
- Continuous epidural analgesia.
- Intubation.

Pneumothorax

Causes

Displaced rib fracture, missile, or stab wounds.

Signs/Symptoms

- Dyspnea, tachycardia, cyanosis.
- Agitation, diaphoresis.
- Neck vein distention, tracheal deviation to contralateral side.
- Maximal cardiac impulse displaced to contralateral side.
- Subcutaneous emphysema.
- Diminished breath sounds.

Diagnosis

- CXR: Upright films not always easily obtained.
- CT: Definitive.

Treatment

- Observation for small pneumothorax.
- 14-gauge angiocatheter in fourth intercostal space in midaxillary line followed by placement of 26–32 Fr chest tube.
- For open pneumothorax: Cover the defect on three sides to allow it to function as one-way valve preventing air entry into pleural cavity but allowing exit during expiration, chest tube placement, intubation.

Anatomy below the rib:

V = *vein*

A = *artery*

N = *nerve*

Hemothorax

Cause

Bleeding intercostal vessels → severe airway deviation.

Treatment

- Drainage with 30–40 Fr chest tube.
- Thoracotomy: Indications—1 L initial drainage of 150–200 mL/hr for 2–4 hrs, "white lung" on CXR, continuous air leak from chest tube.
- Video-assisted thoracoscopic surgery (VATS): Indications—hemodynamically stable patients with persistent bleeding.

Systemic Air Embolism

- Occurs after penetrating lung trauma and blast injuries and involves lacerations of both air passage and pulmonary vein.
- When exacerbated by PPV → air enters into systemic circulation via pulmonary veins.

Signs/Symptoms

Hemoptysis, circulatory and CNS dysfunction after ventilation.

Treatment

- Double-lumen tube.
- Low tidal volumes through single lumen ETT.
- Thoracotomy and clamping hilum of lacerated lung.

Penetrating Cardiac Injury

- Stable: Transthoracic echocardiography (TTE).
- Unstable: Immediate sternotomy or left thoracotomy.

Pericardial Tamponade

Signs/Symptoms

- Tachycardia.
- Beck's triad (distant heart sounds, distended neck veins, hypotension).
- Pulsus paradoxus (10 mmHg decline in BP during inspiration).
- Pulsus alternans.

Diagnosis

TTE/TEE.

Treatment

Intravenous fluids, pericardiocentesis.

Myocardial Contusion

Signs/Symptoms

Angina-like pain, dysrhythmias, right-/left-sided CHF.

Diagnosis

- Echocardiogram.
- Cardiac enzymes.
- Electrocardiogram (ECG): ST/T changes, axis shift, bundle branch block, dysrhythmias.

Thoracic Aortic Injury

Occurs at proximal portion of descending aorta just distal to subclavian artery as aortic arch is fixed while descending aorta is relatively mobile.

Most common dysrhythmias in myocardial contusion are sinus tachycardia with nonspecific ST changes.

Diagnosis

- Widened mediastinum on CXR.
- Spiral CT.
- Ultrasound.

Diaphragmatic Injury

Abdominal herniation occurs at left side with blunt trauma (liver protects right diaphragm).

Signs/Symptoms

Breathing abnormalities, persistent tachycardia, dysrhythmias.

ABDOMINAL AND PELVIC INJURY

- **Penetrating trauma:**
  - Associated with liver injury.
  - Gunshot wounds: Exploratory laparotomy or laparoscopy.
  - Stab wounds → diagnostic peritoneal lavage (DPL) → laparotomy.
- **Blunt trauma**:
  - Associated with splenic rupture.
  - Focused abdominal sonography for trauma (FAST): Identifies intraperitoneal hemorrhage or pericardial tamponade.
    - + Free fluid: Stable—CT; unstable—laparotomy.
    - – Free fluid: Stable—follow-up; unstable: repeat FAST.

Pelvic Fracture

- Causes major hemorrhage in 25% and exsanguinations in 1%.
- Bone fragments disrupt veins.
- Retroperitoneal bleeding:
  - Mostly self-limited secondary to tamponade effect.
  - Eighteen to twenty percent have uncontrolled arterial bleeding.

Treatment

- External pelvic fixation to control blood loss.
- Angiography and embolization of bleeding site.

A femoral fracture can have 1–3 units of occult blood loss.

EXTREMITY INJURIES

- All extremity fractures should be repaired as soon as possible.
- Delayed repair associated with ↑ risk of deep venous thrombosis (DVT), pneumonia, sepsis, fat embolism, and infection.

Fat Emboli

Associated with pelvic and long-bone fractures.

Signs/Symptoms

Pulmonary insufficiency, dysrhythmias, petechiae, mental deterioration.

Diagnosis

Elevated lipase, fat in urine, thrombocytopenia

Compartment Syndrome

Signs/Symptoms

Severe pain in affected extremity, swelling, tenseness, calf pain on dorsiflexion.

Diagnosis

Compartment pressure > 50 cm H_2O.

TREATMENT

Immediate fasciotomy.

BURNS

Classification

Based on depth and size of burn:

- **First degree:** Superficial partial-thickness.
  - Affects epidermis and upper dermis.
  - Red, blanches to touch, sensitive to painful stimuli and heat.
  - Heals spontaneously.
- **Second degree:** Deep partial-thickness.
  - Affects deep dermis.
  - Red, blanches to touch, blisters, sensitive.
  - Requires excision and grafting.
- **Third degree:** Full-thickness.
  - Nonblanching, insensate.
  - Requires excision and grafting.
- **Fourth degree:**
  - Involves muscle, fascia, and bone.
  - Requires complete excision with limited function.
- **"Rule of Nines":**
  - Estimate of size of burned area as fraction of total body surface area (TBSA) in adults (in children the proportions change, depending on age and size; see Figure 15-2).
  - Adult: 9% head, 18% upper extremity (9% for each arm), 36% trunk, 36% lower extremity (18% each leg).
  - 1-year-old: 19% head, 9.5% upper extremity, 32% trunk, 15% lower extremity.
- **Major burns:**

Types of burns:

Electrical

Chemical

Thermal

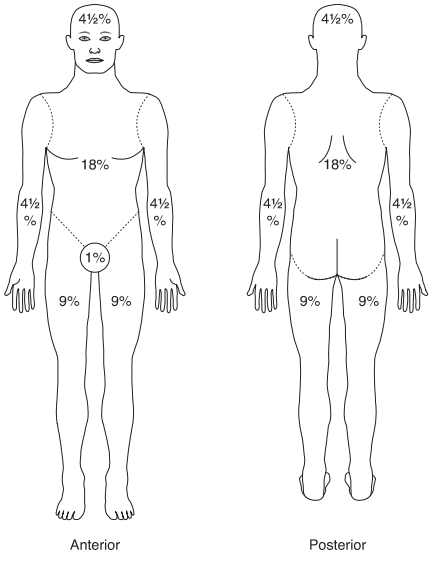

FIGURE 15-2. Rule of Nines

- Second-degree burn involving > 25% of TBSA in adults and > 20% in children/elderly.
- Third-degree burn involving > 10% of TBSA.
- Burns involving face, hands, feet, or perineum.
- Inhalation, chemical and electrical burns.
- Burns in patients with severe preexisting medical conditions.

Airway and Ventilatory Complications

Airway injury to pharynx or trachea can cause respiratory distress.

Signs/Symptoms

- Singed facial hair, facial burns.
- Dysphonia or hoarseness, cough.
- Soot in mouth or nose.
- Swallowing difficulties.
- **Pathophysiology and complications:**
  - ↓ surfactant and mucociliary function.
  - Edema.
  - Secretions → bronchial obstruction, air trapping, and bronchopneumonia.

Management

- Administer high FiO_2 by face mask to spontaneously breathing patients.
- Avoid anesthetics and muscle relaxants in significant airway obstruction or a possible difficult intubation.
- Indications for intubation: Massive burns, stridor, respiratory distress, hypoxemia, hypercarbia, loss of consciousness, altered mental status.
  - **Adults:** Awake fiber-optic with topical anesthesia.
  - **Children:** Inhalation induction; consider prophylactic intubation prior to respiratory distress, as minimal swelling in these patients can occlude airway.
  - Surgical standby for cricothyroidotomy.

Ventilatory Complications

- Acute pulmonary edema: Occurs in first 36 hr.
- Atelectasis, bronchopneumonia, edema: Occur from days 2 to 5.
- Acute respiratory distress syndrome (ARDS), nosocomial pneumonia, pulmonary embolism (PE): After day 5.

Beta agonists may aid in treating bronchospasms due to inhalation injuries.

Fluid Management

- Fluid loss secondary to:
  - ↑ microvascular permeability.
  - ↑ intravascular hydrostatic and interstitial osmotic pressures.
  - ↓ interstitial hydrostatic pressure.
  - ↓ cardiac contractility secondary to ↓ catecholamine response, ↓ coronary blood flow, and ↑ systemic vascular resistance (SVR).
- Replacement fluid types:
  - Isotonic (NS/LR): Most commonly used in thermal injuries; side effect: ↓ plasma protein concentration.

  - Hypertonic: ↓ fluid volume requirements; side effects: hypernatremia, intracellular water depletion.
  - Colloids: Can counteract edema effects of isotonic solutions; side effect: associated with high mortality in critically injured.
- Calculated volumes:
  - Adults and children > 20 kg:
    - Modified Brooke formula: 2 mL LR/kg/%burn/24 hr.
    - Parkland formula: 4 mL crystalloid/kg/%burn/24 hr.
  - Children < 20 kg: Crystalloid 2-3 mL/kg/%burn/24 hr.
- Volume infusion rate:
  - 50% of calculated volume in first 8 hr.
  - 25% in second 8 hr.
  - 25% in last 8 hr.
- Resuscitation goals:
  - UO: 0.5 mL/kg/hr.
  - HR: 80–140.
  - Minimal base deficit: < 2–5 mmol/L.
  - Normal lactate.

Anesthetic Management

- Muscle relaxants:
  - Succinylcholine: Avoid > 24 hr up to 2 years in major burns (proliferation of extrajunctional receptors → hyperkalemia → cardiac arrest).
  - Nondepolarizing neuromuscular blockers: Resistance in burns > 30% TBSA which peaks after 5–6 weeks (drug binds to ↑ extrajunctional acetylcholine receptors and does not cause an effect).
- Narcotics: ↑ requirements secondary to tolerance and ↑ volume of distribution.
- Inhalationals: Hypotension in presence of hypovolemia.
- Thiopental/propofol/etomidate: Hypotension in presence of hypovolemia.
- Ketamine: hypotension secondary to hypovolemia and depleted catecholamine stores.
- Albumin-bound drugs (benzodiazapines and anticonvulsants): Prolonged effects as plasma albumin concentration ↓ after 48 hr.
- Drugs undergoing oxidative metabolism in liver (diazepam): Prolonged effects.
- Drugs undergoing conjugation in liver (lorazapem): No change in effects.

Complications

Carbon Monoxide (CO) Toxicity

When COHb is present, pulse oximetry overestimates oxyhemoglobin saturation.

Two hundred times greater affinity for hemoglobin vs. O_2 → shift oxyhemoglobin dissociation curve to left → tissue hypoxia.

Signs/Symptoms

Classic cherry-red color (only at COHb > 40%), neurologic sequelae (see Table 15-2).

Diagnosis

- Metabolic acidosis.
- ↑ plasma lactate.

TABLE 15-2. Symptoms of Carbon Monoxide Toxicity as Function of Blood COHb Level

Blood COHb (%)	Symptoms
< 15–20	Headache, dizziness, occasional confusion
20–40	Nausea, vomiting, disorientation, visual impairment
40–60	Agitation, combativeness, hallucinations, coma, shock
> 60	Death

(Reproduced, with permission, Barash PG, Cullen BF. *Handbook of Clinical Anesthesia*, 6th ed. Philadelphia PA: Lippincott Williams & Wilkins. 2009: 551.)

Treatment

- High FiO_2: Improves oxygenation and promotes elimination of CO.
  - CO $T_{½}$ at room air = 4 hr.
  - CO $T_{½}$ at FiO_2 of 1.0 = 60–90 min.
- Hyperbaric O_2 treatment.

Cyanide (CN) Toxicity

Inhalation or absorption of cyanide or hydrocyanic acid from incompletely combusted synthetic materials.

Signs/Symptoms

- Generalized cardiovascular depression, cardiac rhythm disturbances.
- Nonspecific neurologic symptoms (agitation, confusion, coma).
- Metabolic acidosis.
- ↑ plasma lactate.

Diagnosis

Blood CN levels: > 0.2 mg/L = toxic; > 1 mg/L= lethal; $T_{½}$: 1 hr.

Treatment

High FiO_2, hyperbaric O_2; amyl nitrate, sodium nitrate, thiosulfate.

INTRAOPERATIVE COMPLICATIONS

Persistent Hypotension

Causes

- Bleeding (most common): External vs. occult (thoracic, abdominal, retroperitoneal).
- Cardiac injury: Myocardial contusion, pericardial tamponade, valvular injury, and septal perforation.
- Tension pneumothorax.
- Others: Citrate intoxication, hypothermia, CAD, allergic reactions, incompatible transfusion.

Reduced doses of etomidate may be necessary in trauma patients as they have ↓ sympathetic tone.

Treatment

- Control bleeding and fluid resuscitation via rapid infuser with LR, PRBC transfusion.
- Neurogenic shock: Hypotension + bradycardia—treat with catecholamine infusion.
- Hypovolemic patients are sensitive to cardiovascular (CV) depressants, catecholamine-inhibiting and baroreceptor depressant effects of IV and volatile anesthetics.
  - CV depression: Propofol > thiopental/midazolam > opioids > etomidate.
  - Baroreceptor depression: Thiopental/propofol/ketamine > midazolam/diazepam/droperidol > etomidate.

Hypothermia

Risk Factors

- Shock.
- Alcohol intoxication.
- Exposure to cold.
- Fluid resuscitation.

Effects

- ↓ CO.
- Cardiac conduction abnormalities.
- ↓ cerebral blood flow (CBF) and renal blood flow (RBF).
- Leftward shift of oxyhemoglobin dissociation curve.
- Altered platelet and clotting enzyme function.
- Abnormalities of K and Ca homeostasis.
- ↑ mortality with ↓ temperatures.

Treatment

- Warm IV fluids: 1 L at 40°C → 7 kcal heat energy gained.
- Countercurrent heat exchanging systems.
- Continuous arteriovenous rewarming.

Coagulation Abnormalities

Risk Factors

FDP

F = fibrin

D = degradation

P = products

- Hemodilution.
- Hypothermia.
- Acidosis.
- Tissue hypoxia and tissue thromboplastin release.
- Hypothermia-induced coagulopathy secondary to altered enzyme activity and platelet adhesion.
- PRBC transfusion of more than one blood volume causes a clinical coagulopathy warranting a transfusion.

Disseminated Intravascular Coagulation (DIC) Diagnosis

- FDP/fdp > 10 mg/mL (> 40 diagnostic).
- Fibrinogen < 150 mg/dL.
- Platelet < 150,000.
- PT > 15.

Treatment

- Platelets: 1 U → 5–10 K/μL ↑.
- FFP: 2 U minimum given within 1 hr.
- Cryoprecipitate: For fibrinogen < 80 mg/dL; 10 U → 100 mg/dL ↑.
- Prophylactic transfusion not warranted.

Primary treatment of DIC is correction of underlying cause.

Electrolyte and Acid-Base Disturbances

Hyperkalemia

Causes

- Massive efflux in irreversible shock.
- Reperfusion of ischemic tissues.
- Rapid transfusion.

Treatment

- 10 U regular insulin with 50 mL 50% dextrose.
- 50 mL sodium bicarbonate.
- Calcium chloride 500 mg for arrhythmias.
- Hemodialysis in severe situations.

Metabolic Acidosis

Causes

- Shock (most common).
- Alcoholic lactic acidosis.
- Alcoholic ketoacidosis.
- Diabetic ketoacidosis (DKA).
- CO/CN poisoning.

Treatment

Effects of treatment of metabolic acidosis with sodium bicarbonate:

- **Negative effects:**
  - Leftward shift of oxyhemoglobin dissociation curve.
  - Hyperosmolar state secondary to sodium load.
  - Hypokalemia.
  - Hemodynamic depression.
  - Alkalosis.
  - Intracellular acidosis if inadequate ventilation or pulmonary blood flow.
- **Positive effects:**
  - Avoids dysrhythmias, myocardial depression, hypotension, catecholamine resistance.
  - Buys time if pH < 7.2.

Osmolality of $NaHCO_3$ = 1800 mOsm/L
50 mEq Na
50 mEq HCO_3

POSTOPERATIVE COMPLICATIONS

Abdominal Compartment Syndrome

Shock-induced inflammatory mediators, fluid rescuscitation, and surgical manipulation → edema of intra-abdominal organs → multisystem organ failure → death.

Systemic Effects

- **Cardiac:** Hypovolemia, ↓ CO, ↓ venous return, ↑ pulmonary artery occlusion pressure and CVP, ↑ SVR.
- **Gastrointestinal:** ↓ celiac/superior mesenteric artery/mucosal blood flow, ↓ intramucosal pH.
- **Hepatic:** ↓ portal blood flow, ↓ mitochondrial function, ↓ lactate clearance.
- **Pulmonary:** ↑ intrathoracic pressure/peak inspiratory pressure/Paw, ↓ PaO_2, ↑ $PaCO_2$, ↑ intrapulmonary shunt, ↑ dead space, ↓ dynamic pulmonary compliance.
- **Renal:** ↓ UO/RBF/glomerular filtration rate (GFR).
- **Abdominal wall:** ↓ compliance, ↓ rectus sheath blood flow.

Refractory elevated CVP may be due to abdominal compartment syndrome even though hypovolemia is present.

Diagnosis

- Tense, distended abdomen.
- Intravesicular pressure > 20–25 mmHg.

Treatment

Abdominal decompression.

Thromboembolism

- Eighteen percent of trauma patients have a proximal femoral vein DVT.
- Less than 2% of trauma patients develop a PE.

Signs/Symptoms

- Most are asymptomatic.
- Leg swelling.
- Hypoxemia.
- Dyspnea.
- Presents within first week of injury.

Diagnosis

- Venography (gold standard).
- Pulmonary angiography (definitive).
- Ventilation/perfusion (V/Q) scan.
- Spiral CT.
- Duplex ultrasound.

Treatment

- Intubation with PPV with FiO_2 1.0.
- Fluids and ionotropes (amrinone or milrinone).
- Anticoagulants (low-molecular-weight heparin).
- Inferior vena cava (IVC) filter.
- Sequential compression devices (SCDs).

Acute Renal Failure

Causes

- Crush syndrome (rhabdomyolysis-induced myoglobin release).
- Prolonged shock.

Diagnosis

- Creatinine clearance (CrCl) < 25 mL/min.
- Free water clearance ≥ –15 mL/hr.
- Elevated blood urea nitrogen (BUN) occurs at least 24 hr later.
- Urine flow rate not a good predictor.

Treatment

IVF.

Creatinine clearance = {(140 – age) × lean body weight in kg}/(72 × serum creatinine) (for males). In females multiply result by 0.85.

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CHAPTER 16

Critical Care

Mechanical Ventilation

- Mechanical ventilation replaces or supports normal pulmonary ventilation due to ventilatory or oxygenation failure and their underlying causes.
- Mechanical ventilation implies positive pressure ventilation (PPV). Inspiratory gas flow is created by a pressure gradient between the machine circuit and alveoli while expiration is passive.
- ↓ functional residual capacity (FRC) leads to hypoxemia. PPV can ↑ FRC and ↑ lung compliance.
- Ventilation/perfusion (V/Q) mismatching → ↓ PaO_2. PPV helps correct V/Q mismatching by the recruitment of alveloli.

Reasons for Mechanical Ventilation

- Arterial O_2 tension < 60–70 mmHg.
- Arterial CO_2 tension > 50–60 mmHg.
- PaO_2/FiO_2 ratio < 300 mmHg.
- PA-aO_2 gradient > 350 mmHg.
- $V_D/V_T > 0.6$.
- Respiratory rate > 35/min.
- Tidal volume < 5 mL/kg.
- Vital capacity < 15 mL/kg.

Adverse Effects of PPV

- Barotrauma: Due to ↑ inspired peak pressures.
- Volutrauma: Due to repetitive collapse and reexpansion of alveoli.
- Altered V/Q relationships: Gas flow is directed to more compliant, nondependent areas of the lung, causing ↑ physiologic dead space.
- ↓ cardiac output (CO):
  - ↑ intrathoracic and mean airway pressures → ↓ ventricular filling during diastole and ↓ ventricular distensibility.
  - ↓ venous return and ↓ right ventricular (RV) ejection fraction → ↓ left ventricular (LV) filling.

Types of Ventilators

- **Volume-cycled ventilators:**
  - Preset volume delivered during inspiration.
  - Inspiratory pressure limit protects against barotrauma. When this pressure limit is reached, the ventilator will cycle to expiration.
  - Some of the set tidal volume (V_T) will be lost to expansion of the breathing circuit—approximately 3–5 mL/cm H_2O. This loss of V_T is inversely proportional to lung compliance.
- **Pressure-cycled ventilators:**
  - Preset airway pressure before changing from inspiration to expiration.
  - V_T and inspiratory time vary as airway resistance and pulmonary compliance vary.
  - A kink in the circuit can cause a ↓ in the circuit compliance, which will → premature cycling and a ↓ in delivered V_T. A ↓ in lung compliance will do the same.

- **Time-cycled ventilators:**
  - Preset time interval before changing from inspiration to expiration.
  - The set inspiratory time and flow rate determine V_T.

Ventilatory Modes

Ventilatory modes are described by the way in which they cycle and whether spontaneous breaths are allowed. Most modes include a pressure limit to avoid barotrauma. Respiratory rate (RR) and V_T can be used to adjust for ventilation. FiO_2 and positive end-expiratory pressure (PEEP) can be used for oxygenation.

- **Controlled mechanical ventilation (CMV) (Figure 16-1):**
  - Fixed V_T and RR give a fixed minute ventilation.
  - The machine cycles after a fixed time interval. Cycling is regardless of patient effort, so patients capable of respiratory effort usually require sedation or paralysis.
- **Assist-control (AC):**
  - Fixed V_T and RR.
  - A patient's inspiratory effort will trigger a full breath (the set V_T). If the patient does not breathe spontaneously or with sufficient effort, the machine will give a mandatory breath, acting like CMV.
  - Time cycled and pressure limited.
  - Risk of breath stacking and respiratory alkalosis.

Dead Space = No Perfusion + Ventilation

Shunt = Perfusion + No Ventilation

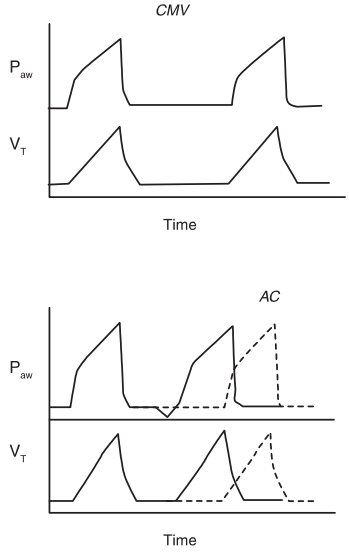

FIGURE 16-1. Control mode ventilation (CMV).

(Top) Graphic representation of the pressure and volume tracings of a patient receiving positive pressure ventilation who is totally passive in control mode ventilation (CMV). The ventilator does all the work, and patient cannot take breaths on their own. (Bottom) Graphic representation of the pressure and volume tracings of a patient receiving positive pressure ventilation via the Assist Control (A/C) mode, also known as the controlled minimum ventilation (CMV) mode. The patient gets a set minimum amount of machine breaths at a set tidal volume (dotted line). If the patient takes a breath in-between these machine breaths, the patient will be given a full tidal volume breath (solid line) and the next machine breath will be given at the appropriate interval following that breath.

Micropressor-controlled ventilators can combine modes simultaneously.

- **Intermittent mandatory ventilation (IMV) (Figure 16-2):**
  - Fixed V_T and RR.
  - A continuous flow of gas allows for spontaneous ventilation by the patient. The V_T and frequency of this independent breath is determined by the patient's pulmonary effort.
  - The set RR is used as a backup for the patient's spontaneous breaths.
  - Risk of breath stacking.
- **Synchronized intermittent mandatory ventilation (SIMV) (Figure 16-3):**
  - Similar to IMV.
  - The mechanical breath is timed with the patient's effort. This attempts to avoid stacking of the ventilator's mechanical breaths over the patient's spontaneous breaths, which would deliver a large V_T (and possible barotrauma).
  - Mode often used for weaning.
- **Pressure support ventilation (PSV):**
  - Patient initiates breath, which determines RR.
  - Patient-initiated breath causes ventilator to deliver preset pressure-augmented V_T.
  - Positive pressure maintained throughout inspiration to overcome resistance in circuit.
  - ↓ work of breathing and augments V_T in spontaneously breathing patients.
  - No backup rate if the patient tires, so PSV is often used with IMV to provide a safety backup RR.
- **Pressure control ventilation (PCV):**
  - Fixed RR, inspiratory time, and maximal pressure.
  - Patient receives breath that ceases when preset maximal pressure is reached.
  - A peak airway pressure limit ↓ the risk of barotrauma and volutrauma.
  - V_T is affected by changes in lung compliance or circuit resistance. Thus, a set V_T is not guaranteed.
  - Can be used with either AC or IMV mode.
- **Inverse I:E ratio ventilation (IRV):**
  - Reverses the normal I:E ratio to greater than 1:1, in an effort to recruit flooded or collapsed alveoli in order to improve oxygenation.
  - Requires heavy sedation or paralysis.
  - Auto-PEEP may be produced if the lung has insufficient time to empty, causing air trapping and ↑ FRC.

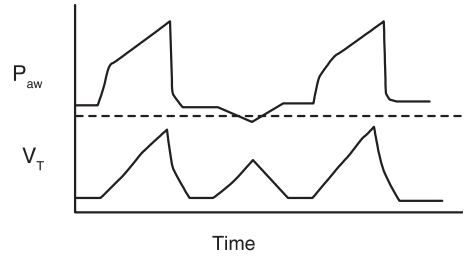

FIGURE 16-2. Intermittent mandatory ventilation (IMV).

Graphic representation of the pressure and volume tracings of a patient receiving positive pressure ventilation via the Intermittent Mandatory Ventilation (IMV) mode. The patient gets mandatory breaths that are determined b the respiratory rate set on the ventilator. In between these machine breaths, the patient can take spontaneous breaths on their own. The frequency and tidal volume of these spontaneous breaths is determined solely by the patient.

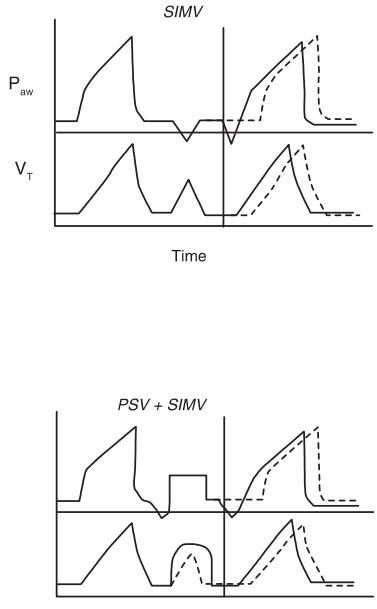

FIGURE 16-3. Synchronized intermittent mandatory ventilation (SIMV).

(Top) Graphic representation of the pressure and volume tracings of a patient receiving positive pressure ventilation via the synchronized intermittent mandatory ventilation (SIMV) mode. With the tracings, it is easy to see the combination of IMV and A/C. In the period of time immediately after a machine breath, the patient may take breaths on their own, at their own tidal volume and respiratory rate, as in IMV. When the next machine breath is due, the ventilator will hesitate a certain period of time, and if a patient breath is taken during that period of time the breath will be a full tidal-volume breath synchronized to the patient's inspiratory effort, as with A/C. (Bottom) Graphic representation of the pressure and volume tracings of a patient receiving pressure support (PS) in the SIMV mode. In the period of time immediately after a machine breath, the patient may take breaths on their own, at their own tidal volume and respiratory rate, as in SIMV, but any breaths that reach the sensitivity threshold will now trigger a supporting pressure that usually is lower than machine-breath PAWP and results in a V_T smaller than the one set for the volume-controlled breaths.

- **High-frequency ventilation (HFV):**
  - Most often high-frequency jet ventilation (HFJV) for laryngeal, tracheal, or bronchial procedures. Can be used for emergency airway management.
  - A pulsed jet of high-pressure gas is delivered via a cannula to give a V_T.
  - The mechanism of gas exchange is unclear. However, elimination of carbon dioxide is generally directly related to drive pressure, and oxygenation is directly related to mean airway pressure.
  - Also available are high-frequency positive pressure ventilation (HF-PPV) and high-frequency oscillation (HFO).

HFJV may be useful in managing patients with bronchopleural fistulas or TEF (when conventional methods of ventilation fail).

PEEP and CPAP

Positive End-Expiratory Pressure (PEEP)

PEEP is the small pressure (greater than atmospheric pressure) remaining at the end of expiration during positive pressure ventilation. A PEEP valve creates a pressure threshold that allows expiratory flow only when that airway pressure is met. Physiologic PEEP is 5 cm H_2O.

- PEEP ↑ FRC through increasing alveolar ventilation by preventing small airway collapse.
- Adding PEEP improves oxygenation. In patients with high degrees of shunt or who require high FiO_2, adding PEEP can help to avoid oxygen toxicity associated with high FiO_2.
- PEEP ↑ CO in normal lungs; however, in lungs with low compliance, PEEP ↓ CO.
- PEEP and lung mechanics:
  - FRC = ERV + RV (functional residual capacity = expiratory reserve volume + residual volume), or, air left in lung after normal tidal expiration.
  - ERV ↓ with small airway collapse, thus decreasing FRC, and ultimately the patient's ability to oxygenate and ventilate.
  - CCV (critical closing volume) = the minimum volume and therefore pressure at which small airways will collapse during expiration.
  - When alveoli collapse, blood is shunted around these alveoli that do not participate in gas exchange.
    - In this case, perfusion is greater than ventilation, such as in pulmonary edema, atelectasis, pneumonia.
    - When ventilation is greater than perfusion, dead space is ↑, as with massive pulmonary embolus.
  - CCV is influenced by compliance:
    - A ↓ in dynamic compliance (↑ peak airway pressures) with no change in static compliance (plateau pressures) indicates an airway resistance problem, such as small airway collapse, bronchospasm, aspiration, or obstruction.
    - A ↓ in both dynamic and static compliance (↑ in both peak and plateau pressures) indicates a compliance (elasticity) problem, such as pneumothorax, excess PEEP, atelectasis, or pulmonary edema.
- **Auto-PEEP:**
  - Occurs when there is airflow at the end of expiration because exhalation has not completed.
  - Associated with obstructed airways, ↑ airway resistance, obstructed airways, and ↓ time for exhalation.
  - Auto-PEEP ↓ the volume of each breath, falsely elevates PCWP readings, and ↑ work of breathing (WOB).
  - Treat with ↓ respiratory rate, ↓ V_T, or ↑ inspiratory flow rate, which will ↑ expiratory time.

Continuous Positive Airway Pressure (CPAP)

- CPAP is the positive pressure (greater than atmospheric pressure) that is applied to both inspiration and expiration during spontaneous breathing.
- The goal is the same as PEEP, which is to improve oxygenation by ↑ FRC:
  1. Partially collapsed alveoli are recruited and stabilized.
  2. FRC is ↑ above CCV.
  3. Lung compliance is ↑.
  4. V/Q mismatching is improved and therefore alveolar shunting is ↓.

Excessive PEEP or CPAP with associated barotrauma is observed at levels > 20 cm H_2O.

Excess CPAP or PEEP

- Barotrauma can → subcutaneous emphysema, pneumomediastinum, pneumothorax, pneumoperitoneum, or bronchopleural fistula. Treat with chest tube placement.

- Overdistended alveoli can ↓ lung compliance, ↑ dead space ventilation, and thus ↑ WOB.
- Compressed alveolar capillaries can ↑ pulmonary vascular resistance and RV afterload, which results in leftward displacement of the interventricular septum, ↓ LV filling, ↓ LV compliance, and ↓ CO.
- ↓ CO → ↓ renal and hepatic blood flow. Antidiuretic hormone (ADH) and angiotensin levels ↑. GFR and urine output ↓.
- High PEEP, ↑ central venous pressure, and ↓ venous return result in ↑ intracranial pressure (ICP).

Complications During Mechanical Ventilation

- **High peak airway pressures:** See above discussion under critical closing volumes.
- **Low tidal volumes:**
  - ↑ resistance.
  - ↓ compliance.
  - Auto-PEEP.
  - ↓ expiratory time.
- **Hypotension:**
  - ↓ venous return.
  - Medications.
  - Cardiac.
  - Infection/shock/sepsis/systemic inflammatory response syndrome (SIRS).
- **Hypoxemia:**
  - Ventilator related: Ventilator-associated pneumonia.
  - Endotracheal tube related: Subglottic stenosis, laryngeal injury, sinusitis.
  - Underlying disease: Adult respiratory distress syndrome (ARDS), pulmonary edema, pneumonia, sepsis.
  - Acute event: Sepsis, pneumonia, pulmonary embolus, atelectasis, aspiration, pneumothorax, bronchospasm.

In ARDS, mortality ↑ with V_T > 10 cc/kg.

Acute Respiratory Failure

Usually a mix between failure of oxygenation and hypoxemia (hypoxic respiratory failure) and failure of CO_2 elimination and hypercapnia (ventilatory failure).

Indications for Intubation

- $PaCO_2$ (ventilatory failure): > 60 mmHg.
- PaO_2 (oxygenation failure): < 70 mmHg.
- Respiratory rate: > 35 breaths/min.
- A-a gradient: > 400 mmHg.
- Airway protection, including:
  - Altered mental status.
  - Severe head injury.
  - High spinal cord injury.
  - ARDS.

Pulmonary Edema

Pulmonary edema marks a progressive, transudative movement of fluid from the pulmonary capillaries into interstitial space, then into alveoli and finally airways. Differentiated between cardiogenic and noncardiogenic pulmonary edema.

- As pulmonary compliance ↓, tachypnea and dyspnea ↑, → ↑ minute ventilation and hypocapnia.
- As pulmonary perfusion continues through flooded alveoli, intrapulmonary shunting results in hypoxemia.
- As airways fill, airway obstruction → hypercapnia with continued hypoxemia.

Protein content of edema fluid.
Low protein = Hemodynamic cause of edema (cardiogenic).
High protein = Fluid due to permeability leak (noncardiogenic).

Cardiogenic Pulmonary Edema

- Represents a net ↑ in hydrostatic pressure across capillaries, by two major mechanisms.
- Pulmonary venous hypertension: Caused by LV failure, mitral stenosis, or left atrial obstruction; elevated pulmonary venous pressure is transmitted backwards to pulmonary capillaries.
- ↑ pulmonary blood flow: Caused by fluid overload, left-to-right cardiac shunts, or severe anemia, where pulmonary blood flow exceeds the capacity of pulmonary vasculature.

Treatment

- Morphine.
- Diuretics (correct fluid overload).
- Vasodilators (nitrates).
- Preload reduction agents (angiotensin-converting enzyme [ACE] inhibitors, brain natriuretic peptide).
- Inotropes (dobutamine).

Noncardiogenic Pulmonary Edema

Three criteria:

1. Bilateral diffuse pulmonary infiltrates.
2. PaO_2/FiO_2 ratio < 300:
   - Acute lung injury (ALI): PaO_2/FiO_2 ratio ≤ 200.
   - ARDS.
3. PAWP ≤ 18 mmHg, which excludes underlying significant LV dysfunction.

Represents ↑ permeability to extravascular lung water due to disruption of the capillary-alveolar membrane. Plasma oncotic forces are lost, resulting in unopposed hydrostatic forces moving fluid into the lungs.

Causes

- Shock.
- Trauma.
- Sepsis/disseminated intravascular coagulation (DIC).
- Necrotizing pancreatitis.
- Pulmonary aspiration.
- Inhalation injury.
- Severe pneumonia.
- Pulmonary embolus.

- Massive transfusion/resuscitation.
- Cardiopulmonary bypass.

Treatment

- Correct the underlying cause and supportive care.
- ↓ fluid in the lungs:
  - Fluid restriction/diuresis.
  - Monitor with low PAWP while not compromising perfusion and LV function. PAWP = pulmonary artery wedge pressure (its not a common pneumonic) = PCWP
- Maintain oxygenation:
  - $FiO_2 < 60\%$ if possible; add PEEP as needed.
  - $SpO_2 > 90\%$ if possible.
  - Hemoglobin (Hgb) > 10 g/dL as indicated.
- Maintain lung volume: PEEP and recruitment of alveoli are important early on in the first several days. After that, maintenance of alveoli with lower airway pressures (< 35 cm H_2O), lower tidal volumes (6 mL/kg), ↓ PEEP, and permissive hypercapnia are more lung protective.
- Permissive hypercapnia:
  - As part of a lung protective strategy as above.
  - Caution in patients with acute head injury, recent cerebrovascular accident (CVA), severe metabolic acidosis.
  - ↑ $PaCO_2$ (see Table 16-1).

Weaning

Extubation Criteria

- $PaCO_2$: < 50 mmHg.
- PaO_2: > 60–70 mmHg on $FiO_2 < 40\%$.
- Negative inspiratory pressure: < –20 cm H_2O.
- Tidal volume: > 5–8 mL/kg.
- Vital capacity: > 10 mL/kg.
- Minute ventilation: < 10 mL.
- RR: < 30 breaths/min.
- Rapid shallow breathing index (RR/V_T): < 100.
- pH: > 7.25.
- Patient alert, able to clear secretions, with intact airway reflexes.
- Correction of underlying medical causes contributing to intubation.
- **SIMV:** Gradual reduction in mechanical breaths to an RR of 2 and pressure support of 5–8 cm H_2O. Monitor with ABG measurements.
- **PSV:** Same as above but with PS alone. Pressure support of 5–8 cm H_2O, RR < 30, V_T 4–6 mL/kg. Monitor with ABG measurements.

Other maneuvers to improve oxygenation:
Add inhaled NO
Add inhaled PGE_1

TABLE 16-1. Effects of ↑ $Paco_2$

↑ sympathetic activity
Cerebral vasodilation
↑ pulmonary vascular resistance
↑ cardiac output
Cardiac arrhythmias
Impaired skeletal muscle function
Rightward shift of Hgb-oxygen dissociation curve

T-Piece or CPAP Trial

- The patient is observed clinically for fatigue, tachypnea, tachycardia, hemodynamic instability, and obvious signs of distress. The patient can be given trials on T-piece or CPAP depending on the duration of intubation and the patient's underlying pulmonary condition.
- A T-piece is attached directly to the endotracheal or tracheostomy tube with sufficient flow of humidified O_2/air mixture blowing from the proximal to distal ends.
- An alternative trial is to add 5–8 cm H_2O of CPAP in order to maintain FRC and prevent atelectasis that may be possible with T-piece alone.

CARDIOVASCULAR CRITICAL CARE

Shock

Definition

Despite adequate fluid resuscitation, a systolic blood pressure < 90 mmHg, mean < 60 mmHg, or a ↓ > 40 mmHg from systolic baseline.

Differential Diagnosis

See Table 16-2.

General Management

Other classifications of shock:
Hypovolemic
Cardiogenic
Obstructive (eg, tamponade, pneumothorax)
Distributive (eg, sepsis, neurogenic)

- Correct underlying causes.
- Maintain tissue perfusion and oxygen delivery with fluids, inotropes, vasopressors, and/or transfusions.
- Supportive treatment of accompanying states:
  - ARDS.
  - Acute renal failure (ARF).
  - DIC.
  - Gastrointestinal (GI) hemorrhage.

Cardiogenic Shock

- Characterized by pump failure.
- Low CO, high pulmonary artery occlusion pressure (PAOP), high systemic vascular resistance (SVR), ↓ mixed venous oxygen saturation (Svo_2).

TABLE 16-2. Differential Diagnosis of Shock

	CI	SVR	PVR	Svo_2	RAP	RVP	PAP	PAOP
Cardiogenic	↓	↑	Normal	↓	↑	↑	↑	↑
Hypovolemic	↓	↑	Normal	↓	↓	↓	↓	↓
Septic	—	↓	Normal	Normal–↓	Normal–↓	Normal–↓	Normal–↓	Normal–↓
Neurogenic	↓	↓		↓				↓

CI, cardiac index; PAOP, pulmonary artery occlusion pressure; PAP, pulmonary artery pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RVP, right ventricular pressure; Svo_2, mixed venous oxygen saturation; SVR, systemic vascular resistance.

- **Compressive:**
  - Cardiac tamponade.
  - Tension pneumothorax.
  - Positive pressure ventilation: $V_T > 12$ mL/kg, PEEP > 10 cm H_2O.
- **Cardiogenic:**
  - Acute myocardial infarction.
  - Pulmonary embolus.
  - Cardiomyopathy.
  - Arrhythmias.
  - Valvular disease.

Treatment Goals

- Improve cardiac performance.
- ↑ contractility (inotropes).
- Improve PAOP (preload).
- ↓ SVR (afterload).

Hypovolemic Shock

- Characterized by low intravascular circulating volume.
- Low CO, low PAOP, high SVR, ↓ SvO_2.

Treatment Goals

- Volume resuscitation.
- Keep the patient warm.

Septic Shock

S = Systemic

I = Inflammatory

R = Response

S = Severe Infection

- Characterized by infection causing systemic loss of resistance.
- More precisely, the term *vasogenic shock* includes different subtypes of shock:
  - SIRS.
  - Septic shock: When SIRS is due to an infectious cause.
  - Anaphylactic shock.
  - Adrenal shock: Due to adrenal insufficiency.
- High CO, low PAOP, low SVR, falsely ↑ SvO_2 in the face of tissue hypoxia.

Mechanism

- Diffuse capillary leak → ↓ circulating volume.
- Vasodilation from ↑ nitric oxide.
- Bacteremia by gram negatives is usual but may not be present.
- Activation of coagulation cascade and platelets.
- Elevated PVR and pulmonary hypertension.
- Cytokine-initiated SIRS.
- Hypovolemia due to transudation of fluid and systemic venodilation.

Treatment Goals

↑ SVR and PAOP.

TABLE 16-3. 12-Lead Electrocardiogram Interpretation

	ECG Lead	Coronary Vessel
Anterior-septal	V_1, V_2	Distal LAD
Anterior	V_1–V_4, poor R wave progression	Proximal LAD
Lateral (high)	I, aVL	Proximal circumflex
Lateral (apical)	V_5, V_6	Distal circumflex
Inferior	II, III, aVF	Terminal branch of LAD or R post descending
Posterior	Tall R and T waves in V_1–V_3	Post descending

Neurogenic Shock

- Characterized by loss of sympathetic nervous and subsequent vascular tone.
- Low CO, low PAOP, low SVR, ↓ SvO_2.

Treatment Goals

↑ SVR and preload.

Acute Myocardial Ischemia and Infarction (MI)

- Myocardial ischemia is the result of an imbalance between myocardial oxygen demand and supply. See Table 16-3 for ECG lead and corresponding vessel to determine origin of ischemia.
- ↓ in coronary blood flow is usually due to a coronary thrombosis at a stenotic atheromatous plaque. ↓ flow can also result from a coronary bypass graft occlusion, coronary spasm, or shock.
  - Transmural infarction occurs distal to a complete occlusion due to thrombosis.
  - Typically ST-segment elevation.
- ↑ myocardial demand can result from ventricular tachycardia or uncontrolled atrial fibrillation.
  - Subendocardial infarction occurs in the setting of sustained, ↑ demand.
  - Typically ST-segment depression.
  - New left bundle branch block (LBBB) suggests acute MI, but it makes interpretation difficult.
  - T-wave inversions are nonspecific.
  - Q waves represent myocardial necrosis.
- "Stunned" myocardium can be seen after brief periods of ischemia resulting in a prolonged period of contractile dysfunction until recovery. Can be seen after cardiopulmonary bypass.
- "Hibernating" myocardium is also characterized by noninfarcted but dysfunctional areas but in the setting of chronic ischemia. Can be seen after myocardial revascularization.

Treatment of Acute MI–

MOAN

Morphine: 2–4 mg IV every 5 min prn pain
Oxygen: 4–6 L/min
Aspirin: 325 mg PO (chew it)
Nitroglycerin: 0.15–0.6 mg sublingual tablet every 5 min × 3 prn

Treatment

- "MOAN": **M**orphine, **O**xygen, **A**spirin, **N**itroglycerin.
- Antithrombotic therapy: Heparin, low-molecular-weight heparins (enoxaparin).
- Beta blockers.
- ACE inhibitors.

- Calcium channel blockers.
- Monitor electrolytes.
- Reperfusion:
  - Angioplasty +/– stenting.
  - Thrombolytic therapy.
- Intra-aortic balloon counterpulsation.
- Temporary pacing.

ACUTE RENAL FAILURE (ARF)

Definitions

- **ARF:**
  - Serum creatinine ↑ of > 0.5 mg/dL over baseline of < 2.0 mg/dL, or ↑ of > 1.0 mg/dL over a baseline of > 2.0 mg/dL.
  - 50% ↓ in creatinine clearance.
- **Oliguria:** Urine output < 400 mL/24 hr.
- **Anuria:** Urine output < 50 mL/24 hr.
- **Nonoliguric:**
  - Urine output > 400 mL/24 hr.
  - Nonoliguric ARF may be less severe than oliguric ARF; however, outcomes with the use of diuretics (Lasix), renal dose dopamine, and mannitol to maintain urine output are debatable.

50–60% of ARF is secondary to renal ischemia.

Treatment

- Supportive.
- Treatment by major symptoms of ARF:
  - **Volume overload:** Convert oliguric to nonoliguric renal failure.
    - Furosemide.
    - Thiazides: Thiazides plus furosemide produce potent diuresis.
    - Dopamine: Efficacy of renal dose dopamine (< 3 μg/kg/min IV infusion) is debatable.
    - Dialysis.
  - **Metabolic acidosis:**
    - Anion gap uremic metabolic acidosis.
    - Alkali requirement is 1 mEq bicarbonate/kg/day.
    - Bicarbonate supplementation.
  - **Hyperkalemia:**
    - Electrocardiogram (ECG) changes.
    - Regular insulin 10 U IV plus 1amp (50 cc) glucose IV.
    - Calcium gluconate 1 amp (1 g) IV.
    - β agonist (albuterol nebulizer).
    - Dialysis.
  - **Hyperphosphatemia:**
    - < 5 mg/dL.
    - Consider oral binders: Calcium carbonate, aluminum hydroxide.
  - **Anemia:** Due to ↓ renal erythropoietin synthesis. Exogenous Epogen injection SC.
  - **Platelet dysfunction:** Treat as needed.

Management

- Renal dosing of medications.
- Avoid nephrotoxins: NSAIDs, ACE inhibitors, contrast dye, aminoglycosides.

Prerenal Azotemia

ARF due to hypoperfusion of the kidneys, usually secondary to a ↓ in perfusion pressure, ↑ in venous pressure, or renal vasoconstriction.

Causes

- Volume depletion/ischemia:
  - Hypovolemia.
  - GI losses (vomiting, diarrhea).
  - Acute blood loss.
  - Overdiuresis.
  - Renal salt-wasting syndromes.
  - Third spacing (trauma, ascites, peritonitis, pancreatitis).
  - Hypoalbuminemia (cirrhosis, nephrotic syndrome, malnutrition).
- ↓ renal perfusion:
  - Nephrotoxins/drugs: Aminoglycosides, amphotericin B, contrast dye, nonsteroidal anti-inflammatory drugs (NSAIDs), ACE inhibitors, vasopressors, immunosuppresants (cyclosporine, cisplatin, tacrolimus FK-506), hemoglobin, myoglobin.
  - Renal artery stenosis.
  - Renal vein thrombosis.
- ↑ venous pressure: Chronic liver disease, sepsis, hepatorenal syndrome.

Treatment

Aimed at correcting volume status and improving cardiac status.

FE_{Na} = excreted Na/filtered Na $\times$ 100 = $(U_{Na} \times P_{Cr} / U_{Cr} \times P_{Na}) \times 100$.

Renal Azotemia

- 30% of ARF.
- Acute tubular necrosis (ATN).
- Ischemic: shock, sepsis, trauma, hypoxia (see Table 16-4).

TABLE 16-4. Prerenal Azotemia vs. ATN

	Prerenal	ATN
Specific gravity	> 1.020	< 0.010
Osmolality (mmol/kg)	> 400	< 350
Urine Na (mEq/L)	< 20	> 40
Urine Osm (mOsm/kg)	> 400	< 400
Urine/serum creatinine	> 40	< 20
Renal failure index	< 1	> 1
Blood urea nitrogen (BUN)	↑	↑
Serum creatinine	Normal	↑
BUN/creatinine	> 20:1	< 20:1
FE_{Na}	< 1	> 1

Values may not be helpful in the elderly and patients on diuretics or with preexisting renal disease.

- Toxic: contrast, aminoglyosides, rhabdomyolysis.
- Glomerulonephritis:
  - Systemic disease: Sarcoidosis, Sjögren's syndrome, lymphoma.
  - Systemic infections: Syphilis, toxoplasma, cytomegalovirus (CMV), Epstein-Barr virus (EBV).
  - Medications: β-lactams, diuretics, NSAIDs.

Postrenal Azotemia

- Ten percent of ARF.
- Postrenal azotemia is the result of obstruction anywhere along the urinary tract.

Causes of Elevated BUN and Creatinine Without ARF

- Elevated blood urea nitrogen (BUN):
  - GI bleeding.
  - High protein intake, total parenteral nutrition (TPN), hypercatabolism.
  - Corticosteroids.
  - Compensation before ARF: Volume depletion, heart failure, urinary obstruction.
- Elevated creatinine: Rhabdomyolysis, cimetidine.

Dialysis

Indications

See mnemonic.

Indications for dialysis—

PHKNAVE

Pulmonary edema
Hypertension
K hyperkalemia
N (nitrogen) uremia
Acidosis
Volume overload
Encephalopathy

Types

- Intermittent hemodialysis (IHD):
  - Easily performed.
  - Hemodynamic instability.
- Continuous venovenous hemodialysis (CVVHD):
  - Labor intensive, expensive.
  - Excellent volume and solute control.
  - Good for hemodynamically unstable patients.
  - Good for hypercatabolism to match nitrogen balance.
- Automated peritoneal dialysis (APD):
  - Easily performed.
  - Needs intact peritoneum.

SPECIAL CONSIDERATIONS IN THE ICU

Infection

Ventilator-Associated Pneumonia (VAP)

- A nosocomial pneumonia in patients on mechanical ventilation for at least 48 hr.
- Likely organisms:
  - Early onset (within 48–72 hr):
    - *Haemophilus influenzae*.

- *Streptococcus pneumoniae.*
- *Staphylococcus aureus*, methicillin-sensitive *S aureus* (MSSA).
- *Escherichia coli*, *Proteus*, *Klebsiella*, *Enterobacter.*

- Late onset (associated with higher mortality):
- Methicillin-resistant *S aureus* (MRSA).
- *Pseudomonas aeruginosa.*
- *Acinetobacter.*

- Aspiration pneumonitis.
- Colonization of oropharynx: Colonization in prolonged, intubated, ICU patients is virtually unavoidable.
  - Indiscriminate use of antibiotic therapy for colonization should be discouraged to avoid developing resistant organisms.
  - This must be balanced by the need for early treatment if clinical suspicion is high.
- Respiratory/sputum cultures will have a low specificity for VAP: Bronchoalveolar lavage (BAL) or bronchoscopic guided protected sampling brush (PSB) may help guide antibiotic choice.

High risk for nosocomial infections: RED CHB

***R**enal failure*

***E**lderly*

***D**evices (prolonged invasive)*

***C**hemotherapy*

***H**ead trauma*

***B**urns*

Line-Associated Bacteremia

Intravascular catheter-associated infection is defined by clinical suspicion, positive blood cultures drawn from the line, and matching positive blood cultures drawn from another site.

- Catheter-associated infection ↑ with duration. Some institutions routinely change all lines after an arbitrary amount of time (ie, 3–7 days); however, others advocate there is no evidence to support this if there is no suspicion for line infection.
- All lines should be placed using strict aseptic technique, including chlorhexidine skin prep, preinsertion hand washing, and full barrier precautions.
- Lines should be changed if placed under nonoptimal emergency situations, especially femoral lines.
- If accompanied by fever, the catheter tip should be sent for culture after change of the line over guidewire.
- Likely organisms:
  - *Staphylococcus epidermidis.*
  - *S aureus.*
  - Enteric gram-negative bacteria.
  - *Pseudomonas.*
  - *Acinetobacter.*

Urinary Tract Infection

The urinary tract is the second most common source of infection in the ICU and includes the kidneys, collecting system, and bladder.

- Likely organisms:
  - *E coli.*
  - *Staphylococcus saprophyticus*, *Staph* species.
  - *Enterococcus.*
  - *Pseudomonas.*
  - *Klebsiella.*
  - *Proteus mirabilis.*
- Incidence ↑ with duration of catheterization.

Fungal Infection

Risk Factors

- Neutropenia.
- Uremia/dialysis-dependent renal failure.
- Antibiotic use: Multiple, broad-spectrum, and/or long duration.
- Steroids.
- Central venous catheters.
- TPN.
- Malnutrition.
- Burns.
- Chronic diabetes.
- Mechanical ventilation.
- *Candida:*
  - Accounts for vast majority of cases in nonimmunocompromised patients.
  - Associated with prolonged mechanical ventilation, UTI, peritonitis, disseminated bloodborne infection.
  - *Candida albicans* accounts for 50% of *Candida* infections.
  - Since fungal cultures and invasive infection grow slowly, a high clinical suspicion may warrant treatment.

Treatment

- Fluconazole: First line.
- Voriconazole/caspofungin: For Candida glabrata and Candida krusei, which are fluconazole resistant.
- Amphotericin B: High toxicity; reserved for refractory, life-threatening infections.

Stress Ulcer Prophylaxis

Causes of erosion gastritis:

Stress

Ulcers

ETOH

ASA

NSAIDs

Steroids

The protective gastric mucosa can break down, → gastritis, ulceration, and ultimately GI bleeding.

Indications

- Mechanical ventilation
- Coagulopathy

Other Risk Factors

- Renal failure
- Burns
- Head injury

Management

- Control of gastric pH.
- Inhibit production of acid:
  - H_2 blocker.
  - Proton pump inhibitor (PPI).
- Neutralize acid with antacid.
- Lowering of gastric pH allows colonization of enteric flora, which may ↑ the risk of aspiration and ventilator-associated pneumonia.
- Cytoprotection:
  - Recreates the protective barrier.
  - Sucralfate can create gastric bezoars.

- Hemodynamic optimization: Maintain blood flow to the gut, avoiding ischemia.
- "Feed the gut": Enteral feedings help prevent stress ulceration.

Deep Venous Thrombosis (DVT) and Pulmonary Embolism (PE)

Venous thromboembolism in the ICU can occur classically from the lower extremities but also can be directly related to upper-extremity venous catheters.

Risk Factors

- High risk:
  - Lower-extremity fracture.
  - Trauma including spinal cord injury.
  - Hip or knee replacement.
- Moderate risk:
  - Central venous lines.
  - Congestive heart failure.
  - Malignancy/chemotherapy.
  - Pregnancy/oral contraceptive therapy.
  - Hormone replacement therapy.
  - Previous DVT.
  - Arthroscopic knee surgery.
- Low risk:
  - Immobility/bed rest.
  - Age.
  - Obesity.
  - Laparoscopic surgery.

Diagnosis

- ECG, plain chest film, arterial blood gas (ABG).
- High clinical suspicion: Unexplained tachycardia, tachypnea, fever, dead space ventilation.
- D-dimer assay:
  - High sensitivity and low specificity.
  - Also elevated with infection, malignancy, and surgery.
  - High negative predictive value: A normal D-dimer helps to exclude 90% of PEs.
- Doppler ultrasound: 50% of patients with a documented DVT also have a PE.
- Spiral CT scan: Good for large, proximal PEs but misses small, distal PEs.
- Transthoracic echocardiography (TTE):
  - Low sensitivity and high specificity.
  - RV hypokinesis with normal apical wall motion signals PE.
- V/Q scan: High-probability scans are helpful but low- or intermediate-probability scans are of little utility.
- Pulmonary angiography: Gold standard.

Prophylactic anticoagulation and intermittent pneumatic compressive devices ↓ incidence of DVTs significantly.

Treatment

- Mainstay in ICU is unfractionated heparin due to titratability and reversibility compared to low-molecular-weight heparin.

- Thrombolytic therapy for massive PE with hemodynamic instability.
- Vena cava filters for those with contraindications to anticoagulation or recurrent PE despite anticoagulation.

Endocrine Issues

Glucose Control

Abrupt dicontinuation of TPN → hypoglycemia. Continued TPN → hyperglycemia.

- Hyperglycemia results from ↑ glucose production and insulin resistance caused by inflammatory and hormonal mediators released in response to injury, steroids, and TPN.
- Tight glucose control for both diabetics and nondiabetics has shown ↓ morbidity and mortality for ICU patients.

Adrenal Function

- Adrenal insufficiency in critical illness caused by inhibition of adrenal stimulation, corticosteroid synthesis by drugs or cytokines, or injury to pituitary or adrenal glands.
- Associated with trauma, burns, sepsis.
- Stim test: Inappropriately ↓ response of serum cortisol after adrenocorticotropic hormone administration.
- Supplemental steroids can reduce dependency on vasopressors.

Thyroid Function

- Thyroid function tests reflect hypothyroidism.
- Not clear whether hormone replacement is beneficial in critical illness.

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CHAPTER 17

Pain Management

PAIN DEFINITIONS

- **Pain:** Caused by actual or potential damage that results in an unpleasant physical and/or emotional experience. Pain can be classified into nociceptive and neuropathic pain.
- **Nociceptive pain:** Results from stimulation of mechanical, thermal, or chemical nociceptors. Can be somatic or visceral.
- **Neuropathic pain:** Results from a lesion in the nervous system or a dysfunction of the nervous system.
- **Allodynia:** Perception of an ordinarily non-noxious stimulus as pain.
- **Analgesia:** Absence of pain perception.
- **Anesthesia:** Absence of all sensations.
- **Anesthesia dolorosa:** Pain in an area without sensation.
- **Dysesthesia:** Unpleasant sensation.
- **Hypoalgesia:** ↓ sensation to noxious stimulation.
- **Hyperalgesia:** ↑ sensation to noxious stimulation.
- **Hyperesthesia:** ↑ response to stimulation.
- **Hypoesthesia:** ↓ sensation to stimulation.
- **Neuralgia:** Pain in the distribution of a nerve.
- **Paresthesia:** Abnormal sensation perceived without stimulation.
- **Radiculopathy:** Functional abnormality of nerve roots (pain associated with numbness).
- **Dependence:** Physiological adaptation to the substance and is associated with withdrawal symptoms during abstinence. Withdrawal symptoms may be relieved by administration of the substance.
- **Addiction:** Psychological dependence that is characterized by craving and compulsion to acquire the drug.
- **Tolerance:** The requirement of increasing dose to achieve the same analgesic effect.

Pain Pathway: Noxious stimuli → Nocioceptors → Substantia gelatinosa (dorsal horn) via A and C fibers → Release of excitatory neurotransmitter (ie, substance P) → Thalamus via spinothalamic tract → Cortex

PHYSIOLOGY AND ANATOMY

- Pain is conducted through the Aδ and C fibers.
- Pain is detected by nociceptors and conducted through various pain pathways. The main ascending pathway is called the spinothalamic tract, which consists of three neurons:
  - **First-order neuron:** Cell body is located in the dorsal root ganglia (DRG). One end of its bifurcating axon is at the peripheral tissues serving as the free-nerve ending. The other end synapses with second-order neuron at the dorsal horn.
  - **Second-order neuron:** After synapsing with first-order neuron at the dorsal horn, its axon ascends and reaches the thalamus to synapse with the third-order neuron. Second-order neurons are either nociceptive-specific neurons or wide dynamic range (WDR) neurons, which receive both noxious and non-noxious input.
  - **Third-order neuron:** Cell body located in thalamus. It sends its axon up to the cortex where perception of pain takes place.
- There are three major classes of opioid receptors:
  - Mu receptor (subtypes μ_1 and μ_2) is primarily responsible for analgesia. It is also responsible for respiratory depression, sedation, gastric dysmotility, nausea/vomiting, pruritus, euphoria and dependence.
  - Kappa receptor (subtypes κ_1, κ_2, κ_3) mediates analgesia, dysphoria, and diuresis. No respiratory depression.
  - Delta (δ_1, δ_2) mediates analgesia. No respiratory depression.

- Nociception involves several processes:
  - Detection: Sensing of noxious stimuli by nociceptors (nerve terminals).
  - Transduction: Translation of physical stimulus into electrical activity by nociceptors.
  - Transmission: Propagation of electrical activity in the nervous system.
  - Perception: Subjective interpretation of electrical activity.
  - Modulation: Modification (enhancement or inhibition) of electrical activity.

Peripheral Modulation

- **Primary hyperalgesia:** Nociceptors are activated by excitatory amino acids (eg, glutamate) and neuropeptides (eg, substance P). The release of potassium, bradykinins, or prostaglandins from damaged tissues sensitizes nociceptors to create greater excitability and frequency of firing.
- **Secondary hyperalgesia:** Mediated by substance P to ↑ the nociceptive field such that noninjured tissues are involved as well.

Central Modulation

- Central enhancement via three mechanisms:
  - **Wind-up phenomenon:** Persistence of action potential due to prolonged depolarization of the neuron despite the discontinuation of the stimulus.
    - Spinal reflexes act as efferents back to the peripheral nociceptive field, which generate more nociceptive afferents.
    - Expansion of receptive fields in dorsal horn.
- Central attenuation via two mechanisms:
  - **Gate theory:** Stimulation of nonpain fibers peripherally inhibits WDR neurons.
  - **Descending antinociceptive pathways:** These neurons originate from periaquedutal gray, reticular formation and nucleus raphe magnus and travel down to synapse in the dorsal horn to inhibit pain.

ACUTE PAIN

Usually associated with a clearly identifiable event (eg, trauma, surgery). It is a protective mechanism that warns the individual of actual or impending tissue damage. It is usually short-lived and resolves as the inciting injury heals. Acute pain can be treated with pharmacologic agents and/or neural blockade.

Treatment

Pharmacologic Agents

- **Nonsteroidal anti-inflammatory drugs (NSAIDs):**
  - **Cyclooxygenase-1 (COX-1) inhibitors:** Consist of several classes, including proprionic acid, anthranilic acid and salicylates. They have analgesic, antipyretic and anti-inflammatory properties. They block prostaglandin synthesis that sensitizes nociceptors. They also disrupt platelet function, may exacerbate bronchospasm and cause GI mucosal irritation.

- **COX-2 inhibitors** (eg, celecoxib): Have anti-inflammatory properties. They do not disrupt platelet function and cause fewer gastrointestinal (GI) side effects. However, they do have an ↑ incidence of cardiovascular complications.

- **Narcotics:**
  - Opioids may be considered "weak" or "strong." Codeine, oxycodone, hydrocodone, propoxyphene, and pentazocine are considered "weak" opioids because their side effects become significant before reaching good analgesic effects. Morphine, hydromorphone, fentanyl, methadone, and levorphanol are considered "strong" opioids.
  - Tramadol is a synthetic opioid-like drug that blocks reuptake of norepinephrine (NE) and 5-hydroxytryptamine (5-HT). It also acts weakly at the mu receptor.
  - Patient-controlled analgesia (PCA): A method of continuous fusion that gives the patient some control in titrating the medication to the desired analgesic level. Advantages include good analgesia efficacy and greater patient satisfaction. There is a lower frequency of side effects such as sedation and respiratory depression as the extreme plasma trough and peak concentration seen in IM or IV routes are avoided.
  - Common equianalgesic doses:
    - PO Morphine–IV morphine: 3:1.
    - Tramadol–morphine: PO—10:1; IV—5:1.
    - Meperidine–morphine: 7:1.
    - Codeine–morphine: 5:1.
    - Hydrocodone–morphine: 1:1.
    - Oxycodone–morphine: 1: 1.5.
    - Methadone–morphine: single dose—1:1.5; multiple doses—1:5.
    - Hydromorphone–morphine: 1:7.
    - Oxymorphone–morphine: 1:10
    - Alfentanyl–morphine: 1:10.
    - Fentanyl–morphine: acute—1:100; chronic—1:40.
    - Remifentanyl–morphine: 1:250.
    - Sufentanyl–morphine: 1:500–1000.
- **Benzodiazepines:** Treat psychogenic component of pain and also act as a strong muscle relaxant.

Neural Blockades

Trigeminal neuralgia is characterized by sharp pain in the trigeminal nerve (V2). Trigeminal neuralgia can be treated with a Gasserian ganglion block.

- **Neuraxial blocks** (spinal, epidurals): Provide good postoperative pain control, which results in earlier ambulation and participation in physical therapy, resulting in fewer postop complications. Local anesthetics result in sympathetic, sensory, and motor blockade. Usage of dilute concentrations of local anesthetics results in less motor blockade. Opioids alone do not cause motor or sympathetic blockade.
- **Peripheral nerve blockade and catheters:** Duration of block depends on the local anesthetic used. Continuous catheters allow for continuous infusions of medications to bring longer period of relief. It also allows for intermittent boluses. One should be cautious of possible motor blockade.
- **Trigeminal nerve block:** Used to treat facial pain, acute zoster, and as an adjunct to pharmacologic treatment of trigeminal neuralgia. Complications include intravascular injection and hematoma.
- **Intercostal nerve block:** Indications include rib and chest wall pain. Complications include pneumothorax.

- **Sympathetic nerve block:**
  - **Stellate ganglion block:** Used to treat acute pain in face and upper extremities secondary to frostbite, herpes zoster, or acute vascular insufficiency. Horner's syndrome is a known side effect of the block. Complications include pneumothorax, difficulty swallowing, hoarseness, and intravascular/subarachnoid/epidural injection and bleeding.
  - **Lumbar sympathetic block:** Used to treat acute pain of the lower extremities from frostbite, herpes zoster, and acute vascular insufficiency. Complications include hypotension, lumbar nerve root damage, and intravascular/subarachnoid/epidural injection.

Stellate ganglion block is important for both diagnosis and treatment of complex regional pain syndrome (CRPS). The stellate ganglion is located at the C6 (Chassaignac's tubercle) and formed from the fusion of the inferior cervical and first thoracic ganglion.

Trancutaneous Electrical Nerve Stimulation (TENS)

- Involves the use of a TENS unit in which a pulse generator delivers stimulation to treat painful areas via electrode stickers on the skin.
- Indications include acute postop or posttraumatic pain and musculoskeletal pain.
- It has also been used in peripheral vascular insufficiency and neuropathic pain.
- Contraindicated in patients with pacemaker, spinal cord stimulator, intrathecal pump, or those who are pregnant.
- There is currently not one single mechanism that can explain the analgesic effect of TENS. However, the gate control theory and the endogenous endorphin/enkephalin release theory can each provide a partial explanation.

Acupuncture

- A technique that is used for pain control through placement of solid needles into acupoints. Acupoints are areas on the skin that has ↓ resistance and ↑ conductance that corresponds to nerve endings.
- Many studies were carried out to study the efficacy of acupuncture but they are poorly designed. However, acupuncture is used in the management of chronic low back pain, neck pain and cancer pain.
- Usually considered a safe technique.
- Some adverse reactions include mild pain, bruising, nausea or vomiting.
- Relatively contraindicated in pregnancy.

CHRONIC PAIN

- Pain that persists longer than expected after an inciting event.
- Disease state in which a single treatment modality provides no benefit to the individual. It is often multifactorial and frequently is associated with psychiatric disorders such as depression and/or anxiety.
- It is a dysfunction of the central nervous system with aberrant pain signal processing.
- Patients suffer from chronic pain secondary to underlying diseases (eg, cancer, AIDS, diabetes mellitus [DM]), known pain syndromes (eg, postherpetic neuralgia, trigeminal neuralgia, migraine), idiopathic causes, or malingering behavior.

Treatment

Systemic Medications

- **NSAIDs:** See Acute Pain.
- **Opioids:** See Acute Pain.
- **Antidepressants:** Block reuptake of 5-HT and/or NE presynaptically. Commonly used in neuropathic pain. Examples: bupropion, amitriptyline, paroxetine, sertraline, venlafaxine.
- **Anticonvulsants:** Block the voltage-gated Na^+ channels, which inhibits the spontaneous neural discharges. Commonly used in neuropathic pain as well. Examples: gabapentin, pregabalin, phenytoin, carbamazepine, clonazepam, lamotrigine, topiramate.
- **Neuroleptics:** Block dompaminergic receptors. Side effects include extrapyramidal symptoms; antimuscarinic, antihistaminic, and α-adrenergic blockade. Used in refractory neuropathic pain. Examples: chlorpromazine, haloperidol.
- **Corticosteroids:** Serve as an anti-inflammatory, analgesic agent.
- **Systemic local anesthetics:** Given as a bolus or as an infusion for central analgesia.
- **α_2-adrenergic agonists:** Activate descending inhibitory pathways. Examples: clonidine, phenoxybenzamine, prazosin, phentolamine.
- **Botulinum toxin:** Blocks acetylcholine release at motor (not sensory) nerve endings to relieve spasm and improve blood flow.

Neural Blockades

- **Neuraxial blocks:** See Acute Pain.
- **Somatic nerve blocks:**
  - **Trigeminal nerve block:** For trigeminal neuralgia, intractable cancer pain of the face.
  - **Cervical paravertebral block:** For neck or shoulder pain.
  - **Thoracic paravertebral block:** For thoracic spine, rib cage, or abdominal wall pain (eg, herpes zoster).
  - **Lumbar paravertebral block:** For lumbar spine pain.
  - **Medial branch nerve block:** For facet joint disease.
  - **Trans-sacral nerve block:** For pelvic or perineal pain.
  - **Pudendal nerve block:** For perineal pain.
- **Sympathetic nerve blocks:** For sympathetic-maintaining pain.
  - **Lumbar sympathetic block:** For pelvis and lower-extremity pain.
  - **Stellate ganglion block:** For head, neck, arm, and upper chest pain.
  - **Celiac plexus block:** For abdominal viscera pain.
  - **Hypogastric plexus block:** For pelvic pain.
  - **Ganglion impar block**: For perineal pain.
  - **Intravenous regional sympathetic block (Bier block):** For pain in extremities. Agents used include guanethidine, reserpin, and bretylium.

*Stellate ganglion block can lead to Horner's syndrome (ptosis, miosis, and anhydriosis). This indicates interruption of sympathetic supply of the head and neck **only**.*

Other Techniques

- **Radiofrequency ablation:** Ablates nerves by passing electricity through a needle with an active electrode, which in turn → heating of the surrounding tissues. The effect generally lasts 3–12 months. A diagnostic block is indicated prior to ablation.

- **Cryoablation:** Neurolysis by freezing tissue through a cryoprobe. The effects generally lasts weeks to months. A diagnostic block is indicated prior to ablation.
- **TENS:** See Acute Pain.
- **Spinal cord stimulation:**
  - Involves the placement of electrode leads in the epidural space and use of a pulse generator, usually implanted in the abdomen or posterior superior gluteal area, to create a paresthesia in the painful area to control pain.
  - The mechanism of action is not understood.
  - It has been used effectively in complex regional pain syndrome, failed back surgery syndrome, angina, and peripheral ischemia.
  - A trial is indicated prior to the permanent implant.
  - Complications include infection, lead migration, and breakage.
- **Chemical neurolysis:**
  - Alcohol or phenol is used to destroy nerves.
  - Used for refractory cancer pain, neuralgia, or peripheral vascular disease.
  - Generally lasts weeks to months.
  - A diagnostic block is indicated prior to neurolysis.

Somatic Pain Conditions

Myofascial Pain

A distinct feature of myofascial pain is the presence of "trigger points."

- Localized muscle pain caused by myofascial trigger points. Trigger points are localized tender points of muscles within a taut muscle band. The trigger points are often found in the head, neck, shoulders, extremities, and low back. Pain should be reproducible upon palpation of the trigger points.
- The localized pain may be associated with referred pain.
- Suggested mechanism appears to be due to pathologic ↑ of acetylcholine release, causing sustained contraction, ischemia, and sensitization of nociceptors from released vasoactive substances, resulting in point tenderness.
- Treatment is physical therapy involving stretching exercise. Trigger point injections (dry needle or local anesthetic) are used in conjunction with physical therapy.

Facet Arthropathy

- A disease of the zygapophyseal joint in which exact pathology is unknown.
- A zygapophyseal joint is composed of the superior and inferior articular processes of two consecutive vertebra. It is innervated by two medial branch nerves, one from that level and one from the level above.
- Patient usually presents with back pain and, at times, also referred pain to the hips, buttocks, thighs or knees.
- On exam, one may find paraspinal tenderness; aggravation of pain upon spinal extension, rotation, or lateral flexion; pain in hip/buttocks/back upon straight leg raising with absence of radiculopathy.
- Diagnostic medial branch block with local anesthetics confirms the diagnosis.
- Once the diagnosis is confirmed, treatment is radiofrequency ablation of the medial branch nerves.

Neuropathic Pain States

Complex Regional Pain Syndrome (CRPS)

CRPS I: Secondary to soft tissue injury with no distinct nerve damage.
CRPS II (causalgia): Secondary to major but incomplete nerve damage.

- Consists of painful conditions that occur after an injury where the pain duration and severity are disproportionate to the injury.
- It is a clinical diagnosis. The criteria is that the condition develops after an injury, there is spontaneous pain or allodynia, evidence of abnormal sudomotor activity (edema, temperature, and skin blood flow abnormalities), and absence of other conditions that would otherwise account for the symptoms.
- The pathophysiology is in the central nervous system (CNS), where there is dysfunction of the sensory, autonomic, and motor system.
- The symptoms are located in the area of injury (nondermatomal) but may spread to other areas of the body.
- CRPS is classified into type I and type II. They are very similar except that type I can result from minor trauma, surgery, sprain, fractures, or dislocation, whereas type II involves injury to a major nerve.

Treatment

- As CRPS can be very debilitating, where patients suffer from extreme pain and motor dysfunction (↓ range and force of active motion, dystonia) of the affected extremity, treatment should be immediate.
- Treatment goal should be adequate pain control, which allows for the patient to participate in physical and occupational therapy.
- Pharmacologic treatment includes NSAIDs, opioids, antidepressants, anticonvulsants, calcium channel blocker (IV lidocaine) and *N*-methyl-D-aspartate (NMDA) receptor blockers (ketamine).
- Interventional therapies are sympathetic ganglion blocks and regional blocks with local anesthetics. The goal of sympathetic blocks is to block the abnormal sympathetic activity that occurs in this disease, which causes the sudomotor changes and trophic changes seen in CRPS patients.
- Patients who are refractory to pharmacologic treatment and blocks may be candidates for neurostimulation (spinal cord stimulation) or epidural clonidine.

Postherpetic Neuralgia (PHN)

- A complication of acute herpes zoster in which pain (dermatomal) persists after resolution of acute zoster rash.
- It occurs mainly in the immunosuppressed population.
- The symptoms can range from mild to debilitating, burning pain severe enough to → suicide.
- There is less chance of developing postherpetic neuralgia if the patient is treated aggressively (with sympathetic neural blocks, antivirals, anticonvulsants, and antidepressants) early on in the acute herpes zoster course.
- Treatments for PHN include antidepressants, anticonvulsants, opioids, topical lidocaine patches, topical capsaicin, and regional nerve blocks. Spinal cord stimulation has been used successfully.

Trigeminal Neuralgia

- A clinical diagnosis characterized by unilateral, paroxysmal, lancinating, stabbing pain lasting for seconds.

- Patients usually have trigger zones in which contact (pressure, wind, change of temperature, brushing) can trigger the pain.
- Etiology is unknown.
- It is classified either as type I (idiopathic) or type II (secondary to irritation, compression, or multiple sclerosis).
- Patients usually have a normal neurologic exam without sensory loss. However, patients with type II often have sensory deficits.

Treatment

- Treatment is often with one or more anticonvulsants (eg, carbamazepine, baclofen, velproate, gabapentin, lamotrigine, topiramate, pregabalin, clonazepam).
- When pain subsides, medication may be tapered off.
- Interventional techniques include glycerol rhizotomy, radiofrequency rhizotomy, balloon compression of the trigeminal nerve, and gamma knife radiosurgery.
- Microvascular decompression is the surgical option of choice.

Peripheral Neuropathy

- Functional disturbance of peripheral nerves that can cause impaired sensory, motor and/or autonomic function.
- There are many causes of peripheral neuropathies and can be classified as those that cause symmetric polyneuropathies (DM, drug toxicity, nutrition-related, cancer-related, infectious), mononeuropathies (nerve entrapment, localized physical damage, infectious) or multiple mononeuropathies (DM, HIV).
- The patient usually presents complaining of various symptoms including pain, numbness, weakness, or clumsiness.
- On exam, one can often see ↓ in sensation, loss of deep tendon reflexes (DTRs), and motor weakness. Before treatment is started, it is important to identify the cause, as the goal of treatment is directed toward treating the underlying disease.

Diabetic Neuropathy

- There are various forms of diabetic neuropathies, the most common form being distal symmetric polyneuropathy.
- Patient have symmetric, gradual onset of paresthesia and pain in their legs and feet.
- Treatment includes tighter glycemic control, antidepressants, and anticonvulsants. Opioids and sympathetic blocks are effective in some cases.

Phantom Limb Syndromes

- **Phantom limb sensation:**
  - Painless; usually described as a warm, tingling sensation.
  - Phenomenon occurs because the cortical representation of the limb remains despite amputation.
  - Occurs in almost all amputees but generally resolves in 2–3 years.
- **Phantom limb pain:**
  - Incidence varies greatly but ↑ with more proximal amputations.
  - Usually intermittent and has been described as burning, aching, sharp, stabbing.

- Pain is likely due to the functional and structural changes in the CNS.
- Pain usually remains unchanged or improved over years.
- Most commonly used drug therapies are antidepressents (eg, amitriptyline) and anticonvulsants (eg, gabapentin). Opioids are also used.
- Nerve blocks are commonly used, including sympathetic blocks, peripheral nerve blocks, and epidural or subarachnoid blocks.

Stump Pain

- Usually localized in the stump, and has been described as pressing, burning, throbbing.
- It is often associated with pathological findings in the stump, such as neuroma, bone spur, infection of skin/underlying tissues.
- Often treated with trigger-point injections, peripheral nerve blocks, or stump revision.

Poststroke Pain

- Patients have regional pain associated with abnormal sensitivity to temperature and noxious stimulation secondary to lesions in the CNS.
- Occurs most frequently to right-sided strokes that affect the left side of the body.
- Patient usually complains of a constant dysesthesia, described as burning, prickly, or achy, located in one part of the body, usually an arm or leg, on the affected side of the body.
- The pain can be aggravated by touch, movement, emotions, or temperature changes (usually cold).
- The onset of symptoms varies from weeks to years after injury and the intensity of pain usually ↑ over time.
- On exam, the patient can usually feel sensation in the affected skin area but can't feel the difference between different types of stimulation (hot vs. cold, sharp vs. blunt).
- Treatment is usually initiated with a tricyclic antidepressant.
- Other agents that may be added to the treatment include gapapentin, lamotrigine, or muscle relaxants.
- Deep brain stimulation, motor cortex stimulation, or spinal cord stimulation may be considered in patients without a good response to medications.

CANCER-RELATED PAIN

- Pain is common in cancer patients. The source of pain may be from the tumor itself, from cancer therapy, or from non-cancer-related sources.
- Fortunately, most patients' pain can be effectively managed with pharmacotherapy alone.
- However, if the pain is due to tumor involvement, anticancer therapy (radiotherapy, surgery, and, less commonly, chemotherapy) should be considered first.
- Addiction is seldomly seen in opioid-using cancer patients.

Treatment

- Systemic medications: Administered via oral, transdermal, or parenteral route.
- World Health Organization (WHO) analgesic ladder: A three-step approach to cancer pain management. WHO recommends to start with nonopioids for mild pain, weak opioids for moderate pain (eg, codeine, oxycodone), and stronger opioids for severe pain (eg, morphine, hydromorphone). Addiction is seldom seen in opioid-using cancer patients.
- Continuous spinal and epidural analgesia: Instituted when patient's pain is uncontrolled with systemic opioids or is suffering from severe side effects. Can be administered via percutaneous intrathecal/epidural catheters or via implanted intrathecal pumps.
- Neurolytic and non-neurolytic blocks: Neurolytic blocks are for terminally ill patients. May result in loss of motor and somatic sensory function. May be performed for intra-abdominal tumors (celiac plexus blocks), pelvic tumors (lumbar sympathetic, hypogastric plexus, ganglion impar or saddle neurolytic blocks), or rib metastasis (intercostal neurolytic blocks).
- TENS: See Acute Pain.
- Acupuncture: See Acute Pain.
- Spinal cord stimulation: If pain is neuropathic in origin. See above.

Celiac plexus is formed from the greater and lesser splanchnic nerves at the L1 vertebral body. Celiac plexus blocks are used for intractable pain caused by malignancies of the pancreas, stomach, liver, and gallbladder. Most common complications of celiac plexus block are hypotension and diarrhea.

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CHAPTER 18

Anesthesia for Ophthalmic and ENT Surgeries

Overall Goals

- Control of intraocular pressure (IOP).
- Avoidance and management of oculocardiac reflex (OCR).
- Motionless eye (akinesia).
- Consideration of adverse effects of ophthalmic drugs.
- Smooth induction and awakening.
- Concerns can differ according to type of ocular procedure (Table 18-1).

Azetazolamide ↓ IOP by inhibiting carbonic anhydrase and ↓ aqueous humor production.

Intraocular Pressure (IOP)

Factors Affecting IOP

- **Normal:** 10–22 mmHg; abnormal: > 25 mmHg.
- **Factors causing ↑ IOP:** Hypertension (HTN), hypercarbia, ↑ venous pressure, laryngoscopy/intubation, straining, vomiting, coughing, overhydration, extended periods of Trendelenberg.
- **Factors causing ↓ IOP:** Hyperventilation, hypothermia, central nervous system (CNS) depressants, ganglionic blockers, acetazolamide.

Effects of Anesthetic Drugs on IOP

- **Agents that ↑ IOP:** Succinylcholine, ketamine.
  - **Succinylcholine:**
    - Controversial use in open eye surgery.
    - Succinylcholine-induced ↑ in IOP might be due to either a drug-induced cycloplegic effect or prolonged contraction of extraocular muscles.

TABLE 18-1. Concerns with Various Ocular Procedures

Procedure	Concerns
Penetrating eye injury	Full stomach IOP control
Strabismus repair	Forced duction testing OCR Oculogastric reflex ? malignant hyperthermia association
Intraocular surgery	IOP control Akinesia Drug interactions Associated systemic disease
Retinal detachment surgery	OCR IOP control Nitrous oxide interaction with air or sulfur hexafluoride

IOP, intraocular pressure; OCR, oculocardiac reflex.

(Adapted, with permission, from Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 990.)

- Possible 5–10 mmHg ↑ in IOP within 1–4 min after IV administration with return to baseline within 7 min.
- IM administration has a longer duration of ↑ IOP.
- ↑ IOP is transient, and it is safe to use it in a majority of ophthalmic surgeries.

- **Ketamine:**
  - Use in eye surgery is questionable.
  - Raises the arterial blood pressure (BP) and does not relax the extraocular muscles.
  - May cause nystagmus, blepharospasm, and possibly an ↑ in IOP.
  - Small doses of ketamine do not ↑ IOP.

- **Agents that ↓ IOP:** Inhaled anesthetics, nitrous oxide (N_2O), most IV anesthetics (propofol, etomidate, benzodiazepines, barbiturates, opioids), nondepolarizing neuromuscular blocking drugs (NMBDs), osmotic diuretics (mannitol).
  - **N_2O:**
    - Use with caution when intravitral injection of air and sulfur hexafluoride (SF_6) is used to form an intraocular bubble during retinal detachment surgery.
    - Can diffuse into the intraocular bubble and ↑ IOP.
    - Rapid discontinuation of N_2O can → another retinal detachment.
    - If N_2O is used, it should be discontinued 15 min before creation of intraocular bubble and should be avoided for 10 days after SF_6 injection (SF_6 is absorbed by the body after 10 days and air bubble is absorbed after 5 days).

Succinylcholine transiently ↑ IOP by 5–10 mmHg through prolonged contracture of the extraocular muscles. Will abnormally cause ↑ in forced duction test–used to evaluate for type of strabismus surgery.

Anesthetic agents that ↓ IOP: Inhaled volatile agents. IV induction agents (except ketamine). Nitrous oxide (except in the presence of intraocular air).

Oculocardiac Reflex (OCR)

- Trigeminal afferent via ciliary and gassarian ganglion to fourth ventricle and vagal efferent.
- **Triggers:** ↑ external pressure on the globe, surgical traction of extraocular muscles, ocular regional anesthesia, hypercarbia, hypoxia, light anesthesia.
- Results in arrhythmias (bradycardia, junctional rhythm, ventricular premature complexes, asystole) and hypotension.
- **Treatment:** Removal of surgical stimulus, atropine (3–5 μg/kg), ↑ anesthetic depth.

*The oculocardiac reflex is most common in the pediatric population. Consists of an **afferent V1 (trigeminal)** and an **efferent vagal** reflex pathway.*

Ophthalmic Drugs

Topical administration of ophthalmic medication may produce adverse systemic effects:

- **Atropine** (cholinergic agonist; miosis):
  - Side effect: Central anticholinergic syndrome.
- **Timolol/betaxolol** (β antagonist):
  - Use: Glaucoma.
  - Side effects: Bradycardia and bronchospasm, especially in existing congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD), and bradycardia.
- **Echothiophate** (anticholinesterase):
  - Use: Glaucoma.
  - Side effects: Bronchospasm, prolonged duration of succinylcholine and mivacurium as it ↓ pseudocholinesterase activity (levels normalize in 4–6 weeks).

- **Phenylephrine** (α-adrenergic agonist; mydriasis):
  - Use: Papillary dilation and capillary decongestion.
  - Side effects: HTN, dysrhythmias.
- **Cyclopentolate** (anticholinergic; mydriasis):
  - Side effect: CNS toxicity (seizures, confusion).
- **Epinephrine** (sympathetic agonist; mydriasis):
  - Side effects: HTN, dysrhythmias.
- **Acetazolamide** (carbonic anhydrase inhibitor):
  - Use: ↓ IOP.
  - Side effects: Hyperchloremic metabolic acidosis, hypokalemia.

Anesthesia Management

General Anesthesia

- Indicated in children, uncooperative patients (mental illness), or patients who are unable to maintain a motionless position.
- Premedication: No contraindication.
- Anxiolytics: Benzodiazepines (diazepam 5–10 mg, midazolam 0.5–2 mg).
- Antiemetics: Metoclopramide, droperidol, ondansetron.

Patients with open globe injuries often have a full stomach, requiring rapid sequence induction.

Induction

- Induction technique depends mainly on patient's medical problems rather than patient's eye disease or type of surgery with the exception of ruptured globe surgery.
- ↑ risk of postoperative nausea and vomiting (PONV) due to OCR.
- Ruptured globe surgery: Prevention of ↑ in IOP crucial.
- Smooth induction and short laryngoscopy under deep anesthesia with aid of paralysis and topical and IV lidocaine (1.5 mg/kg) to blunt airway responses.
- Rapid-sequence induction often necessary.
- NMBDs: Use of succinylcholine is controversial; nondepolarizers can be used safely.

Maintenance, Extubation, and Emergence

- **Maintenance:** Sufficient depth of anesthesia using inhalationals, opioids, and paralytic agents to prevent eye motion, coughing, or straining.
- **Smooth extubation and emergence:** Pharyngeal suctioning while patient is deep, IV lidocaine prior to extubation to blunt airway reflexes, antiemetics to avoid ↑ IOP via Valsalva effect, optional deep extubation.

Regional Anesthesia

- Requires a cooperative and relatively motionless patient.
- **Indications:** Cataract extraction surgery, corneal transplants, and anterior chamber irrigation.
- **Advantages:** Lower incidence of coughing, straining, and PONV; postop analgesia.
- Deep sedation should be avoided due to risk of apnea and unintentional patient movement. Thus, often monitored anesthesia care (MAC) is performed with minimal sedation or no sedation.
- A short-acting anesthetic might be used to ↓ the discomfort during the block injection: midazolam (0.5–2 mg), propofol (10–50 mg), remifentanyl (0.1–0.5 μg/kg), alfentanil (375–500 μg).

Retrobulbar Block

- Local anesthetic is injected behind the eye into the cone formed by extraocular muscles.
- 50:50 mixture of 2–5 mL of 2% lidocaine and 0.75% bupivicaine +/– epinephrine is used.
- **Advantages:** Anesthesia, akinesia.

Sensation to the eye is provided by the trigeminal nerve (V) while the motor is controlled by the oculomotor (III), trochlear (IV), and abducens (VI).

Complications

- OCR.
- Optic nerve trauma.
- Hemorrhage.
- Transient globe compression and ↑ in IOP.
- Globe perforation.
- Trigeminal nerve block.
- IV injection (with seizure and cardiac depression).
- Postretrobulbar apnea syndrome (injection of local anesthetic into the optic nerve sheath with spread into cerebrospinal fluid [CSF] causing unconsciousness and apnea within 20 min).

Contraindications

- Bleeding disorders (risk of retrobulbar hemorrhage).
- Extreme myopia (risk of globe perforation).
- Open eye injury (risk of ↑ IOP and extrusion of intraocular contents).

Peribulbar Block

- The needle is advanced along the inferior orbital floor to depth of 2.5 cm (no needle entry into muscle cone).
- 8–10 mL of local anesthetic is injected; often, hyaluronidase is used to facilitate the spread throughout the cone muscles.
- **Advantages:** Akinesia, less pain on injection, less risk of eye penetration and optic nerve injury.
- **Disadvantages:** Slower onset and ↑ risk of ecchymosis.

The orbicularis oculi muscle is supplied by the facial nerve (VII). It is responsible for closing the eye. (The levator muscles supplied by the oculomotor (III) open the eye).

Sub-Tenon Block

- Local anesthetic is injected beneath the Tenon's facia that surrounds the globe and extraocular muscles, and it diffuses into the retrobulbar space.
- **Complications** (less than with retrobulbar and peribulbar blocks): Globe perforation, hemorrhages, cellulitis, visual loss, anesthetic spread into CSF.

Facial Nerve Block

- Prevents squinting of the eyelids and allows placement of a lid speculum.
- **Complication:** Subcutaneous hemorrhage.

Topical Anesthesia

- **Indications:** Anterior chamber surgeries (cataract extraction and glaucoma surgery), appropriate for a fast surgeon with gentle technique that can work without eye akinesia.

- Topical anesthetic drops are placed every 5 min for five applications, and anesthetic gel is placed into the conjunctival sacs.

OTORHINOLARYNGOLOGIC SURGERY

Special considerations in the anesthesia management:

- Shared airway with the surgeon (often, patient's head is away from anesthesiologist).
- Presence of potentially difficult airway secondary to the disease process, congenital abnormality, trauma, or deformities.
- Selection of an appropriate anesthesia technique and discussing it with the surgeon (type of endotracheal tube [ETT], use of laser, jet ventilation, total intravenous anesthesia [TIVA] vs. volatile agents).
- Recognition of the possibility of airway edema or bleeding at the conclusion of the surgery and planning appropriate tracheal extubation vs. no extubation.
- Use of controlled hypotension to ↓ intraoperative blood loss.
- Administration of epinephrine by the surgeon → cardiac dysrhythmias and systemic HTN.

The extent of airway obstruction must be considered prior to rendering the patient apneic.

Tonsillectomy and Adenoidectomy

- **Indications:** Airway obstruction, recurrent infection, obstructive sleep apnea.
- **Considerations:** Chronic hypoxia, hypercarbia, and acidosis due to airway obstruction.
- **Postoperative complications:** Hemorrhage, respiratory obstruction, aspiration.

Endoscopy

- Allows direct and indirect visualization of pharynx, larynx, trachea, bronchial tree, and esophagus.
- Endoscopy is performed for diagnostic purpose (biopsy) or treatment.
- **Anesthetic goals:**
  - Profound muscle relaxation to suppress cough and laryngeal reflexes and provide relaxed mandible; can be achieved with continuous succinylcholine infusion or intermittent administration of intermediate-duration NMBDs.
  - Minimization of secretions (glycopyrrolate).
  - Adequate oxygenation and ventilation while sharing the airway with the surgeon.
  - Protection of teeth with dental guard.

LARYNGOSCOPY

- Requires general anesthesia (with inhalation agents or TIVA) and muscle paralysis.
- Ventilation can be achieved with:
  - Endotracheal intubation with a small-diameter tube (4.0–6.0 mm). Disadvantage: small tube may be too short for adult trachea and can interfere with surgical field.

- Insufflation of high flows of oxygen via a small catheter placed in the trachea (ventilation is inadequate for longer procedures).
- Intermittent-apnea technique: Periods of ventilation via face mask (or ETT) alternate with periods of apnea. Duration of apnea is determined by patient's oxygen saturation. Risks: hypoventilation, hypercarbia, pulmonary aspiration.
- Jet ventilation through the side port on the laryngoscope or bronchoscope. IV anesthesia and muscle relaxation are required. During inspiration (1–2 sec) high-pressure oxygen (30–50 psi) is directed toward the glottic opening and into the lungs. Expiration is passive. Constant monitoring of chest wall motion and sufficient time should be allowed for exhalation to avoid air trapping and barotraumas. Complications: pneumomediastinum, pneumothorax, gastric aspiration, gastric distention, hypoventilation.

Expiration during jet ventilation is passive. Monitor chest wall excursion to allow sufficient time for exhalation to prevent barotrauma.

- Laryngoscopy is very stimulating and can cause cardiovascular instability (HTN and tachycardia). A moderate baseline anesthesia can be provided with supplementation of propofol or remifentanil as well as a sympathetic antagonist (esmolol) during periods of stimulation.

Bronchoscopy

- **Flexible bronchoscopy** can be performed with topical anesthesia and some sedation. If patient is uncooperative, general anesthesia should be provided with inhalational or TIVA and muscle relaxants. At least 8.0 mm ETT should be used for placement of flexible bronchoscope.
- **Rigid bronchoscopy** with rigid-ventilating or rigid-venturi (Sanders injector) bronchoscope requires general anesthesia. When using Sanders rigid bronchoscope, jet ventilation, TIVA and muscle paralysis should be provided. Patient movement during rigid broncoscopy can → tracheal tear or pneumothorax.

Esophagoscopy

Flexible esophagoscopy is performed with sedation and rigid esophagoscopy requires general anesthesia with a small-diameter ETT and muscle paralysis.

Laser Surgery

- Laser = **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation.
- It is an intense monochromatic, coherent, and collimated beam of light that provides surgical precision with minimal edema, faster healing, and less pain.
- The laser can produce long wavelengths (CO_2 laser, which can also cause corneal injury) or short wavelengths (yttrium-aluminum-garnet [YAG], which can damage the retina). As wavelength ↑, tissue penetration ↓.
- Laser surgery sometimes accompanies endoscopic procedures.
- Hazards associated with laser:
  - Emission of toxic fumes from tissue vaporization.
  - Misdirected laser beam and potential vessel or viscous perforation.
  - Ocular injury (eye protection should be worn by all OR personal).
  - Airway fire.

The ETT cuff can be filled with saline dyed with methylene blue to dissipate heat and signal to cuff rupture.

Precautions Against Airway Fire

- Fire-resistant stainless steel tube can be used with a cuff filled with saline solution.
- FiO_2 should be as low as possible (ideally < 30%): Use mixture of air/O_2 or O_2/helium; avoid N_2O.
- Limited laser intensity and duration.
- Readily available water and moist gauze.

Airway Fire Protocol

1. Remove laser.
2. Stop ventilation/gas flow.
3. Extubate the trachea.
4. Flush pharynx with saline and suction.
5. Ventilate with 100% oxygen.
6. Perform bronchoscopy to assess the damage.
7. Reintubate.
8. Obtain chest x-ray (CXR).

Nasal and Sinus Surgery

Procedures

Sensation to the nasal septum and lateral walls is supplied by branches of the trigeminal nerve (V).

- Polypectomy.
- Endoscopic sinus surgery.
- Rhinoplasty.
- Septoplasty.

Considerations

- Possibility of difficult face mask ventilation secondary to obstruction by nasal polyps or deviated septum.
- Nasal polyps are often associated with asthma or aspirin allergies.
- Possibility of large intraoperative blood loss: Controlled hypotension, epinephrine injection by surgeon, and slight head-up position can minimize blood loss.
- Use of RAE (Ring-Adair-Elwyn) tube vs. regular ETT for intubation (discuss with surgeon).
- Corneal abrasion risk due to proximity of surgical field.
- Use of posterior pharyngeal pack to minimize aspiration of blood.

Anesthesia Technique

- Local anesthesia with sedation or general anesthesia with NMBDs to avoid movement.
- Steroid administration at the beginning of the surgery to ↓ airway edema and PONV.
- Extubation: Remove pharyngeal pack, suction stomach and pharynx, ensure intact upper airway reflexes prior to extubation.

Head and Neck Surgery

Procedures

- Laryngectomy.
- Glossectomy.
- Pharyngectomy.
- Parotidectomy.
- Radical neck dissection including reconstructive surgery with free microvascular flap.

Considerations

- Secure potentially difficult airway. Airway may be difficult secondary to tumor, cancer, or radiation treatment. Consider awake fiber-optic intubation with emergent tracheostomy kit present.
- Consider elective tracheostomy prior to the surgery.
- Patients are typically elderly with a history of alcohol and/or tobacco abuse. Also evaluate for pulmonary, cardiac, and hepatic diseases.
- Manipulation of carotid sinus (right side > left side): Causes wide swings in blood pressure, bradycardia, prolonged QT interval, or asystole. Treatment: stop stimulation, atropine/glycopyrrolate, infiltrate carotid sinus with local anesthetic.
- Risk of pneumothorax and venous air embolism.

Anesthesia Technique

- May require facial or spinal accessory nerve monitoring: Maintain anesthesia with volatile agents and no muscle paralysis; short-acting NMBD can be used to facilitate intubation.
- Minimization of bleeding: Mild controlled hypotension using volatile agents, β blockers, vasodilators, and slight head-up position.
- Avoid vasoconstrictors, hypothermia, and vasodilators (hydralazine and nitroprusside can ↓ perfusion pressure of free flap) with microvascular free flaps.

Postoperative Complications

- Potential vocal cord paralysis: Injury to one recurrent nerve can cause unilateral vocal cord paralysis, which presents with hoarseness and weak voice. Bilateral vocal cord paralysis causes ↑ upper airway obstruction, stridor, inability to phonate, and requires reintubation.
- Neck hematoma and airway obstruction: Immediately evacuate hematoma.

Omission of NMB agents may be necessary to identify nerves and prevent damage by the surgical team.

Craniofacial and Orthognatic Surgery

Surgery required due to facial trauma, developmental malformations, or radical cancer surgery. Orthognatic surgery involves LeFort osteotomies or mandibular osteotomies.

Considerations

- Potentially challenging airway. Consider jaw opening, neck mobility, mask fit, micrognathia, macroglossia, dental pathology, nasal patency, and intraoral lesions and debris. Awake oral/nasal intubation or tracheostomy may be necessary.
- Nasal intubation is often preferred for oral and dental surgeries (contraindicated in LeFort type 3 fractures). Apply topical phenylephrine and well lubricate and soften ETT in warm fluid to minimize damage to turbinates and ↓ bleeding.

Complications of Nasal Intubation

- Epistaxis.
- False passage in mucosa.
- Turbinate damage.
- Nasal necrosis.
- Bacteremia.
- Sinusitis.
- Laryngitis, pharyngitis.

Anesthesia Technique

General anesthesia with controlled hypotension and muscle paralysis.

Ear Surgery

Myringotomy and Tube Insertion

- Usually a very short procedure with no need for tracheal intubation.
- Anesthesia can be managed with mask ventilation with volatile agent and N_2O.

Middle Ear Surgery

Procedures

Tympanoplasty, mastoidectomy.

Middle ear surgery can cause postoperative dizziness (vertigo), nausea, and vomiting. Induction and maintenance with propofol may ↓ PONV.

Considerations

- Facial nerve monitoring and preservation: Avoid paralysis except for a short-acting muscle relaxant for intubation.
- Controlled hypotension to ↓ bleeding.
- Use of epinephrine by the surgeon.
- Effects of N_2O on middle ear:
  - The middle ear is an air-filled nondistensible cavity. As N_2O is more soluble than nitrogen, it enters the space faster than air and ↑ the pressure. If normal venting of the middle ear pressure cannot occur, N_2O can → an ↑ in middle ear pressure. This can cause tympanic membrane rupture, disarticulation of artificial stapes, and disruption of surgical grafts.
  - Acute cessation of N_2O can result in negative middle ear pressure that can → graft dislodgment or serous otitis.
  - If N_2O is used, its maximum inspired concentration should be < 50%, and it should be discontinued about 30 min prior to placement of tympanic graft.
- PONV prophylaxis after middle ear surgery.

CHAPTER 19

Anesthesia for Genitourinary Surgery

CYSTOSCOPY

Cystourethroscopy

- Used to visualize urethra, bladder neck, and bladder.
- Flexible cystourethroscopy can usually be performed with local anesthetic jelly.
- More extensive procedures may require sedation.

Ureteroscopy

- Used to visualize ureters.
- Diagnostic and therapeutic procedures involve biopsies, dilatation of ureters or strictures, ureteral stent placement, removal of calculi, and removal of tumors.
- Often requires general or neuraxial anesthesia.

The lithotomy position can lead to many nerve injuries, especially to the common peroneal nerve, leading to a foot drop.

Intraoperative Considerations

Lithotomy Position

- **Iatrogenic injury risks:**
  - Common peroneal nerve injury causing foot drop if lateral thigh rests on straps.
  - Saphenous nerve injury causing medial calf numbness if legs rest medially on straps.
  - Obturator and femoral nerve compression if thigh is excessively flexed.
  - Compartment syndrome of lower extremities with prolonged time in lithotomy.
- **Physiological changes:** ↓ functional residual capacity (FRC) and ↑ venous return from elevated legs.

Neuraxial Anesthesia

- Must provide T10 level to block discomfort from bladder distention.
- Does *not* block obturator reflex when nerve is stimulated through lateral bladder wall causing external rotation and adduction of thigh.

TRANSURETHRAL RESECTION OF THE PROSTATE (TURP)

- A resectoscope is placed through a cystoscope and removes excess prostatic urethral tissue from benign prostatic hypertrophy (BPH).
- Concurrently, an irrigating solution flows through resectoscope, extending bladder, clearing debris, and providing a clear operative field.
- Neuraxial anesthesia to T10 level (for blocking pain from bladder distention) with minimal sedation to monitor for hyponatremia.

Irrigating Solutions

- The prostate contains a venous plexus that can absorb the irrigating solution if the sinuses are opened and the pressure of the irrigation solution is high.

TABLE 19-1. Advantages and Disadvantages of Irrigating Solutions

Solution	Advantages	Disadvantages
Glycine	Water intoxication less likely	Transient blindness Hyperammonemia, CNS depression
Sorbitol	Water intoxication less likely	Hyperglycemia Osmotic diuresis
Mannitol	Isosmolar solution	Acute intravascular expansion Osmotic diuresis

(Adapted, with permission, from Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 1027.)

- Hypo-osmolar solutions (such as distilled water) can be absorbed into the circulatory system, causing hyponatremia and hemolysis.
- The ideal solution is isotonic and nonhemolytic, nonelectrolyic to minimize current dispersion when electrocautery is used, transparent, nontoxic, and nonmetabolized.
- Various solutes have been added to water to make its osmolality similar to plasma, but these solutes have advantages and disadvantages (see Table 19-1).

Absorption of irrigation fluid is mainly dependent on duration of procedure and height (pressure) of the irrigation fluid.

Complications

Water Intoxication Syndrome

- Amount of irrigation absorbed is related to number and size of venous sinuses opened, duration of procedure (< 1 hr ideal), hydrostatic pressure of irrigation (irrigation bag suspended < 39 cm above OR table ideal) and venous pressure at irrigant-blood interface.

Manifestations

- Hypervolemia: Hypertension (HTN) and bradycardia → left heart failure/pulmonary edema/cardiovascular collapse.
- Hyponatremia: Various electrocardiographic (ECG) and central nervous system (CNS) changes (see Table 19-2).

Rapid correction of hyponatremia can lead to permanent demyelinating lesions (ie, central pontine myelinolysis).

TABLE 19-2. ECG and CNS Changes Associated with Hyponatremia

Serum Na^+ Concentration (mEq/L)	ECG Changes	CNS Changes
120	QRS widening	Restlessness Confusion
115	QRS widening Elevated ST segment	Nausea Somnolence
110	Ventricular tachycardia Ventricular fibrillation	Seizures Coma

Management

- **Mild symptoms:** Fluid restriction, furosemide.
- **Severe symptoms:**
  - Hypertonic saline at < 100 mL/hr and discontinued when $Na^+ > 120$.
  - Oxygenation and circulatory support.
  - Procedure termination with use of normal saline as irrigation for remainder of procedure.
  - ECG and baseline labs (complete blood count [CBC], platelets, electrolytes, coagulation profile).
  - Consider central venous catheter and arterial line.

Bleeding

Causes

- Open venous sinuses or arteries.
- Thrombocytopenia secondary to dilution from irrigant or excessive blood loss.
- Thromboplastin release from prostate cancer cells causing primary fibrinolysis that can be managed with aminocaproic acid. Secondary fibrinolysis can develop involving disseminated intravascular coagulation (DIC), for which aminocaproic acid is contraindicated.

Septicemia

Caused by bacteria spread through open sinuses.

Hypothermia

Caused by cold irrigation.

Bladder Perforation

- **Extraperitoneal** (more common): Pain in periumbilical, inguinal, or suprapubic region.
- **Intraperitoneal:**
  - Sudden abdominal pain referring to shoulder, nausea, vomiting, pallor, sweating, and shortness of breath under neuraxial anesthesia.
  - ↑ airway pressures under general anesthesia as fluid collects in intraperitoneal cavity, causing limited diaphragm movement.
  - Sudden hypotension or HTN with vagally mediated bradycardia.

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY (ESWL)

Patients with a history of cardiac dysrhythmias, pacemakers, and AICDs have an ↑ risk for developing arrythmias due to shock waves during ESWL.

- Used for upper ureteral and renal calculi.
- High-energy sound waves are focused on stone, causing it to fragment and pass through urinary tract.
- Stents are often placed to facilitate passage of stones.
- Shocks are synchronized with R wave to avoid supraventricular arrhythmias.
- Sedation is used for newer lithotripter models.
- Generous IV fluids to maintain brisk urinary flow.

Immersion Lithotripsy

- Immersion causes peripheral venous compression → ↑ central blood volume and cardiopulmonary changes (Table 19-3).
- Hypotension secondary to vasodilation from warm water despite ↑ venous return.
- Epidurals with T6 level can be implemented but as little air as possible should be used for loss of resistance technique, as air in epidural space can dissipate shock waves, causing neuronal injury.
- Spinals are used less commonly as they are associated with hypotension.

Contraindications

- **Absolute:** Bleeding disorder/anticoagulation, pregnancy, obstruction distal to calculi.
- **Relative:** Calcified aortic or renal artery aneurysms, urinary tract infection, pacemaker/automatic implantable cardioverter defibrillator (AICD), morbid obesity.

Complications

- Pulmonary contusions.
- Hematuria secondary to renal parenchymal damage.
- Flank pain and ecchymoses.

RADICAL PROSTATECTOMY

Regional anesthesia should NOT be used in patients with lumbar vertebral metastatic disease.

Preoperative Considerations

- Possible exposure to cyclophosphamide, 5-fluorouracil, cisplatin, or doxorubicin.
- Possible hypercoagulability.
- Neuraxial anesthesia is contraindicated with metastatic disease to lumbar spine.
- Extended time in Trendelenberg is associated with posterior optic neuropathy and can be exacerbated by large-volume fluid replacements and hypotension.

TABLE 19-3. Cardiopulmonary Changes in Immersion Lithotripsy

System	Change
Cardiovascular	↑ central blood volume ↑ central venous pressure ↑ pulmonary artery pressure
Pulmonary	↑ pulmonary blood flow ↓ vital capacity ↓ functional residual capacity ↓ tidal volume ↑ respiratory rate

(Adapted, with permission, from Barash PG et al. *Clinical Anesthesia,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2006: 1032.)

PERINEAL APPROACH

- Performed in lithotomy position with Trendelenberg.
- **Advantages:** Less blood loss, better surgical exposure, shorter recovery time.
- **Disadvantages:**
  - Ischemia of muscles in elevated legs causing rhabdomyolysis/acute renal failure (ARF).
  - Lower-extremity nerve damage secondary to positioning.
  - Ventilation difficulties secondary to compression of abdominal contents against diaphragm.
  - Venous embolism.
- **Anesthetic considerations:** Combined epidural general.

RETROPUBIC APPROACH

- Lower abdominal midline incision performed in a hyperextended supine position with Trendelenberg.
- **Disadvantages:**
  - Potential for blood loss.
  - Injuries to obturator nerve, ureter, and rectum.
  - Incontinence.
  - Impotence.
- **Anesthetic considerations:**
  - General or neuraxial (T6–8 level) with sedation (however, patients may not tolerate prolonged procedure under sedation).
  - Potential for upper airway edema secondary to prolonged Trendelenberg and large amounts of IV fluid.

LAPAROSCOPIC APPROACH

- Performed in a steep Trendelenberg position.
- **Advantages:** Less blood loss, shorter recovery time.
- **Disadvantages:** Longer surgical time, dysrhythmias, CO_2 embolism.
- **Anesthetic considerations:** General anesthesia with controlled ventilation.

Reflex renal vasoconstriction in the nonaffected kidney due to low perfusion can result in postoperative renal dysfunction.

RADICAL NEPHRECTOMY

PREOPERATIVE CONSIDERATIONS

- Degree of renal impairment.
- Anemia.
- Hypercoagulability.
- Underlying chronic obstructive pulmonary disease (COPD), coronary artery disease (CAD), diabetes mellitus (DM), and HTN.
- Anterior subcostal, flank, midline, or thoracoabdominal incision.
- Possibility of acute massive blood loss.
- Retraction of inferior vena cava (IVC) can cause transient hypotension.
- Adequate hydration to maintain perfusion of other kidney.
- Consider arterial line, central venous cannulation, and mannitol infusion to prevent ischemic injury from surgically induced renal vasospasm.

COMPLICATIONS

Pneumothorax, venous air embolism.

CHAPTER 20

Anesthesia for Patients with Liver Disease

FUNCTIONS OF THE LIVER

Glucose Homeostasis

- Hepatocytes store glucose as glycogen.
- Surgical stress → ↑ sympathetic activity → glycogenolysis and perioperative hyperglycemia.

All factors except VIII are synthesized in the liver.

Coagulation

- Hepatosplenomegaly can cause thrombocytopenia.
- Liver synthesizes all clotting factors except for factor VIII.
- Vitamin K–dependent factors are II, VII, IX, and X (which are blocked by coumadin).
- Prothrombin time (PT) tests for factor VII in extrinsic pathway, as well as factors I, II, V, and X of the common pathway.
- Factor VII has the shortest half-life of all clotting factors (4–6 hr) and is the first factor to become deficient in severe hepatic failure. PT is most sensitive to ↓ in factor VII.
- Activated partial thromboplastin time (aPTT) tests for factors VIII and IX of the intrinsic pathway as well as factors I, II, V, and X of the common pathway.

PT is most sensitive to factor VII, which has the shortest half-life.

Drug Metabolism

- Hepatocytes convert lipid-soluble drugs to more water-soluble and less pharmacologically active substances via oxidation and conjugation (most frequent), along with reduction and hydrolysis (less frequent).
- Pseudocholinesterase is produced in the liver, with a plasma half-life of 14 days. ↓ pseudocholinesterase activity will prolong the actions of succinylcholine, mivacuronium, and ester anesthetics.
- Hepatic metabolism occurs in opioids, pancuronium, vecuronium, rocuronium, and sodium nitroprusside.
- Albumin, with a half-life of 23 days, is ↓ in liver failure. Drugs normally bound to albumin, such as benzodiazepines and anticonvulsants, will have an ↑ free fraction, and thus a ↓ dose requirement.

PT tests factors I, II, V, VII, and X.

PTT tests factors I, II, V, VIII, IX, and X.

Bilirubin Formation and Excretion

- Erythrocyte life span is 120 days.
- Bilirubin is produced from breakdown of hemoglobin and is bound to albumin.
- Liver conjugates protein-bound bilirubin with glucuronic acid to make water-soluble bilirubin. Hepatocytes then conjugate bilirubin with glucuronic acid to form more soluble bilirubin glucuronide, which can be excreted into bile.
- Liver produces 1 L of bile per day. Impaired bile production can cause steatorrhea and vitamin K deficiency.

- Blood flow to the liver is 25% of the cardiac output (Figure 20-1).
- Hepatic blood flow consists of:
  - 70% from the portal vein.
  - 30% from hepatic artery.
- Each blood vessel contributes 50% of the liver's oxygen supply.

Determinants of Hepatic Blood Flow

- **Intrinsic regulation** of hepatic blood flow includes hepatic arterial buffer response, autoregulation, and metabolic control.
  - Hepatic arterial buffer response uses adenosine to regulate changes in portal venous flow with reciprocal changes in hepatic arterial flow. ↓ portal venous flow → ↑ adenosine levels → vasodilation → ↑ hepatic artery flow.
  - Autoregulation occurs during postprandial state. Vascular smooth muscles maintain constant blood flow despite changes in systemic arterial pressure.
  - Metabolic control: ↓ pH, ↓ oxygen content, or ↑ P_{CO_2} → ↑ hepatic artery flow.
- **Extrinsic regulation** includes neural control.
  - Hepatic artery: α_1, and dopamine-1 receptor stimulation → ↓ hepatic blood flow; β_2 stimulation → ↑ hepatic blood flow.
  - Portal vein: α_1 and dopamine-1 receptor activation → vasoconstriction.
  - Sympathetic stimulation → vasoconstriction of hepatic artery and mesenteric vessels → ↓ hepatic blood flow.
- Surgical incision sites close to the liver can ↓ hepatic blood flow.
- Positive pressure ventilation (PPV) can ↓ hepatic blood flow.

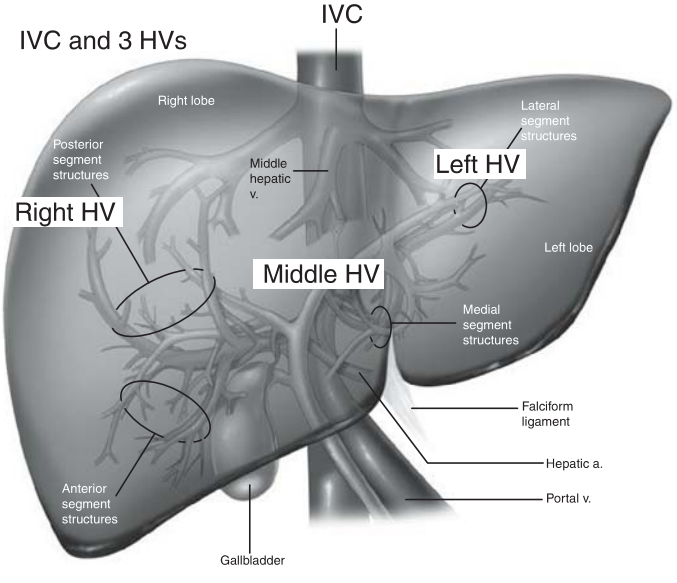

FIGURE 20-1. Hepatic blood flow.

(Reproduced, with permission, from Brunicardi FC et al (eds). *Schwartz's Principles of Surgery*, 9th ed. New York: McGraw-Hill, 2010: 1098.)

Factors that ↓ hepatic blood flow:

Volatiles (halothane most, isoflurane least).

Drugs: β blockers, α_1 agonists, H_2 blockers, vasopressin.

PPV and PEEP.

Hypoxia.

Hypotension from general or neuraxial anesthesia.

Sympathetic activation.

Surgical incision near liver.

- In liver failure, the liver is dependent on hepatic arterial perfusion since portal venous blood flow is ↓.
- Volatile anesthetics ↓ hepatic blood flow by ↓ blood pressure and ↑ hepatic vascular resistance through the release of catecholamines..
- Anesthetic agents that ↓ hepatic blood flow will reduce clearance of drugs with a high perfusion-dependent clearance rate (high extraction ratio). Example: Halothane can ↓ blood flow and reduce clearance of drugs that are highly perfusion dependent, such as fentanyl, verapamil, and propranolol.
- Acute hepatic disease can cause coagulopathy and encephalopathy.
- Fulminant liver failure is the development of encephalopathy within 8 weeks of the onset of symptoms in patients with a previously healthy liver.
- Hepatic encephalopathy with subfulminant liver failure develops between 8 weeks and 6 months.

Etiology

- **Viral:** Hepatitis A, B, C, D + E (especially if pregnant), herpes simplex virus (HSV; immunocompromised), Epstein-Barr virus (EBV), cytomegalovirus (CMV), adenovirus, paramyxovirus, parvovirus B19.
- **Drugs/toxins:** Acetaminophen, halothane, phenytoin, isoniazide, rifampin, amiodarone.
- **Vascular:** Ischemic hepatitis, Budd-Chiari, malignant infiltration.
- **Autoimmune.**
- **Miscellaneous:** HELLP (hemolysis, elevated liver enzymes, low platelets).

ACUTE LIVER FAILURE

- Acute hepatic disease can cause coagulopathy and encephalopathy.
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- Vascular: Ischemic hepatitis, Budd-Chiari, malignant infiltration.
- Autoimmune.
- Miscellaneous: HELLP (hemolysis, elevated liver enzymes, low platelets).

PERIOPERATIVE MANAGEMENT OF LIVER DISEASE

Anesthetic Considerations

- Halothane hepatitis:
  - Halothane is oxidized in the liver by cytochrome P-450 to a trifluoroacetylated metabolite that binds to liver protein. The altered protein is thought to induce humoral or T-cell sensitization, which during subsequent exposure can lead to hepatotoxicity.

- Types of hepatic injury:
  - Mild injury occurs in 20% of adults who receive halothane is characterized by focal necrosis, mildly ↑ alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Self-limited.
  - Severe injury is characterized by ↑ AST, ALT, bilirubin, and alkaline phosphatase. Massive hepatic necrosis with a mortality rate of 50–70%. Incidence rate of halothane hepatitis is 1/10,000.
- The incidence of liver injury caused by fluorinated inhaled anesthetics: Halothane > enflurane > isoflurane > desfurane.
- Methoxyflurane, enflurane, and isoflurane all produce fluoride ions during hepatic metabolism, but only methoxyflurane produces fluoride in quantities associated with renal dysfunction.
- Diminished ability to metabolize narcotics in the liver failure patient.
- Vecuronium undergoes significant hepatic metabolism → potential for prolonged neuromuscular blockade.
- Highly protein-bound drugs will have higher free fraction (pharmacologically active form) because of the reduction in serum albumin associated with liver disease.
- Postpone elective surgery until any acute episode of hepatitis has resolved; there is ↑ perioperative morbidity (12%) and mortality (up to 10% with laparotomy) during acute viral hepatitis.
- Acute alcohol withdrawal may be associated with mortality as high as 50%. Benzodiazepines, thiamine, folate, and glucose are given for acute alcohol withdrawal.
- Hypokalemia and metabolic alkalosis are present due to vomiting. Hypoglycemia and hypomagnesemia are common.
- ↑ PT and INR are indicators of hepatic synthetic function.
- Albumin is a marker for protein synthesis, and slowly ↓ in liver failure.
- Consider fresh frozen plasma (FFP), vitamin K (takes 24 hours for full response), and platelet transfusion if necessary.

Halothane is associated with hepatotoxicity. Methoxyflurane is associated with renal dysfunction.

Cirrhosis

- **Etiologies:** Alcohol, viral hepatitis (chronic HBV, HCV, HDV), autoimmune hepatitis, biliary tract disease, vascular, cryptogenic.
- Fibrosis and nodular regeneration occur secondary to hepatocellular injury.
- **Laboratory studies:** Elevated bilirubin, elevated PT, hypoalbuminemia, aminotransferase may be elevated, hyponatremia, anemia (marrow suppression, hypersplenism, iron/folate deficiencies), neutropenia (hypersplenism), thrombocytopenia (hypersplenism, ↓ thrombopoietin production in liver).
- **Portal hypertension:**
  - Portal venous pressure is > 10–12 mmHg.
  - Ascites is due to ↓ oncotic pressure (albumin < 3.5), ↑ resistance of blood flow in portal system, and ↑ secretion of antidiuretic hormone (ADH). $T_{1/2}$ of albumin is 23 days. Patients are at risk for spontaneous bacterial peritonitis. ↑ volume of distribution for highly ionized drugs (eg, neuromuscular blocking agents) may require larger loading doses.
  - Gastroesophageal varices and possible upper gastrointestinal bleed (UGIB) can be managed with nonselective β blockers, nitrates, somatostatin, vasopressin to reduce the rate of blood loss. Endoscopic sclerosis, ligation of varices, transjugular intrahepatic portosystemic shunt (TIPS) are additional approaches.

- **Hepatic encephalopathy** is due to failure of liver to detoxify ammonia (NH_3) and other noxious substances. Common precipitants include excess dietary protein, UGIB, infection, hypokalemia, renal failure, hepatic failure, and diuretics. Encephalopathy can be treated with antibiotics such as neomycin that ↓ colonic concentration of ammoniagenic bacteria, or lactulose, which reduces colonic bacterial load.
- **Hepatorenal syndrome** is associated with azotemia, oliguria, no response to volume challenge, and UNa < 10 mE/L.
  - The syndrome can be precipitated by:
    - UGIB.
    - Overdiuresis.
    - Paracentesis.
    - Aminoglycosides.
    - Nonsteroidal anti-inflammatory drugs (NSAIDs).
    - Treat with octreotide, midodrine, norepinephrine, albumin, and possibly TIPS.
- **Portopulmonary hypertension** is an uncommon complication of portal hypertension. Diagnostic criteria include mean pulmonary artery pressure > 25 mmHg, pulmonary vascular resistance > 120 dyn sec cm^{-5}, and pulmonary capillary wedge pressure > 15 mmHg. Portopulmonary hypertension carries high perioperative morbidity and mortality and does not resolve after liver transplantion.

CHAPTER 21

Anesthesia for Patients with Endocrine Disease

- Thyroid hormone production involves the uptake of iodine from diet, reduction to iodide in gastrointestinal (GI) tract, and binding of iodide with tyrosine residues to form various iodotyrosines that are eventually bound to thyroglobulin proteins and stored as colloids to be released by the thyroid gland.
- Thyrotropin-releasing hormone from the hypothalamus stimulates release of thyroid-stimulating hormone (TSH) from the anterior pituitary, which releases bound and unbound triiodothyronine (T_3) and thyroxine (T_4).
- Eighty percent of T_3 is produced from T_4.
- Unbound product is more potent.
- T_3 is more potent then T_4.
- T_4 is elevated in 90% of patients with hyperthyroidism, and low in 85% of patients with hypothyroidism.
- Half-life of T_3: 24–30 hours.
- Half-life of T_4: 6–7 days.

Diagnosis

- Hyperthyroidism: Normal/↓ TSH, ↑ T_3/T_4.
- Primary hypothyroidism: ↑ TSH, normal/↓ T_3/T_4.
- Secondary hypothyroidism: ↓ TSH, ↓ T_3/T_4.
- Starvation, fever, stress, and corticosteroids depress TSH levels.

Hyperthyroidism

Clinical Features

- Major manifestations: Weight loss, diarrhea, muscle weakness, heat intolerance, nervousness.
- Cardiovascular manifestations: ↑ left ventricular contractility/ejection fraction, ↑ systolic blood pressure, ↓ diastolic blood pressure.
- Other manifestations: hypercalcemia, thrombocytopenia, anemia.

Etiology

- **Graves' disease:**
  - Most common cause of hyperthyroidism.
  - Usually occurs in women ages 20–40.
  - Frequently have thyroid-stimulating autoantibodies.
- **Thyroid adenoma:** Second most common cause (cold adenomas more likely to be malignant).
- **Thyroid storm:**
  - Life-threatening exacerbation of hyperthyroidism during stress or illness.
  - Patients exhibit hyperthermia, tachycardia, dysrhythmias, myocardial ischemia, congestive heart failure (CHF), agitation, and confusion. Differential includes pheochromocytoma, malignant hyperthermia, light anesthesia.
  - Treat with fluid, sodium iodide, PTU, hydrocortisone, propranolol, cooling blanket, acetaminophen.
- Other causes:
  - **Subacute thyroiditis:** Frequently follows respiratory illness.
  - **Hashimoto's** (chronic autoimmune disease): Can produce hyperthyroidism in early stages.
  - **Amiodarone:** Iodine rich and can cause iodine-induced thyrotoxicosis.

Treatment

- Goal is to make patient euthyroid.
- Inhibit organification of iodide and synthesis of thyroid hormone:
  - PTU and methimazole: ↓ hormone synthesis. Takes several weeks to manifest effects. Also ↓ peripheral conversion of T_4 to T_3.
  - β blockade: Blocks sympathetic overactivity and impairs peripheral conversion of T_4 to to T_3.
  - Dexamethasone: ↓ peripheral conversion of T_4 to T_3.
  - Inorganic iodide: ↓ hormone synthesis and secretion over several weeks; can precipitate thyrotoxicosis so PTU must be given first.
- Radioactive iodine: Avoid in pregnancy as it crosses placenta and could effect fetal thyroid.
- Myasthenia gravis has higher incidence among hyperthyroid patients, and muscle relaxants should be reduced.

MAC is unchanged in hyperthyroidism.

Anesthetic Considerations

- Potentially difficult airways:
  - Consider CXR, CT, flow volume loops to evaluate potential extrathoracic or intrathoracic obstruction.
  - Avoid pancuronium, ketamine, epinephrine, and vasoactive drugs provoking catecholamine release.
  - Anesthetic requirements (MAC) are not increased but ↑ cardiac output slows rate of rise of alveolar concentration.
- Postoperative complications:
  - Superior laryngeal nerve injury: Can cause aspiration.
  - Recurrent laryngeal nerve injury: Unilateral damage can cause hoarseness; bilateral damage can lead to airway obstruction necessitating intubation.
  - Hypocalcemia: Caused by removal of parathyroid glands, appears 24–72 hours after surgery; can cause laryngospasm.
  - Airway compression: Caused by hematoma or tracheomalacia.
  - Hypothyroidism.

Hypothyroidism

- **Clinical features:**
  - Bradycardia.
  - ↓ cardiac output.
  - ↑ peripheral resistance.
  - Accumulation of pericardial fluid may produce low voltage electrocardiogram (ECG).
  - Depressed ventilatory responses to hypoxia and hypercapnia which can be potentiated by sedatives and opioids.
  - Also associated with lethargy, anemia, coagulopathy, hypothermia, hyponatremia, and gastroparesis.

Etiology

- Failure of thyroid gland function.
- Medical or surgical treatment of hyperthyroidism.
- **Myxedema coma** is a severe form of hypothyroidism, characterized by stupor/coma, hypoventilation, hypothermia, hyponatremia (medical emergency—high mortality: 25–50%) requiring aggressive therapy with intubation, controlled ventilation, levothyroxine, hydrocortisone, fluid and electrolyte repletion, and conservation of body heat without warming blankets.

Treatment

Synthroid: Half life of 7 days.

Anesthetic Considerations

- No increase in complications in patients with mild to moderate hypothyroidism.
- Severely hypothyroid patients require partial treatment prior to surgery.
- Ketamine useful for induction.
- Volatiles can depress the myocardium.

PARATHYROID DISEASE

- Normal adult has 1–2 kg of Ca^{++}, 99% of which is in the skeleton.
- Calcium forms: Protein bound (50%), ionized (45%, active form), nonionized (5%).
- Serum Ca^{++} levels are controlled by parathyroid hormone (PTH) and vitamin D.
- PTH ↑ Ca^{++} levels via:
  1. Bone resorption.
  2. Distal renal tubule resorption.
  3. Synthesis of 1,25-dihydroxyvitamin D.
- Acute low magnesium stimulates PTH; chronic low magnesium inhibits proper function of parathyroid gland.
- Vitamin D stimulates Ca++ and phosphate absorption in bones, kidneys, and GI tract; ↓ Vit D can cause ↓ absorption of Ca^{++} and cause secondary hyperparathyroidism.
- Vitamin D is absorbed by GI tract or produced enzymatically by ultraviolet light, hydroxylated to inactive form in liver, and then hydroxylated to active form in kidneys.

Hyperparathyroidism

Etiology

- Primary hyperparathyroidism due to adenoma (90%), hyperplasia (9%, involves all four glands), carcinoma (1%) including multiple endocrine neoplasia (MEN) syndrome.
- Secondary hyperparathyroidism is caused by chronic hypercalcemia/ hypophosphatemia seen in renal failure and GI malabsorption.
- Hyperparathyroidism in pregnancy has 50% mortality rate for mother and fetus.

Clinical Features

- Nephrolithisis.
- Bone demineralization.
- Muscle weakness.
- Constipation.
- Depression.
- Psychosis.
- Hypertension.
- Enhanced cardiac function enhanced in early stages.
- ECG: Shortened QT, prolonged PR and QRS, heart block.

Treatment

- Hydration with normal saline.
- Furosemide.
- Mithramycin: Inhibits PTH activity.
- Calcitonin: Inhibits bone resorption.
- Glucocorticoids: Inhibits bone resorption and GI absorption.
- Hemodialysis.

Anesthetic Considerations

- Unpredictable responses to neuromuscular blockers.
- The use of general anesthesia is common, but a cervical plexus block can be performed for a parathyroidectomy.
- Postoperative complications: Recurrent laryngeal nerve injury, bleeding, transient/complete hypoparathyroidism.

Hypoparathyroidism

Etiology

- Most common cause is inadvertent removal of parathyroid glands.
- Other causes: Iodine therapy, neck trauma, granulomatous disease, malignancies, amyloidosis.

Clinical Features

- Neuronal irritability.
- Skeletal muscle spasm.
- Tetany.
- Seizures.
- Chvostek's or Trousseau's sign.
- Fatigue, depression, paresthesias, cramps, CHF, hypotension, prolonged QT interval.

Treatment

- Calcium chloride or calcium gluconate for acute treatment.
- Calcium supplements, vitamin D, phosphate binders for chronic therapy.

Anesthetic Considerations

Avoid alkalosis or hyperventilation as this can further depress Ca^{++} levels.

DISEASES OF THE ADRENAL GLAND

Adrenal Cortex

- Secretes three types of hormones: glucocorticoids, mineralocorticoids, and androgens.
- Hormone release triggered by ACTH release from anterior pituitary

Cortisol

- Release stimulated by ACTH and inhibited by cortisol.
- Maximal activity after awakening.

Steroid potency:

Cortisol	*1*
Hydrocortisone	*1*
Prednisone	*4*
Methylprednisone	*5*
Dexamethasone	*25*

- Effects: Elevates blood glucose, promotes glycogen synthesis, enhances muscle degradation, suppresses growth hormone secretion, stabilizes lysosomes, promotes capillary integrity, reduces white cell adherence to endothelium, facilitates free-water clearance, maintains blood pressures, promotes appetite, stimulates hematopoiesis.

Aldosterone

- Release stimulated by hyperkalemia, hyponatremia, hypotension, ACTH, angiotensin II.
- Effects: Resorbs sodium and secretes potassium by binding to distal convoluted tubules.

Cushing's Syndrome

Cushing's: HTN, K^+

Conn's: HTN, hypokalemic metabolic alkalosis

Addison's: Hypotension, K^+

- Causes: Excess cortisol from pituitary tumors (ACTH secreting), adrenal cortical/lung/testis/prostate/pancreas tumors, exogenous steroids.
- Manifestations: Hypertension, hypokalemic alkalosis, hyperglycemia, truncal obesity, osteoporosis, psychosis, infections, muscle weakness.
- Diagnosis:
  - Urinary cortisol and 17-hydroxycorticosteroid levels.
  - Plasma ACTH.
  - Dexamethasone suppression test: Dexamethasone given to patient with subsequent measurement of cortisol and ACTH levels.
- Management: Diuretics, insulin, K^+.
- Anesthetic concerns:
  - Treat HTN and hyperglycemia; normalize intravascular volume and K^+.
  - Carefully position osteopenic patients.
  - Patients undergoing adrenalectomy should receive steroids and may require mineralocorticoids 5 days postoperatively.
  - Etomidate can inhibit steroid synthesis and can be a temporizing measure for Cushing's disease.

Edema is seen in secondary hyperaldosteronism, not Conn's disease.

Conn's Disease (Primary Hyperaldosteronism)

- Cause: Excess aldosterone caused by adrenal adenoma/hyperplasia (secondary hyperaldosteronism results from renin-angiotensin system activation).
- Manifestations: HTN, hypokalemic metabolic alkalosis, hypernatremia, polyuria, polydipsia, muscle weakness, no edema.
- Diagnosis: Low serum renin.
- Management: Spironolactone, amiloride/triamterene, K^+.

Addison's Disease (Primary Adrenal Insufficiency)

- Glucocorticoid and mineralcorticoid deficiency.
- Cause: Autoimmune destruction (exogenous adrenal suppression, tuberculosis, pituitary insufficiency cause secondary adrenal insufficiency).
- Manifestations:
  - Hypotension, hypovolemia, hyperkalemia, hyponatremia, fatigue, muscle weakness, anorexia, nausea/vomiting, diarrhea, hyperpigmentation.

- Acute crisis: Abdominal pain, severe vomiting and diarrhea, hypotension, loss of consciousness, shock.
- Management: Hydration, glucocorticoid (prednisone), adrenocorticoid (fludrocortisone), K^+.

Rate-limiting step of tyrosine to epinephrine or norepinephrine is conversion of tyrosine to dopamine via tyrosine hydroxylase.

Adrenal Medulla

- Embryologically from neuroectodermal cells.
- Secretes 80% epinephrine, 20% norepinephrine.
- Rate-limiting step of epinephrine/norepinephrine production: Conversion of tyrosine to dopamine via tyrosine hydroxylase.
- Half-life of epinephrine/norepinephrine: 10–30 seconds.
- Metanephrine and vanillylmandelic acid (VMA) are the major end of catecholamines.

Pheochromocytoma

- Occurs in < 0.2% of hypertensive population.
- **Rule of 90/10:** Surgery is curative in 90% of cases, 90% solitary tumors, 10% in extra-adrenal sites, 90% in abdomen, 10% malignant spread.
- Associated with inherited familial autosomal-dominant traits: MEN IIa and IIb, von Recklinghausen's neurofibromatosis, von Hippel–Lindau disease.

Clinical Features

Paroxysmal hypertension (which can lead to cerebrovascular hemorrhage, myocardial infarction, heart failure), orthostatic hypotension (from hypovolemia), headache, diaphoresis, palpitations, nausea/vomiting.

Diagnosis

Serum catecholamines and urinary catecholamine metabolites.

Preoperative Management

- Correction of hypovolemia and hypertension which can take up to 2 weeks.
- α blockers (phenoxybenzamine or prazosin): Started prior to β blockade as unopposed α vasoconstriction occurs if β blocker given first; side effects include orthostasis and tachycardia.
- β blockers (metoprolol, propranolol): Treats tachycardias or tachydysrhythmias, not always necessary.
- α-methyl tyrosine: Inhibits tyrosine hydroxylase; used in patients who are not surgical candidates.

Intraoperative Management

- Keep patients deep during laryngoscopy to minimize sympathetic stimulation.
- Intraoperative blood pressure lability common—HTN occurs before ligation of vein, and hypotension results after ligation.
- Hypertensive crisis: Titrate shorter-acting drugs such as nitroprusside, phentolamine, and esmolol.
- Avoid halothane (sensitizes myocardium to dysrhythmias), sympathomimetics (ketamine), histamine-releasing drugs (atracurium, tubocurarine, droperidol, pancuronium, morphine, meperidine), and succinylcholine (fasciculations can ↑ intra-abdominal pressure and release catecholamines from tumor).

Drugs to Avoid with Pheochromocytoma—

Kindly Avoid These Drugs in Pheo So You Avoid M&M

Ketamine
Atracurium
Tubocurarine
Droperidol
Pancuronium
Succinylcholine
Anticholinergics
Morphine
Meperidine

Insulin

Life-threatening complications with DM: DKA, nonketotic hyperosmolar coma, hypoglycemia.

- Functions:
  - Glucose: Facilitates glucose entry into cells, ↑ glycogenolysis.
  - Fat: ↑ triglyceride synthesis and storage, ↑ lipolysis.
  - Protein: ↑ protein synthesis.
- Metabolized by liver and kidneys.
- Does not affect glucose transport in brain and liver.

Physiological Effects of Diabetes Mellitus

- Endocrine: Hyperglycemia, diabetic ketoacidosis (DKA), hyperosmolar coma, hypoglycemia.
- Cardiovascular: Coronary artery disease (CAD), peripheral vascular disease (PVD).
- Neurologic: Autonomic neuropathy (resting tachycardia, orthostasis, silent myocardial ischemia, gastroparesis), peripheral neuropathy.
- Renal: Nephropathy.
- Musculoskeletal: Stiff joints.
- Immunologic: Impaired wound healing.
- Ophthalmologic: Cataracts, retinopathy.

Diabetes Types

- Type I: Insulin-dependent, prone to ketoacidosis.
- Type II: Non-insulin-dependent, associated with obesity.

Management

- Oral hypoglycemics: Sulfonylureas (glyburide, glipizide).
- Biguanides (metformin), rosiglitazone (Avandia), pioglitazone (Actos).
- Insulins: See Table 21-1 for a comparison of the different medications.

TABLE 21-1. Summary of Bioavailability Characteristics of the Insulins

	Insulin Type[2]	Onset	Peak Action	Duration
Short-acting	Lispro	10–20 min	30–90 min	4–6 h
	Regular, Actrapid, Velosulin	15–30 min	1–3 h	5–7 h
	Semilente, Semitard	30–60 min	4–6 h	12–16 h
Intermediate-acting	Lente, Lentard, Monotard, NPH, Insulatard	2–4 h	8–10 h	18–24 h
Long-acting	Ultralente, Ultratard, PZI	4–5 h	8–14 h	25–36 h

[1]There is considerable patient-to-patient variation.

[2]NPH, neutral protamine Hagedorn; PZI, protamine zinc insulin.

(Reproduced, with permission, from Morgan GE et al. *Clinical Anesthesiology,* 4th ed. New York: Appleton & Lange, 2006: 807, Table 36-5.)

Complications

Diabetic Ketoacidosis (DKA)

- Etiology: Often triggered by infection, insulin deficiency → hyperglycemia and ketone production → osmotic diuresis, dehydration, metabolic acidosis.
- Manifestations: Nausea/vomiting, polyuria, tachypnea, hypotension, stupor, abdominal pain.
- Diagnosis: Anion gap acidosis, hyperglycemia (rarely > 500 mg/dL), ketosis.
- Management: IV fluids, insulin, potassium, D5W infusion, bicarbonate.

Nonketotic Hyperosmolar Coma

- Etiology: Elderly patients with Type II and poor PO intake have enough insulin to prevent ketosis but not hyperglycemia.
- Manifestations: Nausea/vomiting, dehydration, altered mental status, seizures, coma.
- Diagnosis: Severe hyperglycemia (often > 1000 mg/dL), no ketones.
- Management: IV fluids, insulin, potassium.

Hypoglycemia

- Nondiabetic patients may have blood sugars as low as 50 mg/dL without being symptomatic, but diabetic patients are more sensitive to low sugars.
- Manifestations: Light-headedness, seizures, coma, tachycardia/diaphoresis/tremor from sympathetic activation; symptoms masked with intubation, β blockade, or other sympatholytic drugs.
- Patients who are given sulfonylureas and insulin without supplemental glucose are at higher risk for hypoglycemia, warranting frequent intraoperative blood glucose levels.

Anesthetic Considerations

Preoperative

- Consider comorbid diseases such as CAD, PVD, and renal insufficiency.
- Treat as full stomach due to gastroparesis.
- Check morning glucose level.
- Insulin regimen:
  - Withhold morning dose of oral hypoglycemics.
  - Give half of morning dose of intermediate-acting insulin.

Intraoperative

- Possible difficult laryngoscopy secondary to stiff joint syndrome, causing problems with the temporomandibular joint and cervical spine mobility.
- Risk of intraoperative hypothermia due to autonomic neuropathy.
- Ischemic peripheral nerves are more prone to injury during positioning; hyperglycemia exacerbates neuronal ischemic damage.
- Check glucose levels regularly and maintain between 80 and 110 mg/dL as it ↓ risk for sepsis, renal failure, neuropathy, and mortality.

Postoperative

Continue to monitor glucose levels.

- Neurologic symptoms from tumor compression:
  - Lateral visual defects from compression on optic chiasm.
  - Hydrocephalus from obstruction.
  - Cranial nerve palsies from compression (cranial nerves III, IV, V).
- Endocrinologic symptoms from hypopituitarism or hormone hypersecretion.
  - Causes of hypopituitarism: Gland compression, postpartum hemorrhagic shock (Sheehan's syndrome), hypophysectomy.
  - Causes of hypersecretion: Adenomas.

Anterior Pituitary

Anterior pituitary secretes FLAT PiG:

FSH
LH
ACTH
TSH
Prolactin
Growth hormone

- Secretes: FSH, LA, ACTH, TSH, prolactin, growth hormone.
- FSH: Stimulates normal female sexual development; produces inactive adenomas.
- LH: Stimulates normal male sexual development; produces inactive adenomas.
- ACTH: Stimulates cortisol release; excess causes Cushing's disease.
- TSH: Stimulates T_3 and T_4 release; excess causes hyperthyroidism.
- Prolactin: Stimulates breast development during pregnancy; excess causes amenorrhea and galactorrhea, bromocriptine or octreotride as treatment.
- Growth hormone: Stimulates tissue growth, protein synthesis; excess leads to acromegaly, which is characterised by an overgrowth of skeletal, soft, and connective tissues.
  - Anesthetic concerns in acromegaly:
    - Difficult airway: Enlarged mandible, nose, and lips make mask ventilation difficult; large tongue and excessive pharyngeal tissue make intubation difficult; ↓ tracheal diameter.
    - Other: HTN, CAD, V/Q mismatching, glucose intolerance, osteoporosis.

Posterior Pituitary

- Secretes: Oxytocin and ADH.
- Oxytocin: Elicits uterine contraction and milk letdown.
- ADH: Maintains extracellular fluid volume and plasma osmolality.
  - Stimulated by: ↓ intravascular volume, pain, positive pressure ventilation, ↑ plasma osmolality.
  - Facilitates free-water absorption via distal renal tubules → ↓ plasma osmolality and urine output.
  - Promotes hemostasis by ↑ levels of von Willebrand factor and factor VIII. Desmopressin (DDAVP) reduces transfusion requirements in cardiopulmonary bypass and can reverse coagulopathy of renal failure.

Diabetes Insipidus (DI) vs. Syndrome of Inappropriate ADH Secretion (SIADH)

See Table 21-2 comparing DI and SIADH.

TABLE 21-2. DI vs SIADH

	DI	SIADH
Etiology	Central: Loss of ADH secondary to posterior pituitary destruction (trauma, neoplasm) Nephrogenic: Failure of renal tubules to respond to ADH	Surgery Tumors (brain and lung) Hypothyroidism Porphyria Drugs (cyclophosphamide, clofibrate)
Clinical manifestations	Large volume of dilute urine ↑ serum osmolality Hypernatremia Hypovolemia	Concentrated urine ↓ serum osmolality Hyponatremia
Management	Fluid replacement IM ADH for central causes Chorpropamide for renal insensitivity Clofibrate Carbamazepine Thiazide diuretics	Fluid restriction Furosemide Demeclocycline: Antagonizes ADH effects on renal tubules Hypertonic saline for severe symptomatic hyponatremia—risk of central pontine myelinolysis with rapid correction

OBESITY

- Body mass index (BMI) = weight (kg) / [height (m)]^2. See Table 21-3 for the relationship between BMI and obesity classification.
- Obesity is associated with multiple endocrine and other problems, including:
  - Hypertension (10 times more prevalent in morbidly obese).
  - High low-density lipoprotein (LDL) and triglycerides; low high-density lipoprotein (HDL).

TABLE 21-3. Relationship Between BMI and Obesity Classification

BMI	Status
18.5–24.9	Normal
25.0–29.9	Overweight
30.0 and above	Obese
> 35	Morbid obesity

- DM and associated gastroparesis.
- Family history of premature heart disease, CAD.
- Hypothyroidism.
- Physical inactivity, degenerative joint disease.
- Obstructive sleep apnea, Pickwickian sydrome (obesity hypoventilation syndrome).
- Gallbladder disease (elevated liver function tests, cholelithiasis)
- Cigarette smoking, psychological and socioeconomic impairment.

Physiologic Changes

- Respiratory:
  - ↑ O_2 consumption and CO_2 production.
  - ↓ functional residual capacity (FRC), vital capacity, expiratory reserve volume, total lung capacity.
  - Unchanged tidal volume, residual volume, closing capacity.
  - V/Q mismatch.
- Cardiovascular:
  - ↑ cardiac output.
  - Hypertension.
  - Pulmonary hypertension.
- Gastrointestinal:
  - Hiatal hernia, gastroesophageal reflux (GERD).
- Endocrine
  - Diabetes mellitus.
- Hematologic
  - DVT and PE.

Anesthetic Considerations

Preoperative

- Aspiration prophylaxis as GERD is common.
- Nutritional deficiencies are associated with bariatric surgery.

Intraoperative

- Anticipate difficult airway and rapid desaturation from ↓ FRC.
- Neck circumference as good predictor of problematic intubation in obese patient.
- Laryngoscopy position: Elevate head, upper body, and shoulders above chest.
- Supine position may cause restrictive lung disease resulting in hypoxemia and hypercarbia.
- Preoxygenation vital.
- PEEP may ↓ V/Q mismatching and hypoxia.
- Rhabdomyolysis, ↑ creatinine, and ↑ creatine phosphokinase can be seen with prolonged cases.

- Anesthetic agents:
  - Water soluble drugs: Use ↑ doses.
  - Fat soluble drugs: Use larger doses initially but fewer maintainance doses required due to large volume of distribution.
  - ↑ dose of succinylcholine due to ↑ pseudocholinesterase activity.
  - Inhalational agents which are fat soluble may lead to delayed emergence (isoflurane > sevoflurane > desflurane).
  - Nitrous oxide is fat insoluble but may ↑ pulmonary vascular resistance which can worsen right ventricular dysfunction in patients with pulmonary hypertension.
  - Neuraxial anesthesia: Placement may be difficult, use smaller local volumes.

POSTOPERATIVE

- Respiratory insufficiency: Consider oberservation in monitored setting, supplememtal oxygen.
- Epidurals for pain management: ↓ DVT risk, improved analgesia, ↓ O_2 consumptions, ↓ left ventricular stroke work.

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CHAPTER 22

Anesthesia for Organ Transplantation

Rule out reversible causes of unresponsiveness prior to declaration of brain death:
Drugs/ toxins
Metabolic derangements
Hypothermia
Hypotension

Brain Dead Donors

Declaration

- Clinical brain death is declared when irreversible loss of brain function ensues.
- Reversible causes of unresponsiveness must be ruled out: Drugs/toxins, metabolic derangements, hypothermia, hypotension.
- Electroencephalogram (EEG), transcranial Doppler, and brain perfusion scans can aid in declaring death.
- Brain stem reflexes must be absent.
- Spinal reflexes may be intact and spontaneous limb movement may occur.

Preoperative Assessment and Preparation

- Protocol for clinical brain death and consent for donation should be clearly documented.
- Hemodynamic instability ensues with brain death.
- Common regimens for preservation of organ function include infusion of triiodothyronine (T_3), methylprednisolone, desmopressin (DDAVP), insulin, mannitol, antibiotics, and vasopressors.

Intraoperative Management

- Maintain euvolemia with crystalloids and colloids with central venous pressure (CVP) between 6 and 12 mmHg.
- Maintain oxygen carrying capacity with hematocrit > 30%.
- Maintain International Normalized Ratio (INR) < 1.5 with fresh frozen plasma (FFP).

Living Donors

Kidney

- Donors must be free from significant disease with normal renal function.
- Donors often require no invasive monitoring but a CVP line may guide fluid replacement.
- Open donor nephrectomy requires a large incision, which can be painful.
- Laparoscopic approach is well tolerated but pneumoperitoneum by CO_2 will ↓ renal blood flow. Maintain urine output > 100 mL/hr.
- Donors are often extubated in the OR.
- Intravenous patient-controlled analgesia (IV PCA) is usually sufficient for postoperative analgesia.
- Complications: Atelectasis, pneumothorax, wound infection, urinary tract infection.

Liver

- Left lobe donation is often performed for parent-to-child donation.
- Right lobe donation has a higher mortality and is a larger undertaking.
- Large resections require hepatic venous exclusion resulting in > 50% ↓ in venous return.
- ↑ endogenous catecholamine release maintains blood pressure.

- Anesthetic technique can vary:
  - Low CVP technique (CVP < 5) reduces blood loss and allows for better venous exclusion.
  - Volume-loading technique maintains hemodynamics and causes hemodilution to avoid blood transfusion.
- Blood loss is usually limited, but blood should always be available.
- Most patients are extubated in the OR.
- Pain management is usually adequate with intraoperative loading of narcotics and an IV PCA.
- Epidural catheters are often avoided as there is controversy regarding the timing of their removal due to coagulopathy.
- Complications: Air embolism, atelectasis, pneumonia.

RENAL TRANSPLANTATION

Preoperative Assessment

- Patients are medically optimized with a focus on cardiovascular risk factor modification.
- Preoperative tests: Electrocardiogram (ECG), echocardiogram, pulmonary function tests (PFTs), viral serologies.
- Patients on dialysis can be dialyzed prior to transplantation as cadaveric kidneys can be preserved for 24–48 hr.
- Correction of existing coagulopathy.

Intraoperative Management

- Central venous access guides fluid therapy to maintain CVP > 10.
- Arterial cannulation: Performed to maintain systolic blood pressure (SBP) > 90, which is achieved with crystalloid and colloid administration and without use of vasopressors.
- Rapid-sequence induction due to potential gastroparesis from diabetes.
- Neuromuscular blockade: Any nondepolarizing muscle relaxant can be used, but cisatracurium is most common.
- Immunosuppressive therapy may be initiated in the OR.
- Grafted kidney is placed in the right iliac fossa with the anastamosis of the renal vessels to the iliac vessels and the ureter to the bladder, while the native kidney is usually not removed.
- Heparin is given prior to clamping of the iliac vessels.
- Calcium channel blocker injection into arterial circulation of graft helps protect against reperfusion injury.
- After the first anastamosis is made, mannitol and/or furosemide may given for diuresis.
- Hyperkalemia from the preservative solutions is possible after the release of vascular clamp.
- Monitor electrolytes, as the transplanted kidney is unable to concentrate urine and reabsorb sodium.
- Patients are usually extubated in the OR.

Maintain SBP through fluids, not vasopressors, during a renal transplant.

Postoperative Management

- Postoperative analgesia: IV PCA; avoid nonsteroidal anti-inflammatory drugs (NSAIDs).
- Recovery in postanesthesia care unit (PACU) or intensive care unit (ICU) for a 24-hr period.
- Complications: Vascular thrombosis, ureteral obstruction, fistula formation, wound infections.

Preoperative Assessment

- Common indications:
  - Adults: Nonalcoholic cirrhosis, primary biliary cirrhosis, sclerosing cholangitis.
  - Children: Biliary atresia, inborn errors of metabolism, postnecrotic cirrhosis.
- Patients with end-stage liver disease have multisystem dysfunction.
- Preoperative tests: Stress echocardiogram to examine ischemia, valvular abnormalities, and severity of pulmonary HTN to determine candidacy and risk stratification; PFTs to establish extent of hepatopulmonary syndrome and hypoxia.

Intraoperative Management

- Rapid-sequence induction due to gastroparesis, ↑ intra-abdominal pressure from ascites or recent upper GI bleed.
- Arterial cannulation for pressure monitoring, central venous access with a large-bore introducer with or without a pulmonary artery (PA) catheter, and several large-bore peripheral IVs.
- Use of epidural catheters is discouraged due to perioperative coagulopathy.
- Postinduction hypotension due to low systemic vascular resistance and hypovolemia may be treated with vasoconstrictors.
- Veno-venous bypass is not employed in many centers, but there is always a possibility that it may be necessary.
- A rapid-infusion system should be in the OR with large volumes of FFP and PRBCs available.
- Keep INR < 1.5 with FFP, platelet count > 50,000/mm^3 and fibrinogen > 150 mg/dL.
- Rapid transfusion of FFP and PRBCs will cause citrate binding of calcium. Low-dose infusion of $CaCl_2$ will aid in maintenance of a normal ionized calcium.
- Three phases of surgery:
  1. **Dissection phase:**
     - Wide subcostal incision with dissection of liver.
     - Very high blood loss during this phase.
  2. **Anhepatic phase:**
     - Hepatic excision occurs with clamping of the IVC above and below the liver as well as clamping of the hepatic artery, portal vein, and common bile duct.
     - The donor liver is anastamosed to the IVC and portal vein.
     - Venous return ↓ by >50% with IVC cross-clamping.
     - Many patients can be managed with volume loading and vasopressors.
     - Veno-venous bypass may be employed to restore venous return and improve hemodynamic stability and minimize intestinal edema, acid buildup, and renal dysfunction. Risks: Air embolism, arm lymphedema, vascular injury.
     - Physiological derangements: Hypocalcemia (and subsequent myocardial depression), acidosis, hypo/hyperglycemia.
  3. **Neohepatic phase**
     - Caval clamps are removed and hepatic artery and portal vein are anastamosed and then the common bile duct is anastamosed.

Metabolic derangements during liver transplant: Hypocalcemia, acidosis, hypo/hyperglycemia, hyperkalemia.

- Portal vein reperfusion results in hemodynamic instability.
- $NaHCO_3$ and $CaCl_2$ can be given as portal unclamping is begun to counteract the increasing acid and potassium load (from the preservative solution).
- Sudden ↑ in blood volume can cause arrhythmias.

Postoperative Management

- Patients are observed in the PACU and/or a unit designated for liver transplantation.
- Complications: Persistent hemorrhage, volume overload, metabolic abnormalities, renal and respiratory failure, anastamosis thrombosis and leak, encephalopathy, intracranial hemorrhage, right phrenic nerve injury, infection, rejection.

LUNG TRANSPLANTATION

Preoperative Assessment and Considerations

- Common indications: Cystic fibrosis, COPD, α_1-antitrypsin deficiency, idiopathic pulmonary fibrosis, pulmonary HTN.
- Cor pulmonale does not necessarily require combined heart-lung transplant since right ventricular heart function can stabilize once pulmonary artery pressures normalize.
- Transplant options: Single, en bloc double, sequential double, heart-lung transplantation.
  - Double-lung transplantation is performed for bullous disease, cystic fibrosis and vascular disease.
  - Single-lung transplantation is reserved for COPD.
- Patients usually have dyspnea at rest with resting hypoxemia ($PaO_2 < 50$ mmHg) and CO_2 retention.
- Preoperative tests: PFTs, echocardiography, cardiac catheterization, viral serologies.
- Preoperative medications: Continue steroids, bronchodilators and vasodilators; immunosuppressants may be started preoperatively.
- Postoperative pain management: Thoracic epidural (if CPB unlikely).

Intraoperative Management

- Review NPO status and consider rapid-sequence induction, as cases are not delayed in order to minimize graft ischemia time.
- Slow induction undertaken with etomidate, ketamine, and opioids as hypotension may be observed.
- Avoid histamine-releasing muscle relaxants.
- Maintain anesthesia through a combination of opioids and an inhalational agent.
- PA catheter placement necessary with the catheter pulled back prior to lung resection.
- Hypotension treated with vasopressors (dobutamine) rather than fluid boluses.
- Methylprednisolone given prior to release of vascular clamps.
- Avoid hypoxia or hypercarbia to prevent pulmonary artery pressure fluctuations.

Single-Lung Transplantation

- Often performed without CPB via a posterolateral thoracotomy.
- A double-lumen ETT or single-lumen ETT with a bronchial blocker is used for lung isolation.
- CPB may be initiated if the patient is unable to tolerate one-lung ventilation with resultant hypoxemia and ↑ pulmonary artery pressures.
- Nitric oxide (NO), prostaglandin E_1, milrinone, nitroglycerin, and dobutamine, are used to control pulmonary HTN.
- After pneumonectomy, the pulmonary artery, atrial cuff, and bronchus are anastamosed.
- Ventilation and perfusion to the donor lung are resumed and flexible bronchoscopy performed to check the suture line.
- TEE is useful to evaluate the extent of cardiac dysfunction.
- The double-lumen ETT is exchanged for a single-lumen ETT, as patients remain intubated.

Double-Lung Transplantation

- Performed with CPB via a clamshell sternotomy in the supine position.
- In an en bloc procedure, a single-lumen ETT can be used.
- Sequential transplantation requires a double-lumen ETT and is the preferred procedure, as a tracheal anastomosis is unnecessary and blood loss is less.

Postoperative Management

- Minimize peak inspiratory pressures.
- Maintain $FiO_2 < 60\%$.
- HPV remains normal, while the cough reflex of the transplanted lung may be blunted.
- Extubation should be performed as early as possible.
- Postoperative complications: Acute rejection, infections, renal and hepatic dysfunction, reperfusion injury, pulmonary edema secondary to loss of lymphatic drainage, ischemic breakdown of suture line secondary to loss of bronchial circulation, damage to phrenic, vagus, and left recurrent laryngeal nerves.

CHAPTER 23

Anesthesia for Laparoscopic Surgery

PHYSIOLOGICAL EFFECTS OF LAPAROSCOPY

Cardiovascular Effects

CV effects:
↑ SVR
↓ preload
↑ MAP
↑ filling pressures
↓ cardiac index
Unchanged HR

- The hemodynamic response to laparoscopy depends on the intra-abdominal pressure, patient positioning, neurohumoral response, intravascular volume, and preexisting cardiac status.
- Hemodynamic changes:
  - ↑ systemic vascular resistance (SVR) due to compression of arterial vasculature, catecholamine release ↓ preload (due to compression of venous vessels and ↓ venous return in reverse Trendelenburg), and vasopressin production.
  - ↑ mean arterial pressure (MAP).
  - ↑ filling pressures.
  - Transient ↓ cardiac index (proportional to intra-abdominal pressure).
  - Unchanged heart rate (HR).

Respiratory Effects

Respiratory effects:
↓ FRC
↓ compliance
↑ peak pressures
↑ atelectasis
↑ V/Q mismatch
↑ $Paco_2$

- The changes in pulmonary function during laparoscopy are due to mechanical effects and CO_2 absorption.
- Mechanical effects:
  - ↓ functional residual capacity (FRC).
  - ↓ compliance.
  - ↑ peak airway pressures.
  - ↑ atelectasis.
  - ↑ ventilation/perfusion (V/Q) mismatch.
- CO_2 effects:
  - High blood solubility and pulmonary excretion reduce risk of adverse effect in case of gas embolism.
  - Absorption is greater in extraperitoneal vs intraperitoneal insufflation.
  - $PaCO_2$ plateaus at 15–30 min of insufflation.

Regional Blood Flow

- ↑ cerebral.
- ↓ renal.
- Unchanged splanchnic.

ANESTHETIC TECHNIQUE

Airway Management

- Endotracheal intubation and controlled ventilation:
  - Reduces $PaCO_2$ and avoids ventilatory compromise.
  - Reduces risk of aspiration if reflux occurs due to high intra-abdominal pressure.
  - Tracheobronchial shift upward may cause tube to migrate into right mainstem bronchus.
- Epidural/spinal anesthesia:
  - High level required for complete muscle relaxation.
  - Difficult to ↑ respirations to maintain normocarbia.

Monitoring

- Standard ASA monitors.
- Check ABGs if patient is at risk for acidosis.

Analgesia

- Laparoscopy is associated with intra-abdominal, incisional, and shoulder pain.
- Multimodal analgesia is necessary:
  - Opioids.
  - NSAIDs.
  - Local anesthetic infiltration.

INTRAOPERATIVE COMPLICATIONS

Intra-abdominal Injuries

- Vascular injury to major vessels or abdominal wall vasculature (can cause bleeding into retroperitoneal space indicated by unexplained hypotension—requires conversion to open procedure to repair injury).
- Gastrointestinal tract perforations (decompression of stomach with nasogastric tube may ↓ risk of inadvertent puncture by trochar).
- Hepatic or splenic tears.
- Mesenteric lacerations.

Cardiac Arrhythmias

- Bradycardia (caused by ↑ vagal tone).
- Asystole.

CO_2 gas embolism:

↑ $ETCO_2$

Hypotension

Hypoxemia

Asystole

Extraperitoneal Insufflation

Subcutaneous emphysema (can cause hypercapnia and respiratory acidosis—no intervention required).

Pneumothorax/Pneumoperitoneum/Pneumopericardium

- Unexplained ↑ airway pressures:
  - Hypoxemia.
  - Surgical emphysema.
- Hypotension.

Gas Embolism

- ↑ $ETCO_2$ with use of CO_2, for insufflation (↓ $ETCO_2$ with use of air and/or nitrogen for insufflation).
- Hypotension.
- Hypoxemia.
- Asystole.

POSTOPERATIVE CONCERNS

- **Diaphragmatic dysfunction:** Follows laparoscopic cholecystectomy for 24 hr.
- **Postoperative nausea and vomiting:** Administer antiemetics at end of procedure.
- **Venous stasis and thromboembolism:**
  - Sequential compressive devices should be used.
  - Early postoperative ambulation.
- **Bile duct injury:** Pain and jaundice are initial symptoms (may require surgical drainage).

CHAPTER 24

Anesthesia for Orthopedic Surgery

PREOPERATIVE ASSESSMENT

History

Includes preexisting medical problems, previous anesthetic complications, potential airway difficulties, and intraoperative positioning.

Airway Management

Atlantoaxial instability:
Rheumatoid arthritis
Down syndrome
Anklyosing spondylitis
Mucopolysaccharidosis

- Orthopedic patients with potentially difficult airways:
  - Ankylosing spondylitis: Fusion of cervical spine.
  - Juvenile rheumatoid arthritis: Ankylosis of cervical spine, hypoplasia of mandible.
  - Adult rheumatoid arthritis: Multiple deformities, ankylosis and instability of the cervical spine.
  - Prior spinal fusion: Ankylosis and limited extension of the cervical spine.
- Congenital deformities of the cervical spine:
  - Epiphyseal dysplasia.
  - Dwarfism (achondroplasia).
  - Fractured cervical spine: Limited motion, risk of quadriplegia.
- Atlantoaxial (C1–C2) instability:
  - Rheumatoid arthritis.
  - Down syndrome.
  - Ankylosing spondylitis.
  - Mucopolysaccharidosis (eg, Morquio's disease).
- Rheumatoid arthritis and ankylosing spondylitis are musculoskeletal diseases in which there are specific airway management and perioperative anesthetic concerns.

Rheumatoid Arthritis

- Arthritis may affect joints involved in airway management:
  - Temporomandibular joint—limited opening of mouth requiring a fiberoptic intubation.
  - Cricoarytenoid joint—associated with hoarseness or stridor necessitating the use of a smaller endotracheal tube.
  - Cervical spine—atlantoaxial subluxation can compromise cerebral blood flow and injury to brainstem and spinal cord. > 5 mm posterior atlanto-odontoid distance on lateral C spine film necessitates fiberoptic intubation to prevent flexion of the neck.
- Using regional anesthesia with the patient minimally sedated and the neck stabilized is a reasonable perioperative alternative.
- Other anesthetic concerns:
  - **Cardiovascular:** Pericardial thickening and effusion, myocarditis, coronary arteries, conduction defects, vasculitis, cardiac fibrosis, aortic regurgitation, and coronary artery disease (CAD) from steroid use (consider perioperative β blockers); preoperative assessment of cardiorespiratory status may be difficult due to limited exercise history (consider stress test), calcified arteries compounded with wrist flexion deformities may contribute to difficult intra-arterial catheterization.
  - **Pulmonary:** Pleural effusion, pulmonary nodules, and interstitial fibrosis may be present.

- **Hematopoietic:** Anemia, eosinophilia, aspirin-induced platelet dysfunction, thrombocytopenia, and immune dysfunction.
- **Endocrine:** Adrenal insufficiency from chronic steroid use.
- **Dermatologic:** Thin, atrophic skin.
- **Immunologic:** Immunosuppression from steroids and anti-inflammatory medications.

- Postoperative considerations of rheumatoid arthritis patients:
  - Development of airway obstruction postoperatively from narcotics or sedatives; judicious use of narcotics or epidural analgesia for pain relief should be considered, considering possible poor cardiopulomary reserve.
  - Administration of nasal oxygen and pulse oximetry.
  - Cardiopulmonary resuscitation may be difficult, given possible cervical neck instability and cardiopulmonary status.
  - Emergency tracheotomy is almost impossible in severe cases. Jet ventilation by means of a percutaneous catheter through the cricothyroid membrane may be required.
  - Carpal tunnel syndrome may predispose to recurrent symptoms after intra-arterial cannulation.

> 5 mm posterior atlanto-odontoid distance on lateral C spine film necessitates fiber-optic intubation.

Ankylosing Spondylitis

Ossification of ligaments attachment to bone, including joint cartilage at disk space of the axial skeleton. Arthritis and ankylosis may also develop in the hips, shoulders, and costovertebral joints.

Anesthetic Concerns

- Consider fiber-optic techniques for tracheal intubation.
- Impaired lung function from the development of rigidity of the rib cage (vital capacity is minimally reduced if diaphragmatic activity is preserved).
- Careful positioning in the operating room is important as there is an ever-present risk of spine fracture and cervical spine instability in these patients; therefore, position patient while the patient is awake.
- Aortic regurgitation and bundle branch block may develop, necessitating aortic valve replacement or pacemaker insertion.
- Lumbar epidural or spinal anesthesia may be difficult or impossible as vertebral column is usually fused, but in patients who can still move the neck, fusion of the lumbar spine may be incomplete, enabling epidural or spinal anesthesia to be performed successfully.
- Caudal anesthesia can be readily obtained (usually reserved for children, though possible on adults).

POSITIONING FOR ORTHOPEDIC SURGERY

Patients are placed in a variety of positions for orthopedic procedures. Improper positioning may result in intraoperative or postoperative problems.

Anesthetic Considerations

- Air embolisms can occur when the operative field is above the level of the heart as in shoulder surgery in the sitting position or total hip replacement in the lateral decubitus position.

- Stretch or malposition of joints may occur during anesthesia and can account for a variety of nonspecific postoperative discomforts in the back or the extremities patients with rheumatoid arthritis, osteoporosis, osteogenesis imperfecta, or contractures must be carefully positioned to avoid ligamentous or bony injury.
- Direct pressure, especially over bony prominences, may cause tissue ischemia or necrosis, particularly after prolonged surgery when hypotensive anesthesia is used.
- Direct pressure over other peripheral nerves may result in postoperative neurapraxia.

Sites of Peripheral Nerve Injury in Orthopedics

Upper Extremity

- Brachial plexus: Caused by abduction, external rotation, extension of shoulder (usually resolves within several months), or traction of shoulder.
- Ulnar nerve injury: Pressure at the elbow or traction of nerves over the first rib results in numbness of ring and fifth fingers.
- Radial nerve: Pressure behind arm results in wrist drop.
- Anterior interosseous nerve: Pressure from bandages on lateral aspect of distal elbow.

Lower Extremity

- Lateral femoral cutaneous nerve: Pressure over anterior iliac crest in lateral position or over lateral thigh results in numbness of the lateral aspect of the thigh and knee.
- Femoral nerve: Pressure to the groin of the dependent limb in the lateral decubitus position results in numbness of the anterior thigh and medial aspect of lower leg.
- Common peroneal nerve: Pressure below the head of the fibula which may be caused by compartment syndrome results in foot drop.

Vessel Compression

- An axillary roll placed beneath the upper thorax in a patient in the lateral decubitus position alleviates venous obstruction at the axillary vein.
- Stabilizing posts must be positioned carefully over the groin to prevent interference with venous return at the level of the femoral vein.
- Use a pulse oximeter or palpate the pulse of a distal artery to check arterial obstruction of a limb.
- Venous obstruction → compartment syndrome, with edema, neuropraxia, elevation of creatine phosphokinase, and myoglobinuria.

Positioning of the Rheumatoid Arthritis Patient

- Avoid excessive flexion of the neck. Regional anesthesia is particularly suitable for these patients, because neck stability can be maintained by the patients themselves, particularly if only light or moderate sedation is given.
- Other joints should not be moved beyond the normal range of motion. A conscious patient can prevent excess motion from occurring unlike an anes-

thetized patient who is at risk for neurapraxia, joint dislocation, stretch, or muscle trauma.
- Malpositioning of the extremities may cause various stretch or compression-induced neurapraxia.
- Prone positioning for surgery should be avoided in patients at risk for postoperative visual loss.

CHOICE OF ANESTHETIC TECHNIQUE

- In most cases, the choice of regional (RA) or general anesthesia (GA) in orthopedics depends on some or all of the following factors:
  - Patient's preference.
  - State of health of the patient.
  - Expertise of the anesthesiologist.
  - Duration of the procedure.
  - Surgeon's preference.
  - Practice pattern in the hospital.
- In general, most extremity procedures can be performed using RA alone with light sedation. Alternatively, combined techniques using continuous regional anesthesia supplemented with light GA using a laryngeal mask airway may be particularly useful (benefits of RA with a sedated patient and a secured airway).

Advantages of RA

- Avoidance of GA and associated problems.
- Improved pain control intra- and postoperatively.
- Attenuation of stress response.
- Improved postop pulmonary function.
- ↓ incidence of deep venous thrombosis (DVT) possibly related to reduction of perioperative hypercoagulable state.
- Hypotensive epidural anesthesia may improve the quality of the interface between methyl methacrylate and bone.
- ↓ blood loss (especially for hip arthroplasties).
- Less risk of transfusion-related morbidity and cost.
- Improved patient satisfaction, early discharge.
- Questionable improvement of cardiac outcomes.

Disadvantages of RA

- In an emergency, RA can make it more difficult to administer appropriate airway management in event of a high level of blockade, inadequate analgesia, or oversedation.
- Other disadvantages of RA include local anesthetic toxicity, infection, hematoma, paralyisis, and neurological deficits.

MAJOR ORTHOPEDIC PROCEDURES

Major operations require special preparation on the part of the anesthesiologist, ↑ attention to the details of intraoperative monitoring and fluid management, and active participation whenever possible in postoperative pain management.

Hip and Knee Surgeries

Total Hip Replacement

Average duration of procedure: 1–4 hr.

Anesthetic Management

- Invasive hemodynamic monitoring is suggested in the elderly or medically compromised patient undergoing total hip replacement particularly with the involvement of complex or revision surgery:
  - Arterial cannula.
  - Central venous pressure and Foley catheter.
- Limited ability to exercise making assessment of cardiopulmonary function difficult.
  - Underlying systemic disease.
  - Risk of developing hypoxemia or pulmonary edema due to pulmonary endothelial injury from fat or bone marrow emboli and from ventilation/perfusion (V/Q) mismatching.

Cement Fixation

- Hypotensive anesthesia improves the quality of cement bone fixation because it reduces bleeding from bone.
- Cement fixation poses other risks:
  - Bone cement implantation syndrome: Hypoxia, hypotension, dysrhythmias, ↓ cardiac output (CO), ↑ peripheral vascular resistance (PVR).
  - Emboli during insertion of prosthesis.
  - Gradual loosening of the cement postoperatively (years later).

Blood Loss

Extracapsular (base of femoral neck, intertrochanteric, subtrochanteric) fractures have more associated bleeding than intracapsular (subcapital, transcervical) fractures. Blood loss can be ↓ with hypotensive anesthesia techiques.

Postoperative Pain Management

- Use of the epidural catheter for patient-controlled epidural analgesia is extremely effective and easy if the catheter has been properly placed for surgery.
- Lumbar plexus block significantly improves pain scores and ↓ postoperative narcotic requirements.
- Prophylactic anticoagulant therapy and management of epidural catheters need to be coordinated to prevent formation of epidural hematomas.

Total Knee Replacement

Average duration of procedure: 1.5–3 hr.

Anesthetic Management

- Unilateral versus bilateral total knee replacement: Patients undergoing bilateral total knee replacement have a higher rate of postoperative complications, such as postoperative confusion, cardiopulmonary complications, and ↑ need for banked blood. Outcomes are good if patients have appropriate

hemodynamic monitoring, postoperative epidural analgesia, and a 24- to 48-hr period of more intensive surveillance.
- Suggested monitoring:
  - Autologous blood donation.
  - Arterial cannula.
  - Foley catheter.

Cement Fixation

- Acute hemodynamic responses seldom follow acrylic cement application.
- Lesser degrees of femoral reaming may ↓ the incidence of embolic events.
- Pressures in the femoral canal of 300 mmHg or more do not appear to adversely affect arterial oxygen or pulmonary artery pressures.

Blood Loss

- Negligible with intraoperative use of tourniquets, but postoperative drainage averages 500–1000 mL per knee.
- Postoperative monitoring, possibly in the postanesthesia care unit (PACU), for 24 hr or more may be necessary for high-risk patients until wound drainage slows.
- Bilateral procedures are at additional risk for becoming hypovolemic during the first few hours after the operation.
- Preoperative autologous blood donation and intervention with drugs such as erythropoietin should be considered before surgery (less risk of complications with allogenic blood transfusion).
- Consider use of antifibrinolytic agents to minimize blood loss after total knee replacement (fibrinolytic activity ↑ with tourniquet use).
- Hypotensive epidural anesthesia in total knee replacement without a tourniquet has revealed ↓ total blood loss and need for transfusion.

Postoperative Pain Management

- Total knee replacement is associated with significantly more pain than total hip replacement, and the use of continuous passive motion devices or early mobilization of the knee ↑ the pain.
- Regional analgesia options include various combinations of epidurals and single shot/continuous femoral and sciatic blocks.
- These regional techniques provide better pain relief and faster rehabilitation than intravenous patient-controlled analgesia.
- Intraarticular injections of local anesthetic and narcotic have also been used.

Knee Arthroscopy and Anterior Cruciate Ligament (ACL) Repair

- Can be performed under local with sedation, or a neuraxial anesthetic. Consider lower-extremity block postoperatively.
- Consider use of a tourniquet to minimize blood loss and provide a bloodless operating field. In general, cuff pressure 100 mmHg above patient's systolic blood pressure for thigh.

Shoulder Surgery

Most commonly performed: Shoulder arthroplasty, rotator cuff repair.

Anesthetic Management

- Regional, general, or both.
- Regional: Interscalene or supraclavicular.
- Preoperative block:
  - Pros: Preemptive analgesia and ↓ requirements of volatile agents and opioids.
  - Cons: Inability to perform postoperative evaluation of neurologic function until block resolution, ipsilateral diaphragmatic paresis in patients with severe pulmonary disease.

Positioning

- Typically, "beach chair" with flexed hips and knees and 10–20% reverse Trendelenburg.
- Keep the head and neck in neutral position. Avoid pressure on the eyes and ears.
- Keep all airway connections tightened and reinforced due to limited access to the head.

Possible Complications

- Significant blood loss (tourniquet cannot be used).
- Brachial plexus injury (head turned away from operative site).
- Hypotension and bradycardia.
- Arterial cannulation may be helpful to monitor intraoperative hemoglobin and hematocrit.
- Venous air embolism: Operative site higher than the heart, use of precordial Doppler in right-to-left shunt patients.
- Cerebrovascular accident (CVA) and blindness secondary to hypotension and ↓ cerebral perfusion.

COMPLICATIONS IN ORTHOPEDIC SURGERY

Bone Cement Implantation Syndrome

- Hypotension, hypoxia, dysrhythmias, and pulmonary hypertension.
- Methyl methacrylate (MM) hardens causing ↑ intramedullary pressure → embolization of fat and bone marrow → release of vasoactive mediators.
- Prevention:
  - Lavage of femoral shaft to remove debris.
  - Allow cement to become viscous before placement.
  - Vent in distal femur to reduce intramedullary pressure.
  - Use an uncemented femoral component.
- Management:
  - Hydration to minimize hypotension.
  - ↑ FiO_2 to minimize hypoxemia.
  - Avoid nitrous oxide due to risk of air entrapment.

Fat Embolus Syndrome (FES)

Fat embolus syndrome:

1. *Dypsnea*
2. *Confusion*
3. *Petechiae*

- Risk factors: Male gender, age between 20 and 30 years, hypovolemic shock, rheumatoid arthritis, intramedullary instrumentation, hip arthroplasty using cementing femoral stems, bilateral knee replacements.
- Incidence: 3–4% with long bone fractures.
- Mortality: 10–20%.
- Symptoms: Occur 12-40 hr after injury.
  - Triad of dyspnea, confusion, petechiae (conjunctiva, axilla, upper thorax).
  - Desaturation, increased $ETCO_2$ and pulmonary hypertension intraoperatively.
  - Coma, ARDS, and DIC can occur.
- Management: Supportive therapy with ventilation and CPAP/PEEP, consider steroids.

Criteria for Diagnosis of FES

- Major:
  - Axillary and subconjunctival petechiae.
  - Hypoxemia ($PaO_2 < 60$ mmHg: $FiO_2 < 0.4$).
  - Central nervous system depression.
  - Pulmonary edema.
- Minor:
  - Tachycardia (> 110 bpm).
  - Hyperthermia.
  - Retinal fat emboli.
  - Urinary fat globules.
  - Unexplained ↓ platelets/hematocrit.
  - ↑ erythrocyte sedimentation rate.
  - Fat globules in sputum.

Physiological Changes Associated with Tourniquets

Effects of tourniquet release:

↓ *core temperature.*

Metabolic acidosis.

↓ *venous oxygen tension.*

↓ *pulmonary and systemic arterial pressures.*

↑ $ETCO_2$.

↑ *oxygen consumption.*

- Neurologic effects:
  - Abolition of somatosensory evoked potentials and nerve conduction occurs within 30 min.
  - Application for > 60 min causes tourniquet pain and hypertension.
  - Application for > 2 hr may result in postoperative neurapraxia.
  - Evidence of nerve injury may occur at a skin level underlying the edge of the tourniquet.
- Muscle changes:
  - Cellular hypoxia develops within 8 min.
  - Progressive cellular acidosis occurs.
  - Endothelial capillary leak develops after 2 hr.
  - Limb becomes progressively colder.
- Systemic effects of tourniquet inflation: Arterial and pulmonary artery pressures become elevated.
- Systemic effects of tourniquet release:
  - Transient fall in core temperature.
  - Transient metabolic acidosis.
  - Transient fall in central venous oxygen tension (but systemic hypoxemia unusual).

- Transient fall in pulmonary and systemic arterial pressures.
- Transient ↑ in end-tidal carbon dioxide ($ETCO_2$).
- ↑ oxygen consumption.

Complications of Tourniquet Use

- Damage to vessels, muscles, and nerves with > 2 hr of inflation time.
- Tourniquet pain: Usually after 45 min of inflation, may be observed during GA or RA. The definitive treatment is tourniquet release, use of double tourniquet or GA.
- Tourniquet-induced hypertension: Stimulation of C-fibers, NMDA receptor activation ↑ BP 11–66% after 30–60 min of inflation. More common with GA than RA.
- Tourniquet deflation: Transient systemic metabolic acidosis, ↑ of $PaCO_2$ level, particularly of concern in a patient with underlying acidosis and history of CVA.
- Arterial thrombosis.
- Pulmonary embolism (PE) following deflation:
  - Mechanism: ↓ fibrinolysis or release of plasminogen activators secondary to metabolic conditions distal to tourniquet.
  - Sudden, unexplained hypotension, hypoxemia, and bronchospasm.
  - ↑ central venous pressure (CVP), pulmonary HTN, and acute ischemia from coronary embolization.
  - Patients at risk: Advanced age, bed rest, prior deep venous thrombosis (DVT) or PE, morbid obesity, venous insufficiency, oral contraceptive use, and malignancy, (fat, marrow, or air) from high-volume and pressurized cement use, especially in femoral canal.

Contraindications to Tourniquet Use

- Absolute:
  - Sickle cell disease or other hemoglobinopathies.
  - Severe peripheral vascular disease with arterial spasm and vasculitis.
  - Ongoing ischemia in operative limb.
- Relative:
  - Existing peripheral neuropathy.
  - Chronic vascular insufficiency of the limb.
  - Vascular graft for dialysis (risk of thrombosis).
  - Venous insufficiency from mastectomy, vein harvesting, or radiation therapy.
  - Proven infection in the operative extremity.
  - An extremity with the tumor.
- DVT and thromboembolism:
  - Venous thromboembolism is a major cause of death (0.1–8%) after surgery to lower extremities.
  - Incidence is 40–80% without prophylaxis, and 1–20% show clinical or laboratory evidence. Incidence higher with hip fracture.
- Thromboprophylaxis is based on identification of risk factors.

CHAPTER 25

Anesthesia Outside of the Operating Room

OFFICE-BASED PRINCIPLES

Office-based anesthesia (OBA) is anesthesia given at an unaccredited or out-of-hospital location that also houses nonsurgical needs such as consultation or practice administration.

History

- Mortality rates from anesthesia have been declining: 1/10,000 in 1990 to 1/250,000 for hospitals and 1/400,000 for ambulatory surgery centers (ASCs) in 2000.
- Mortality rates in OBA are approximately 1/57,000.
- This discrepancy in mortality rates can be attributed to anesthetic complications, prolonged surgeries, drug overdose, malfunctions of anesthetic machines, inadequate resuscitation equipment, inadequate monitoring, and lack of experience.
- Injuries that occur in OBA tend to be greater in severity than in ASCs.
- 1994: 8.5% of all procedures were OBA.
- 2000: 75% were outpatient procedures: 17% in ambulatory care setting, 14–25% in OBA.

Advantages

- ↓ facility fee.
- Insurance incentives for OBA.
- Scheduling ease.
- ↓ in nosocomial infections.
- Improved patient privacy.
- Continuity of care.

Disadvantages

Patient safety.

Patient Selection

Poor candidates for OBA include:

- Procedures with high estimated blood loss (EBL).
- Pain and procedures known to be extremely painful.
- History of substance abuse.
- Seizure disorder.
- Malignant hyperthermia.
- Difficult airway, morbid obesity.
- Obstructive sleep apnea (OSA).
- NPO < 8 hr.
- No escort.
- History of adverse outcome with anesthesia.
- Significant drug allergies.
- Pulmonary aspiration risk.

Office Safety

- Office must have appropriate equipment for the patient population:
  - Scavenging system must be active if inhalation anesthesia is to be performed, along with air testing.
  - Offices without anesthesia machines or ventilators should have a device that can deliver positive pressure ventilation.

- Either oxygen via pipeline or H and E cylinders should be available.
- Standard ASA monitors must be present.
- All equipment for a difficult airway algorithm should be present, along with **initial treatment plan for malignant hyperthermia (MH)**, which includes 12 bottles of dantrolene.
- As per DEA rules and regulations, drugs must be double locked and should be accounted for per state and federal regulations.

- Manuals for responsibilities of staff members and infection control policy.
- Basic life support training is a minimum for staff.
- Destination for patient in need of a hospital must be identified.
- Contingency plan available in case of power supply failure.
- Patient records should be kept for a minimum of 5 years.

Anesthetic Technique

- Each procedure has its own risks; the anesthesiologist should be aware of the surgical risks and safely be able to manage them.
- Anesthetic agents should be short acting and high in safety profile, as well as cost effective.
- There are four levels of sedation in OBA:
  - Minimal sedation (anxiolysis): Patient responds to verbal command, cognitive/motor function may impaired.
  - Moderate sedation (conscious sedation): Same as above, but patient will respond after tactile stimuli.
  - Deep sedation: Same as above, but patient will require painful stimuli for response; may have impaired ventilation.
  - General anesthesia: Drug-induced loss of consciousness; patient is unarousable via painful stimuli, may require positive pressure ventilation; cardiovascular function may be impaired.

Postanesthesia Care

- Following office-based procedures, patients should be in a monitored environment.
- The use of **pulse oximetry** is imperative.
- It is essential that these patients have **escorts** prior to being discharged.
- Medication for postoperative pain and nausea/vomiting should be readily available; a multimodal strategy is recommended.
- Adequate hydration is advised (up to 20 mL/kg).

ELECTROCONVULSIVE THERAPY (ECT)

- A procedure in which electric current is passed through the brain in a controlled fashion to produce a seizure lasting < 1 min.
- Commonly used to treat refractory depression with psychotic features, suicidal ideation, mania, or schizophrenia.
- Usually a series of treatments.
- Electroconvulsive shock may be applied to one or both cerebral hemispheres.
- Associated with progressive memory loss with ↑ number of treatments.
- Initial shock causes parasympathetic discharge (bradycardia, asystole, PVCs, hypotension), followed by longer sympathetic discharge (tachycardia, PVCs, bigeminy, trigeminy, hypertension).
- Other physiological effects: ↑ cerebral blood flow, ↑ intraocular pressure, ↑ gastric pressure, ↑ ACTH/cortisol/catecholamine release.

Contraindications

- Absolute contraindications: Recent MI (< 3 mo), recent cerebrovascular accident (CVA; < 1 mo), ↑ intracranial pressure (ICP).
- Relative contraindications: Pheochromocytoma, intracranial mass with normal ICP, intracranial aneurysm, angina, congestive heart failure (CHF), untreated glaucoma, osteoporosis, thrombophlebitis, pregnancy, retinal detachment, pacemakers.
- Place a magnet over pacemakers and convert to a fixed pacing mode prior to ECT.

Tonic-clonic contracture can cause long-bone fracture without the use of muscle relaxants.

Anesthesia for ECT

- Have emergency drugs ready.
- Apply standard ASA monitors. Use additional monitoring based on patient's condition (ie, invasive monitoring for patients with severe cardiac dysfunction).
- Control the seizure and ↓ the incidence of musculoskeletal injuries.
- Monitor for cardiovascular changes.
- Manage the airway: Hyperventilation can ↑ seizure duration.
- Place a bite block during induced seizure.
- Terminate seizures > 3 min with propofol (20–50 mg IV).
- Commonly used drugs:
  - Methohexital (0.5–1.0 mg/kg IV).
  - Succinylcholine (0.5–1.0 mg/kg IV).
  - Glycopyrrolate or atropine in case of bradycardia.
  - β blocker in case of hypertension or tachycardia.
- Common drugs used to treat depression may interact with ECT/anesthetic drugs:
  - Monoamine oxidase inhibitors (MAOIs): ↑ availability of norepinephrine at postsynaptic receptors.
    - Orthostatic hypotension or hypertension based on autonomic response.
    - Avoid meperidine if patient is on MAOIs, as it can cause serotonin syndrome and death.
  - Tricyclic antidepressants (TCAs): Prevent norepinephrine and serotonin reuptake.
    - Orthostatic hypotension
    - Sedation
    - Dry mouth
    - Urinary retention
    - Tachycardia
  - Selective serotonin reuptake inhibitors (SSRIs).

Drugs that ↑ Seizure Threshold and ↓ Seizure Duration—

Please Beware with ECT

Propofol
Benzodiazepines
Etomidate

Lithium is associated with post-ECT delirium and interferes with ECT success.

MAGNETIC RESONANCE IMAGING (MRI)

- Patients with head injury, anxiety, claustrophobia, or an inability to remain still, or children may need anesthesia for MRI.
- Use standard ASA monitors, provide hearing protection to the patient, and have emergency drugs and equipment available.

- Must use special equipment:
  - No ferromagnetic objects should be near the MRI magnet (including aneurysm clips and pacemakers).
  - Cannot use ordinary electrocardiogram (ECG) leads or pulse oximetry wires.
  - Need extra long cables, circuit, and tubing.
  - Use lithium batteries instead of zinc batteries.
- Limited access to and visibility of the patient; thus, an $ETCO_2$ monitor is helpful.
- Watch for acute reactions to contrast media (gadolinium, etc.):
  - Risk factors: Previous reaction, asthma, severe hay fever, use of β blockers or interleukin-2.
  - Look for skin reactions, airway obstructions, signs of anaphylaxis, hemodynamic instability with 30 min of exposure.
  - Treat as anaphylaxis.
  - Use *N*-acetylcysteine to ↓ contrast-induced nephropathy, especially in patients with compromised renal function.
- Move patient out of MRI field if resuscitation is required.

Avoid looping monitor cables in an MRI as it can cause thermal burns.

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CHAPTER 26

Special Considerations in Anesthesia Practice

- Hereditary defect of the calcium-release channels of the sarcoplasmic reticulum of skeletal muscles.
- Autosomal dominance with variable penetrance, meaning it can skip generations. Genes on chromosomes 1, 3, 7, 17, and 19 have been linked to MH.
- This defect causes sustained contraction of skeletal muscles when exposed to triggering agent (succinylcholine and all volatile anesthetic agents—halothane, enflurane, isoflurane, desflurane, and sevoflurane).
- Muscle biopsy and contracture testing with caffeine and/or halothane is the only reliable way to diagnose MH. Serum creatine kinase (CK) levels are chronically elevated in 50–70% of susceptible individuals.
- More prevalent in children. The Midwest has the highest incidence in the United States.

Anesthestic Management

Preoperative

- History:
  - Previous adverse anesthetic experiences.
  - Family history of anesthetic complications.
  - Intolerance to caffeine-containing foods.
  - Unexplained fevers or muscle spasms.
- Muscle biopsy and contracture testing with caffeine and/or halothane in suspected susceptibility.
- Serum CK levels are ↑ in 70% of susceptible patients.
- Avoid triggering agents (volatile anesthetic agents, succinylcholine).
  - Flush the anesthesia machine with air or oxygen for 4 hr.
  - Dantrolene prophylaxis is not recommended.
- Consider regional anesthesia or nontriggering agents:
  - Opioids.
  - Benzodiazepines.
  - Propofol.
  - Etomidate.
  - Ketamine.
  - Barbiturates.
  - Nitrous oxide.
  - Local anesthetics.
  - Nondepolarizing neuromuscular relaxants.

Early signs of MH:

Tachycardia

↑ $ETCO_2$.

Intraoperative

Watch for signs of MH:

- Unexplained tachycardia (early) and possible tachyarrhythmias.
- ↑ end-tidal CO_2 (early).
- Hypertension.
- Masseter spasm causing inability to perform direct laryngoscopy. (It can also be due to myotonia, but it is safer to assume that those patients are susceptible to MH.)
- Arterial hypoxemia.
- Metabolic and respiratory acidosis.
- Myoglobinemia and myoglobinuria.
- ↑ serum CK levels.

- ↑ body temperature.
- Renal failure.
- Disseminated intravascular coagulation (DIC).
- Hyperkalemia.
- Sudden death.

Treatment

Once suspected, immediately:

- Discontinue triggering agents.
- Give dantrolene 2–3 mg/kg every 5–10 min until symptoms subside, or total dose of 10 mg/kg—give early and rapidly.
- Symptom control:
  - Hyperventilate with 100% oxygen.
  - Cool the patient down.
  - Sodium bicarbonate to correct acidosis.
  - Insulin and glucose to correct hyperkalemia.
  - Antiarrhythmic agents.
  - Vasopressors.
- Induce diuresis to prevent acute renal failure from myoglobinuria.
- Do not give any calcium channel blockers in the presence of dantrolene—may cause myocardial depression.
- Monitor arterial blood gas (ABG), serum CK levels, and vital signs.

Avoid calcium channel blockers with dantrolene—the combination can cause myocardial depression.

Postoperative

- Continue dantrolene if started for treatment, as MH can recur within the first 24 hr after an episode.
- Continue supportive care in monitored setting.

MYASTHENIA GRAVIS

- Weakness and easy fatigability of skeletal muscles due to autoimmune destruction or inactivation of postsynaptic acetylcholine (ACh) receptors in the neuromuscular junction.
- More common in young females.
- 65% associated with thymic hyperplasia and 15% with thymoma. It is also associated with other autoimmune disorders in 10% of patients.
- Pregnant women with myasthenia gravis will get weaker during the last trimester. Babies born to myasthenic mothers may experience transient myasthenia due to transplacental transfer of ACh receptor antibodies.
- Disease course is marked by exacerbations and remissions which might be complete or partial.
- Weakness might involve one group of muscles or generalized. Ocular muscles are usually involved.
- Respiratory failure and cardiomyopathy can occur.
- Can resemble Lambert-Eaton syndrome but has a different pathology (Table 26-1).

Succinylcholine—resistance in (untreated) myasthenia gravis and sensitivity in Lambert-Eaton.

Nondepolarizing muscle relaxants—sensitivity in myasthenia gravis and Lambert-Eaton.

Treatment

- Cholinesterase inhibitors (pyridostigmine).
- Immunosuppressants.
- Plasmapheresis.

TABLE 26-1. Myasthenia Gravis vs. Lambert-Eaton Syndrome

	Myasthenia Gravis	Lambert-Eaton Syndrome
Common gender affected	Females.	Males.
Age of onset	20–40.	50–70.
Common muscle groups affected	Ocular, bulbar, facial.	Proximal muscles.
Activity	↑ weakness.	↑ muscle strength.
Electromyographic response	Voltage decrement to repeated stimulation, with good response to anticholinesterase.	Voltage increment to repeated stimulation, with poor response to anticholinesterase.
Pathology	Thymus gland pathology common.	Small cell lung cancer usually present.
Response to muscle relaxants	Succinylcholine—resistant in untreated; variable response in treated. Sensitive to nondepolarizing muscle relaxants.	Sensitive to succinylcholine and nondepolarizing muscle relaxants.
Antibodies	To acetycholine receptors at postsynaptic neuromuscular junction.	To calcium channel–associated protein synaptogamin.
Treatment	Steroids, plasmapheresis, immunosuppressants.	Steroids, plasmapheresis, immunosuppressants.

- Intravenous immunoglobulin (IVIG).
- Thymectomy (improves symptoms, especially in young patients).

Anesthetic Management

Preoperative

- Achieve optimal control of myasthenic symptoms preoperatively.
- Avoid premedication with respiratory depressant drugs.
- Aspiration prophylaxis.
- Check for electrolyte imbalances, especially hypokalemia, which may exacerbate weakness.

Intraoperative

- Avoid muscle relaxants. Defasciculating dose can result in prolonged paralysis.
- Response to succinylcholine is unpredictable.
- Due to their muscle weakness, the relaxant effect of inhalational agents is often enough for endotracheal intubation and surgical procedure.
- If muscle relaxants are necessary, small doses of short-acting nondepolarizing muscle relaxant should be used (eg, mivacurium or cisatracurium) with neuromuscular monitoring.

Postoperative

- Patients are at high risk for postoperative respiratory failure.
- Postoperative ventilatory support may be needed if:
  - Disease duration is > 6 years.
  - Concomitant pulmonary disease.
  - Peak negative inspiratory pressure < –25 cm H_2O.
  - Vital capacity < 4 mL/kg.
  - Pyridostigmine dose > 750 mg/day.
- Postoperative pain should be controlled with regional methods, if possible, and avoidance of respiratory depressants.

CHOLINERGIC CRISIS VS. CENTRAL ANTICHOLINERGIC SYNDROME

- Cholinergic crisis:
  - Cause: Pyridostigmine excess in myasthenia gravis treatment; neostigmine overdose.
  - Edrophonium test: Differentiates myasthenic crisis from cholinergic crisis, ↑ muscle strength in myasthenic crisis, and ↑ weakness in cholinergic crisis.
  - Treatment: Atropine (anticholinergic).
  - Manifestations: Bradycardia, salivation, diarrhea, miosis.
- Central anticholinergic syndrome:
  - Cause: Atropine > scopolamine (**not** glycopyrrolate—does not cross blood-brain barrier).
  - Treatment: Physostigmine—crosses blood-brain barrier.
  - Manifestations: Tachycardia, restlessness/confusion, flushing, ↑ temperature, mydriasis, photophobia.
- Anticholinergic inhibitor overdose:
  - → muscarinic effects and nicotinic effects (paralysis).
  - Nerve gas: Atropine (for muscarinic effects) and ventilation (for nicotinic effects).
  - Organophosphates: Atropine (for muscarinic effects), pralidoxime (for nicotinic effects), cholinesterase inhibitor antagonist.

DUCHENNE'S MUSCULAR DYSTROPHY

Manifestations

- X-linked recessive.
- Manifests in boys 2–5 years old.
- Plasma CK ↑ 30–300 times normal due to muscle breakdown.
- Musculoskeletal:
  - Progressive muscle weakness.
  - Calf pseudohypertrophy secondary to fatty infiltration.
  - Waddling gate, difficulty climbing stairs.
  - Kyphoscoliosis.
- Cardiovascular:
  - Mitral regurgitation from papillary muscle dysfunction.
  - ECG—tall R waves in V_1, Q waves in limb leads, short PR, sinus tachycardia.
- Pulmonary:
  - Respiratory muscle weakness.
  - ↓ coughing ability and respiratory reserve.
- Muscle biopsy shows necrosis and phagocytosis of muscle fibers.
- Death occurs at age 15–25 due to CHF, pneumonia, or both.

Anesthetic Management

- Full stomach precautions due to delayed gastric emptying.
- Muscle relaxants:
  - Succinylcholine—causes potassium release.
  - Nondepolarizing muscle relaxants—↑ sensitivity.
- Volatiles—enhanced myocardial depression.
- Postoperative respiratory dysfunction possible.
- Consider regional anesthesia if possible.
- Questionable association with malignant hyperthermia.

MULTIPLE SCLEROSIS (MS)

Autoimmune disease involving demyelination of central nervous system.

Manifestations

- Waxing/waning pattern of neurologic symptoms unrelated to single anatomic system.
- Visual disturbances, paresthesia, weakness, bowel/bladder dysfunction.
- Brain-stem involvement manifests as autonomic dysfunction, dysrhythmias, and apnea.
- Symptoms improve in pregnancy.
- Management:
  - Spasticity: Benzodiazapine, Dantrolene and Baclofen.
  - Pain: Tricyclic antidepressants.
  - Exacerbations: Steroids.
  - Immunosuppressants.
  - Plasmapheresis.

Anesthetic Management

- Potential perioperative triggers: Stress, infection, hyperpyrexia.
- Temperature: An ↑ of 0.5° C can block conduction in demyelinated fibers.
- Muscle relaxants: ↑ sensitivity to nondepolarising relaxants, which could be potentiated by baclofen; avoid succinylcholine if experienceing prolonged paralysis.
- Volatiles: May contribute to underlying autonomic dysfunction.
- Steroids: Consider stress dose if undergoing major surgery and steroid use within past year.
- Peripheral nerve blocks appear safe.
- Neuraxial anesthesia:
  - Epidurals: Unlikely to cause neurologic injury.
  - Spinals: Conflicting evidence associating spinals with exacerbations.

Stress, infection, hyperpyrexia, and spinals may trigger MS exacerbations.

MYOTONIA

- Hereditary group of diseases of skeletal muscles characterized by persistent contracture after stimulation due to abnormal calcium metabolism causing failure to return calcium to the sarcoplasmic reticulum.
- General and regional anesthesia fail to relax the contracted muscles. Local muscle infiltration may cause relaxation.

- ↑ ambient temperature reduces the severity of contraction.

Types

Myotonia Dystrophica

Avoid succinylcholine and reversal agents in myotonia to prevent contractures and hyperkalemia.

- Most common and most severe type.
- Autosomal dominant.
- Onset in second or third decade of life.
- Multisystem disease with prominence of skeletal muscle involvement.
- Early facial weakness, wasting of the sternocleidomastoid, ptosis, dysarthria, and inability to relax hand grip.
- Characteristic triad of frontal baldness, mental retardation, and cataract formation.
- Endocrine insufficiency (diabetes, thyroid dysfunction), central sleep apnea, cardiomyopathy, dysrhythmias.

Myotonia Congenita

- Widespread skeletal muscle involvement.
- No other systemic involvement.
- No disease progression.
- Normal life expectancy.

Paramyotonia

- Very rare.
- Same as myotonia congenita, but apparent only on exposure to cold.

Anesthetic Management

- Aspiration precautions as patents have delayed gastric emptying.
- Muscle relaxants and reversals
  - Succinylcholine/reversals—avoid as it can cause sustained contractions.
  - Nondepolarizing relaxants—normal response.
  - Nerve stimulator may produce contractions.
- Volatiles—sensitivity to respiratory and cardiac depression effects.
- Consider regional or local anesthesia for pain control.
- Keep patient warm as shivering can cause contractions.
- Cardiac conduction defects warrant careful monitoring and possible pacing.
- No definitive association with malignant hyperthermia.

DOWN SYNDROME (TRISOMY 21)

The abnormality is due to the presence of an extra chromosome 21. The risk of having this abnormality ↑ with maternal age.

Manifestations

- Flat facies with oblique palpebral fissures.
- Single palmar crease.
- Dysplastic middle phalanx of the fifth finger.

- Asymptomatic atlantoaxial instability in 20%.
- Mental retardation.
- Chronic pulmonary infections.
- Seizures.
- **Airway:**
  - Narrow nasopharynx.
  - Small mouth.
  - Short neck.
  - Irregular dentition.
  - Large tongue, tonsils, and adenoids.
  - Subglottic stenosis.
- **Cardiac:**
  - 40% of patients have cardiac abnormalities.
  - Endocardial cushion defect.
  - Ventricular septal defect.
  - Tetralogy of Fallot.
  - Patent ductus arteriosus (PDA).

Anesthetic Considerations

- Use anticholinergic drugs to ↓ airway secretions.
- A small dose of intramuscular ketamine may be necessary to control an uncooperative patient.
- Difficult airway.
- Trachea is usually smaller than calculated by patient's age.
- Forceful neck flexion during intubation should be avoided due to atlantoaxial laxity.
- Avoid bubbles in the IV line due to frequent presence of right-to-left shunts.
- Patients are prone to postoperative pulmonary complications.
- IV access is usually difficult due to obesity.
- Choice of anesthetic is influenced by the presence of congenital heart disease.

ANAPHYLAXIS

- Degranulation of mast cells and basophils with release of histamine and other vasoactive mediators, in response to antigen antibody interaction.
- Prior exposure to the allergen is required for the production of antigen-specific IgE.
- This is different from anaphylactoid reaction in which vasoactive mediators are released from basophils in response to certain drugs that have the ability to displace them from basophils. This reaction is independent of antigen-antibody reaction.

Manifestations

- Cutaneous: Pruritis, urticaria, flushing, edema.
- Cardiovascular: Tachycardia, dysrhythmias, hypovolemia (secondary to extravasation of up to 50% of intravascular volume), hypotension (may be only manifestation under anesthesia).
- Pulmonary: Laryngeal edema, pulmonary edema, bronchospasm, wheezing.
- Gastrointestinal: Abdominal pain, cramping, diarrhea.
- Initial ↓ followed by an ↑ in plasma IgE levels.

- ↑ in plasma tryptase.
- Anesthetic drugs mask may alter vasoactive mediator release, possibly delaying early recognition.
- Blockade of the innervation of the adrenal glands could accentuate the symptoms of anaphylaxis by preventing release of endogenous catecholamines.

Allergic reaction triggers:

Latex

Muscle relaxants

Antibiotics

Blood products

Colloids

Anesthetic Considerations

- Early recognition is important in successful treatment of anaphylaxis.
- The main goals are treatment of vascular collapse, reversal of arterial hypoxemia and prevention of further release of vasoactive mediators.
- Supplemental oxygen.
- Balanced salt solution to replenish the intravascular space.
- Intravenous epinephrine acts by ↑ intracellular cyclic adenosine monophosphate (cAMP), thus ↓ membrane permeability and ↓ release of vaso-

TABLE 26-2. Normal Aging Process

Cardiovascular	
↓ arterial elasticity	↑ afterload/SBP, LVH
↓ β-adrenergic response	↓ resting HR/max HR/baroreceptor reflex
Pulmonary	
↓ elasticity	↓ alveolar surface area/Pao_2
↑ chest wall rigidity	↑ residual volume/closing capacity/V/Q mismatch
Blunted response to hypoxia + hypercapnia	↓ cough/max breathing capacity
Renal	
↓ renal mass	↓ renal plasma flow/glomerular filtration rate
↓ renal blood flow	Impaired Na/fluid handling
↓ tubular function	↓ concentrating/diluting ability/drug excretion
↓ renin-aldosterone response	Impaired K excretion
Gastrointestinal	
↓ hepatic blood flow	↓ albumin production/biotransformation rate
↓ liver mass	↓ plasma cholinesterase levels
↑ gastric pH/emptying time	
Central nervous system	
↓ brain mass/CBF	Preserved autoregulation of cerebral blood flow
Dosing	↓ requirements for local anesthetics, longer duration of anesthetic agents
Spinal	↑ cephalad spread, ↓ duration of action/motor block
Epidural	↑ risk for postoperative mental status changes
General anesthesia	
Musculoskeletal	
Stiff joints	Difficulty positioning
↓ muscle mass + frail skin	Just be careful all around

TABLE 26-3. Pharmacokinetic Changes Associated with Aging

Water-soluble drugs	↓ volume of distribution (total body water) → ↑ plasma concentration → ↑ elimination half-life
Lipid-soluble drugs	↑ volume of distribution (fat) → ↓ plasma concentration → ↓ elimination half life
↓ albumin	Binds acidic drugs Opioids, benzodiazepines, barbiturates ↑ plasma (unbound drugs) = less available for metabolism, excretion
↑ α_1-Acid glycoprotein	Binds basic drugs Local anesthetics ↓ plasma (unbound drugs)

active mediators and by relaxing bronchial smooth muscles. If the anaphylaxis is not life threatening, subcutaneous epinephrine is the standard of care (0.3–0.5 mg).

- Antihistamines compete with histamine for receptors, but the effect of antihistamines once the mediators have been released is questionable.
- Corticosteroids are often given intravenously to patients suffering from life-threatening anaphylactic reaction. They enhance the β effect of other drugs and inhibit the release of arachidonic acid responsible for the production of leukotrienes and prostaglandins.

ELDERLY

Normal Aging Process

See Table 26-2.

Pharmacologic Changes

- Pharmacokinetics: Relationship between drug dose and plasma concentration (see Table 26-3).
- Inhalational anesthetics:
  - > 40 years of age: ↓ 4% MAC/decade.
  - ↓ cardiac output: ↑ onset of action.
  - V/Q mismatch: ↓ onset of action.
  - Worsens myocardial depressant effects.
  - Blunted heart rate response to isoflurane + desflurane.
  - Prolonged recovery:
    - ↑ volume of distribution.
    - ↓ hepatic clearance.
    - ↓ alveolar ventilation.
- Nonvolatile anesthetics:
  - ↓ dose requirements (mostly due to pharmacokinetics).
  - ↑ sensitivity to benzodiazepines (due to pharmacodynamics).
- Muscle relaxants:
  - Same response to depolarizing and nondepolarizng agents.
  - ↑ onset: ↓ CO/slow muscle blood flow.
  - ↑ elimination half-life: ↓ hepatic/renal excretion.

SECTION III

COMMON ORAL BOARD TOPICS

HYPOTENSION

Hypovolemia	Dehydration Hemorrhage
Cardiac	MI Ventricular failure Tamponade Arrhythmias LV outflow obstruction
Drugs	Histamine releasing (vancomycin, protamine, morphine, codeine, meperidine, thiopental, atracurium, mivacurium, atracurium, tubocurarine, rapacuronium, succinylcholine) β blockers ACEI/ARBs Propofol Volatiles
↓ venous return	PEEP ↑ PIP Aortocaval obstruction Reverse Trendelenburg
Metabolic	DM Hypothyroidism Adrenal insufficiency Acidosis Hypoxemia Anaphylaxis Sepsis
↓ sympathetic tone	Clonidine Regional block Neuraxial block Sympathetic block

HYPERTENSION

Preexisting disease	HTN Early acute MI Aortic dissection Elevated ICP Autonomic hyperreflexia
Surgical causes	Prolonged tourniquet time Aortic cross clamping Post myocardial revascularization Post carotid endarterectomy
Anesthetic causes	Inadequate analgesia/anesthesia Hypervolemia Hypercarbia Shivering Improperly sized blood pressure cuff
Medications	Rebound HTN (clonidine, β blockers, methyldopa) Vasoconstrictors

Other	Bladder distention Hypothermia

BRADYCARDIA

Causes of Bradycardia	Treatment
Hypoxia	Ensure oxygenation
Vagal reflexes (occurs with hypotension)	Stop vagal stimulation Atropine Ephedrine
↑ ICP	Hyperventilation Diuretics Burr hole
β blockade	Stop drug Atropine Isoproterenol
Anticholinergics	Atropine
Digitalis	Check serum concentration Digibind Lidocaine Phenytoin/procainamide Potassium Ventricular pacing
Succinylcholine in children	Avoid succinylcholine Atropine
Conditioning in athletes	No intervention necessary if patient is asymptomatic

TACHYCARDIA

Preoperative	Intraoperative	Postoperative
Infection Anxiety Hypovolemia	Light anesthesia Desflurane Ephedrine Atropine/glycopyrrolate Glucagon Isoproteronol, epinephrine, dobutamine, dopamine, ketamine Malignant hyperthermia Hypovolemia	↑ sympathetic system (hypoxemia, hypercarbia, hypotension, hypoglycemia, MI, ↑ ICP) Pain Distended bladder Anxiety Hypovolemia

HYPOXEMIA

Causes

- ↓ mixed venous O_2
  - ↑ O_2 consumption
  - ↓ CO

- ↓ alveolar PO_2
  - ↓ FiO_2
  - ↑ altitude
  - Hypoventilation
- ↑ A-a gradient
  - Shunt
  - V/Q mismatch
  - Diffusion limitations
- ↓ O_2 carrying capacity
  - ↓ hemoglobin
  - Carbon monoxide toxicity

DEAD SPACE

Causes of ↑ Dead Space

- Age/anticholinesterases.
- Bronchodilators.
- Hypotension/hypovolemia/hypothermia.
- Pulmonary disease (PE, emphysema).
- Smoking.
- Upright position.
- PPV.
- PEEP.
- ↓ cardiac output.

SHUNT

Causes of ↑ Shunt

- Thebesian and bronchial veins.
- Bronchospasm.
- PTX/PNA.
- Right-to-left intracardiac shunt.

HYPERCARBIA

Causes of ↑ $PETCO_2$	$PaCO_2$-$PETCO_2$ Gradient
Rebreathing (↑ circuit dead space, exhausted CO_2 absorbent)	Normal
↑ cardiac output	Normal
Hypoventilation (↓ compliance, ↓ respiratory drive from anesthesia, ↑ airway resistance)	Normal
↑ CO_2 production (↑ O_2 consumption, ↑ temperature, MH, shivering, hyperthyroidism, catecholamines, TPN, CO_2 insufflation)	Normal
Right-to-left cardiac shunt	↑
Endobronchial intubation	↑

Effects

- Acidosis.
- Arrhythmias.
- Right shift of O_2-Hb curve.

- Intracerebral steal.
- ↑ PA pressure.
- Epinephrine/norepinephrine release, causing cutaneous vasoconstriction.
- Apnea: CO_2 ↑ 6 mmHg for first min with an ↑ of 3 mmHg for every min thereafter.

HYPOCARBIA

Causes of ↓ $PETCO_2$	$PaCO_2/PETCO_2$ Gradient
↓ cardiac output	↑
↑ dead space (PE, air embolism, bronchospasm)	↑
Disconnect, ETT leak	↑
Hyperventilation	Normal

Effects

- Apnea.
- Alkalosis.
- Airway constriction.
- V/Q mismatch.
- ↓ CO/ CBF/ Ca^{2+}/coronary blood flow.
- Left shift of O_2-Hb curve.

HYPOTHERMIA

Heat Loss Mechanisms

Redistribution	Core body temperature drops 1–2°C within first hr of general anesthesia, as anesthesia-induced vasodilation warms the extremities at the expense of the core
Radiation	Temperature difference between OR and patient Major contributor of perioperative heat loss
Convection	Air flow around patient Major contributor of perioperative heat loss
Conduction	Direct skin contact to wet/cold surfaces Minor contributor to perioperative heat loss
Evaporation	Exhaled gases and exposed viscera Minor contributor of perioperative heat loss

Clinical Implications

Temperature < 33°C	Temperature < 28°C
Hypertension	Hypotension Hypovolemia (secondary to diuresis from cold suppressing ADH)
↑ O_2 consumption by 300–500% secondary to shivering	↓ O_2 consumption/$CMRO_2$
Tachycardia	Bradycardia

Hyperventilation	↓ PaO_2 (secondary to ↑ V/Q mismatch and ↓ HPV) Left shift of O_2-Hb dissociation curve secondary to metabolic acidosis Hyperglycemia (secondary to ↓ insulin) ↑ ACTH/ cortisol ↑ Epinephrine/norepinerphrine ↑ viscosity Thrombocytopenia secondary to splenic sequestration ↓ drug metabolism ↓ MAC

Treatment

- Warming blankets
- Meperidine
- Clonidine

HYPERTHERMIA

Intraoperative Causes

- Malignant hyperthermia.
- Neuroleptic malignant syndrome.
- Thyroid storm.
- Sepsis.

Postoperative Causes

- Underlying infection (ie, from abscess or appendicitis).
- Atelectasis.
- Atropine.
- Transfusion reaction.

Effects

↑ MAC, tachycardia.

HYPOKALEMIA

Causes of Hypokalemia	ECG Changes	Management
↓ Total body K^+ GI loss (diarrhea, vomiting, NGT) Loop diuretics (furosemide) Altered distribution between extracellular (5%) and intracellular (95%) sites Alkalosis (respiratory or metabolic) Hyperglycemia, DKA ↑ sympathetic system Epinephrine Activates β_1 and β_2 receptors causing K+ to shift into cells Causes hyperglycemia, lipolysis, and glycogenolysis	↑ PR interval ↑ T interval Widened QRS Flattened T waves Tachycardia Ventricular fibrillation	Avoid hyperventilation and glucose-containing solutions Replace K^+ at 0.2–0.5 mEq/kg/hr

HYPERKALEMIA

Causes of Hyperkalemia	ECG Changes	Management
↑ Total body K^+ Acute renal failure ↓ aldosterone K^+ sparing diuretics (spironolactone, triamterene) **Altered distribution between extracellular (5%) and intracellular (95%) sites** Acidosis (respiratory or metabolic) Trauma Succinylcholine (↑ serum K^+ 0.5 mEq/L) Diabetes (insulin deficiency) Digitalis Malignant hyperthermia, familial paralysis **Lab-related** Hemolyzed blood simple Thrombocytosis (platelets > 100,000) with leukocytosis (WBC >100,000) Lab error	Peaked T waves Primary AV block QRS widening ST depression Sine wave	**Acute management** D50 and insulin: Moves K^+ intracellularly, which ↑ cell membrane threshold $CaCl_2$ (500 mg over 10 min): Stabilizes myocardium membranes by making the membrane threshold less negative Bicarbonate (1 amp) and hyperventilation: Causes alkalosis, which shifts K^+ intracellularly **Chronic management** Diuretics, kayexalate

HYPONATREMIA

Causes of Hyponatremia	Characteristics	Management
SIADH	Concentrated urine Associated with craniotomy, lung cancer, hypothyroidism, porphyria	Fluid restriction Hypertonic saline Demeclocycline
Addison's (adrenal insufficiency)	Hypotension, hyperkalemia, hypovolemia	Cortisol

HYPERNATREMIA

Causes of Hypernatremia	Characteristics	Management
DI	Dilute urine Associated with head trauma, posterior pituitary surgery, lithium	DDVAP (ADH deficiency) Chlorpropramide (renal insufficiency)
Cushing's (adrenal hypersecretion)	Polyuria HTN, hypokalemic alkalosis hyperglycemia Associated with iatrogenic causes or pituitary/adrenal cortical/lung/testis/prostate/pancreas tumors	Diuretics Insulin Ranitidine K^+

HYPOCALCEMIA

Causes of Hypocalcemia	Characteristics	ECG Changes	Management
Liver disease/↓ albumin ↓ PTH Renal insuffiency Pancreatitis Citrate chelation secondary to blood transfusion	Neuromuscular irritability—tetany, laryngospasm, hyperreflexia, Chvostek's sign (facial nerve spasm), Trousseau (carpopedal spasm) Seizures Paresthesias	Prolonged QT Inverted T waves	Avoid hyperventilation $CaCl_2$

HYPERCALCEMIA

Causes of Hypercalcemia	Characteristics	ECG Changes	Management
Endocrine	Weakness	Prolonged PR	Goal to ↑ volume and ↑ urine output
Primary hyperparathyroidism (adenoma, MEN I, MEN II)	Renal failure	Widened QRS	NaCl
Secondary hyperparathyroidism (renal failure)	Renal stones		Furosemide
Hyperthyroidism—PTH stimulates bone reabsorption, ↓ kidney Ca2+ excretion, ↑ conversion to vitamin D	Cardiac conduction abnormalities		Correct acidosis
			Calcitonin
			Etindronate
			Plicamycin
Tumors			
Myeloma			
Breast			
Lung			
Drugs			
Milk alkali syndrome			
Thiazides			
Vitamin D			
Granulomas			
Tuberculosis			
Sarcoidosis			
Coccidioidomycosis			
Other			
Immobilization			
Hyperalimentation			

Nutritional Deficiency Anemias

Iron deficiency (microcytic, hypochromic)
- Caused by lack of intake or fast RBC turnover
- Associated with CHF, thrombocytopenia, respiratory distress

B_{12} deficiency (megaloblastic)
- Vitamin B_{12} absorption depends on intrinsic factor (IF) made by gastric parietal cells
- Lower levels of IF seen in chronic gastritis and gastric atrophy
- Associated with extended periods of N_2O exposure
- Causes spinal cord degeneration of lateral and posterior columns → symmetric loss of proprioception/vibration

Hemolytic Anemias

G6PD deficiency
- 1% population
- Lack of NADPH → less reduced glutathione → RBCs more susceptible to oxidation → shorter RBC life span (about 60 days vs. 120 days)
- Avoid PCN, ASA, streptomycin, chloramphenicol, methylene blue, INH, sulfas

Immune-Mediated

Autoimmune hemolysis
- Cold and warm antibody hemolysis
- Associated with collagen vascular diseases, cancer, infection
- Avoid hypothermia in cold antibody hemolysis

Drug-induced hemolysis
- Associated with PCN, quinidine, quinine, sulfas, isoniazid, phenacetin, acetaminophen, cephalosporins, tetracycline, hydralazine, HCTZ

Alloimmune hemolysis
- Hemolytic disease of the newborn

Hemoglobinopathies

Sickle cell anemia
- β chain substituted for glutamic acid → insoluble deoxyhemoglobin → sickling and hemolysis
- Crises: Vaso-occlusion, splenic sequestration, aplastic anemia
- Avoid crisis by preventing hypoxia, hypothermia, hypovolemia, hypotension, infection, acidosis, venostasis, ↑ viscosity
- Treatment: O_2, IVF, exchange transfusion to maintain Hct 30% and Hg S 30–40%

α and β thalassemias
- Symptoms: Microcytic, hypochromic anemia; hemolysis; bone marrow hyperplasia

Management:
- Preoperative echo
- Possible difficulty in laryngoscopy secondary to hyperplasia of facial bones
- Possible bleeding into epidural space

Folic acid deficiency (megaloblastic)	**Hereditary spherocytosis**
Associated with pregnancy, alcoholism, malabsorption, methotrexate, phenytoin	RBC membrane defect causes a more round-shaped RBC, which is more susceptible to hemolysis Treatment: Splenectomy (after age 6 to ↓ incidence of infections)

Compensatory Mechanisms in Chronic Anemia

- ↑ cardiac output
- ↑ 2,3-DPG
- ↑ P50
- ↑ plasma volume
- ↓ blood viscosity

PLATELET DISORDERS

Causes of Thrombocytopenia

- Inadequate production: Chemotherapy, radiation, drugs (thiazides, sulfas, alcohol), infection (TB, hepatitis B, sepsis), chronic disease (uremia, liver disease).
- ↑ consumption: Process causing denuding of endothelium, which impairs normal process of hemostasis (burn/crush injury, nonendothelialized vascular grafts), DIC.
- ↑ destruction: Drugs (heparin, cephalosporins), autoimmune diseases (SLE, RA, TTP), alloimmunization from previous transfusion.
- Dilution: Massive transfusion.
- Sequestration: Cirrhosis, sickle cell disease.

Causes of Platelet Dysfunction

- Uremia.
- Antiplatelet drugs.
- COX inhibitors: ASA, COX-2 inhibitors.
- Phosphodiesterase inhibitors: Dipyridamole, caffeine, aminophylline, theophylline.
- ADP receptor antagonists: Ticlodipine, clopidogrel.
- Glycoprotein IIb/IIIa receptor antagonists: Abciximab, tirofiban, eptifibatide.
- Herbal medications/vitamins: Ginkgo, ginseng, garlic, ginger, vitamin E.

ELEVATED PT/PTT

	PT	PTT
Measures activity of:	Extrinsic pathway: Tissue factor; factors VII, X, V, II, (prothrombin) and I (fibrinogen) Most sensitive to ↓ in factor VII Least sensitive to changes in factor II	Intrinsic pathway: HMWK; prekallikrein; factors XII, XI, IX, VIII, X, V, II, and I Most sensitive to changes in factors VIII and IX
Causes of elevation:	Heparin Early coumadin Early vitamin K deficiency Early liver disease Massive transfusion DIC	Heparin Late coumadin Late vitamin K deficiency Late liver disease Massive transfusion LMWH Hemophilia von Willebrand's disease Antiphospholipid antibody

HIGH INSPIRATORY PRESSURE

Causes of ↑ PIP with ↑ Plateau Pressure

- ↑ V_T
- ↓ pulmonary compliance
- Pulmonary edema
- Tension pneumothorax
- Endobronchial intubation
- Pleural effusion
- Trendelenburg position
- Ascites
- Abdominal packing
- Laparoscopy

Causes of ↑ PIP with Unchanged Plateau Pressure

- ↑ inspiratory gas flow rate.
- ↑ airway resistance secondary to kinked ETT, bronchospasm, secretions, foreign body aspiration, airway compression, ETT cuff herniation.

INTRAOPERATIVE WHEEZING

Possible Causes

- Obstruction of ETT: Kinking, secretions.
- Inadequate anesthesia depth: Patient makes expiratory efforts.
- Endobronchial intubation.
- Acute asthma attack.
- Aspiration.
- Pulmonary edema.
- Pulmonary embolus.
- Pneumothorax.

American Society of Anesthesiologists

DIFFICULT AIRWAY ALGORITHM

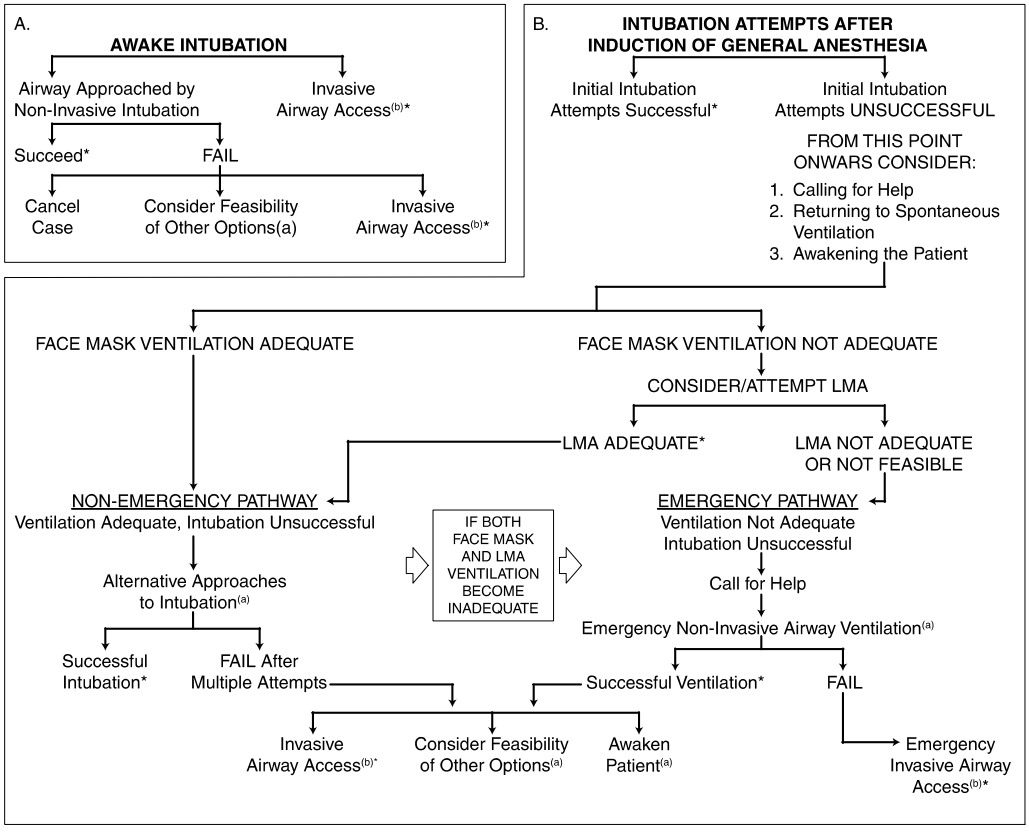
1. Assess the likelihood and clinical impact of basic management problems:
   - A. Difficult Ventilation
   - B. Difficult Intubation
   - C. Difficulty with Patient Cooperation or Consent
   - D. Difficult Tracheostomy

2. Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management

3. Consider the relative merits and feasibility of basic management choices:

A.	Awake Intubation	vs.	Intubation Attempts After Induction of General Anesthesia
B.	Non-Invasive Technique for Initial Approach to Intubation	vs.	Invasive Technique for Initial Approach to Intubation
C.	Preservation of Spontaneous Ventilation	vs.	Ablation of Spontaneous Ventilation

4. Develop primary and alternative strategies:



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PATIENT ASPIRATES

Fluid Type

- Blood: Causes obstruction, creates fibrinous changes in air spaces.
- Acidic fluid: Destroys alveoli and capillaries, causing a chemical pneumonitis characterized by interstitial edema, hemorrhage, and atelectasis.
- Nonacidic fluid: Destroys surfactant, causing alveolar collapse and atelectasis.
- Particulate matter: Causes obstruction and inflammatory response with areas of atelectasis and hyperexpansion.

Manifestations

- Fever.
- Tachypnea.
- Cough, cyanosis, wheezing.
- Hypoxia.
- Hypercapnia.
- Bronchospasm.
- Atelectasis.

Management

- Lateral head placement and Trendelenburg position.
- Suction airway.
- Intubation.
- Suction ETT prior to PPV to avoid dissemination of material.
- Monitor for 24–48 hr for development of pneumonititis.
- Monitor temperature, WBC, ABG, CXR.
- PEEP.
- Bronchoscopy for removal of obstructing pieces of particulate.
- Steroids and lavage **not** indicated.
- Antibiotics indicated if aspirated fluid is fecally contaminated.

Complications

- Bronchospasm.
- PNA.
- ARDS.
- Lung abscess/empyema.

PULMONARY HYPERTENSION

Pulmonary systolic pressure 25+ mmHg and diastolic pressure 12+ mmHg.

Intraoperative Management

- Diuresis.
- Vasodilation: NTG, NTP, prostacyclin, prostaglandin E_1, isoproterenol.
- Nitric oxide: 2–80 ppm; side effects include platelet inhibition, methemoglobinemia, and nitrate metabolite toxicity.
- Volatile anesthetics.
- Avoid factors that can ↑ PVR: Hypoxemia, hypercarbia, acidemia, hypothermia.

PATIENT WITH AORTIC STENOSIS FOR NONCARDIAC SURGERY

Intraoperative Goals

- Avoid ↑ HR: Can ↓ diastolic perfusion and ↑ myocardial O_2 demand.
- Avoid ↓ SVR: Can ↓ perfusion pressure.
- ↑ LV systolic pressures → ↑ wall tension → concentric ventricular wall hypertrophy and ↓ diastolic compliance.
- Ventricle with ↓ compliance requires that preload be maintained (via avoidance of hypovolemia) and atrial fibrillation avoided, as the patient is dependent on atrial kick.

PATIENT WITH MITRAL STENOSIS FOR NONCARDIAC SURGERY

Intraoperative Goals

- Avoid ↑ HR: Can ↓ diastolic perfusion and ↑ myocardial O_2 demand.
- Avoid ↓ SVR: Can ↓ perfusion pressure.
- Associated with atrial fibrillation and ↑ PAP, which can → pulmonary edema and RV failure.

HYPOTHYROIDISM

- Prone to intraoperative hypothermia.
- ↓ ventilatory response to hypoxia and hypercarbia.
- MAC unchanged.
- **Myxedema coma:** Precipitated by sepsis or cold exposure.

Symptoms

- Loss of deep tendon reflexes.
- Hypothermia.
- Hypoventilation.
- Cardiovascular collapse.
- Coma.

Treatment

T_3, IVF, hydrocortisone.

HYPERTHYROIDISM

- Assess for upper airway obstruction due to goiter.
- Deep anesthesia to avoid sympathetic response to stimulation.
- Avoid drugs that stimulate sympathetic nervous system (pancuronium, ketamine).
- MAC unchanged.
- Treat hypotension with direct acting vasopressors.
- Possible sensitivity to muscular relaxants if patient has comorbid myasthenia gravis.
- **Thyroid storm:**
  - May occur 6–18 hr postop.
  - Precipitated by infection, trauma, DKA.

Symptoms

- Hyperthermia
- Tachycardia
- CHF
- Shock
- Dysrhythmias

Treatment

- Propranolol/labetalol
- Cortisol
- Sodium iodide
- PTU
- IVF
- Acetaminophen

HYPOALDOSTERONISM

Causes

- Congenital.
- Adrenalectomy for aldosterone-secreting adenoma.
- Heparin.
- Renin deficiency.
- Indomethecin-induced prostaglandin deficiency.
- Associated with renal failure and diabetes.

Manifestations

- Hypotension.
- Hyperkalemia.
- Hypercloremic metabolic acidosis.
- Heart block.

Management

- Liberal sodium intake.
- Fludrocortisone.
- Furosemide.
- Avoidance of NSAIDs that can inhibit prostaglandin synthesis and further ↓ renin.

HYPERALDOSTERONISM

Types

- Primary hyperaldosteronism (Conn's syndrome): Adrenal adenoma/carcinoma; associated with pheochromocytomas, primary hyperparathyroidism, and acromegaly.
- Secondary hyperaldosteronism: Results from ↑ renin production from renovascular HTN.

Manifestations

- HTN.
- Hypokalemic metabolic alkalosis.
- Muscle weakness.
- Fatigue.

Management

- Spironolactone
- Supplemental potassium

Anesthestic Considerations

- Hypokalemia may cause altered responses to nondepolarizing muscle relaxants.
- Avoid hyperventilation, as it can further ↓ potassium levels.

CARCINOID SYNDROME

- Enterochromaffin cell tumor (mostly in GI system) releases vasoactive hormones, which reach liver via portal vein and are inactivated unless metastases reaches liver or lungs to produce symptoms.
- Twenty percent of patients with carcinoid tumors have carcinoid syndrome.

Symptoms

- Cutaneous flushing
- Abdominal pain
- Vomiting
- Diarrhea
- BP changes
- Bronchospasm
- Hyperglycemia
- Carcinoid heart disease:
  - Seen in 60% of patients with carcinoid sydrome.
  - Right-sided heart involvement → regurgitation and dysrythmias.
  - Vasoactive substances do not reach left side of heart secondary to pulmonary metabolism.

Management

- Preoperative anxiolytics: ↓ stress-triggered serotonin release.
- Octreotide: A somatostatin analogue that suppresses release of serotonin and other vasoactive substances from tumor.
- Avoid histamine-releasing drugs.
- H_1- and H_2-blocking drugs particularly for gastric carcinoids.
- Diphenhydramine and steroids inhibit bradykinin.
- Treat hypotension with fluids (avoid ephedrine and epinephrine).
- Treat HTN with labetalol.

PHEOCHROMOCYTOMA

- Catecholamine-releasing tumor with HTN.
- 90% are benign/solitary/unilateral.
- Associated with hereditary diseases: MEN IIA (thyroid medullary CA and hyperparathyroidism), MEN IIB (thyroid medullary CA and neuromas), von Hippel–Lindau disease (CNS hemangiomas).

Symptoms

- Triad of diaphoresis, tachycardia, headache with HTN.
- Death from cardiac-related causes (MI, CHF, dilated cardiomyopathy) and intracranial bleed.

Diagnosis

- Twenty-four-hour urine collection for catecholamines.
- CT scan.

Preoperative Management

- α antagonism: Phenoxybenzamine, doxazosin, terazosin, prazosin.
- β blockade: Occasionally added *after* α blockade is established to avoid unopposed α vasoconstriction.
- α-methyl tyrosine: Inhibits catecholamine synthesis; used for patients in which surgery is contraindicated.

Intraoperative Management

- BP lability, particularly HTN, after tumor manipulation, and hypotension after adrenal vein ligation secondary to removal of α constriction.
- Avoid muscle relaxants associated with histamine release (atracurium, mivacurium, curare, metocurine) or $\uparrow$ sympathetic outflow (pancuronium).
- HTN can be managed with NTP, esmolol, phentolamine.
- Hypotension can be managed with IVF and $\downarrow$ anesthetic agents.

MULTIPLE SCLEROSIS

- Characterized by episodes of inflammation and demyelination in areas of the brain and spinal cord.
- Succinylcholine use can $\rightarrow$ hyperkalemia.
- Resistant to nondepolarizing neuromuscular blockers.
- May require intraoperative steroids.
- Modest body temperature $\uparrow$ (by 1°C) may cause exacerbations.

Neuraxial Anesthesia

- Epidural placement tolerated.
- Spinal placement controversial as there have been reports of postoperative exacerbations of MS possibly due to demyelinated areas being more sensitive to local anesthetics.

General Anesthesia

- Autonomic dysfunction can exacerbate hypotension caused by anesthetic agents.

- Postoperative mechanical ventilation may be necessary if patient has respiratory weakness.

MYASTHENIA GRAVIS

- IgG antibodies attack ACh receptors in skeletal muscle.
- Untreated patients are resistant to succinylcholine and more sensitive to nondepolarizing muscle relaxants.
- Patients treated with pyridostigmine or plasmapheresis are sensitive to succinylcholine and less sensitive to nondepolarizing muscle relaxants.
- May require intraoperative corticosteroids.
- Limit narcotic use to prevent postoperative respiratory depression.
- Postoperative ventilation likely in:
  - Disease for 6+ years.
  - Comorbid COPD.
  - Daily dose of pyridostigmine 750+ mg.
  - Properative vital capacity of < 2.9 L.

GUILLAIN-BARRÉ SYNDROME

Autoimmune condition triggered by an infection that targets Schwann cells, → demyelination and axonal degeneration in a caudad to cephalad manner.

- Symptoms:
  - Autonomic dysfunction reflected in blood pressure lability and cardiac conduction abnormalities.
  - Ascending bilateral muscular weakness.
- Avoid succinylcholine due to risk of hyperkalemia.
- Consider mechanical ventilation and monitor in ICU.

PARKINSON'S DISEASE

- Degenerative CNS disease with destruction of dopaminergic neurons in the substantia nigra of the basal ganglia causing depletion of dopamine.
- Continue levodopa the morning of surgery, as it has a short $T_{½}$.
- Extrapyramidal symptoms (EPS): Bradykinesia, muscular rigidity, resting tremor, imbalance.
- Avoid drugs that can cause EPS: Droperidol, promethazine, thiethylperazine, metoclopramide.

ELEVATED INTRACRANIAL PRESSURE

- Normal ICP: 5–15 mmHg.
- The intracranial compliance curve shows the effect of ↑ intracranial volume with ICP remaining constant until reaching the upward curve of the slope where small ↑ in intracranial volume can → life-threatening ↑ in ICP, which can → intracranial ischemia and herniation.

Indications for ICP Monitoring

- Head trauma
- Brain mass/tumor
- Hydrocephalus

Treatment

- Head up.
- Hyperventilate with $PaCO_2$ goal 25–30 mmHg.
- ↓ systemic BP as CPP = MAP – ICP.
- Diuretics (furosemide, mannitol).
- Pentothal.
- Muscle relaxant.
- CSF drainage via external ventricular drainage (EVD) placement—contraindicated in large posterior fossa tumors.
- Dexamethasone.
- Fluid restriction.

Anesthetic Management

- All intravenous agents (except ketamine) ↓ CBF and $CMRO_2$.
- Ketamine and nitrous oxide ↑ both CBF and $CMRO_2$.
- **Note:** Rapidly infused mannitol can induce cerebral vasodilation and ↑ cerebral blood volume and consequently ↑ the ICP.

AUTONOMIC HYPERREFLEXIA

- Occurs after spinal shock in association with return of spinal cord reflexes in lesion at T7 or above about 1–3 weeks after initial injury.
- Greater response with ↑ distance between level of stimulus and lesion.
- Lack of supraspinal inhibition permits sympathetic outflow below lesion to act unopposed.

Characteristics

- Vasodilation above lesion secondary to parasympathetic stimulation: HTN, compensatory bradycardia, arrhythmias, flushing, sweating, headache.
- Vasoconstriction below lesion secondary to sympathetic stimulation: Blanching.

Complications

SAH, CVA, confusion, seizures, pulmonary edema, MI.

Management

- Spinal preferable over deep general anesthesia.
- NTP for intraoperative HTN.

PENETRATING EYE INJURY

- Goal: Prevention of elevated IOP (normal 10–22 mmHg) and extrusion of ocular contents.
- Avoid retrobulbar block as it can cause extrusion of ocular contents.
- Avoid coughing/premature laryngoscopy.
- Succinylcholine can ↑ IOP slightly.
- Avoid N_2O as it ↑ IOP.
- Anesthetic drugs that ↓ IOP: Volatile anesthetics, barbiturates, lidocaine, narcotics, nondepolarizing muscle relaxants.
- Patient may have a full stomach, requiring a modified rapid sequence using rocuronium.

PATIENT REFUSES BLOOD PRODUCTS

Cell Saver

- Contraindicated in presence of infection, malignancy, bowel contents and amniotic fluid.
- 50% of blood recovered through salvage process.
- Dilutional coagulopathy as clotting factors and platelets removed by washing process.

Autologous Donation

- Donation of 1 unit per 4 days up to 3 units occurring > 72 hr prior to surgery.
- Contraindicated in severe aortic stenosis, coronary disease, and anemia.
- Can be supplemented by iron and erythropoietin administration.

Acute Normovolemic Hemodilution

- Candidates have preoperative Hct 30 without coronary or cerebral vascular disease with < 1500 mL EBL expected.
- Remove blood before or after induction of anesthesia and restore with IVF.
- Store blood in sterile bag or container with anticoagulants.
- **Advantages:** Blood contains platelets and coagulation factors; ↓ blood viscosity → better tissue perfusion and less intraoperative RBC loss.
- **Disadvantages:** ↑ HR and ↑ CO → ↑ myocardial oxygen consumption and possible myocardial ischemia.

Jehovah's Witnesses

- Usually accept supplemental erythropoietin and iron.
- Some consent to albumin.
- Many will accept acute normovolemic hemodilution and cell saver as long at the tubing is connected to the patient at all times.
- Consider keeping the patient ventilated and sedated postoperatively to minimize oxygen consumption.

HYPOTENSION FOLLOWING TRANSFUSION

- Hemolytic transfusion reaction.
- Characteristics under general anesthesia: Hypotension, tachycardia, hemoglobinuria, DIC.
- Additional characteristics in awake patient: Chills, fever, chest pain, nausea, vomiting, diarrhea.

Diagnosis

↑ haptoglobin.

Management

- Stop transfusion.
- IVF, pressors, and inotropes to maintain hemodynamic stability.
- Maintain urine output at > 100 mL/hr: Give furosemide/mannitol, $NaHCO_3$ to alkalinize urine.
- Draw new blood specimen for type and cross, platelets, coagulation.

LOCAL ANESTHETIC TOXICITY

Manifestations

CNS effects present before cardiac effects.

- CNS: Light-headedness, tinnitus, perioral numbness → muscle twitching, hallucinations → seizure, unconsciousness, respiratory arrest.
- Cardiac: HTN, tachycardia → hypotension → bradycardia, dysrhythmias, circulatory arrest.

Management

- Stop injection of local.
- Maintain oxygenation and hyperventilation: Toxicity is enhanced by hypoxemia, hypercarbia, and acidosis; consider intubation.
- CNS toxicity: Seizures can ↑ body metabolism and cause hypoxemia, hypercarbia, and acidosis; propofol and midazolam can terminate seizure activity, while succinylcholine can terminate muscular activity from seizures and facilitate intubation.
- CV depression:
  - Mild CV depression: Ephedrine, atropine.
  - Profound CV depression with dysrhythmias: Cardioversion; epinephrine, vasopressin, amiodarone; intralipid 4 mL/kg, then 0.5 mL/kg/min for 10 min.

PATIENT ON MAOIs

- MAOIs: Phenelzine (Nardil), isocarboxazid (Marplan), tranylcypromine (Parnate), pargyline (Eutonyl).
- Side effects: Agitation, hallucinations, hyperpyrexia, convulsions, HTN, hypotension.
- MAOIs can be continued until surgery despite earlier recommendations to discontinue them 14 days prior.
- Avoid sympathetic stimulation involving hypoxemia, hypercarbia, hypotension, indirect-acting vasopressors (ephedrine), ketamine, and pancuronium.
- For hypotension use direct-acting drugs (phenylephrine) in smaller doses.
- Avoid adding epinephrine to local anesthetic solutions.
- Avoid meperidine as it has caused fatal excitatory reactions involving HTN, hypotension, seizures, coma, and hyperthermia.
- Use a ↓ dose of succinylcholine as serum cholinesterase levels may be ↓.

CHEMOTHERAPY SIDE EFFECTS

- Doxorubicin/adriamycin: Cardiomyopathy, red urine.
- Bleomycin: Pulmonary fibrosis.
- Vinicristine: Peripheral neuropathy.
- Cisplatin: Peripheral neuropathy.
- Cyclophosphamide: Hemorrhagic cystitis, SIADH.

HYPOTENSION FOLLOWING PROTAMINE ADMINISTRATION

Protamine Reactions

Type I	Rapid infusion of protamine Hypotension due to histamine-induced vasodilatation Patients with low LV function might not tolerate hypotension
Type II	IgE-mediated anaphylaxis (secondary to previous exposure from protamine, NPH insulin, vasectomy) Anaphylaxis with hypotension, ↓ SVR, flushing, edema, bronchospasm
Type III	Pulmonary vasoconstriction → right heart failure with complement activation and thromboxane release Pulmonary HTN, pulmonary vasoconstriction, bronchospasm

PATIENT WITH HELLP SYNDROME

Form of severe preeclampsia:

- **H**emolysis
- **E**levated **L**iver enzymes
- **L**ow **P**latelets

PREECLAMPSIA/ECLAMPSIA

Diagnosed after 20 weeks' gestational age.

Classification	Characteristics	Management
Mild preeclampsia	BP > 140/90 Proteinuria > 0.3g/day Edema	Hydralazine controls BP and ↑ renal and uterine blood flow
Severe preeclampsia	BP > 160/110 Proteinuria > 5 g/day CNS changes (headaches, visual changes) Edema (airway edema, pulmonary edema → CHF) Oliguria < 500 mL/day DIC	Hydralazine Magnesium
Eclampsia	Above symptoms Seizures	Hydralazine Magnesium Intubation

Magnesium Effects

- Anticonvulsant.
- Tocolytic: ↓ uterine activity and ↑ uterine blood flow.
- Sedation: Enhances effects of opioids and sedatives.
- Vasodilation.
- Enhanced neuromuscular block: ↓ presynaptic release of ACh and ↓ sensitivity of postjunctional membrane.

Magnesium Levels

4–6 mEq/L	Therapeutic
5–10 mEq/L	ECG changes: ↑ PR interval, QRS complex widening
10 mEq/L	Loss of DTRs
15 mEq/L	Respiratory depression, SA and AV blocks
25 mEq/L	Cardiac arrest

THIRD-TRIMESTER BLEEDING

	Placenta Previa	Abruptio Placenta	Placenta Accreta	Uterine Rupture
Characteristics	Placenta near internal cervical os Painless bleeding around week 32	Placenta separates from uterine wall after week 20 Hypotension Painful contractions +/– bleeding Fetal distress	Placenta adheres to myometrium (but does not penetrate uterine muscle)	Severe abdominal pain Loss of fetal heart tones Shock
Risk factors	Prior C/S ↑ maternal age Multiparity	↑ maternal age Multiparity Cocaine, smoking Trauma HTN History of abruption	Prior C/S	Prior C/S Rapid spontaneous delivery Excessive oxytocin Multiparity
Management	Double setup (for both SVD and C/S) in OR for vaginal exam	GA with ketamine or thiopental		Emergent C/S

POSTPARTUM BLEEDING

	Retained Placenta	Uterine Atony
Characteristics	Placenta not delivered within the hour	Most common cause of postpartum hemorrhage Can → hypotension several hours after delivery
Risk factors		Prolonged labor Multiparity Multiple gestations Macrosomnia Polyhydramnios Retained placenta Tocolytics (β_2 agonists, magnesium, volatiles)
Management	Manual removal under neuraxial anesthesia GA with volatiles to relax the uterus for placental extraction	Uterine massage Oxytocin Methylergonovine Prostaglandin $F_{2\alpha}$ (Hemabate) Hysterectomy with internal iliac artery ligation

PEDIATRIC PATIENT WITH A URI

- Anesthesia in a pediatric patient with a URI is associated with an ↑ risk of laryngospasm, bronchospasm, postoperative croup and atelectasis, and hypoxia.
- Risk factors that suggest postponing elective procedures: Asthma, bronchopulmonary dysplasia, age < 1 year old, sickle cell disease, smoking in household.
- Bronchial hyperactivity can remain for 7 weeks after a URI, and children can average 5–8 URIs per year; thus, it is impractical to postpone all children with colds for elective procedures.
- Uncomplicated URIs (sore throat, laryngitis, sneezing, rhinorrhea, congestion, malaise, nonproductive cough, temperature < 38°C) in a child without pulmonary disease might receive anesthesia, while complicated URIs (nasopharyngitis, purulent sputum, productive cough, wheezes/rales) are postponed.
- Consider a mask anesthetic as it a has lower rate of complications compared to ETT in children with URIs.

PEDIATRIC PATIENT FOR MAJOR SURGERY–VOLUME REPLACEMENT SCHEMES

Fluid Types

- D5½NS for maintenance.
- NS or LR for deficit and third-space loss.

Fluid Replacement

	1st hour	2nd hour	3rd hour
Maintenance First 10 kg × 4 = 40 cc/h Second 10 kg × 2 = 20 cc/h Additional kg × 1 = ____			
Deficit = maintenance × #h NPO Give half the first hour and other half the second hour			
Third-space loss Intra-abdominal 6–15 mL/kg Intrathoracic 4–7 mL/kg Intracranial/cutaneous 1–2 mL/kg			
EBL For each mL of blood lost, give 3 mL of fluids			
Total			

CONCERNS FOR PREMATURE INFANT REQUIRING HERNIA REPAIR

Retinopathy of Prematurity

- Risk factors: Weight < 1000 g, < 28 weeks' gestation.
- Use a lower FiO_2 to minimize risk of developing retinopathy of prematurity but maintain PaO_2 50–80 mmHg or O_2 saturation > 95%.
- Risk is minimal after vascularization has been completed.

Postoperative Apnea

- Risk factors: Lower postconceptional age; history of apnea, bradycardia, and mechanical ventilation; chronic lung disease.
- Admit for 24-hr monitoring if infant < 60 weeks' postconceptional age.
- Use local or regional anesthesia in lieu of general anesthesia and narcotics if possible.

PAIN MANAGEMENT SCHEMES IN PEDIATRIC PATIENTS

- Caudal blocks:
  - Often used with perineal, abdominal, and lower-extremity procedures in conjuction with general anesthesia.
  - 1 mL/kg of 0.25% bupivacaine provides 4–6 hr of analgesia without motor paralysis.
  - Fewer opioids are required postoperatively.
- Acetominophen: Can be given 10–15 mg/kg every 4 hr PO or 35–45 mg/kg PR.
- Ketorolac: Can be given 0.75 mg/kg IM or 1 mg/kg IV with 0.5 mg/kg every 6 hr for maintenance.

- IV PCAs:
  - Can be used in developmentally normal children over age 5.
  - Alternatively nurse controlled analgesia can be used.

PEDIATRIC PATIENT FOR CLEFT LIP/PALATE SURGERY

- **Cleft lip:** Repaired at 2–3 months old to prevent feeding problems, aspiration, and URIs
- **Cleft palate:** Repaired at 18 months old to aid in phonation.

PEDIATRIC PATIENT WITH STRIDOR

Causes

- Postintubation laryngeal edema: 1–4 years old; associated with traumatic intubation, ETT without an air leak, and coexisting URIs.
- Croup: < 2 years old.

Management

- Humidified oxygen
- Steroids
- Racemic epinephrine

CORNEAL ABRASION

Risk Factors

- Loss of pain sensation.
- Loss of corneal reflexes.
- ↓ tear production.
- Sedated patient rubbing eyes.
- Status post blepharoplasty.

Prevention

Tape eyelids closed, petroleum-based ointments.

Symptoms

- Foreign body sensation.
- Pain exacerbated by blinking and ocular movement.
- Tearing.
- Photophobia.

Management

- Ophthalmology consult.
- Patch eye.
- Topical antibiotic.
- Topical NSAID, **not** steroid drops.

ANESTHESIA IN THE MRI SCANNER

- Contraindicated in patients with implanted metal such as pacemakers or nontitanium cerebral aneurysm clips.
- Use MRI-compatible anesthesia machines and equipment such as laryngoscopes with lithium batteries.
- Gas cylinders cannot be brought into MRI suite.
- All monitor cables should run in straight paths as loops can cause thermal injury.
- Place ECG electrodes close together toward center of magnetic field to avoid artifact and insulate the leads from the patient's skin as they can cause thermal injury.
- Provide hearing protection for the patient.

SURGEON PULLS ON THE GUT

- Mesenteric or gallbladder traction, rectal distention, and stimulation of respiratory tract receptors can cause vasovagal reflex.
- Manifestations: Bradycardia, hypotension, apnea.

SURGEON COMPRESSES THE BRAIN STEM

- Vital respiratory, circulatory centers, cranial nerves and nuclei can be affected.
- Manifestations: Acute changes in blood pressure, HR, and heart rhythm.
- Communication with the surgeon is of utmost importance.

LINE ISOLATION MONITOR ALARMS

- Line isolation monitor checks the integrity of an isolated or ungrounded power system.
- Alarms if faulty piece of equipment is attached to system but allows piece to continue to function normally.
- Identify faulty equipment by unplugging each piece until alarm ceases.
- Offers no protection against macroshock.

FIRE IN THE TRACHEA

1. Extubate.
2. Stop flow of all airway gases.
3. Remove other flammable material from airway.
4. Pour saline into airway.
5. Reestablish ventilation.
6. Examine ETT to ensure no fragments were left in airway.
7. Consider bronchoscopy.

Practice Advisory for the Prevention and Management of Operating Room Fires, A Report by the American Society of Anesthesiologists Task Force on Operating Room Fires. *Anesthesiology* 2008; 108: 786–801.

FIRE ON THE SURGICAL FIELD

1. Stop the flow of all airway gases.
2. Removes drapes and flammable materials.
3. Flood surgical field with saline.
4. Use CO_2 extinguisher if fire not extinguished on first attempt.
5. Maintain ventilation.
6. Assess of inhalational injury if patient is not intubated.

Practice Advisory for the Prevention and Management of Operating Room Fires, A Report by the American Society of Anesthesiologists Task Force on Operating Room Fires. *Anesthesiology* 2008; 108: 786–801.

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