Anesthesia Student Survival Guide
A Case-Based Approach
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Preface

This book was written by residents, fellows, and faculty who specialize in medical education and would like to share with you their excitement for the field of anesthesiology. Our goal in writing the Anesthesia Student Survival Guide: A Case-Based Approach was to provide a concise, easy to use, and up-to-date introduction to the practice of anesthesiology.

Any student of anesthesia will find this unique book useful – it covers both basic and advanced topics and includes case studies designed to help apply theoretical knowledge to real patient situations. In order to get the most out of the book we suggest you first read the case associated with each chapter, followed by the chapter, and then try to answer the questions in the case on your own before reading our sample answers found at the end of the chapter. This will help you focus your reading and retain as much of the key information as possible because each case will provide a context in which the material is presented. The case studies without answers can be downloaded at extras.springer.com and can also be found on pages xvii–xxxv of the book. A selection of the case studies with answers is also available at extras.springer.com as downloadable PDF files and can be used as educational handouts or for individual study.

As educators, we are indebted to generations of students at Harvard Medical School who inspired us to write this practical “survival” guide, and we are thankful for the support and expertise of our contributors at the Massachusetts General Hospital, Brigham and Women’s Hospital, and beyond.

We are especially indebted to a number of individuals whose unending support and encouragement made this work possible. These include Drs. Jeanine Wiener-Kronish, Charles Vacanti, and Warren Sandberg. We would also like to thank Dr. Joseph Garfield and Ms. Eva Cassedy for their outstanding editorial contributions, and Dr. Katharine Nicodemus for her tireless support, encouragement, and guidance. Finally, a special thanks to our parents and families.

As you discover the exciting world of anesthesiology, we hope that you find the Anesthesia Student Survival Guide: A Case-Based Approach an essential tool!

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As anesthesiologists and Harvard Medical Student educators, we have met few people more dedicated to the art of teaching and the experience of learning than Drs. Ehrenfeld, Urman, and Segal. Now we have the privilege of introducing their exciting new textbook of anesthesiology written for medical students. *Anesthesia Student Survival Guide: A Case-Based Approach* is a wonderful synthesis of the broad scope and key concepts of anesthesiology. The book is presented in an easy format for a medical student to learn and absorb during the typically brief 1–4 week exposure to the specialty.

Students come to their anesthesia rotation with a basic science foundation, and little to no familiarity with the types of clinical challenges facing the anesthesiologist. They typically have even less exposure to the thinking and behaviors required to successfully meet those challenges. Drs. Ehrenfeld, Urman, and Segal have created a textbook which not only delivers concise and logical anesthesiology content, but demonstrates the connection between the student’s basic knowledge of anatomy, physiology, pharmacology, and the clinical art and science of anesthesiology. The educational format enables students to move up the taxonomy of learning behaviors by helping them synthesize and apply what they learn to sample cases.

The book begins with a historic overview and introduction of the medical specialty of anesthesiology. In addition, the introduction instructs students on how to practically get the most out of their anesthesia rotation. The pharmacologic principles on intravenous and inhalational anesthetic agents, local anesthetics, muscle relaxants, and sedatives are presented in the next five chapters. The all important preoperative patient evaluation, airway evaluation and monitoring are covered in the following three chapters. The all important preoperative patient evaluation, airway evaluation and monitoring are covered in the following three chapters. Pharmacology is then put together with the patient history and physiology to help the student understand the choice of anesthetic techniques, fluid management, common anesthetic problems, and subspecialty management. Postoperative PACU and ICU care with an emphasis on pain and organ system derangement are reviewed. Lastly, the book discusses the complex and contemporary topics of professionalism, teamwork, quality assurance, and ethics in anesthesia in a clear and forthright manner.

Drs. Ehrenfeld, Urman, and Segal clarify and solidify perioperative concepts with their use of case-based studies at the end of each chapter. The cases
are practical and help to contextualize anesthesia principles. As medical student educators, we know that case studies are indeed one of the best strategies to help students transition from the classroom to the clinical environment. These cases are illustrative, thought provoking, and a stimulus for further discussion that will help students gain the most from their exposure to anesthesia practice.

The topics are judiciously chosen and are widely applicable to patient care both within and out of the operating room. They will help all students develop the necessary skills to become better perioperative caregivers. This book is a valuable guide for all students, whether or not they become anesthesiologists, because they will come away with an appreciation of how anesthesiologists apply their understanding of human physiology and pharmacology to provide safe and effective medical care to patients.

Boston, MA

Michele Szabo, MD
Roger Russell, MD
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In order to focus your reading and retain as much of the key information as possible, please read each of the following case studies immediately before beginning the corresponding chapter. The answers to the case studies are found at the end of each chapter.

Chapter 1 Case Study
You are preparing to provide general anesthesia for a 40 year-old woman undergoing an abdominal hysterectomy. She is otherwise healthy. She had two uncomplicated vaginal deliveries in the past, both with uncomplicated epidural labor analgesia. She had uneventful general anesthesia for a laparoscopic tubal ligation four years ago. Your attending is willing to let you perform as much of the anesthetic as you are able to describe in detail.

- Upon meeting the patient in the preop area and reviewing the history and physical, you find no important new information. What steps will you take to prepare the patient for surgery prior to any interventions?
- You have engaged the patient and checked all the paperwork and you are ready to begin preparing the patient for surgery. What are the next steps?
- You have brought the patient into the OR. Describe the steps you will take prior to induction of anesthesia.
- How will you induce anesthesia?
- Following induction, what else will you do prior to the beginning of the surgical procedure?
- How will you maintain anesthesia?
- What other adjunctive drugs might you give in addition to anesthetics?
- The operation has gone well and is ending. How will you conclude the anesthetic?
- What will you do on arrival to the PACU?

Chapter 2 Case Study
The year is 1900 and you are a student spending time with surgeons during medical school. You are excited because you are to go to the operating room for the first time this morning. To your surprise, you will not be watching the procedure, open removal of a kidney stone, from afar, but instead will be taking an active role! The surgeon has asked you to administer the anesthesia. You are
told to bring the patient into the operating theater (where there are indeed stadium seats occupied by numerous observers of the famous attending surgeon). An orderly has shown you where the anesthetic supplies are kept.

- Which anesthetics are you most likely to use?
- Which intravenous agents will you administer?
- How will you administer the anesthetic? How will you manage the airway?
- How will you monitor the patient?
- Will you keep an anesthetic record?

Chapter 3 Case Study

You are finished with a radical cystectomy with creation of an ileal pouch neobladder on an otherwise healthy, 80 kg, 60 year-old man with bladder cancer. The operation began six hours ago and the patient has not yet emerged from general anesthesia. You experienced no major untoward events during the case and you believe the problem to be pharmacologic. The patient received 4 mg of midazolam in divided doses during the preoperative period to facilitate placement of an arterial line. Anesthesia was induced with thiopental and succinylcholine. You maintained anesthesia with isoflurane, nitrous oxide, vecuronium, and fentanyl. Hydromorphone was given during the last hour of the case. You administered ondansetron during closure as antiemetic prophylaxis. You gave neostigmine and glycopyrrolate a few minutes ago. The isoflurane vaporizer is turned off and the patient is being ventilated with 100% oxygen.

- Which classes of drugs are most likely to be responsible for his delayed emergence? Which are less likely?
- Among the most likely possible causes, do you suspect a pharmacokinetic problem? A pharmacodynamic problem?
- How could you narrow the differential diagnosis using history, physical examination, clinical monitors, or pharmacologic probes?
- If you conclude that isoflurane is responsible for the patient’s delayed awakening, how will you proceed?

Chapter 4 Case Study

You are asked to provide general anesthesia for an otherwise healthy 30 year-old woman undergoing pelviscopy. She has a history of endometriosis and chronic pelvic pain. Her brother had a near-fatal episode of malignant hyperthermia as a child and she has been counseled to avoid triggering anesthetics. You decide to manage the case with total intravenous anesthesia, avoiding...
inhalation anesthetics altogether. You have appropriately removed the vaporizers from your anesthesia machine and flushed it with 100% oxygen according to published recommendations.

- Which classes of intravenous agents will you need?
- Which drug will use to produce and maintain unconsciousness? How will you know you’ve given enough? Will the dose needed change during the surgery?
- Which opioid would be most appropriate for intraoperative use? The case is booked for 2 hours. Will you change to a different agent for postoperative analgesia?
- Which neuromuscular blocking drug(s) will you choose, if any?
- At the end of the case, how will you conduct the emergence?

**Chapter 5 Case Study**
You are asked to induce anesthesia for an ENT procedure in which the surgeon wishes to inspect the airway during spontaneous respiration without the presence of an endotracheal tube or laryngeal mask airway. The patient is otherwise healthy and has a normal appearing airway, and you judge that maintaining the airway by mask will be successful. You agree to induce anesthesia by inhalation. The patient has an IV and standard monitors are in place.

- Which inhalation agent will you choose?
- Would a combination of more than one inhaled agent offer any advantage?
- What are the factors you can control which will speed induction of anesthesia?
- You have an end-tidal gas monitor to measure exhaled agent. How will you know when you have the patient deeply anesthetized enough to allow the surgeon to perform laryngoscopy?

**Chapter 6 Case Study**
A 70 kg otherwise healthy male patient is undergoing bilateral inguinal herniorrhaphy under local anesthesia administered by the surgeon and intravenous sedation you are giving. The surgeon is planning to infiltrate the skin with lidocaine prior to skin incision.

- The patient reports a history of an “allergic reaction” to Novocain (procaine) which she received during a dental procedure. Is it safe to administer the planned local anesthetics?
- The surgeon is planning to use 2% lidocaine with epinephrine for initial infiltration, followed by bupivacaine, 0.5% for longer lasting analgesia. How can she enhance the onset of the block?
• After infiltration with lidocaine, the surgeon is prepared to infiltrate further with bupivacaine and perform some deep nerve blocks to enhance analgesia. She asks you how much of a 0.5% solution she can safely use. How will you respond?
• The surgeon begins infiltration with bupivacaine. After about 15 mL have been injected, the patient complains of lightheadedness and then his eyes roll back and he loses consciousness. The patient develops tonic-clonic movements of his extremities. How will you respond?
• Despite your initial efforts, the patient remains unresponsive. The electrocardiogram shows ventricular tachycardia. You cannot palpate a pulse. How will you proceed?

Chapter 7 Case Study
You are asked to provide anesthesia for a woman undergoing needle-directed breast biopsy. She has had several past anesthetics and has not had good experiences. She explains that she has had severe nausea after all her general anesthetics, and that she has been very somnolent after general anesthesia as well as after monitored anesthesia care (local anesthetic plus sedation). Review of her medical record shows that she received reasonably ordinary general anesthesia, with a potent inhaled agent, nitrous oxide, and fentanyl. For her MAC case, she received intravenous boluses of midazolam and fentanyl. After both anesthetics, she recalls experiencing significant pain but could not tolerate oral opioids prescribed for her. She is motivated to avoid general anesthesia and would like you to develop an anesthetic plan that reduces her risk of excessive somnolence and nausea. She is otherwise healthy, exercises regularly, does not smoke or drink and takes no medication regularly. She has fasted overnight.

• The surgeon believes that she can perform the procedure under local anesthesia plus intravenous sedation (MAC). What drugs will you select for sedation?
• What strategy will you follow to control her pain?
• What strategy will you follow to avoid postoperative nausea?

Chapter 8 Case Study
You are seeing a 64 year-old man in the preop clinic. He is to undergo an open suprapubic prostatectomy a week from today. His past medical history is notable for an inferior non-q wave MI 2 years ago. He was managed at that time by placement of a bare-metal stent. He has smoked a pack of cigarettes a day for 35 years and sometimes gets shortness of breath during exertion, in cold weather, and when he has a URI. He has had hypertension for many years. Five years ago he was diagnosed with type 2 diabetes mellitus. He works as a carpenter, carrying boards
around the job site, and he does his own yard work. His medications at present are aspirin 81 mg once per day, atenolol 100 mg daily, metformin, exenatide (Byetta), as well as an albuterol inhaler and sublingual nitroglycerin as needed.

- What ASA physical status class is this patient?
- How would you assess his risk and prepare him for surgery from a cardiovascular standpoint?
- How would you assess his risk and prepare him for surgery from a pulmonary standpoint?
- He asks you if he should quit smoking before the surgery. How would you respond?
- How should his diabetes be managed for surgery? Would your recommendation be different if he were taking insulin?
- What other information would you like to obtain to complete your preoperative evaluation?

Chapter 9 Case Study
You are preparing to anesthetize a 50-year-old man for abdominal hernia repair with mesh. He is 68 inches tall and weighs 260 pounds. He has a full beard and mustache. He has no other major comorbidities. He underwent general anesthesia 20 years ago for arthroscopy of his knee and is not aware of any problems with the anesthetic. You are planning general endotracheal anesthesia.

- What factors in this patient worry or reassure you regarding his airway management?
- How will you further assess his airway?
- You decide to proceed with induction of anesthesia. After administering propofol you attempt mask ventilation. You find it difficult to obtain a good mask fit and mask ventilation is difficult. How will you proceed?
- You are now successfully ventilating the patient. You administer rocuronium to facilitate intubation. After ventilating the patient for 3 minutes, you perform direct laryngoscopy with a Macintosh 3 blade. You can only visualize the tip of the epiglottis. How will you proceed?
- Your initial efforts are still yielding only a view of the epiglottis. You decide to use an alternative airway device to assist you. What are some of your options?

Chapter 10 Case Study
You are working with your attending on a busy day. She tells you to go set up the room for your first case. You are familiar with the preparation of the airway equipment and have previously discussed the drugs you will be using. As you
walk towards the OR, your attending calls out to you to “remember to check the anesthesia machine.” You walk into the OR and discover to your dismay that the machine is an older model that does not feature an automatic machine checkout like the more modern ones that you have been using. (Note that this case will be easier if you have read the supplemental Internet material referenced in the chapter).

- You begin by inspecting the hoses attached to the machine from the gas outlets on the wall. How can you tell if they are properly connected and functional?
- How can you tell if you have adequate backup gas supplies should the hospital supply fail?
- How can you test to make sure the machine will prevent administration of a hypoxic gas mixture?
- Later you are doing the case, which began uneventfully. The patient is intubated and being mechanically ventilated. You note on the capnograph that there appears to be inspired CO2. Given your understanding of the anesthesia machine, why might this be occurring (see figure 10.2)? Which of the causes should you have been able to pick up during the machine checkout?

**Chapter 11 Case Study**

[Editor’s note: this case is primarily about monitoring, though figuring out the entire scenario will require your knowledge from other chapters].

You are providing anesthesia for a healthy young woman having a laparoscopic tubal ligation, your last case of a busy day of short gynecology cases. You induced anesthesia with propofol and succinylcholine and artfully intubated the woman’s trachea. You have maintained anesthesia with sevoflurane and fentanyl. The case is now over and you are preparing to wake the patient up. You have discontinued sevoflurane, increased oxygen flows, and have expected to see the patient open her eyes by now. She remains apneic (ventilator dependent, no spontaneous respirations), unresponsive to verbal stimuli, and does not react when you suction her mouth. Your attending comes into the room and asks you why you’re not already on your way to the PACU.

- How do you know she is apneic? Which monitors can verify this for you?
- You conclude that the patient is indeed apneic. Two minutes into your examination, the pulse oximeter shows the saturation to be 99%. How is this possible? Do you suspect a malfunction?
- How can you tell if you have allowed enough time for the anesthetics to be eliminated?
Although you believe that enough time has indeed elapsed, you would like to confirm whether or not she is “asleep.” What other monitors can help you?

On the basis of these investigations, you are convinced that the patient’s anesthetics have been eliminated, and that she is not anesthetized. What else might explain her failure to awaken? What monitor could help you verify the diagnosis?

Chapter 12 Case Study
A 78-year-old ASA III male with a Mallampati class III airway presents for a cerebral angiogram due to a recent episode of severe headache and transient neurological deficit. He has a history of stable coronary artery disease, poorly controlled hypertension, hyperlipidemia and type II diabetes mellitus. He is a former heavy drinker and smoker but quit both last year. He has no known drug allergies and takes atorvastatin, lisinopril, metoprolol and rosiglitazone (Avandia). You plan monitored anesthesia care (MAC).

The case will be done in the angiography suite, not the OR, and you plan MAC, not general anesthesia. How will this alter your anesthetic equipment set up?

What drugs will you select for the case?

After imaging the patient, the radiologist discovers an aneurism and small intracerebral hemorrhage and wishes to coil embolize it to prevent further bleeding. She requests that you alter conditions to completely immobilize the patient for the procedure. What are your options?

Suppose you select general anesthesia. How will you induce and maintain anesthesia? Do you need to intubate the patient and control ventilation?

How will you monitor the patient after you induce general anesthesia? Will your plan change, relative to the monitored anesthesia care phase of the case?

How do your recovery (PACU) plans change with the decision to change to general anesthesia?

Chapter 13 Case Study
A 58 year old man is to undergo right total knee replacement (TKR). After a thorough H&P and consultation, he elects to have the procedure under regional anesthesia. He is otherwise healthy, though he smokes a pack of cigarettes a day and does not exercise regularly due to his arthritic knee. He takes a NSAID daily for pain and lately has been taking oxycodone and acetaminophen for worsening pain.

Which dermatomes or nerves will you need to block to perform a total knee replacement comfortably?
CASE STUDIES

Which regional anesthetic techniques are suitable for total knee replacement? Which will you choose?

If you choose epidural analgesia, how will you locate the epidural space? What precautions will you take to avoid toxicity?

After verifying proper position of the epidural catheter, what drugs will you use?

Will you continue to use your epidural after the procedure?

Chapter 14 Case Study

A 25-year-old otherwise healthy woman is to undergo radical resection of a pelvic sarcoma with prosthetic reconstruction to attempt to salvage the hip joint and thigh. The surgeon estimates blood loss will be 2-5 liters, depending on the findings at operation and extent of major vascular involvement. The estimated surgical time is 6 hours. She has a peripheral 14 G IV, a three-lumen central venous catheter in the right internal jugular vein, and a 20G right radial arterial line. She has 4 units of packed red cells available. She weighs 60 kg. Her preoperative hemoglobin and hematocrit are 12 and 36 respectively. She has fasted overnight and is scheduled for the first case in the morning.

How will you estimate her basic fluid requirements for the case?

How low will you let her hemoglobin drop?

What is her acceptable blood loss?

How will you assess and correct other blood product requirements?

What options do you have for reducing transfusion requirements?

Chapter 15 Case Study

A 35 year-old woman comes to the OR for emergency laparoscopic resection of a ruptured ectopic pregnancy. She was admitted to the emergency department with abdominal pain and was found to have a positive beta-HCG, a mass on abdominal ultrasound in her right Fallopian tube, and an empty uterus. Her last menstrual period was approximately 8 weeks ago. She states that she is otherwise healthy. She ate dinner approximately 4 hours ago but had little appetite at the time so states that it was “just a little.” She has a 20 G antecubital IV in place, which is slowly infusing lactated Ringer’s.

Is the existing IV sufficient for this case? How will you decide whether or not you need better IV access?

Exhaustive search for other veins yields no obvious prospects for additional access. The patient states that she has always been “a tough stick.” How will you proceed?
You plan a rapid sequence induction with propofol and succinylcholine.  60 seconds after injecting propofol, the patient has not lost consciousness. You have not yet injected succinylcholine. How will you proceed?

Can you induce anesthesia by inhalation instead?

You decide that you will need another IV to proceed. What options do you have to establish access?

Chapter 16 Case Study

A 52 year-old man is undergoing proctocolectomy for rectal cancer. He was admitted this morning for the operation after undergoing a bowel prep at home the day before. Anesthesia was induced with thiopental and vecuronium and intubation was uneventful. You have placed a peripheral IV, a right internal jugular central line, and a right radial arterial line. You are infusing cefazolin prior to incision.

Five minutes after induction, the blood pressure has decreased to 82/50. What is the differential diagnosis? What will your initial steps be to manage his blood pressure?

Your intervention is successful and the case begins. The patient develops tachycardia in the first few minutes. What is your differential diagnosis and initial response?

The patient's hemodynamic status has stabilized and the case is proceeding. Fifteen minutes later the patient's oxygen saturation begins to decrease and is now 90%. The patient is breathing 50% oxygen and 50% air by volume controlled ventilation. What is your differential diagnosis? What will be your response?

Your initial response to hypoxia has raised the saturation to 92% on 100% oxygen. Auscultation of the lungs reveals bilateral wheezes on exhalation. What steps will you take?

Wheezing resolves but the patient develops tachycardia and ST segment depressions. How will you respond?

Chapter 17 Case Study

A 48 year-old woman presents for resection of extensive rectal hemorrhoids. She first developed the condition during pregnancies in her late 30s and now has had unremitting symptoms of pain, itching, and occasional bleeding. Her surgeon also plans to perform a “tension free vaginal tape” (TVT) procedure for moderate stress urinary incontinence. She has a history of rheumatic heart disease and has had progressively worsening mitral stenosis. She takes digoxin and a baby aspirin daily.
• How will you assess the severity of her mitral valve disease?
• You conclude that she has moderately severe mitral stenosis with moderately reduced systolic function. What are your hemodynamic goals for the perioperative period?
• Her cousin had a very similar procedure performed recently and had spinal anesthesia. She had spinal anesthesia herself for a cesarean section and was very pleased with it. She asks you if she can have this form of anesthesia for her current procedure. How will you respond?
• Does she need antibiotic prophylaxis?
• You decide to administer general anesthesia. What drugs will you avoid? Which will you choose?
• What other special precautions will you take in the intra- and post-operative periods?

Chapter 18 Case Study
A 20-year-old male is attending a company picnic. After lunch, the attendees play softball. Your patient is struck in the head by a hit ball. He immediately loses consciousness and paramedics are called to the scene. He is transported to the hospital where a CT scan shows an acute subdural hematoma requiring surgical evacuation. He is awake but confused and sluggish and does not respond appropriately to verbal commands. He does withdraw purposefully to painful stimuli. He does not have any other injuries. His friends tell you he has “never been sick a day in his life.” He is 6 feet, 185 pounds. BP 185/90, HR 55, SpO2 96% on room air.
• Do you believe his intracranial pressure (ICP) to be elevated? What signs, symptoms, or tests can help you decide? Does it matter when deciding how to induce anesthesia?
• What determinants of ICP can you influence prior to induction? Will you lower his blood pressure prior to induction?
• What other considerations are there in deciding how you will induce anesthesia?
• Given all of the above considerations, what drugs will you choose for induction of anesthesia?
• What will you do if you are unsuccessful in intubating him?
• Once you have successfully induced anesthesia and secured the airway, what anesthetic considerations do you have for the remainder of the case?

Chapter 19 Case Study
A 30 year-old otherwise healthy woman presents at 39 weeks gestation with elevated blood pressure for induction of labor. You are consulted when she is 4 cm dilated, contracting regularly, and requesting labor analgesia.
What other information will you seek during your preoperative interview?

Your preop shows that she is pregnant with her first child and has intact membranes. Her platelet count is 165 x 10^9/L. Other laboratory studies are negative. Her previous medical history is negative and her anesthetic history is unremarkable. Her blood pressure on admission was 150/90 and has remained stable. The FHR shows a reassuring pattern. What is your anesthetic plan?

You select epidural analgesia. Describe the technique and your initial choice of drugs?

How will you maintain analgesia once established?

After three hours, you are paged because the patient is experiencing discomfort in the perineal area. She has tried pushing the PCEA button. How would you respond?

The patient has reached full cervical dilation and begins pushing. Shortly thereafter, you are paged urgently because of decelerations noted on the FHR tracing. What are your immediate steps?

Vital signs are normal and the patient is comfortable, but the FHR tracing does not improve. The obstetrician wishes to perform a cesarean section. How do you extend the epidural block for the operation?

Chapter 20 Case Study

A 38 year-old female is scheduled for laparoscopic Roux-en-Y gastric bypass. She is 5 feet, 6 inches tall and weighs 300 pounds. She has tried various diet and exercise plans to lose weight without success. She has hypertension treated with an ACE inhibitor. She wheezes on exertion or in hot weather and uses an albuterol inhaler as needed. She snores loudly while sleeping but has not had a formal sleep study and is not interested in CPAP at home due to a poor experience related by a friend. She does not exercise regularly but she is able to walk on level ground for a few minutes at a time in her work as an office postal worker. She has been told she has “borderline diabetes” but is not currently taking any medication for it. Preoperatively, her examination shows BP 180/95, HR 90, RR 24, scattered end expiratory wheezes which clear with cough, airway Mallampati class II, thyromental distance 4 fingerbreadths.

How severe is her obesity? Does it matter? Can any other obesity measures help you characterize her health risk further?

What concerns do you have about her respiratory status? How will these impact your anesthetic plan?

How will you monitor her during the anesthetic? Will your plan differ from a normally proportioned patient having laparoscopic surgery?
• How will you induce and maintain anesthesia?
• How will you manage postoperative pain? Would your plan differ if the procedure were an open Roux-en-Y?

Chapter 21 Case Study
A 68 year-old man has symptoms of benign prostatic hypertrophy and is to undergo transurethral resection of the prostate (TURP). He has hypertension and hyperlipidemia and takes an ACE inhibitor and atorvastatin (Lipitor). He is physically active and has no symptoms of angina or heart failure.
• What else will you investigate in the preoperative assessment?
• Will you recommend regional or general anesthesia? What are the relative merits of each?
• After discussion with the patient, you decide on general anesthesia. How will you induce and maintain anesthesia?
• The procedure takes longer than expected due to a very large amount of prostatic tissue requiring resection. At the end of the operation, you extubate the patient and take him to the PACU. He is hypertensive, confused and agitated. How will you assess him?
• If you believe he has TURP syndrome, how will you treat him?

Chapter 22 Case Study
A five year-old boy has been vomiting and had little or no appetite for two days. He has taken limited amounts of liquids by mouth. He has now developed abdominal pain and is suspected of having acute appendicitis. The surgeons plan a laparoscopic appendectomy. The child is a healthy product of a full-term delivery. Vital signs are HR 120, BP 95/50, RR 24.
• How will you assess his volume status prior to surgery? What metabolic derangement would you suspect him to have?
• The child is anxious and teary. How can you help during the preparation for and induction of anesthesia?
• Would you perform an inhalation or intravenous induction?
• If you decide on an intravenous induction, how can you facilitate placement of the IV in this frightened child?
• How will you induce and maintain anesthesia? What size endotracheal tube will you use?
• How will you know when you are able to extubate the patient at the end of the procedure?
Chapter 23 Case Study
An 82 year-old female suffered a fall, fractured her right hip, and is to undergo open reduction and hemiarthroplasty. She has no other injuries and did not lose consciousness. She is a smoker with a 60 pack-year history, but currently smokes just 2-3 cigarettes per day. She has chronic hypertension and an electrocardiogram from last year showed a right bundle branch block and a left anterior hemiblock with a sinus rhythm and rate of 55. She is a retired professor of pathology, a medical school dean, and still serves on your hospital's faculty council on promotions. She is in mild-moderate pain, which is much worse with movement of the right leg. She has expressed some concern regarding the effects of anesthetics on postoperative cognitive function.

- **What preoperative assessment will you perform before deciding on an anesthetic plan? How would it differ from the preop you'd perform if the patient were having an elective cataract surgery?**
- **How will you address her concern about postoperative cognitive dysfunction?**
- **Will you favor regional or general anesthesia?**
- **Will you premedicate the patient prior to anesthesia?**
- **If you and the patient agree on regional anesthesia, what type will you perform?**

Chapter 24 Case Study
A 20 year-old woman is scheduled for breast augmentation surgery. She attends college and works part time as a waitress and in the college library. She is strongly motivated to have the procedure performed as an outpatient and to return to work and minimize her time away from school and work. She is generally healthy, though she notes that she has seasonal allergies and occasional wheezing for which she takes an antihistamine and uses a metered dose inhaler (albuterol) as needed. She does not smoke, drinks alcohol on the weekend (3-4 drinks once per week), and does not use recreational drugs. She takes oral contraceptives and also has a history of motion sickness.

- **Is it appropriate to do this case in an outpatient surgery center? What other information do you need to decide?**
- **Is she at high risk of postoperative nausea and vomiting (PONV)?**
- **How will you induce and maintain anesthesia?**
- **How will you manage postoperative pain?**
- **How will you reduce the risk of PONV?**
- **Anesthesia and emergence are uneventful and you take the patient to the PACU. When can she go home?**
Chapter 25 Case Study
A 23 year-old male was an unrestrained driver in an automobile crash in an older car without airbags. He and his friends had recently left a party where he had consumed “a couple of beers.” He hit the steering wheel on impact and has multiple contusions on his chest and complains of chest pain with respiration. His left shoulder is dislocated. He also has a broken tibia and is suspected of having a splenic injury. He did not lose consciousness at the scene. His breath smells of alcohol and he is snoring loudly. He awakens with vigorous shouting and is somewhat combative and confused. He complains of pain in the affected injured area when examined and can move all four extremities on command. He is an otherwise previously healthy college student.

- What is his Glasgow Coma Scale score?
- The patient arrives from the emergency department with two upper extremity peripheral IVs in place infusing room temperature lactated Ringer’s. Do you need additional access? How will you modify the resuscitation strategy in the OR?
- Studies of the aorta have led the surgeons to observe rather than operate for this injury. The cervical spine was found free of fractures or dislocations on head and neck CT scan. The patient is still wearing a cervical collar placed at the scene. He does not complain of neck pain. Can you now remove it prior to facilitate management of the airway?
- The patient has not consumed solid food for eight hours and last drank liquids more than 2 hours ago. How will you induce anesthesia and secure the airway?
- What other goals will you have during anesthesia for the case?

Chapter 26 Case Study
A 32 year-old woman seeks consultation with you in the pain management clinic. Six months ago she sprained her left elbow and wrist in a fall while roller blading. After recovering uneventfully with splinting of her wrist and wearing a sling for four weeks, she has developed severe pain again. She describes it as burning and constant. She describes tingling, “electric shock” sensations over the affected area. It covers the dorsum of her hand, both sides of her forearm, and the posterior aspect of the elbow and lower arm. She notes that she cannot type with her left hand and that she cannot lift her backpack with her left arm. She finds showering painful and keeps the arm out of the water; she avoids long-sleeved shirts because the fabric rubbing against her skin is painful. On examination the limb is purplish and mottled, edematous, and cool to the touch. There is less hair than on comparable regions of her right arm. The nails
of her left hand are thickened, discolored, and longer than those on her right. Lightly stroking the dorsum of her hand with a fingertip causes pain.

- **You perform the initial evaluation with your attending. You are asked to dictate the note describing the patient’s pain presentation. Which of the four main types of pain will you characterize hers as?**
- **Which pain descriptors will you use to describe her symptoms?**
- **What is your working diagnosis? How could you verify it?**
- **What treatment would you offer her?**

### Chapter 27 Case Study

A 45 year old woman has just undergone total abdominal hysterectomy. She is generally healthy, does not smoke or drink alcohol, and has not had general anesthesia ever before. She emerged from general anesthesia (thiopental, vecuronium, sevoflurane, fentanyl) uneventfully. You accompany the patient to the PACU, assist the nurse with settling the patient, and obtain initial vital signs on arrival: BP 148/90, HR 77, SpO2 98% on facemask oxygen at 6 L/min.

- **Describe the elements of the report you will now give to the PACU nurse.**
- **After completing your report you leave the bedside to complete your paperwork. Before you return to the operating room, approximately 5 minutes after your initial arrival in the PACU, the nurse calls you back to the bedside. The patient is agitated, thrashing around in bed and not answering questions or following instructions to lie back and relax. What will be your initial steps in assessing the patient? What is the differential diagnosis?**
- **You exclude emergencies and conclude the patient is experiencing emergence delirium. How will you respond?**
- **The patient improves. One hour later you are called back to the PACU. The patient is complaining of pain. How will you assess the patient? What intervention will you recommend? Would your approach be different if the patient had undergone laparoscopic myomectomy and was scheduled to be discharged home later today?**
- **The pain is under control 30 minutes later but the patient now complains of nausea. How will you respond?**
- **When can the patient be discharged from the PACU? How would your criteria differ if the patient were being discharged home after laparoscopy instead?**

### Chapter 28 Case Study

You are called to the PACU emergently to see a 57 year old patient who has just undergone an aorto-bifemoral bypass graft. On arrival at the bedside, the nurse
inform you that the case proceeded uneventfully and the patient arrived in the PACU one hour ago. The patient underwent general endotracheal anesthesia and was extubated in the OR. Vital signs on arrival had been normal, but the blood pressure had been progressively declining and heart rate had been rising since then. Five minutes ago, the patient’s blood pressure had been 68/40 and heart rate 128. Now the nurse notes she cannot obtain a blood pressure and cannot feel a pulse. The patient has a peripheral IV infusing lactated Ringer’s and an arterial line in the right radial artery. No blood pressure is seen on the arterial tracing.

- What will be your initial response (first 30 seconds) on arrival?
- The patient is found to be apneic and pulseless. What will you do next?
- The patient is found to be in ventricular fibrillation. What will you do next?
- After your initial intervention, sinus rhythm reappears. Inspection of the arterial tracing shows minimal pulsatile activity, and manual blood pressure measurement confirms that the blood pressure remains unobtainable. What are your next steps?

Chapter 29 Case Study
Peter is your favorite anesthesiology resident. He is amazingly confident, skillful, and aggressive. He loves “big” cases and always volunteers for trauma, cardiac, or messy “whomps.” You’ve seen him at a couple of social events, and he is the life of the party, joking with everyone, positively lighting up the room. He drives a sports car, regales his friends with stories of his travel adventures, and dates a model. He recently took up skydiving and is working on his private pilot’s license. But he is also amazingly generous. He has covered other residents’ call several times, and he offers to stay late and finish late cases so others can go home. Today, you witnessed an event that seemed totally out of character. One of his assigned cases, one of those big cases he loves, was moved to another room because the first case in his room was running late. He was irritable as he dropped off his patient in the PACU. Then he sought out the floor leader and lambasted him (an attending with 20 years of seniority) for “taking my case away.” Then he sought out the resident in the room where the case was transferred and demanded to switch assignments (they had put a breast biopsy in his room). This resident had already begun working with the patient and refused. Peter told the patient that he was more experienced and a better anesthesiologist than the resident now assigned to him, and asked the patient if he wouldn’t prefer Peter as his anesthesiologist. The frightened patient was
speechless. Peter stormed out of the preoperative area and told the floor leader that he was sick and needed to be sent home.

- **What lapses in professionalism have you witnessed?**
- **Later, you are discussing the event with another resident and a nurse in the PACU. Both tell you that they are not surprised. “Peter has been pretty volatile lately,” they agree. Another resident says that Peter has recently ended his relationship with his girlfriend and “is always at the hospital. He sleeps here even when he isn’t on call. And he has a great apartment.” How does this knowledge influence your view of the event you witnessed?**
- **Despite your suspicions, no action is taken against Peter. Several weeks later, he is on call with you and he is paged for a case. He does not respond to several pages. You are sent to his call room to wake him up and ask him to come to the OR. You knock on his door with no response. You knock more loudly and finally enter the room with your own key. You find Peter in bed, apparently asleep, with the lights and television on. You wake him with great difficulty and when rising he is groggy and somewhat incoherent. He sits up and quickly gathers his belongings into his backpack while muttering something about being exhausted. You believe you have seen several glass ampoules in his bag. What will you do?**
- **Peter is later found to have fentanyl and hydromorphone in his bag and tests positive for opioids in his urine. He admits to having been diverting drugs from the OR for about three months, beginning after his relationship began to unravel. Would random drug testing of all residents have prevented this situation?**
- **Is this problem more common in anesthesiology?**
- **Peter undergoes several weeks of inpatient detoxification and rehabilitation. Should he re-enter the operating room as an anesthesia resident?**

**Chapter 30 Case Study**

An anxious 48 year-old patient is in the preoperative holding area awaiting outpatient surgery under general anesthesia. With her is her husband, an expert on risk assessment in non-medical industries, and her father, a retired surgeon in his late 70’s. She is anxious because her father has told her stories of surgery in the 1950s and 60s, when he remembers significant numbers of patients dying or suffering significant morbidity. Her husband has worked in aviation, industrial process design, and is a “six sigma black belt.” All three acknowledge your assurance that the practice of anesthesia is remarkably safer now, but ask you to explain some of the safety advances that characterize anesthesiology today and explain the improvements.
You have just finished setting up the operating room for this case. What safety features of the modern anesthesia machine can you point to in reassuring the patient and her family? What are some of the monitoring developments since the 1950’s that have improved safety? What drug-related advances and procedures have you employed that have enhanced safety? What communication procedures will you employ that enhance safety? What other safety procedures are routine for all anesthetics in modern practice? The patient’s husband asks if anesthesia is “six sigma?”

Chapter 31 Case Study
An 80-year-old man has terminal colon cancer. He has metastatic disease with liver and brain metastases. As his condition worsened over the preceding year, he had several conversations with his family and physicians about his end of life care. He has a signed and witnessed advanced directive indicating his desire to be treated as “DNR/DNI” (do not resuscitate, do not intubate). He has now developed bowel obstruction and was admitted with severe abdominal pain. His surgeons have recommended a diverting colostomy for palliative care. They obtained consent for the operation from the patient last night but anesthesia consent has not yet been obtained. The patient was medicated with hydromorphone and is now somnolent and falls asleep immediately upon waking. The surgeons are eager to operate before the bowel ruptures.

Can you obtain informed consent from the patient? Is surgical consent sufficient? What options do you have? How should you interpret the patient’s DNR/DNI order for the operation, assuming you have obtained consent? You are planning general endotracheal anesthesia for the operation. If you proceed with surgery with general endotracheal anesthesia, and you are unable to extubate the patient at the end of the case, what will you do? Are you liable for a malpractice claim?

Chapter 32 Case Study
It’s the last day of your rotation. You are doing a case completely by yourself in the simulator. You are surprised by how nervous you felt in the beginning, as if the patient you are caring for is not the mannequin in front of you but a real patient. But there is no attending guiding you, and you’ve heard that sometimes
things go very wrong in the simulator. You’re not being graded, but you are being videotaped, and you know that your fellow students and the instructors will be reviewing your performance. But so far it’s been a quiet case. Your “patient” is undergoing an abdominal operation under general anesthesia. You handled the application of monitors, induction of anesthesia, mask ventilation and endotracheal intubation like a pro. The patient is being mechanically ventilated. You are using desflurane, nitrous oxide, fentanyl, and vecuronium for anesthesia. You are using standard monitors and have a peripheral 18 G IV in place. Blood loss has been about 100 mL but the surgeons anticipate more later in the case, and you have blood available in the blood bank. You’re feeling pretty good about yourself, thinking you might enjoy anesthesiology as a career. After all, you’ve learned a ton of the basics in your month, and here you are doing a case pretty much by yourself!

Suddenly, all the lights in the room go off and the room falls into an inky blackness and eerie quiet.

- It doesn’t stay quiet for long. The surgeon shouts that he has just incised a structure and is concerned that the patient may be bleeding. He is screaming for light and help and accusing you and the circulating nurse of causing a power failure. The circulator is screaming back at the surgeon that she didn’t do anything (and that neither had you). What are your first steps in assessing the situation?
- The surgeon says that the operation is at a critical juncture but that if he can work for 5-10 minutes, he will be at a stable stage and could end the operation with a quick closure. He is still concerned that the patient may be bleeding. How can you get him enough light to continue?
- You recognize that both the ordinary hospital power supply and the emergency power have failed. Your ventilator is still functioning on battery backup. All of your monitors are not functional except for the BIS brain monitor, which is running on battery power. How will you alter your anesthetic?
- How will you monitor the patient?
- The battery backup on your ventilator has now run out of power and the ventilator stops. The oxygen flowmeter drops to zero and you realize that the pipeline oxygen supply has failed. How will you proceed?
Section I

Introduction to Anesthesiology
Chapter 1

How to Be a “Star” Student, Career Options, and the Match

Roy G. Soto

For maximum impact, it is recommended that the case study and questions found on page xvii are reviewed before reading this chapter.

Introduction

If your favorite place in the world is the operating room, be a surgeon. If your favorite place in the hospital is the operating room, be an anesthesiologist. For many, the practice of anesthesiology is the perfect blend of science, medical management, procedural skills, variety, and fun. Where else can you care for critically ill patients, listen to music, socialize with surgeons, and wear your pajamas all at the same time?

This chapter will outline:

- setting goals for your medical student anesthesia rotation
- career options within anesthesiology
- the match
- a run-through of a typical case

How to Be a “Star” Student

Although most medical student rotations are only 2 weeks in length, it is still possible to structure your rotation for maximum educational value. It is important, however, to have a list of educational objectives prior to starting your rotation – many programs are moving away from the “show up and we’ll stick you somewhere” method of teaching. If you arrive with (1) a basic understanding of the important physiologic and pharmacologic concepts, and
(1) **Understand basic physiology/pharmacology:**

- Review respiratory and cardiac physiology (Chap. 17)
- Review autonomic nervous system pharmacology (Chap. 3)
- Review cholinergic, anticholinergic, cholinesterase actions / Interactions (Chap. 7)
- Review opiate pharmacology (Chap. 5)
- Review local anesthetic pharmacology (Chap. 6)

(2) **Formulate specific goals:**

- Become proficient at mask ventilation
- Intubate 7 patients successfully
- Place LMA's in 3 patients
- Observe at least 1 epidural
- Observe at least 1 spinal
- Observe at least 1 peripheral nerve block
- Spend at least one day in a pain clinic or work with a pain team

(2) a list of specific goals, then you’ll stand out from the crowd and get the most from your rotation. This means that you will have to:

**Career Options**

As an anesthesiologist, you will have a variety of career options to choose from. Some physicians choose to stay in academic medicine – focusing on research, teaching, or advancing clinical practice. Others choose to go into private practice – most often working for a private group that contracts with a hospital, or less frequently becoming a direct hospital-paid employee.

Within the specialty, individuals may opt to complete advanced training or fellowships after residency in one of three ACGME approved areas: cardiothoracic anesthesia, pain management, or critical care. There are also nonaccredited opportunities for additional training in pediatric anesthesia, obstetric anesthesia, regional anesthesia, and neuroanesthesia, among others. Many individuals choose to engage in research training – either during or after their residency. Currently, the American Board of Anesthesiology will allow some residents to enter into the “clinical scientist” pathway – which provides for a 6 month research experience during the final (CA-3) year of residency.

The location, size, and type of practice you choose will ultimately affect the kind of anesthesia you end up practicing. In some states and regions, there is an increased reliance on physician extenders including Anesthesia Assistants...
(AAs) and Certified Registered Nurse Anesthetists (CRNAs). This occurs just because there are not enough anesthesiologists to go around, and nurse/AA supervision in the “anesthesia care team model” is a safe, effective, and efficient way to provide care. That being said, there are a number of variations on the theme, with physician-only practices still popular, and supervision ratios varying widely from 2:1 to 4:1, depending on the setting.

CRNAs are registered nurses who have completed masters-level training in nurse anesthesia following nursing work in a critical care environment. Anesthesiologist Assistants (AAs) have also completed masters-level training in anesthesia, with an undergraduate degree typically in premed or a similar science major. AAs are currently licensed to work in approximately 20 states, with only five training programs nationwide when compared to the approximately 100 programs for CRNAs.

Ultimately, most practitioners find themselves happy, successful, and satisfied with the safe care provided in a team environment, and the future of the specialty continues to be bright.

The Match
Anesthesia has had its ups and downs as a popular specialty, and is now considered one of the most sought after fields. Many seek out the challenge of solving complex problems in real-time, the ability to work in the operating room environment, or the satisfaction of placing endotracheal tubes, invasive monitors, and/or advanced nerve blocks. In addition, the flexible schedule and ability to balance clinical practice with other interests (e.g. teaching, research) are other appealing features of the practice of anesthesiology. The match is currently very competitive, with hundreds of applicants applying for, on average, a dozen positions at popular programs. Figure 1.1 outlines typical milestones for a medical student interested in pursuing anesthesia.

As with most specialties, applicants must use the ERAS system, although they can apply “outside the match” for PGY2 (aka CA1) positions following an internship/residency year in another specialty. All programs formed as of 2008 must have an integrated internship (predominately medicine, surgery, and critical care), and chances are that all programs will move in this direction eventually. Couples matching is supported, and there is no early match system.

Programs may sometimes give priority to students from their own hospital system, or at least to students who have rotated with them. As a student, try to get as much “face time” as possible during your 3rd and 4th years with the programs in which you are interested. Although there is no official “cut-off”
of USMLE score for prospective residents, given the current competitive nature of the anesthesiology match, many programs have set USMLE step-1 score limits for granting interviews, often in the 200–230 range. Again, having personal experience in a department can be a great way to gain an advantage over competing candidates.

**A Typical General Anesthesia Case**

Although the anesthesiologist needs to consider various patient and procedure factors when administering anesthesia care for a patient, there are some routine actions that are commonly performed in the pre-op holding area, in the operating room, and in the recovery room (PACU) during a typical general anesthetic. Figure 1.2 outlines the phases of a typical general anesthetic case.

Now, let us discuss the flow of a routine general anesthetic:

Josh is a 33-year-old man with cholecystitis who needs his gallbladder removed.

**Preoperative Evaluation**

Unlike the standard internal medicine history and physical, ours is much more focused, with specific attention being paid to the airway and to organ systems that are at a potential risk for anesthetic complications. The type of operation and the type of anesthetic will also help us focus our evaluation. Prior problems with anesthesia are noted, and physical exam should focus on the heart, lung, and airway evaluation (to assess ease of intubation). Josh has a history of hypothyroidism, but takes his medications and recent TSH values are normal.
He has no drug allergies, has a good mouth opening, excellent neck extension/flexion, and good dentition. He had an appendectomy 10 years ago, and reports no problems other than postoperative nausea.

**Sedation**

Although many patients appear to be cool, calm, and collected, anxiety about surgery (as well as pain, prognosis, and being naked in front of strangers) is high and understandably common. We frequently sedate patients with midazolam (a benzodiazepine) and/or fentanyl (an opioid) prior to travel to the OR, with the goal of achieving sedation, amnesia (although this is not predictable), while maintaining normal breathing and airway protective reflexes.

Josh seems relaxed, but his palms are sweaty and his resting heart rate is 90 bpm. Administering 2 mg of midazolam has calmed him right down, and he jokingly asks if he can have it for his kids as he giggles his way into the OR.

**Monitoring**

Standard required intraoperative monitoring (general, regional, or sedation) includes continuous ECG, blood pressure (at least every 5 min), continuous pulse oximetry, and capnography in cases of intubation. Additional monitors may include temperature, invasive blood pressure (arterial line), central venous pressure, pulmonary artery, TEE (transesophageal echocardiography), and processed EEG (electroencephalography) monitoring, all at the discretion of the provider and guided by the patient’s health status and type of procedure. Given Josh’s good health and the minimally invasive nature of his operation, no monitoring beyond the minimal standard is required.
**Induction and Intubation**

Following preoxygenation, general anesthesia is induced with a variety of hypnotic and paralytic medications. Propofol is the most widely used induction agent today, with rapid and predictable loss of consciousness in about 20 sec, amnesia, and depression of airway reflexes. Other agents include thiopental (a barbiturate) which was the mainstay of anesthetic induction prior to the introduction of propofol, ketamine, which is reserved for those needing a sympathetic boost (e.g. trauma patients), and etomidate, which has minimal cardiac depressant properties and is typically reserved for patients with heart failure or shock. Paralytics come in two flavors: depolarizing and nondepolarizing – with succinylcholine being the only available example of the former. Succinylcholine produces the most rapid paralysis (45 sec), but can be associated with hyperkalemia, malignant hyperthermia, and muscle pain. The nondepolarizers are slower and longer acting, but are the most predominantly used agents (vecuronium, rocuronium, cisatracurium, and less frequently pancuronium), with each agent having its own unique advantages and disadvantages.

Intubation is performed following preoxygenation, loss of consciousness, and onset of paralysis using a rigid laryngoscope and a plastic endotracheal tube. The actual mechanics of intubation are much better taught on actual patients, and will not be discussed here, but suffice it to say that the more intubations you do, the better you get, and the tube will either make it into the right hole (trachea), or the wrong hole (esophagus). The key to success is rapidly determining which it is, and correcting a mistake quickly. A number of alternate airway techniques are available, including awake fiberoptic techniques, laryngeal masks, indirect visualization devices such as the Glidescope, and blind techniques such as the Light Wand.

Josh has a normal-appearing airway, is otherwise pretty healthy, and his operation requires approximately 1 h of paralysis to ensure appropriate abdominal relaxation for pneumoperitoneum (gas insufflation of the abdomen). We will perform a typical induction using propofol (2 mg/kg) and rocuronium (0.6 mg/kg) and intubate him using a Macintosh 3 blade and a 7.5-mm endotracheal tube. We will confirm tube placement by visualizing chest rise, “misting of the tube,” checking for end-tidal CO₂, and listening for bilateral breath sounds.

**Maintenance**

Maintenance of general anesthesia is usually achieved with inhalation of potent volatile agents such as sevoflurane, isoflurane, or desflurane (each with their unique potential advantages and disadvantages). The concept of “balanced anesthesia”
proposes that giving drugs from multiple classes will allow for less of any one to be given, thereby reducing the chance of side effects. Therefore, in addition to volatile agents, we frequently add nitrous oxide, opioids, intravenous hypnotics, and paralytics to the mix. If desired, volatile anesthetics can be avoided completely using a total IV anesthetic (TIVA) technique which is technically more difficult to perform, but can be used to great advantage in certain patients (e.g. patients with risk of malignant hyperthermia).

For Josh, we will choose isoflurane (1.1% exhaled concentration), fentanyl (1–2 mcg/kg every 20 min as needed, titrated to heart rate and blood pressure which we can use as a proxy for response to pain), and rocuronium (5–10 mg every 30 min as determined by nerve monitoring). In addition, we will address the issue of postoperative nausea and vomiting (he is at high-risk given his age, prior history, and procedure) by giving him a dose of dexamethasone (4 mg) at the start of the case and ondansetron (4 mg) at the end.

**Emergence**

The case is nearing its end, and it is time to start thinking about emergence. To be extubated, a patient must be hemodynamically stable, be oxygenating and ventilating well, be relatively normothermic, and have return of neuromuscular function. Volatile agents are rapidly exhaled once inspired vapor is turned off, and most intravenous agents have a short enough half-life, ensuring rapid awakening. Paralytics usually are actively reversed with cholinesterase inhibitors (increasing acetylcholine available to compete with the paralyzing agents), and antimuscarinics are given in tandem to counteract their side effects. Again, antiemetics are frequently given at this point as are pain medications.

Josh has had an uneventful procedure, is breathing on his own with excellent spontaneous minute ventilation and oxygenation, and is hemodynamically stable. He received neostigmine and glycopyrrolate to fully reverse his paralysis, and incremental doses of morphine are titrated to respiratory rate (the goal is the rate in the 10–20 range) to achieve a smooth, pain-free wake-up.

**PACU Management**

Anesthetic management does not end as soon as the tube comes out! The recovery period can be marked with challenges big and small, and as always, being properly prepared and expecting the unexpected can improve patient safety and satisfaction. Pain, nausea, and shivering are probably the most common complaints (in that order), but other frequently encountered problems
include delirium, airway obstruction, bronchospasm, hypertension, hypotension, tachycardia, postsurgical bleeding, and oliguria. Furthermore, some patients cannot be extubated in the OR, and PACU care, therefore, can include continued ventilation of the patient through a breathing tube and sedation management.

Josh has done well, but upon arrival to the PACU complains of pain and nausea despite your best intraoperative efforts. You prescribe doses of metoclopramide (for nausea) and hydromorphone (for pain), and he ends up meeting discharge criteria in 30 min… another successful anesthetic!

Summary
Enjoy your time during your anesthesia rotation! Make sure to come to the clerkship with your own goals and objectives in mind. You will likely enjoy getting procedures, but also pick the brains of those that you are working with to get the most out of your time. Do not want to be an anesthesiologist? That is OK… just direct your efforts in those aspects of anesthesia that most affect your career choice and pique your interest: obstetric, pediatric, cardiac anesthesia, etc. Anesthesiologists are experts in physiology, pharmacology, and clinical monitoring. They have to establish patient rapport rapidly, allay fear, and educate their diverse patients. Have fun and ask questions.

Case Study
You are preparing to provide general anesthesia for a 40-year-old woman undergoing an abdominal hysterectomy. She is otherwise healthy. She had two uncomplicated vaginal deliveries in the past, both with uncomplicated epidural labor analgesia. She had uneventful general anesthesia for a laparoscopic tubal ligation 4 years ago. Your attending is willing to let you perform as much of the anesthetic as you are able to describe in detail.

Upon meeting the patient in the pre-op area and reviewing the history and physical, you find no important new information. What steps will you take to prepare the patient for surgery prior to any interventions?
You will greet the patient and her family and answer any questions they may have about the procedure and planned anesthetic. You should review the remainder of the chart, paying special attention to any laboratory studies that may have returned since her pre-op clinic visit, including the
You have engaged the patient and checked all the paperwork and you are ready to begin preparing the patient for surgery. What are the next steps?
You will start an IV, probably a single 18–20 G cannula. Most anesthesiologists use a skin wheal of 1% lidocaine at the entry site before placing the IV. You will begin an infusion of IV fluid, typically lactated Ringer’s solution. If the patient is anxious, you may consider sedation prior to surgery. Not all patients require sedation and asking the patient whether she would like it or not can help you decide. If desired, midazolam, 1–2 mg with or without fentanyl, 50–100 µg, is a reasonable choice. It is prudent to place a pulse oximeter and to consider supplemental oxygen by mask or nasal cannula, especially if you are leaving the bedside.

You have brought the patient into the OR. Describe the steps you will take prior to induction of anesthesia.
You will check your machine, airway equipment, suction, and drugs if you have not already done so (many anesthesiologists do this before greeting the patient). You will position the patient comfortably, making certain her gown is not tied at the neck or in back, and that all pressure points are well padded. You will apply standard monitors (discussed in Chap. 11), including an electrocardiograph, pulse oximeter, and noninvasive blood pressure cuff, and verify that all are working properly. You will then “preoxygenate” the patient (more precisely, “denitrogenate”) by having her breathe 100% oxygen by facemask for several minutes to replace the room air in her FRC with oxygen. If the rest of the surgical team (surgeon, circulating nurse, scrub nurse or technician) is ready, you can induce anesthesia.

How will you induce anesthesia?
Intravenous induction is most common in adults. A short acting hypnotic, typically propofol or thiopental, is given to induce unconsciousness. Next, you will ensure that you can ventilate the patient by mask by giving a few breaths and observing chest movement, exhaled carbon dioxide, and noting a reasonable
tidal volume on the ventilation monitor. A neuromuscular blocking drug is then given to facilitate endotracheal intubation. Succinylcholine is rapid acting and reliable, though some anesthesiologists prefer the nondepolarizing type (rocuronium or vecuronium), which take longer to reach peak effect but may have fewer side effects. After about a minute (succinylcholine) or 2–3 min (nondepolarizers), you will intubate the trachea. A laryngoscope is inserted, carefully avoiding trauma to the lips, tongue, and teeth. The vocal cords are visualized and a cuffed endotracheal tube, 7.0 or 7.5 mm internal diameter is inserted until the cuff is below the cords. The cuff is inflated, the tube is connected to the anesthesia machine circuit, and positive pressure breaths are given by hand. If \( \text{CO}_2 \) is seen on the capnograph, intubation is verified. Auscultation of bilateral breath sounds verifies appropriate depth of the tube, which is then secured with tape. The patient can then be ventilated mechanically by activating the ventilator on the anesthesia machine.

**Following induction, what else will you do prior to the beginning of the surgical procedure?**

You will tape the patient’s eyes closed to prevent corneal injury. You will reposition the patient for surgery, if necessary, and check pressure points again. You may add additional monitors (peripheral nerve stimulator to monitor neuromuscular blockade, esophageal temperature probe, processed EEG or consciousness monitor [e.g., BIS]). Often, you will employ a convective air warming device to help maintain normothermia. Prophylactic antibiotics are best given less than 60 minutes before incision, so you will start these now if you haven't given already. In some operations, a nasogastric or orogastric tube may be useful (but probably not in this case). In others, you might want a second IV, a fluid warmer, or a blood administration set. Since you do not expect large fluid shifts or blood loss, you will forego these for now. You will participate in a “safety pause,” “time-out,” or a more extensive “surgical safety checklist” with the other members of the OR team. You will also begin your maintenance anesthetics.

**How will you maintain anesthesia?**

There are numerous ways to maintain a general anesthetic, which will be discussed in future chapters. A common one is the “balanced technique” which combines a volatile anesthetic with or without nitrous oxide, an
opioid, and a nondepolarizing neuromuscular blocking drug. A reasonable combination would be sevoflurane, fentanyl, and vecuronium. Sevoflurane is rapidly eliminated after discontinuation, so nitrous oxide is not necessary to reduce the amount of sevoflurane given as it might be for a more slowly eliminated drug like isoflurane (see Chap. 5).

**What other adjunctive drugs might you give in addition to anesthetics?**

A healthy young woman such as our patient is at reasonably high-risk of postoperative nausea and vomiting (PONV). Prophylactic antiemetics are often given, and a reasonable combination would be dexamethasone and ondansetron. You may be asked to give other drugs to facilitate the operation, for example, methylene blue to check for integrity of the urinary bladder. You will also consider longer acting opioids (for example, morphine or hydromorphone) before the end of the procedure to provide longer-lasting analgesia in the postoperative period.

**The operation has gone well and is ending. How will you conclude the anesthetic?**

As the surgical stimulation lessens during closure, you will lighten the anesthetic. After the fascia is closed, you can reverse neuromuscular blockade with a cholinesterase inhibitor (e.g., neostigmine) and an anticholinergic (e.g., glycopyrrolate). You can prepare for emergence by suctioning the patient’s mouth, untaping the eyes, and turning off the volatile agent and increasing fresh gas flow of oxygen to help wash out residual anesthetic in the circuit. If the room is cool, you will increase the temperature; if the patient’s gown is soiled, it may be changed. Once the surgical instrument and sponge counts are completed, the wound is closed, and the dressing is in place, you can wake up the patient. You will watch for return of spontaneous respiration, switching off the ventilator and allowing the patient to breathe on her own when she is ready. You will ask the patient to open her eyes and to follow a simple command (e.g., “Squeeze my fingers”). Once you are satisfied that she is awake, breathing adequately spontaneously, and strong enough to protect her airway, you will extubate her by deflating the cuff and removing the endotracheal tube. You will observe spontaneous respiration via a mask for a few moments, and then place a simple oxygen mask. After disconnecting the monitors, moving the patient, IV’s, urinary catheter
What will you do on arrival to the PACU?
Depending on the local procedures at your hospital, you may assist the PACU nurses in getting the patient “settled” by reestablishing hemodynamic monitoring, verifying adequate pain control and absence of nausea, and checking for stable vital signs. You will give a brief report of the procedure and your anesthetic course, fluid totals, analgesics and antiemetics to the PACU nurse. You will ensure that orders are present for maintaining analgesia, and rescue orders for breakthrough pain or nausea.

Congratulations on completing your first anesthetic!

Suggested Further Reading
Medical Student Anesthesia Primer: http://www.anesthesia-education.com/primer.doc

Society for Education in Anesthesia (SEA): www.seahq.org
History of Anesthesia and Introduction to the Specialty

David C. Lai

For maximum impact, it is recommended that the case study and questions found on page xvii are reviewed before reading this chapter.

Introduction

Welcome to the exciting and fast-paced specialty of Anesthesiology! As a medical student rotating through anesthesia, you are fortunate in being able to learn about anesthesia without having to give it on your own. In the past, despite having little or no prior experience, medical students were often the ones administering the anesthetics. The time honored tradition of “See one, do one, teach one” would not be invoked. Instead, a bottle of ether would be provided, and you would “pour one.” This is, in fact, what happened in 1894 to a third-year student at the Massachusetts General Hospital. He was called down from the seats in the famed hospital amphitheater, sent to a side room with a patient and an orderly, and told to put the patient to sleep for a surgical demonstration. Knowing nothing about the patient whatsoever, he proceeded the best he could under the orderly’s directions. The patient was finally brought into the amphitheater after an interminable amount of gagging. Once the operation began, there was a great gush of fluid from the patient’s mouth, most of which he inhaled, and he died. Despite this unfortunate turn of events, the operation was completed and was deemed a success. That evening, the student went to see the surgeon to atone for his sins – planning to then find a different occupation. He was told that he was not responsible for the man’s death, as he had a strangulated hernia, had been vomiting all night anyway,
and that sort of thing happened frequently. The student was Harvey Cushing, who went on to become one of the world’s greatest neurosurgeons.

Cushing’s experience, unfortunately, was all too common. Harold Griffith describes the early days of anesthesia as “… all too frequently associated with bubbling, gurgling, retching, regurgitation, tongue-biting, wild threshing about, and sometimes deadly asphyxia.” Euphemisms for anesthesia and/or surgical complications included “patient took the anesthesia poorly” or “status lymphaticus.” Matters were compounded because there were no recovery rooms – patients were taken directly to their ward beds. Today, even with high-risk patients undergoing increasingly complex operations and procedures, safe anesthesia is almost taken for granted.

Patients often want to know what kind of anesthesia they will be receiving. The more important question is who is giving the anesthesia. Historically, the anesthetist was the most junior person present (i.e. the medical student). This person also had the most interest with the operation at hand, so their focus was on the operative field, not the patient. If a surgeon did his own spinal before operating, then no one even pretended to watch the patient. Macintosh noted: “for the surgeon the spinal ends with the injection of the agent; for the anesthetist it begins with the injection of the agent.” Keep in mind that all anesthetic options should be discussed with the patient, despite the fact that the surgeon may have already “requested general anesthesia.” As for ether and chloroform, they are still in use. All of the modern inhalational agents (except halothane) are ether derivatives, and chloroform is a low-level contaminant of both enflurane and desflurane.

The Story of Ether

Before the anesthetic properties of ether were understood, various techniques and agents were employed to control surgical pain. Ancient humans collected herbs to be used as sedatives. Many remedies were either ineffective or presented a significant risk to the patient. Drugs long available for relief of pain included alcohol, opium, hyoscine, cannabis, and cocaine. Non-drug methods of acute pain control included ice, head concussion, carotid compression, nerve compression, hypnosis, and bloodletting.

Ether anesthetics had been successfully administered to patients from the early 1840s by the likes of William E. Clarke and Crawford W. Long for procedures ranging from teeth extraction to neck tumor excision. However, its impact on surgical anesthesia practice solidified after the first successful public demonstration of ether by a Boston dentist, William T.G. Morton
This demonstration occurred at the Massachusetts General Hospital on October 16, 1846 inside what is known today as the “Ether Dome.” After administering the ether, Morton said to Dr. John Collins Warren, the surgeon: “Your patient is ready, sir.” Under general anesthesia, Dr. Warren removed a congenital vascular malformation from 20-year-old Edward Gilbert Abbott’s neck. After the surgery, the patient replied, “I did not experience pain at any time, though I knew that the operation was proceeding.” Dr. Warren then famously remarked, “Gentlemen, this is no humbug.” This demonstration was indeed a landmark in the history of anesthesia. Before anesthesia, surgery was considered a terrifying last resort, and without adequate pain control available (agents such as alcohol, morphine, herbals were used with variable success), surgeons were judged by their speed and patients had to be strapped down and endure excruciating pain. Anesthesia made it possible for surgeons to take more time to do a better job, and to perform more complex operations.

The success of ether anesthesia was welcomed by the public as the “greatest gift ever made to suffering humanity.” Dedicated to the discovery
of ether and reliable anesthesia, “The Ether Monument” was constructed in Boston Public Garden in 1868 to commemorate the first public demonstration of inhalational anesthesia (Fig. 2.2).

**Chloroform and the Queen**

In addition to ether, another agent, chloroform, was introduced into clinical practice by James Simpson in 1847. Although chloroform was more potent and easier to use, it had significant side effects. However, it was John. Y. Snow who
popularized the use of chloroform for obstetric anesthesia. He was one of the first physicians to study and calculate dosages for the use of ether and chloroform, and personally administered chloroform to Queen Victoria when she gave birth to the last two of her nine children.

By the end of the nineteenth century, many other advances in the field of anesthesia followed, as shown in Table 2.1. With the discovery of the local anesthetic properties of cocaine, infiltration anesthesia, nerve blocks, spinal, and epidural techniques were introduced. By the turn of the century, there were many advances in the area of airway management, such as orotracheal tubes used for intubation, laryngoscopes, and bag-mask ventilation devices. Soon, various intravenous induction agents were introduced, allowing patients to go off to sleep quickly, resulting in a more pleasant experience. Newer and better muscle relaxants became widely available, followed by safer and more clinically useful inhalational agents. Today, anesthesia is very safe, with mortality as low as 4–5 deaths per million of anesthetic administrations. This improvement in safety is in large part due to better patient monitoring, modern anesthetic drugs and equipment, and constant vigilance by the anesthesia provider.

**Monitors**
The earliest monitor was simply a finger on the pulse. Virginia Apgar (an anesthesiologist at Columbia who developed the now famous Apgar Score for newborns) had a case where she didn’t realize that the pulse she was feeling was her own, and the patient unfortunately had a poor outcome. When ECG machines came into being, they were not available in every room. One hospital in Sheffield, England had to borrow one from the university hospital a mile down the road – and someone to interpret the ECG as well!

When President Kennedy was brought into the Parkland emergency room after being fatally shot, Buddy Giesecke (who later became Chairman of Anesthesia at the University of Texas) had to go to the anesthesia workroom to get the ironically named “bullet” cardioscope, which required him to push needle electrodes into the President. At the same time, Jim Carrico (a surgery resident who had just finished an anesthesia rotation) intubated the President’s trachea, but had neither pulse oximetry nor capnography at his disposal. Pepper Jenkins (who was Chair of Anesthesiology at UT Southwestern in Dallas at the time) helped recreate these scenes in Oliver Stone’s movie JFK, where Jenkins plays himself.
Table 2.1  Historical dates of note.

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>1500 BC</td>
<td>The use of opium-like preparations in anesthesia recorded in the Ebers Papyrus</td>
</tr>
<tr>
<td>1275</td>
<td>Ether discovered by Spanish chemist Raymundus Lullius</td>
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<tr>
<td>1540</td>
<td>The synthesis of ether was described by German scientist Valerius Cordus</td>
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<tr>
<td>1665</td>
<td>First intravenous injection of an opiate through a quill</td>
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<tr>
<td>1773</td>
<td>Joseph Priestly introduces nitrous oxide</td>
</tr>
<tr>
<td>1842</td>
<td>Crawford W. Long successfully uses ether during neck tumor excision in Jefferson, Georgia</td>
</tr>
<tr>
<td>1845</td>
<td>Horace Wells publicly demonstrates the use of nitrous oxide in Boston – however, it is labeled a “failure”</td>
</tr>
<tr>
<td>1846</td>
<td>William T.G. Morton shows first successful public demonstration of ether anesthesia at Massachusetts General Hospital, Boston.</td>
</tr>
<tr>
<td>1847</td>
<td>James Young Simpson uses Chloroform for labor pain</td>
</tr>
<tr>
<td>1853</td>
<td>John Snow administers chloroform to Queen Victoria during childbirth</td>
</tr>
<tr>
<td>1878</td>
<td>William MacEwan introduces oro-tracheal intubation with a flexible brass tube</td>
</tr>
<tr>
<td>1884</td>
<td>Carl Koller discovers local anesthetic properties of cocaine</td>
</tr>
<tr>
<td>1889</td>
<td>August Bier describes spinal anesthesia for surgery</td>
</tr>
<tr>
<td>1894</td>
<td>Anesthetic charts introduced</td>
</tr>
<tr>
<td>1905</td>
<td>Long Island Society of Anesthetists founded</td>
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<tr>
<td>1921</td>
<td>Fidel Pagés describes a lumbar approach to epidural anesthesia</td>
</tr>
<tr>
<td>1932</td>
<td>First barbiturate, hexobarbital, used clinically</td>
</tr>
<tr>
<td>1941</td>
<td>Robert Miller and Sir Robert Macintosh introduce “Miller” and “Macintosh” blade concepts, respectively</td>
</tr>
<tr>
<td>1942</td>
<td>Harold Griffith uses curare for the first time during an appendectomy</td>
</tr>
<tr>
<td>1956</td>
<td>Michael Johnstone introduces halothane, a halogenated inhalational agent</td>
</tr>
<tr>
<td>1960s</td>
<td>Fentanyl, ketamine and etomidate synthesized</td>
</tr>
<tr>
<td>1977</td>
<td>Propofol is synthesized</td>
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<tr>
<td>1970s</td>
<td>Pulse oximeter is developed and becomes widely available for use in 1980s</td>
</tr>
<tr>
<td>1980s</td>
<td>Halothane gradually replaced by enflurane and isoflurane</td>
</tr>
<tr>
<td>1983</td>
<td>Archie I.J. Brain introduces Laryngeal Mask Airway (LMA)</td>
</tr>
<tr>
<td>1986</td>
<td>ASA House of Delegates passes “Standards for Basic Anesthetic Monitoring” resolution</td>
</tr>
<tr>
<td>1990s</td>
<td>Sevoflurane and Desflurane introduced into clinical practice</td>
</tr>
<tr>
<td>2000s</td>
<td>Anesthesia Information Management Systems (AIMs) come into widespread use</td>
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</tbody>
</table>

Airway Management

Everyone rotating through anesthesia wants to learn how to intubate, but it is **more important** to learn the way to effectively bag-mask ventilate a patient. Early general anesthetics were given by “open drop” masks and required great
skill and judgement to titrate (Fig. 2.3). Only since the end of WWII has endotracheal intubation become a common and accepted practice. Prior to this, surgeons feared laryngeal damage from tracheal intubation, and many would not allow it. Lumbar discectomies in the prone position, trachealesophageal fistula repairs and lobectomies were some of the most challenging surgeries anesthesiologists had to suffer through with mask anesthesia. In fact, the desire to use endotracheal tubes was considered by some surgeons to be laziness and unwillingness to work to maintain the airway by more “conventional” means.

Early endotracheal tubes were originally cut from rubber tubing. Harold Griffith (a pioneer in the use of muscle relaxants) utilized French silk-woven urethral catheters. He was amused when the manufacturer’s representative visited from Paris and said that “the girls in his factory asked him to take a look at those Canadians who needed a size 36 catheter.” Blind nasal intubation, which did not require a laryngoscope, was a common technique. Ivan Magill, whose forceps are still used today, described the ideal sniffing position of the patient’s head as “draining a pint of beer.” His nasal intubations were facilitated by up to 20% topical cocaine. Although J. Alfred Lee (who published his famous A Synopsis of Anaesthesia in 1947) had to purchase his own laryngoscope during WWII, because it was a new and unnecessary technique, he still enjoyed

Figure 2.3   Ether Mask & Bottles (Photo J. Ehrenfeld, M.D.).
teaching the practical skill of blind nasal intubation. Lee even intubated his own trachea under local anesthesia prior to his thyroidectomy under general anesthesia.

Noel Gillespie remarked in his 1941 book *Endotracheal Anaesthesia*: “Intubation is a difficult proceeding which calls for the services of a person with special training, skill, and experience. The beginner can acquire the last two qualifications only by constant practice. He must at the outset face the fact that learning to intubate is a *via dolorosa*: that he will often inflict trauma and that he will undergo much embarrassment, vexation, and humiliation in the process of learning. Surgeons should realize this and should have as much forbearance with the young anesthetist as they do with the early ineptitude of their own residents.”

**CPR**

Peter Safar was a pioneer in cardiopulmonary resuscitation and in the development of modern intensive care units. Asmund Laerdal from Norway visited him in 1958 while he was at Baltimore City Hospital. Within a few months, they had developed the *Resusi-Annie* manikin. A copy of the death mask of the “Girl of the Seine” was in Laerdal’s parents’ home. This became the face you look into when you ask “Annie, Annie, are you OK?” The concept for the original Ambu bag was developed in 1953 by Henning Ruben. It incorporated his Ruben Valve, which was so ubiquitous that he was once mistakenly addressed as Dr. Valve.

**Autonomy**

From a historical perspective, anesthesia has not always been held in high regard. In the early 1900s, it was simply a service, like laundry, dietary, or housekeeping. More often, it was under the jurisdiction of the department of surgery. Everybody wants to be able to do their own thing. The child grows up. The medical student becomes an intern. The resident becomes an attending. But to have an entire department under the aegis of another? The inability to gain independent status tormented many heads of anesthesia sections in the department of surgery to the point where they resigned. Often, a new chief could be recruited only on the condition of independent departmental status.

When the Chair of Surgery at the University of Arizona was let go, Burnell Brown, as senior member of the surgical faculty, became the acting Chair of Surgery. As a result, at least one class of graduating surgical residents had certificates signed by an anesthesiologist. The Dean was so pleased with
Brown’s performance that he recommended the division of anesthesia become a separate department. The acting Chair of Surgery did not object.

The American Society of Anesthesiologists was founded in 1905 to advance the art and science of anesthesia as a medical specialty and to raise standards through education and research. Today, most anesthesia departments are autonomous, and anesthesiologists have been Medical School Deans at Columbia, Johns Hopkins, Louisville, South Carolina, SUNY Upstate, Vanderbilt, Texas College of Osteopathic Medicine, and Florida. Their way was paved by Stuart Cullen (UCSF 1966–1970), Manny Papper (Miami 1969–1981) and James Eckenhoff (Northwestern 1970–1983).

**A Slumber of Anesthesiologists**

Choosing a specialty is no easy task. It will likely define your future professional career, your colleagues, the meetings you attend, and your work environment. The following list of medical groups generated at the 1972 American Society of Anesthesiologists (ASA) Board of Directors meeting may help you decide which medical clique best suits you: a slumber of anesthesiologists, a slash of surgeons, a rash of dermatologists, a brace/cast of orthopedists, a hassle of psychiatrists, a dribble/pool of urologists, an aerie of ophthalmologists, a gaggle of laryngologists, a stiff of pathologists, a clot of hematologists, a push of obstetricians, a family of GPs, a warren of gynecologists, a beat of cardiologists, a shadow of radiologists, a cavity of dentists. The appeal of anesthesia is that you get to interact with the whole menagerie of medical and surgical specialties, or you may choose to limit your practice or to specialize.

**Growing Prestige**

When Leroy Vandam entered medical school in 1934, few medical students, himself included, opted for a future in anesthesia. Vandam himself practiced surgical pathology and surgery, until recurrent retinal hemorrhages resulting in left eye enucleation brought him to anesthesia. He later oversaw anesthesia during the first successful human kidney transplant in 1954 and published hundreds of papers on the practice of anesthesia.

As the specialty began, there were few role models, and personal experience would have been open drop ether at home by the family practitioner, or at the hospital by the surgical intern or a nurse anesthetist. For a long time, the best students were going into internal medicine, the next level into the surgical specialties, and anesthesia was getting what was left. Commenting on the perception of anesthesia in the 1940s, John Bonica (founder of the field of pain
medicine) remarked, “When I went into the specialty, people thought you went into anesthesia because you couldn’t do anything else. Now it has become one of the most prestigious specialties.”

This change in prestige was not confined to the United States. A South African doctor in 1991 reflected on the improved status of anesthetists: “I have had to revise drastically the opinion I formed of anesthetists forty years ago when I first began to practice. Then they were at the bottom of the professional hierarchy, with a high proportion of dimwits, no-hopers and drunks. Now, they are near the top, with a range of professional skills that make the obstetrician a member of the lumpen proletariat.”

**Life Outside Anesthesia**

Medical school admission committees often look for well-rounded applicants. Once accepted, many things may have to be scaled back or even put on hiatus in order to survive medical school and residency. But how long must you sacrifice the activities other than medicine that bring meaning to your life? No doubt, anesthesia is a high-risk specialty, but if you are up to the challenge, you can find both professional as well as personal fulfillment.

**Conclusion**

Anesthesia is an incredible specialty practiced by a wide range of interesting people. Whereas it was once an art possessed by a few, then a science that could be taught to many – in its most elegant and refined form, it is both an art and a science. As Frances Foldes (founding chairman of the Department of Anesthesiology at Montefiore Medical Center in the Bronx) used to say, “Anesthesia is awfully simple or simply awful.”

**Case Study**

The year is 1900 and you are a student spending time with surgeons during medical school. You are excited because you are to go to the operating room for the first time this morning. To your surprise, you will not be watching the procedure, open removal of a kidney stone, from afar, but instead will be taking an active role! The surgeon has asked you to administer the anesthesia. You are told to bring the patient into the operating theater (where there are indeed stadium seats occupied by numerous observers of the famous attending surgeon). An orderly has shown you where the anesthetic supplies are kept.
Anesthesia in 1900 probably means general anesthesia. Although spinal anesthesia had been demonstrated by the late nineteenth century, it was not in common use. Ethyl ether and chloroform were the most popular anesthetics and the only ones in widespread clinical practice. Some 600 compounds had been proposed for anesthetic action, but many were toxic, ineffective, or violently explosive. Nitrous oxide had been tried unsuccessfully in the nineteenth century and had not become a routine drug because its lack of potency and tendency to hypoxia were known but still unsolved problems.

Probably none! Hypodermic needles were known, but intravenous anesthetics were not in clinical use. Some experiments with injection of agents such as morphine, chloral hydrate, or scopolamine had produced sedative effects but were not mainstream in 1900. Attempts to inject a plethora of drugs, herbs, and other chemicals intravenously had led to numerous complications (likely including sepsis).

You will likely induce anesthesia by mask inhalation. For many procedures, a gauze lined mask was used and ether was administered by the open drop technique. Some very early anesthesia delivery systems were in use by 1900, however, and depending on the sophistication of your hospital, you may use one of these, which might feature a “draw over” chloroform vaporizer. Pressurized oxygen was available in some locations, but frequently at this time you would administer no supplemental oxygen. Spontaneous breathing was nearly universal at this time, so you will use your skills at maintaining a natural airway. It will be tricky in the lateral position required for this operation!

Likely, you will use your senses only. You will observe the patient's respiration and color to evaluate the pulmonary system. You will palpate the pulse and perhaps listen to the heart and lungs intermittently. Blood pressure monitoring had been invented by 1900, but you would only have access to it in the most sophisticated hospitals. You will observe the patient's pupils and body movements to monitor the depth of anesthesia.
Will you keep an anesthetic record?
You might! Harvey Cushing, a neurosurgeon, had introduced recording of pulse and other observations by this time. A diligent and compulsive student, you will attempt to record some basic observations of the patient’s condition during your anesthetic.

Suggested Further Reading


Leake CD (1947) Letheon: the cadenced story of anesthesia. The University of Texas Press, Austin, TX


Section II

Pharmacology
Chapter 3

Pharmacology Principles

Jerome M. Adams and John W. Wolfe

For maximum impact, it is recommended that the case study and questions found on page xviii are reviewed before reading this chapter.

Key Learning Objectives

- Understand the basic principles of pharmacokinetics such as drug absorption, distribution, metabolism, and excretion
- Learn the basic principles of pharmacodynamics such as drug potency, efficacy, and therapeutic index
- Discuss the concept of context-sensitive half-time

Basic Pharmacologic Principles

An understanding of pharmacologic principles is important for effective anesthetic management. These principles are commonly divided into two groups:

1. **Pharmacokinetics** describes the fate of drugs once they have been administered to a patient. This process can generally be divided into three phases:
   (a) drug administration
   (b) drug distribution into body
   (c) drug metabolism and excretion

2. **Pharmacodynamics** describes the actions that a drug has on the body. This mainly consists of drug actions in which cellular receptors are enhanced or antagonized and includes the relationship between drug concentration and effects.
Pharmacokinetics

Absorption
The first step in drug delivery is absorption of the drug into the systemic circulation. A drug’s bioavailability is the fraction of the dose administered that reaches the plasma in an active form. Major factors affecting the bioavailability include:

- **Route of administration:** Most anesthetic drugs are administered via intravenous or inhaled routes, providing rapid and reliable blood concentrations of drug and high bioavailability. Other routes for administration include intramuscular or subcutaneous injection, oral or rectal administration, transcutaneous absorption (i.e. a fentanyl patch), and transmucosal absorption (i.e. sublingual nitroglycerin, nasal midazolam).

- **First pass metabolism:** Drugs administered via the gastrointestinal tract pass through the portal venous system prior to entry into the systemic circulation. As a result, drugs that are extensively metabolized by the liver must be administered in larger doses via the oral route versus the IV route in order to achieve similar blood concentrations.

- **Ionization:** The pH of the environment at the site of absorption (i.e. acidic conditions in the stomach) may affect the efficiency of drug absorption. In general, the nonionized fraction of a drug crosses the gastric mucosa more easily. Drugs that are weak acids, such as barbiturates, exist in a nonionized state at low pH and cross the gastric mucosa relatively easily. The opposite is true for drugs that are weak bases, such as opioids.

Distribution
Once the drug has entered the systemic circulation, it is distributed to various sites in the body, including the target organs. Factors affecting distribution include:

- **Free fraction and protein binding:** Many drugs exist in the plasma in an equilibrium of free drug and drug bound to various plasma proteins. In many cases, the drug is more than 90% protein-bound (midazolam, propofol, bupivacaine, etc). The portion of the drug that is protein bound is therapeutically inactive, and the free, unbound fraction is active. In cases where plasma protein levels are decreased, the free fraction of the drug (and the therapeutic effect of a given dose) is increased. Some conditions, such as hepatic or renal disease, can decrease the affinity of plasma proteins for drugs, again increasing the free fraction of the drug.
- **Volume of distribution (Vd):** The volume of distribution is defined as the total dose of drug given divided by the plasma concentration of drug. Drugs which are highly hydrophilic or protein-bound and stay in the plasma have a Vd close to the plasma volume. Those that are highly lipid-soluble will redistribute from the plasma to adipose tissue, leading to a low plasma concentration and a high apparent volume of distribution.

- **Redistribution:** This phenomenon describes a rapid fluctuation of drug concentration in highly perfused tissues that is most commonly seen with very lipid-soluble drugs (e.g., thiopental). It consists of the following stages:
  - After injection, the free fraction of the drug rapidly enters highly perfused tissues such as the brain and the heart, and more slowly enters into less perfused tissues such as adipose tissue.
  - As plasma drug levels drop because of continued entry of the drug into adipose tissue, the drug distributes back from the highly perfused tissues into the plasma. This typically terminates its therapeutic effect.
  - The drug then continues to distribute into adipose tissue, where it is stored.

- **Storage:** If doses of highly lipid soluble drugs such as thiopental are given repeatedly, the storage sites in adipose tissue may become saturated. The termination of the drug’s therapeutic effect then becomes dependent on metabolism and excretion, which are typically much slower than redistribution.

### Metabolism and Excretion

Drug effects are terminated by metabolism and excretion. Factors affecting this process include:

- **Mechanisms of metabolism:** Most anesthetic drug metabolism and excretion occurs at the liver, kidneys, and lungs. The major mechanisms can be summarized as below:
  - **Hepatic:** The liver eliminates drugs primarily by metabolizing them to inactive or less active compounds. The end products of hepatic metabolism are typically polar, water-soluble compounds that are suitable for renal excretion. Some excretion of drugs and drug metabolites into the biliary system also occurs.
  - **Renal:** The kidneys primarily eliminate drugs by excretion of water-soluble drugs or drug metabolites into the urine. Some direct drug metabolism also occurs in the kidneys.
  - **Pulmonary:** The lungs are the primary site of elimination of inhalational anesthetics, which are absorbed from the plasma and exhaled.
- **Zero-order pharmacokinetics**: A few drugs are eliminated via processes that obey zero-order kinetics, in which the drug is metabolized at a fixed rate, regardless of its concentration. (see Fig. 3.1)

- **First-order pharmacokinetics**: Most drugs are metabolized via processes that obey first-order kinetics, meaning that the rate of drug metabolism is proportional to the concentration of the drug (see Fig. 3.1). The rate of elimination is usually described in terms of the drug’s half time, which is the time in which metabolism and excretion reduce the plasma concentration of the drug to 50% of its starting value. As further time progresses, the process continues as detailed in Table 3.1. Note that after 5 half-times have passed, 96.9% of the drug has been eliminated, and for practical purposes, the drug has been fully eliminated.

- **Clearance**: The clearance of a drug is defined as the theoretical volume of blood that is completely cleared of drug per unit time. It is analogous to the creatinine clearance rate of the kidneys. Different pathways of clearance for a drug (i.e. renal and hepatic) are additive, and a decrease in a major pathway of clearance will prolong the effect of drugs that use that pathway for elimination (e.g., administration of a drug that is mainly cleared by the kidneys to a patient with impaired renal function will result in a relatively long duration of action).

![Zero vs. first order kinetics](Image Courtesy J. Ehrenfeld).
Context-sensitive half-time: As discussed above, some drugs are eliminated from the plasma by redistribution to adipose tissue. As the adipose tissue acquires more drug, the diffusion gradient from plasma to tissue decreases, and the rate of redistribution decreases. This leads to the phenomenon of context-sensitive half-times, in which the time to 50% reduction in drug concentration increases with increasing total doses of the drug or duration of infusion. Drugs that are highly redistributed but metabolized relatively slowly, such as thiopental, are affected more than drugs with rapid metabolism, such as sufentanil. The context sensitive half-times as a function of duration of drug infusion are shown in Fig. 3.2.
Pharmacodynamics
Factors relating to the actions that a drug has on the body include:

- **Potency**: A drug’s potency refers to the dose of the drug required to achieve a therapeutic effect. A smaller dose of a more potent drug will achieve the same effect as a larger dose of a less potent drug (see Fig. 3.3).

- **Efficacy**: A drug’s efficacy refers to the maximum effect achievable with the drug. Once a drug’s maximum effect has been reached, giving more will not result in increased effects (see Fig. 3.3).

- **Toxicity**: Drug toxicity occurs when undesirable side-effects of its administration occur.

- **Therapeutic index**: The therapeutic index of a drug is the ratio of the dose producing a toxic effect to that producing a therapeutic effect. A drug with a high therapeutic index requires a much higher dose to do harm than to achieve a desired effect, giving a relatively high margin of safety.

- **Actions on receptor systems**: Most drugs used in anesthesia exert their effects by binding to and modulating cellular receptor systems. In general, these effects can be categorized as being agonistic (enhancing the receptor system) or antagonistic (decreasing the receptor system). Some drugs are partial agonists, meaning that they have a relatively low efficacy and cannot produce a maximal effect on a receptor system, even at very high doses.

![Figure 3.3 Drug dose response relationship (Image Courtesy J. Ehrenfeld).](image-url)
- **Competitive vs. noncompetitive antagonism:** Competitive antagonists bind reversibly to cellular receptors, but do not activate them. The antagonist molecules compete with agonist molecules for access to the receptors. The effect of a competitive antagonist can be overcome by administering a high dose of an agonist. Noncompetitive antagonists bind to sites on the receptor molecule that are separate from the agonist binding site, decreasing the receptor’s affinity for agonist molecules or preventing the receptor from responding to the presence of an agonist. Because they bind to a separate site on the receptor, noncompetitive antagonists cannot be overcome by increased doses of agonist.

- **Stereospecificity:** Many drugs are supplied as a mixture of enantiomers (left/right stereoisomers). The levo- and dextro- variants of the drug may have different pharmacologic properties, and based on this, some drugs are supplied as pure levo- or dextro- formulations (e.g., levobupivacaine, ropivacaine).

- **Additive and synergistic responses:** Drugs with similar physiologic effects may interact with additive effects (i.e. Drug A plus Drug B gives the sum of their expected effects). In some cases, the interactions are synergistic, meaning that the combined effect is larger than would be expected from the additive effects of the drugs given.

- **Tolerance and physiological dependence:** Repeated administration of a drug can result in changes in its target receptor system as the body adjusts to the presence of the drug. Tolerance occurs when progressively larger doses of drug are required to produce the same physiologic effect. Physiological dependence occurs when a subject’s receptor systems have adjusted to the presence of a drug, and withdrawal symptoms occur when it is stopped (e.g., with opioids or benzodiazepines). Physiological dependence is distinct from addiction, which is characterized by psychological craving for a substance and its pursuit despite actual or potential negative consequences.

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**Case Study**

You are finished with a radical cystectomy with creation of an ileal pouch neobladder on an otherwise healthy, 80 kg, 60-year-old man with bladder cancer. The operation began 6 h ago and the patient has not yet emerged from general anesthesia. You experienced no major untoward events during the case and you believe the problem to be pharmacologic. The patient received
4 mg of midazolam in divided doses during the preoperative period to facilitate placement of an arterial line. Anesthesia was induced with thiopental and succinylcholine. You maintained anesthesia with isoflurane, nitrous oxide, vecuronium, and fentanyl. Hydromorphone was given during the last hour of the case. You administered ondansetron during closure as antiemetic prophylaxis. You gave neostigmine and glycopyrrolate a few minutes ago. The isoflurane vaporizer is turned off and the patient is being ventilated with 100% oxygen.

Which classes of drugs are most likely to be responsible for his delayed emergence? Which are less likely?

The patient received a short acting **benzodiazepine** and a single dose of a **barbiturate** induction agent with a short biologic action many hours ago. Although the terminal elimination of both drugs is many hours, it is unlikely that either is responsible. He received a **depolarizing neuromuscular blocking agent** during induction. Under ordinary circumstances, this drug (succinylcholine) would have lasted only 3–5 min and would therefore be an unlikely cause of delayed awakening. If the patient had a genetic deficiency in pseudocholinesterase, he would not have been able to metabolize it, and this would have vastly prolonged its effect. Most anesthesiologists would not have administered vecuronium, the longer-acting **nondepolarizing neuromuscular blocking drug**, if the patient had not shown signs of recovery from succinylcholine. Nitrous oxide, an **inhalation anesthetic**, is very rapidly eliminated after discontinuing its administration, making it an unlikely cause. The **anticholinesterase** neostigmine, the **anticholinergic** glycopyrrolate, and the **serotonin antagonist (antiemetic)** ondansetron do not cause sedation and are not causes of delayed emergence. This leaves the inhalation agent isoflurane, the **opioids** fentanyl and hydromorphone, and the neuromuscular blocking agent vecuronium as possible causes.

Among the most likely possible causes, do you suspect a **pharmacokinetic problem**? A **pharmacodynamic problem**?

Sensitivity to inhalation anesthetics does not vary markedly between otherwise healthy individuals who are not at the extremes of age. Therefore, if isoflurane is responsible for this patient’s slow emergence, it is likely due to a kinetic problem. Long periods of inhalation anesthesia
can slow emergence more than proportionately, because of the shape of the elimination curve. Conversely, opioid sensitivity varies significantly between individuals, and even if given based on body weight, unexpectedly intense effects may be observed. In addition, the clearance of some opioids can exhibit cumulative effects, particularly after prolonged infusions. (fentanyl shows this effect). In addition, the relatively long duration opioid hydromorphone was given recently, also suggesting a kinetic problem. Vecuronium is metabolized hepatically, and in the absence of liver disease, prolonged elimination (pharmacokinetic effect) is unlikely. However, if the effect of neostigmine is incomplete, either due to insufficient dose or time elapsed since administration, vecuronium may still be active. This would represent a combination of a pharmacodynamic effect of vecuronium and possibly a pharmacokinetic effect of neostigmine, which takes several minutes to produce its full effect.

How could you narrow the differential diagnosis using history, physical examination, clinical monitors, or pharmacologic probes?

The presence of isoflurane should be detected by an agent monitor, which measures the concentration of inhaled anesthetics in the expired gas. Generally, patients should awaken when the end-tidal concentration falls to less than 0.1–0.2 MAC, which would be about 0.1–0.2 % for isoflurane. The presence of opioids may lower this value for “MAC awake.” The peripheral nerve stimulator can diagnose residual neuromuscular blockade. Four strong twitches on train-of-four stimulation, or more accurately, sustained (>5 s) tetanus in response to 50–100 Hz stimulation, rules out residual vecuronium action. Alternatively, an additional dose of neostigmine (up to 5 mg total) can be given to ensure full antagonism. However, nerve stimulation is more reliable. A processed EEG monitor (e.g., BIS) can differentiate a sedated patient from a paralyzed but “awake” patient. Opioid effects are more difficult to diagnose. The history of dose and timing of administration may be helpful. For example, one should check to see if a large dose of opioids was recently given, or if a prolonged fentanyl infusion was only recently discontinued. The presence of pinpoint pupils is a sign of mu-opioid agonism, but papillary signs are considered only partially reliable under general anesthesia. However, if isoflurane has been eliminated and neuromuscular blockade has been reversed, then the physical sign may be helpful.
Slow respiratory rate may also indicate excessive opioid effect. In some cases, careful titration of naloxone, an opioid antagonist, can be used to reverse opioids. But care must be taken not to be overzealous with this drug. Sudden reversal of deep narcosis can lead to hypertension and pain. Moreover, due to its short duration of action, vigilance for return of opioid effects in the PACU is essential.

*If you conclude that isoflurane is responsible for the patient’s delayed awakening, how will you proceed?*

Isoflurane must be eliminated by exhalation. You can raise the fresh gas flow of 100% oxygen to 10 L/min or more to ensure that the patient does not rebreathe any isoflurane. Modest hyperventilation, or at least avoidance of hypoventilation with the use of controlled ventilation or careful attention to end-tidal CO₂ during hand ventilation, may increase the rate of elimination. However, care must be taken not to hyperventilate to the point of cerebral vasoconstriction, which may counteract any enhanced elimination by reducing egress of drug from the brain. Beyond these maneuvers, only time will terminate the action of isoflurane. In some cases, postoperative ventilation in the PACU may be necessary.

**Suggested Further Readings**


Chapter 4

Pharmacology of Intravenous Anesthetic Agents

Jerome M. Adams and John W. Wolfe

For maximum impact, it is recommended that the case study and questions found on page xviii are reviewed before reading this chapter.

Key Learning Objectives

- Learn the relative advantages of each of the commonly used intravenous induction agents (propofol, etomidate, ketamine, thiopental)
- Discuss the pharmacokinetic properties of each of the commonly used intravenous opioids (fentanyl, morphine, hydromorphone, remifentanil)
- Understand the differences between depolarizing and nondepolarizing neuromuscular blockers

Ideal anesthetic agents are typically easy to administer (even in patients who are noncooperative), act quickly, and have limited durations of action and side effects. Inhalational and intravenously administered drugs tend to share these characteristics, in contrast to oral, intramuscular, and subcutaneous agents. It is for this reason that inhalational and IV drugs are used most frequently during a general anesthetic. A breakdown and description of the principal types of IV drugs encountered during a typical general anesthetic follows.

General anesthesia is the process of rendering a patient unconscious for the purpose of performing a surgical operation or other procedure. A good general anesthetic should facilitate airway management, including endotracheal intubation, if necessary. A general anesthetic will ensure that the patient is unconscious and amnesic throughout the procedure, optimize surgical conditions,
maintain hemodynamic stability, and will not negatively impact the patient’s intraoperative course or recovery. There is no one drug that can accomplish all these things in every patient, so multiple drugs are typically utilized in concert. This concept is known as “balanced anesthesia.” The anesthesiologist strives to maximize the positive actions of various drugs, while minimizing negative side effects.

Neuraxial (spinal and epidural) and peripheral nerve blockade are anesthetic techniques requiring drug delivery to very precise locations along the body’s neural transmission pathways. Local anesthetic drugs are primarily used for these techniques. A full description of both neuraxial blockade and peripheral nerve blockade appears in subsequent chapters.

The intravenous route is the primary means of delivery for most drugs during a typical anesthetic case, owing to the ease of administration and rapidity of transit to the drugs’ sites of action. We will consider several of the most commonly used intravenous drugs according to their pharmacological classes and their clinical application. The five most commonly used classes of drugs for a typical anesthetic are benzodiazepines, opioids, induction agents, neuromuscular blockers (NMBs), and sympathomimetics.

Benzodiazepines
The benzodiazepines utilized in anesthesia include midazolam (Versed), diazepam (Valium), and lorazepam (Ativan) all of which exert their sedative and hypnotic effects by enhancing GABA transmission (an inhibitory neurotransmitter). The most commonly used perioperative benzodiazepine is midazolam which has an elimination half-life of 3 h. With a typical sedative IV dose of 1–2 mg, the clinical effect typically lasts for 20–30 min owing to redistribution. Benzodiazepines are used for sedation, anxiolysis, and amnesia. A beneficial side effect of these drugs is their anticonvulsant activity, which can help raise the seizure threshold in susceptible patients (e.g. patients receiving nerve blocks are at risk for local anesthetic toxicity). Benzodiazepines do not provide analgesia and can be very long-acting when used in large doses. This is why benzodiazepines are usually used jointly with other agents during the course of an anesthetic.

Some patients, particularly children, are so anxious that the anesthesiologist deems it prudent to administer a benzodiazepine for anxiolysis prior to entering the operating room. Midazolam (0.25–0.5 mg/kg orally in children) can be administered in these situations. It is important to remember that loss of balance and respiratory depression can occur after administration of benzodiazepines (particularly when combined with opioids). Patients given
a benzodiazepine preoperatively should not be allowed to ambulate without assistance, and should always be monitored.

Intraoperatively, benzodiazepines can be used for sedation in instances where the patient does not receive a general anesthetic (often referred to as monitored anesthesia care, or MAC), or to provide sedation and/or amnesia as part of a balanced anesthetic technique. The amnestic properties of benzodiazepines are particularly useful in patients with poor hemodynamic status, who may not tolerate enough inhaled anesthetic agent to ensure complete unconsciousness.

If a patient becomes oversedated, or exhibits delayed emergence from general anesthesia, and the cause is suspected to be due to benzodiazepines, flumazenil (Romazicon) can be administered. Flumazenil is a pharmacologic antagonist which acts at the benzodiazepine receptor and effectively reverses the sedation from benzodiazepines. The drug is titrated in boluses of 0.1 mg every 5 min in adults. Because flumazenil only lasts about an hour and causes or produces incomplete reversal of respiratory depression, resedation can occur after administration (especially when used with diazepam, which has a half-life of approximately 20 h).

**Opioids**

Commonly used opioids include morphine, hydromorphone (Dilaudid), fentanyl and its derivatives, and meperidine (Demerol). These drugs provide sedation and analgesia, but do not provide reliable amnesia. They act on receptors in the brain (periaqueductal gray area) and spinal cord (substantia gelatinosa) via the mu (μ), kappa (κ), and delta (δ) receptors by mimicking endogenous endorphins. Opioid receptor activation is considered to lead to neurotransmitter inhibition via inhibition of acetylcholine and substance P release. Table 4.1 shows opioid receptor subtypes and effects.

<table>
<thead>
<tr>
<th></th>
<th>μ/δ</th>
<th>κ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analgesia</td>
<td>Supraspinal/spinal</td>
<td>Spinal</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>↓↓</td>
<td>↓</td>
</tr>
<tr>
<td>GI motility</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>Sedation</td>
<td>↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>Dependence</td>
<td>↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>Other effects</td>
<td>Euphoria</td>
<td>Dysphoria</td>
</tr>
</tbody>
</table>

**Table 4.1 Opioid receptor subtypes & effects.**
mainly for pain control intraoperatively, longer-acting opioids such as morphine, hydromorphone, or meperidine are usually used for postoperative pain. In addition to varying durations of action, it is their degree of binding to different opioid receptors and consequent side effect profiles that help in choosing the appropriate opioid for each patient and situation. Table 4.2 shows the relative dose, time to peak effect, and duration for the most commonly used IV opioids.

**Fentanyl** is a rapid-acting synthetic opioid which is about 100 times more potent than morphine. It is often given (dose 50–150 mcg for a 70-kg adult) during the induction of anesthesia to blunt the sympathetic response during intubation. It can cause apparent chest wall rigidity in high doses (1,000 mcg), which in rare cases may impair or prevent adequate ventilation.

**Sufentanil** and **Alfentanil** are both analogues of fentanyl. When compared with sufentanil and fentanyl, alfentanil is an ultra short-acting opioid (5–10 min), about 25% as potent as fentanyl, but has significantly faster onset than fentanyl (1–2 min). Sufentanil is approximately 5–10 times more potent than fentanyl. Both opioids may be used for induction and maintenance of anesthesia.

**Morphine** is the least lipid-soluble opioid and the most likely agent to accumulate in the presence of renal failure. It can cause bradycardia and histamine release in some patients. Morphine has a slower peak onset (30 min) when compared with fentanyl. Along with hydromorphone, morphine is the most commonly used long-acting opioid for postoperative pain control. Usually, either 5–15 mg of morphine or 1–2 mg of hydromorphone is given during a typical general anesthetic case.

### Table 4.2  Dose, time to peak effect, and duration of analgesia for commonly used perioperative opioids.

<table>
<thead>
<tr>
<th>Opioid</th>
<th>Dose (mg)</th>
<th>Peak (min)</th>
<th>Duration (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>10</td>
<td>20–30</td>
<td>3–4</td>
</tr>
<tr>
<td>Meperidine</td>
<td>80</td>
<td>5–7</td>
<td>2–3</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>1.5</td>
<td>15–30</td>
<td>2–3</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.1</td>
<td>3–5</td>
<td>0.5–1</td>
</tr>
<tr>
<td>Sufentanil</td>
<td>0.01</td>
<td>3–5</td>
<td>0.5–1</td>
</tr>
<tr>
<td>Alfentanil</td>
<td>0.75</td>
<td>1.5–2</td>
<td>0.2–0.3</td>
</tr>
<tr>
<td>Remifentanil</td>
<td>0.1</td>
<td>1.5–2</td>
<td>0.1–0.2</td>
</tr>
</tbody>
</table>

*Approximately equianalgesic dosages.*
Meperidine is structurally similar to atropine (may increase heart rate) and is metabolized to an active agent, normeperidine. It has useful antishivering properties and may be used postoperatively for this effect. It can accumulate in patients with renal failure leading to oversedation and/or seizures, and can cause release of histamine. It should be avoided in patients on type A monoamine oxidase inhibitors, as it may lead to hyperthermia, seizures, and even death. There is a well-known case involving the death of a patient named Libby Zion who received meperidine, although she had been taking phenelzine (Nardil), a type A MAO inhibitor. This error was found to result from overworked physicians who overlooked the drug reaction and ultimately led to the 80-h workweek limitation for residents.

Remifentanil has a potency similar to fentanyl, but is much shorter-acting (context sensitive half-time is about 4 min). It is broken down by nonspecific plasma esterases, and does not accumulate in patients after prolonged infusion, or in patients with renal or hepatic failure. It is almost always used as a continuous infusion, but can also be given as a bolus to facilitate intubation or nerve blocks.

Figure 4.1 shows how the context-sensitive half-time is a function of the length of time that the agent is administered. For opioids that exhibit accumulation (i.e., fentanyl), the context-sensitive half-time increases markedly with long
durations of administration. Opioids which are enzymatically degraded as fast as they are administered (i.e., remifentanil) do not show this effect.

Opioids can be used alone for sedation cases but have several dose-dependent adverse side effects. Consequently, opioids are more commonly used in combination with other agents for MAC cases or as part of a balanced general anesthetic.

The major adverse side effect of opioids is respiratory depression. This is due to both a decrease in the hypoxic drive to breathe, and an increase in the apneic threshold (the CO₂ level above which patients are stimulated to breathe). If a patient is nonresponsive and/or hypoventilating from opioid overdose, this effect can be reversed with naloxone (Narcan, 0.04–0.4 mg every 2min) which antagonizes mu receptors. Other adverse side effects of opioids include pruritus, bradycardia, arterial and venous vasodilation, nausea and vomiting, urinary retention, miosis, muscle rigidity (mainly with fentanyl), and decreased gastric motility/constipation.

There are also peripheral opioid receptors located in the gastrointestinal tract and other organs. Methylnaltrexone is an investigational peripheral opioid receptor antagonist and a quaternary derivative of naltrexone. Unlike naloxone, methylnaltrexone offers the therapeutic potential to block or reverse the undesired side effects of opioids that are mediated by receptors located in the periphery (e.g., in the gastrointestinal tract), without affecting analgesia or precipitating the opioid withdrawal symptoms that are predominantly mediated by receptors in the central nervous system.

Blunting of the endocrine stress response is a side effect of opioids that can be beneficial, especially during surgery. Because of their ability to decrease the stress response and minimal effects on baseline cardiovascular status, high-dose opioids are favored over other anesthetics in cases where hemodynamic instability is anticipated, or in patients where such changes would not be well tolerated.

**Induction Agents**

Induction is the process of starting a general anesthetic, or “putting the patient to sleep.” An ideal induction agent should be quick in onset, but should also be short-acting in case problems are encountered and the patient has to be awakened, or if the procedure is short in duration. Any IV medication that causes a patient to become unconscious can be considered an induction agent, and both benzodiazepines and opioids have been used in this capacity. However, due to their unpredictable onset time and long durations of action when used in doses high enough for induction, neither class is commonly used alone.
Typical induction agents include propofol, thiopental, etomidate, and ketamine. The agent chosen is usually determined by each drug’s side effect profile in relation to the patient or the case. Table 4.3 summarizes intravenous drugs and their dosages commonly used in anesthesia practice.

Propofol is the most commonly used induction agent and acts by enhancing transmission at the GABA-A receptor. Because of its rapid onset, titratability and short duration of action, propofol is also frequently utilized as an IV infusion to provide sedation for MAC or sedation cases, or as part of a balanced general anesthetic.

Propofol is a water insoluble agent that can only be administered intravenously. It is prepared as 1% emulsion with egg lecithin, glycerol, and soybean oil.

Propofol’s initial distribution half-life is 2–8 min, and it undergoes rapid hepatic metabolism to water soluble metabolites which are excreted by the kidneys. Remarkably, few pharmacokinetic changes are noted in the elimination of propofol for patients with liver or renal disease.

Propofol is a potent cardiovascular and respiratory depressant, and it should only be used by persons qualified and prepared to maintain the patient’s airway and hemodynamic stability. Propofol is often avoided in cases where

### Table 4.3 Recommended drug dosages for common IV agents.

<table>
<thead>
<tr>
<th><strong>Benzodiazepines</strong>&lt;sup&gt;b&lt;/sup&gt;</th>
<th><strong>Induction agents</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Midazolam 1–4 mg</td>
<td>Propofol 2–2.5 mg/kg (induction), 25–200 mcg/kg/min (infusion)</td>
</tr>
<tr>
<td>Diazepam 2.5–10 mg</td>
<td>Thiopental 3–5 mg/kg</td>
</tr>
<tr>
<td>Lorazepam 1–4 mg</td>
<td>Etomidate 0.2–0.5 mg/kg</td>
</tr>
<tr>
<td><strong>Opioids (Bolus)</strong>&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Ketamine 1–2 mg/kg IV, 3–4 mg/kg IM (induction or bolus), 1–2 mg/kg/h infusion</td>
</tr>
<tr>
<td>Morphine 1–5 mg</td>
<td><strong>Neuromuscular blockers</strong>&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Hydromorphone 0.2–0.5 mg</td>
<td>Succinylcholine 1–2 mg/kg, 20 mg bolus for laryngospasm</td>
</tr>
<tr>
<td>Fentanyl 25–100 mcg</td>
<td>Rocuronium 0.6 mg/kg</td>
</tr>
<tr>
<td>Meperidine 25–50 mg</td>
<td>Vecuronium 0.1 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Cisatracurium 0.15 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Pancuronium 0.1 mg/kg</td>
</tr>
</tbody>
</table>

<sup>a</sup> Adult dosages: Always start at low end of range and titrate up.

<sup>b</sup> Titration ranges for premedication or intraop/post op bolus dosing.

<sup>c</sup> Intubating dosages: Divide by 3 for ED95, divide by 5 for maintenance bolus dosing.
the maintenance of spontaneous ventilation is required, the patient is already hypotensive, or the patient’s ability to sustain hemodynamic stability is in question for any reason. Propofol decreases the body’s normal response to both hypoxia and hypercarbia, and up to 35% of patients experience apnea after an induction dose. Propofol decreases blood pressure by decreasing cardiac contractility, systemic vascular resistance, and preload. It is generally thought of as having the most profound cardiodepressant effects of all the induction agents.

From a neurologic standpoint, propofol has moderate anticonvulsant activity. It reduces both intracranial pressure and cerebral blood flow. However, due to greater effects on systemic blood pressure, propofol can actually decrease cerebral perfusion pressure when given in large doses. Another advantage of propofol is that it is generally thought of as affording less residual cognitive disarray when compared to other induction agents.

In addition to negative side effects of propofol already mentioned, pain on injection is seen in up to 67% of patients. Pain can be lessened with concomitant administration of 1% lidocaine. In addition, patients may experience mild muscle twitching and hiccupping. Favorable propofol side effects include antipruritic and antiemetic properties.

**Thiopental** is a barbiturate that shares many characteristics with propofol (enhances GABA transmission). It is rapid in onset, and has both cardiovascular and respiratory depressant properties. Many favor propofol because of the prolonged cognitive disarray observed in some patients after administration of thiopental. Thiopental is also possibly cerebro-protective, and it is used in many brain surgery cases. Thiopental solution is very alkaline, and can form a precipitate that will occlude IV catheters if mixed with acidic solutions or drugs (such as paralytic agents). Thiopental induces the enzyme ALA synthetase (the rate limiting step in porphyrin synthesis), and is therefore contraindicated in patients with inducible porphyrias. Repeated doses may result in a delayed emergence because of high protein binding and a low hepatic extraction ratio.

**Etomidate** is an imidazole which increases GABA transmission and has the advantages of minimal cardiac and respiratory depression. Its onset and duration of action are similar to propofol, but etomidate is considered a safer drug to use for patients in a compromised hemodynamic state. Trauma patients, elderly patients, and patients who are severely volume depleted or are on vasopressors are typical candidates for an etomidate induction. After a single bolus, the clinical effect of etomidate is terminated by redistribution and rapid hepatic metabolism. A concern exists regarding transient adrenal suppression after
use of etomidate, due to enzyme inhibition. The drug should therefore be used with caution or in concert with corticosteroid administration in those patients demonstrating adrenal insufficiency. Other side effects include myoclonus, pain on injection, and a high incidence of postoperative nausea and vomiting.

**Ketamine** is a dissociative anesthetic agent that is related to PCP (phencyclidine) and acts as an NMDA receptor antagonist. Its major drawback is the consequent perceptual distortions and illusory phenomena patients experience after administration. It is the only induction agent that is a cardiovascular stimulant, owing to inhibition of norepinephrine reuptake at sympathetic nerve endings, and also has minimal effects on respiratory drive. Of additional benefit is the fact that ketamine is both a potent analgesic and a bronchodilator (it is often administered in the emergency room to patients in status asthmaticus). Ketamine is ideal for many trauma inductions (sedation, analgesia, amnesia, and cardiovascular support), and for use in pediatrics (where perceptual distortions are not as frequently viewed with apprehension by the patient). It is typically avoided in situations where cardiac stimulation could be deleterious (arrhythmias, hypertension), and in cases where the patient is expected to emerge from anesthesia soon after administration (again due to expected deleterious psychological effects and cognitive disarray). Further side effects include increased salivation and intracranial pressure elevations (relative contraindication in patients with intracranial hypertension). Table 4.4 shows the cardiovascular effects of the most commonly used IV induction agents.

**Neuromuscular Blocking Agents**

Neuromuscular blockers (NMBs) or “paralytics” are frequently utilized during the administration of a general anesthetic. They are used to facilitate intubation and to improve surgical conditions by inducing relaxation of skeletal muscle.

---

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mean arterial pressure</th>
<th>Systemic vascular resistance</th>
<th>Cardiac output</th>
<th>Contractility</th>
<th>Heart rate</th>
<th>Intracranial pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propofol</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Thiopental</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Etomidate</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Ketamine</td>
<td>↑</td>
<td>↑</td>
<td>–</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

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**Table 4.4 Cardiovascular effects of IV induction agents.**
There are two major classes of NMBs, depolarizing and nondepolarizing. The classes are differentiated based on their action at the neuromuscular junction. Adequacy of relaxation can be determined by use of a nerve stimulator (see Chap. 11 on equipment). Nerve stimulator testing of a typical blockade with nondepolarizing NMBs demonstrates tetanic fade, posttetanic facilitation, train of four ratio less than 30%, and the ability to be reversed with anticholinesterases. In contrast, a typical depolarizing block does not display these characteristics – unless a Phase II block is present (see depolarizing NMBs below). The appropriate NMB for a given situation is chosen based on desired onset time, duration, elimination, and side effects.

**Depolarizing NMBs**

Succinylcholine is the only commercially available depolarizing NMB. Like acetylcholine, it works as an agonist on acetylcholine receptors at the neuromuscular junction. This causes depolarization, and prolonged binding of succinylcholine to the receptor prevents junctional repolarization because the drug is not hydrolyzed by true acetylcholinesterase. It is during this period that the muscle becomes relaxed.

Succinylcholine has the quickest onset (≈30–45 s) and shortest duration (≈5 min) of any available NMB, and it is the drug of choice for “rapid sequence” inductions. Because of its short duration, succinylcholine is used almost exclusively during intubation, and only rarely for maintenance of relaxation during a procedure. Should repeated doses of succinylcholine be administered (4–6 mg/kg in total), phase II blockade may occur leading to a slow recovery. This occurs when prolonged end-plate depolarization leads to conformational changes within the acetylcholine receptor.

Pseudocholinesterase is the enzyme responsible for breaking down succinylcholine. Some people have a partial or total deficiency of this enzyme and can therefore exhibit slightly prolonged (20–30 min for heterozygotes) or significantly prolonged (6–8 h for homozygotes) paralysis when the drug is administered.

One important side effect of succinylcholine is an elevation in serum potassium levels after administration. Because of this effect, succinylcholine must be used with caution in patients with elevated K⁺ levels and is usually avoided in patients with burn or denervation injury as these patients have an upregulation of postjunctional acetylcholine receptors and a consequently exaggerated response to the drug that may lead to a fatal arrhythmia.
Bradycardia, owing to a resemblance to acetylcholine and subsequent action on muscarinic receptors, and malignant hyperthermia (a rare hypermetabolic state that can occur in the skeletal muscle of susceptible individuals) are other side effects of note. Table 4.5 shows contraindications to the use of succinylcholine. Because of its mechanism of action, succinylcholine cannot be “reversed” by acetylcholinesterase inhibitors. In fact, attempting reversal can actually make neuromuscular blockade prolonged and more intense.

**Nondepolarizing NMBs**

There are several types of nondepolarizing NMBs, with the four in most common use being rocuronium, vecuronium, cisatracurium, and pancuronium (see Table 4.6). They can be subdivided according to their chemical structure into benzylisoquinoliniums (cisatracurium), and steroidals (rocuronium, vecuronium, and pancuronium). NMBs exert their effects by competitively

<table>
<thead>
<tr>
<th>Table 4.5 Contraindications to succinylcholine use.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated serum potassium levels (&gt;5.5 meq/L)</td>
</tr>
<tr>
<td>History of burn injury</td>
</tr>
<tr>
<td>History of denervation injury</td>
</tr>
<tr>
<td>Known or suspected myopathy</td>
</tr>
<tr>
<td>Known or suspected risk for Malignant Hyperthermia</td>
</tr>
<tr>
<td>Known pseudocholinesterase deficiency</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 4.6 Neuromuscular blocking drugs.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drug</strong></td>
</tr>
<tr>
<td><strong>Onset</strong></td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td>Succinylcholine</td>
</tr>
<tr>
<td>Cisatracurium</td>
</tr>
<tr>
<td>Vecuronium</td>
</tr>
<tr>
<td>Pancuronium</td>
</tr>
<tr>
<td>Rocuronium</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Notes</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma cholinesterase</td>
</tr>
<tr>
<td>Hoffman degradation</td>
</tr>
<tr>
<td>Liver/renal</td>
</tr>
<tr>
<td>Tachycardia &amp; hypertension</td>
</tr>
</tbody>
</table>
antagonizing acetylcholine from binding at the postsynaptic nicotinic receptor in the neuromuscular junction. The result of this competitive antagonism is an inhibition of junctional depolarization.

Onset time and duration of action are as follows: rocuronium < vecuronium < cisatracurium < pancuronium. Because of their longer durations of action as compared to succinylcholine, NMBs are commonly used to maintain muscle relaxation during surgery. NMBs are also used to facilitate intubation, but the time to achieve equivalent and ideal intubating conditions is significantly longer than with succinylcholine.

Reversal of NMBs is accomplished by the administration of an acetylcholinesterase inhibitor (e.g. neostigmine), which prevents breakdown of acetylcholine at the neuromuscular junction. The subsequent excess of acetylcholine can then out-compete the NMB for junctional binding, and allow for muscle depolarization. An anticholinergic, such as glycopyrrolate, must be simultaneously administered to prevent muscarinic overactivity such as severe bradycardia, asystole, or bronchospasm.

Most of the commonly used NMBs are metabolized to some degree, but they rely mainly on biliary and renal excretion for termination of action. Cisatracurium is the exception, as it is degraded in the plasma (Hoffman elimination). Cisatracurium is therefore commonly used in patients who have renal or hepatic dysfunction.

Side effects of NMBs are rare, with tachycardia (pancuronium), and hypotension (cisatracurium) being the most frequently encountered. Allergic reactions to anesthetics are rare, but are most commonly from NMBs.

**Acetylcholinesterase Inhibitors**

Neostigmine and edrophonium are acetylcholinesterase inhibitors that are primarily used to reverse neuromuscular blockade. They work by preventing breakdown of acetylcholine at the neuromuscular junction, thereby allowing the competitive inhibition of nondepolarizing NMBs to be overcome. Major side effects are bradycardia and excessive salivation. These are due to sudden and substantial increases in acetylcholine concentrations. Concomitant administration of an anticholinergic (such as glycopyrrolate) is required to prevent these side effects.

A “cholinergic crisis” may result from an overdose of acetylcholinesterase inhibitors or when the agent is given without a concomitant anticholinergic drug. Symptoms include bradycardia, bronchospasm, vomiting, miosis, and muscle weakness. Many nerve gases used in warfare are acetylcholinesterase inhibitors that can lead to a severe cholinergic crisis.
Anticholinergics

Atropine and glycopyrrolate are both anticholinergics that are used perioperatively for several purposes. As their name implies, they are used to counteract harmful cholinergic responses that can occur during paralytic reversal with anticholinesterase inhibitors, particularly bradycardia and parasympathetic side effects. Both agents are also antisialogues, and they are often used to facilitate intubating conditions. Anticholinesterase inhibitors (see above) increase the amount of acetylcholine available in the body. This excess of acetylcholine can act on the heart to cause severe bradycardia. If atropine or glycopyrrolate is administered along with the anticholinesterase, bradycardia can be tempered or avoided. Neostigmine and glycopyrrolate (slower onset, longer acting) are used in concert for neuromuscular blockade reversal, while edrophonium is paired with atropine (quicker onset, shorter acting). This specific pairing is due to the comparable durations of action of the combinations, as outlined in Table 4.7.

A central anticholinergic syndrome may result from an overdose of atropine (which, unlike glycopyrrolate, crosses the blood brain barrier). Symptoms include delirium, excitation, fever, flushing, and tachycardia. Treatment is with physostigmine (a centrally acting acetylcholinesterase inhibitor) which acts to restore blocked cholinergic activity in the CNS.

<table>
<thead>
<tr>
<th>Table 4.7 Reversal of neuromuscular blockade.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neostigmine 50 mcg/kg paired with Neostigmine 10 mcg/kg</td>
</tr>
<tr>
<td>Edrophonium 500 mcg/kg paired with Atropine 20 mcg/kg</td>
</tr>
</tbody>
</table>

**Case Study**

You are asked to provide general anesthesia for an otherwise healthy 30-year-old woman undergoing pelviscopy. She has a history of endometriosis and chronic pelvic pain. Her brother had a near-fatal episode of malignant hyperthermia as a child, and she has been counseled to avoid triggering anesthetics. You decide to manage the case with total intravenous anesthesia, avoiding inhalation anesthetics altogether. You have appropriately removed the vaporizers from your anesthesia machine and flushed it with 100% oxygen according to published recommendations.
Which classes of intravenous agents will you need?
As with any anesthetic, you need to provide all three components of complete anesthesia: hypnosis, analgesia, and muscle relaxation. No one intravenous anesthetic can provide all three, as can inhalation anesthetics in high enough doses. As you do with balanced anesthesia, you will likely use a combination of drugs that provide primarily one of the three components. You will need a sedative-hypnotic, an opioid analgesic, and a neuromuscular blocking drug.

Which drug will use to produce and maintain unconsciousness? How will you know you have given enough? Will the dose need change during the surgery?
The most commonly used drug in this class for TIVA is propofol, due to its short acting properties and relatively rapid administration after even prolonged administration. Unfortunately, unlike inhalation anesthetics, there is no equivalent of end-tidal concentration to directly monitor effect site concentration. Mathematical models have been developed, however, which closely model this concentration and can be used to control infusion pumps or guide a human operator. In Europe, but not yet in the United States, target controlled infusion pumps exist and can be programmed directly in terms of the desired brain concentration of propofol. When using a manual pump, the dose will indeed be reduced over time in order to maintain such a constant effect site concentration.

Which opioid would be most appropriate for intraoperative use? The case is booked for 2 h. Will you change to a different agent for postoperative analgesia?
As shown in Fig. 4.1, opioids differ markedly in their context-sensitive half times (CSHT; the time required for a 50% decrease in plasma concentration after discontinuing a constant-dose infusion). Therefore, if not using a computerized pump that holds a constant effect site concentration by decreasing the infusion rate over time, it would be most appropriate to select a drug with a relatively flat CSHT curve. This would include sufentanil, alfentanil, or best remifentanil. The latter, though expensive, is often favored for TIVA because even very high doses (requiring even more than a 50% decrease in concentration at the
end of the case) are rapidly eliminated after discontinuation. At the end of the case, you should consider a longer acting drug such as fentanyl, morphine, or hydromorphone to provide postoperative analgesia. The choice may depend on whether the patient will be staying overnight in the hospital (favoring longer acting drugs) or having day surgery (favoring fentanyl).

**Which neuromuscular blocking drug(s) will you choose, if any?**
You will avoid succinylcholine because it is a trigger for malignant hyperthermia. In general, you will intubate and control ventilation in patients undergoing pelviscopy. Therefore, you will use a short-acting and rapid-onset nondepolarizing neuromuscular blocking drug such as vecuronium, rocuronium, or cisatracurium. Given the duration of the case (2 h), any would be a reasonable choice. For shorter cases, rocuronium is somewhat shorter acting, though more expensive, than the other choices.

**At the end of the case, how will you conduct the emergence?**
This can be the greatest challenge of a TIVA. Because you cannot monitor the concentration of the drugs in the patient’s body, and because there is no well-characterized equivalent of MAC, you must have an understanding of the pharmacokinetics of the drugs in order to allow the patient to awaken promptly at the end of the surgery. You will reverse neuromuscular blockade and discontinue the opioid infusion. If you are using remifentanil, you will consider a small dose of a longer acting drug to provide early postoperative analgesia. Propofol elimination is rapid but not instantaneous; the CSHT is 11 min for a 1 h infusion plus 4 min per additional hour for propofol, so you will have to carefully monitor the procedure and discontinue it at the appropriate time. Moreover, a 50% decrease in concentration may or may not be sufficient for the patient to awaken, so more or less time may be required. You can monitor the depth of anesthesia with clinical signs (BP and heart rate, signs of sympathetic activation such as tearing or grimace) and with a consciousness monitor such as BIS. You may also decrease the rate of infusion somewhat as surgical stimulation decreases during surgical closure to facilitate emergence once the infusion is halted.
Suggested Further Reading


Pharmacology of Inhalational Anesthetics

Jerome M. Adams and John W. Wolfe

For maximum impact, it is recommended that the case study and questions found on page xix are reviewed before reading this chapter.

Key Learning Objectives

- Learn the pharmacokinetic factors affecting the rate of induction and emergence with inhalational anesthetics
- Understand the concept of Minimum Alveolar Concentration (MAC)
- Know the key characteristics of the four most commonly used inhalational agents (nitrous oxide, isoflurane, desflurane, sevoflurane)

The inhalational anesthetics (nitrous oxide and various volatile halogenated ethers) play a key role in current anesthetic practice. They provide rapid induction of anesthesia, rapid titratability during the anesthetic, and rapid emergence at the conclusion of the anesthetic. At clinically relevant doses, the volatile anesthetics provide reliable amnesia, immobility, a modest degree of muscle relaxation, and blunting of the adrenergic response to surgical stimulation.

Pharmacokinetics of Uptake, Distribution and Elimination

Induction: In order to have an effect on the patient, inhalational anesthetics must be:

1. Inspired after having been delivered from the breathing circuit
2. Absorbed from the alveoli into the blood
3. Transported from the lungs to the target tissue
4. Absorbed from the blood into the target tissue (i.e. the brain)
Emergence: The sequence of events from the induction of anesthesia is reversed (i.e. the agent is absorbed from the target tissue into the blood, transported to the lungs, and then expired into the breathing circuit).

A useful analogy for induction and emergence from inhalational anesthetics is to imagine that a reservoir is being filled during induction and emptied during emergence. When the reservoir is empty, the partial pressure of the anesthetic in target tissues is zero, and the patient is awake. When the reservoir is full of drug, the partial pressure of the anesthetic in target tissues is therapeutic, and the patient is anesthetized.

Factors affecting the rapidity of induction and emergence include:

- **Tissue and blood solubility**: Agents that are more soluble in blood and tissues effectively have a larger reservoir that must be filled before adequate tissue partial pressures are reached to achieve an anesthetic effect. On emergence, the more soluble agents have a larger reservoir of drug that must be emptied. By the same mechanism that induction is slowed (owing to the larger reservoir that has to be filled), emergence with the more soluble agents typically takes longer. For example, all other factors being equal, induction and emergence with isoflurane is slower than with desflurane. (See Table 5.1, Physical Characteristics of Inhalational Agents)

- **Inspired concentration**: A high inspired concentration of the anesthetic speeds induction by providing a large gradient between the partial pressure of the agent in the alveoli and the blood. This concentration gradient increases the arterial concentration of the agent, thereby speeding induction of the anesthetic effect. The reverse of this phenomenon is seen on emergence,

### Table 5.1 Physical characteristics of inhalational anesthetics.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Vapor pressure (20°C)</th>
<th>Blood: gas partition coefficient</th>
<th>Fat: blood partition coefficient</th>
<th>Metabolism</th>
<th>Pungency&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>N₂O</td>
<td>38,770 mmHg</td>
<td>0.46</td>
<td>2.3</td>
<td>0</td>
<td>None</td>
</tr>
<tr>
<td>Desflurane</td>
<td>669 mmHg</td>
<td>0.42</td>
<td>27</td>
<td>0.02%</td>
<td>High</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>157 mmHg</td>
<td>0.65</td>
<td>48</td>
<td>5%</td>
<td>Low</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>238 mmHg</td>
<td>1.46</td>
<td>45</td>
<td>0.2%</td>
<td>High</td>
</tr>
</tbody>
</table>

<sup>a</sup>The low blood:gas partition coefficients (i.e. low solubility in blood) of nitrous oxide, desflurane, and sevoflurane speed induction and emergence.

<sup>b</sup>Due to their low pungencies, nitrous oxide and sevoflurane are excellent agents for inhalational induction of anesthesia by mask.
when an inspired concentration of zero favors passage of volatile agent out of the blood into the alveoli. (see Fig. 5.1)

- **Fresh gas flow rate:** A higher fresh gas flow rate into the anesthesia machine circuit speeds induction. By more completely and rapidly replacing expired gases (which contain less anesthetic agent), a consistently high inspired concentration is provided. Similarly, a high inflow rate of anesthetic-free fresh gas during emergence quickly flushes the anesthetic agent out of the circuit, enhancing the elimination of inhaled anesthetic from the lungs.

- **Minute ventilation:** High minute ventilation (respiratory rate x tidal volume) increases the rate of induction and emergence by rapidly providing fresh inhalational agent during induction and rapidly removing it during emergence. This is clinically relevant during inhalational inductions and during the emergence of most patients. For example, a patient with high minute ventilation (i.e. an infant) will have a faster inhalational induction and emergence than a patient
with lower minute ventilation (e.g., an elderly patient). Ventilation has a greater effect on high-solubility agents (such as diethyl ether) and a lesser effect on relatively insoluble agents (such as nitrous oxide). Since most of our commonly used inhalational agents have low to intermediate solubilities, this effect is therefore of a moderate significance.

Theories of Inhalational Anesthetic Action

The mechanism of action of the inhalational anesthetics remains incompletely understood. Anesthetic effects have been demonstrated at the levels of the spinal cord, brain stem, and cerebral cortex.

Theories explaining the mechanism of action of inhalational anesthetics include:

- **The Meyer-Overton Rule**: It has been observed that the potencies of inhalational agents correlate with their lipid solubilities. Extrapolating from this observation, it has been theorized that inhalational anesthetics act by dissolving at hydrophobic sites, formerly assumed to be in the lipid bilayers of cell membranes, but currently thought to be in the relatively hydrophobic regions of one or more proteins.

- **GABA enhancement**: Many inhaled anesthetic agents enhance activity of the gamma-aminobutyric acid (GABA) system, which is also enhanced by intravenous anesthetic agents such as benzodiazepines, propofol, and etomidate. It has been observed that the potencies of inhalational agents correlate with their potentiation of the GABA system, leading to the theory that GABA enhancement may be a key element of inhalational anesthetic activity.

- **Other receptors systems**: Inhalational anesthetic agents have been shown to interact to varying degrees with a wide variety of cellular receptors, including NMDA and acetylcholine receptors.

Depth of Anesthesia and MAC

The minimum alveolar concentration (MAC) is a commonly used method for describing the dose of inhalational anesthetics. MAC, as used by anesthesiologists, is a specialized example of an ED$_{50}$, where a MAC of 1 is the alveolar concentration of a drug at which movement in response to a surgical incision will be absent in 50% of subjects. By referring to the MAC of a volatile agent being delivered, one can normalize the different potencies of the various agents when comparing them. In addition, MAC values for inhalational agents are additive (a patient receiving 0.5 MAC of one agent and 0.4 MAC of another has a total anesthetic dose of 0.9
Pharmacology of Inhalational Anesthetics

Multiples of the MAC for inhalational anesthetics can be used to describe differing depths of anesthesia, although MAC multiples are not linear because the dose–response curves for different agents do not parallel. Nevertheless, some useful dose levels are:

- 0.3–0.4 MAC is associated with awakening from anesthesia in the absence of other agents (referred to as MAC-awake).
- 1.3 MAC is known to prevent movement in 95% of patients in response to a surgical incision (making 1.3 MAC an inhalational anesthetic analog to an ED95 dose used for intravenous agents).
- 1.5 MAC typically blocks the adrenergic response to the surgical stimulus.

Please note that the MAC values cited above for inhalational anesthetics are values for normal adults. Table 5.2 lists MAC values for commonly used inhalational agents.

The MAC value for an inhalational anesthetic may be increased or decreased in individual patients by a variety of factors, as outlined in Table 5.3:

### Nitrous Oxide

Nitrous oxide is a colorless, nonpungent gas with a slightly sweet odor and taste. It is the only inorganic chemical in current use as an anesthetic. The vapor pressure of nitrous oxide at room temperature is 745 PSI. Therefore, it exists as a gas at atmospheric pressure, because its critical temperature (the temperature below which a gas cannot be liquefied, no matter how high the applied pressure) is in the range of ambient operating room temperatures. It is stored as a compressed liquid. Note that due to its low potency (MAC = 105%).

<table>
<thead>
<tr>
<th>Table 5.2 Minimum alveolar concentration (MAC) values.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Agent</strong></td>
</tr>
<tr>
<td>Desflurane</td>
</tr>
<tr>
<td>Sevoflurane</td>
</tr>
<tr>
<td>Isoflurane</td>
</tr>
<tr>
<td>Halothane</td>
</tr>
<tr>
<td>Nitrous Oxide</td>
</tr>
</tbody>
</table>
An administration of 1 MAC of nitrous oxide at atmospheric pressure is not possible – as this would lead to asphyxia from a lack of oxygen. In practice, the highest MAC of nitrous oxide that can be delivered on most anesthesia machines is 0.67 (corresponding to an inspired concentration of 70%).

- **Cardiovascular effects:** Nitrous oxide depresses myocardial contractility, but this effect is usually offset by its stimulation of the sympathetic nervous system. Blood pressure and heart rate are generally unchanged by administration of nitrous oxide in the absence of surgical stimulation.

- **Respiratory effects:** Nitrous oxide causes an increase in respiratory rate and a decrease in tidal volume. These effects are balanced, so that minute ventilation is minimally changed. Hypoxic ventilatory drive is markedly diminished, so that patients may remain apneic despite marked hypoxemia. Nitrous oxide may increase pulmonary vascular resistance and is generally avoided in patients with pulmonary hypertension.

- **Cerebral effects:** Nitrous oxide increases cerebral blood flow, blood volume, and oxygen consumption. Intracranial pressure is mildly increased.

- **Diffusion into gas filled spaces:** Nitrous oxide can diffuse from the patient’s blood into gas-filled spaces within the patient (bowel gas, pneumothorax, etc.) more rapidly than other gases (e.g. nitrogen) can diffuse out. This is because nitrous oxide is 20 times more soluble in blood than nitrogen. This diffusion continues until the partial pressure of nitrous oxide in the space equals that in the blood. The accumulation of nitrous oxide can lead to expansion of the gas-filled space, causing distention of the bowel or expansion of a pneumothorax.

- **Methionine synthetase inhibition:** Nitrous oxide oxidizes the cobalt atom in vitamin B12, inactivating vitamin B12-dependent enzymes, such as

---

### Table 5.3 Factors affecting MAC.

<table>
<thead>
<tr>
<th>Increased MAC</th>
<th>Decreased MAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children (from infancy to adolescence)</td>
<td>Premature infants and the elderly</td>
</tr>
<tr>
<td>Hypernatremia</td>
<td>Hypothermia</td>
</tr>
<tr>
<td>Cocaine or amphetamine intoxication</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>Chronic alcohol use</td>
<td>Acute alcohol intoxication</td>
</tr>
<tr>
<td>MAO inhibitors</td>
<td>Opiates, benzodiazepines, barbiturates, clonidine, dexametomidine</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td></td>
</tr>
</tbody>
</table>

---
methionine synthetase. Prolonged exposure to nitrous oxide causes bone marrow depression and neural toxicity similar to that seen with vitamin B12 deficiency. It is unknown whether short perioperative exposures cause clinically important sequelae by this mechanism.

- **Teratogenicity:** Nitrous oxide has been implicated as a possible teratogen in animal studies and is usually avoided in pregnant patients.
- **Nausea and vomiting:** Nitrous oxide has been implicated as a possible cause of postoperative nausea and vomiting. This effect is thought to be less prevalent in children.
- **Diffusion hypoxia:** At the conclusion of an anesthetic, when nitrous oxide is discontinued, it will diffuse out of the blood into alveoli in large volumes over a period of 2–3 min. Due to the fact that nitrous oxide is usually administered at concentrations around 50% inspired, appreciable quantities of the gas can dissolve in body tissue, often as much as 12–14 L over a long case. At the beginning of emergence, if the patient is allowed to breathe room air, large quantities of nitrous oxide diffuse out through the alveoli, significantly reducing the alveolar PO$_2$ by a dilutional effect. This can lead to a phenomenon known as diffusion hypoxia, but it may be prevented by administering 100% O$_2$ for several minutes at the beginning of the emergence phase as nitrous oxide is discontinued.

**Concentration Effect**

The concentration effect explains why higher inspired anesthetic agent concentrations lead to faster rises in arterial concentrations. Because the volume of gas entering the pulmonary capillaries is higher than the amount of nitrogen entering the alveolus, the result is a decrease in alveolar volume. This decrease in alveolar volume leads to a higher fractional concentration of anesthetic agent, somewhat analogous to the creation of a vacuum within the alveolus whereby additional agent enters rapidly in response. This effect is most significant with nitrous oxide, as it is the least soluble gas.

**Second Gas Effect**

The second gas effect is an extension of the concentration effect and a theoretical concept which may occur when nitrous oxide is combined with an inhalational agent (e.g. isoflurane). Just as we see with the concentration effect, despite its relatively low solubility, large volumes of nitrous oxide may be rapidly absorbed into arterial blood during induction, creating a vacuum of sorts
within the alveoli. This, in turn, leads to an increase in the uptake of the second agent, which enters the alveoli more readily in response to the partial vacuum created by rapid absorption of nitrous oxide.

**Volatile Anesthetics (Isoflurane, Sevoflurane, and Desflurane)**

The volatile anesthetic agents used in current anesthetic practice share many similar characteristics and side effects:

- **Cardiovascular effects:** The volatile anesthetics depress myocardial contractility and cause peripheral vasodilation (the various agents differ somewhat in the balance of these two effects). The effect on heart rate is variable. Arterial blood pressure is decreased in a dose-dependent fashion.

- **Respiratory effects:** Tidal volume is decreased by the volatile anesthetics. Respiratory rate increases slightly or remains stable, leading to decreased minute ventilation. The responses to hypercapnia are blunted (i.e. an anesthetized patient will increase minute ventilation in response to hypercapnia less than an awake patient and will remain apneic at a higher PCO$_2$ than an awake patient). As with nitrous oxide, hypoxic ventilatory drive is markedly diminished. Volatile anesthetics also produce bronchodilation.

- **Cerebral effects:** The volatile anesthetics reduce cerebral oxygen consumption. At doses above 1 MAC, cerebral blood flow and consequently intracranial pressure are increased. Hyperventilation reverses the cerebral vasodilation seen with these agents.

- **Musculoskeletal effects:** The effects of neuromuscular blockers are potentiated by volatile anesthetics.

- **Obstetric effects:** The volatile anesthetics produce a dose-dependent reduction in uterine smooth muscle contractility.

- **Renal and hepatic blood flow:** All agents decrease renal blood flow, glomerular filtration rate, and urinary output. They also decrease hepatic blood flow.

- **Nausea and vomiting:** The volatile anesthetic agents are known to cause postoperative nausea and vomiting.

- **Malignant hyperthermia:** The volatile anesthetic agents are triggers for malignant hyperthermia *(note: nitrous oxide is not a triggering agent).*

- **Cardiac preconditioning:** Exposure of cardiac tissue to volatile anesthetics may be protective against the effects of subsequent ischemia and reperfusion.
Specific characteristics of the volatile anesthetics include:

**Isoflurane**
- *Hepatic effects:* Although total hepatic blood flow is reduced during isoflurane anesthesia, isoflurane may preserve hepatic blood flow to a greater degree than the other inhalational anesthetics.

**Sevoflurane**
- *Fluoride:* Sevoflurane is metabolized at an overall rate of 5%, which is much higher than the metabolism rates of isoflurane (0.2%) or desflurane (0.02%). Inorganic fluoride is an end-product of sevoflurane metabolism. No association has been demonstrated between this fluoride production and postanesthetic renal dysfunction (such an association was previously made with the volatile anesthetic methoxyflurane, which is also metabolized to inorganic fluoride).
- *Compound A:* Sevoflurane can degrade in the presence of soda lime to produce a known nephrotoxin called Compound A. Higher levels of Compound A are associated with high respiratory gas temperature, low-flow anesthetic techniques, high sevoflurane concentrations, and sevoflurane anesthetics of long duration. Due to concern about Compound A production, the package insert for sevoflurane recommends that fresh gas flows be maintained at least 1 L/min. Some anesthesiologists avoid sevoflurane in patients with known renal impairment.

**Desflurane**
- *Cardiovascular effects:* High concentrations and rapid increases in the concentration of desflurane can cause a transient period of sympathetic activation, with tachycardia and hypertension.
- *Vapor Pressure:* Desflurane’s high vapor pressure (669 mmHg at 20°C) is close to atmospheric pressure, so it almost boils at room temperature. As a result, the desflurane vaporizer is constructed differently than the vaporizers for isoflurane and sevoflurane. The desflurane vaporizer heats and pressurizes the anesthetic gas, then delivers a fractional concentration into the fresh gas flow.

**Case Study**
You are asked to induce anesthesia for an ENT procedure, in which the surgeon wishes to inspect the airway during spontaneous respiration without the presence of an endotracheal tube or laryngeal mask airway.
The patient is otherwise healthy and has a normal appearing airway, and you judge that maintaining the airway by mask will be successful. You agree to induce anesthesia by inhalation. The patient has an IV and standard monitors are in place.

Which inhalation agent will you choose?
The ideal agent would have several properties. It would be relatively potent, so that a high multiple of the minimum alveolar concentration (MAC) could be delivered by the vaporizer during induction. It would have low solubility, so that the “tank” needed to be filled before the brain concentration reaches that needed for anesthesia would be small. Importantly for inhalation induction in an awake patient, it would be pleasant smelling and would not irritate the airway. Of the available drugs in clinical practice today, halothane, nitrous oxide, and sevoflurane are not pungent, and are therefore potentially suitable for such “mask” induction. Nitrous oxide is not potent and indeed at 1 atmosphere the MAC exceeds 100%, meaning it is not possible to fully anesthetize a patient with nitrous oxide alone. It is, however, insoluble and thus has a rapid uptake into the brain. Halothane is potent but much more soluble than the other agents, making inhalation induction slow. Sevoflurane is relatively potent (a commercial vaporizer can deliver approximately 4 MAC inhaled agent) and is of low solubility, making it the preferred choice for most anesthesiologists.

Would a combination of more than one inhaled agent offer any advantage? Theoretically, adding nitrous oxide should help speed the induction with sevoflurane. This is because of the two-part “second gas effect.” First, the rapid uptake of nitrous oxide from the alveoli will concentrate sevoflurane there, effectively increasing the inhaled concentration. Second, this same uptake will entrain more gas from the trachea (which contains sevoflurane in the case of inhalation induction), effectively increasing alveolar flow of this “second” gas. These physiologic effects have been conclusively demonstrated in research studies. However, in practice, their effect on clinical induction is minimal. Indeed, randomized trials comparing inhalation induction with sevoflurane in oxygen vs. in N₂O plus oxygen have demonstrated no difference in the rate of induction.
What are the factors you can control which will speed induction of anesthesia? After picking a low solubility agent like sevoflurane, you can also speed induction by increasing the inspired concentration and fresh gas flow. The former causes the gradient across the pulmonary capillary (from the alveolus to the pulmonary vein) to be higher, increasing the amount of drug crossing into the bloodstream. The latter ensures that expired gas, which will contain very little sevoflurane at the beginning of the anesthetic uptake, will not dilute the inspired gas. Although you cannot directly control it during spontaneous respiration, you can ask the patient to breathe deeply, increasing minute ventilation and increasing transfer of drug from the lung to the pulmonary venous blood. With sevoflurane, these factors can be combined to achieve single-breath induction: the patient breathes out to residual volume and then takes a vital capacity breath of high concentration of sevoflurane (6–8%, with very high fresh gas flow set on the machine). The patient holds the breath as long as possible, increasing uptake into the blood from the high alveolar concentration. Many patients will lose consciousness with this first breath, but will also resume spontaneous respiration shortly thereafter.

You have an end-tidal gas monitor to measure exhaled agent. How will you know when you have the patient deeply anesthetized enough to allow the surgeon to perform laryngoscopy? Once you have achieved induction of anesthesia and the patient is unconscious, you will continue to have the patient breathe sevoflurane at relatively high inspired concentration as the brain completely equilibrates with the alveolar concentration. While the induction is taking place, these two concentrations are not the same (the brain lags about 2 min behind the alveolus). At equilibrium, the alveolar concentration, as estimated by the end tidal concentration, should reflect the vessel-rich group concentration, which includes the brain and spinal cord. These are the structures that need to be anesthetized for the surgery to begin. The concentration should be somewhat higher than 1 MAC, which is the concentration at which 50% of patients will move in response to surgical stimulation. At 1.3 MAC, 95% will not move. For sevoflurane, with an MAC of 1.7–2%, this means you should strive for an end-tidal concentration of about 2.2–2.6%. Since the goal in this case is to maintain spontaneous
respiration, you will likely not add opioids or neuromuscular blocking drugs to enhance anesthesia. However, since anesthetic delivery will be interrupted during the surgeon's examination of the airway, you will need to have intravenous agents ready should the patient react, and you and the surgeon will have to maintain close communication during this part of the procedure. In some cases, it is possible to use jet ventilation (directing a high pressure jet of gas from the laryngoscope down the airway) with oxygen and sevoflurane.

**Suggested Further Reading**


Chapter 6

Pharmacology of Local Anesthetics

John W. Wolfe and Jerome M. Adams

For maximum impact, it is recommended that the case study and questions found on page xix are reviewed before reading this chapter.

Key Learning Objectives
- Understand the basic mechanisms of local anesthetic action and metabolism
- Appreciate the differences in the properties among commonly used local anesthetics
- Learn the signs of local anesthetic toxicity and its treatment

History of Local Anesthetics
Cocaine was the first local anesthetic to be discovered after isolation from coca leaves by Albert Niemann in the 1860s. Cocaine was first used clinically in 1884 by Sigmund Freud, who used it to wean a patient from morphine addiction. Freud and Karl Kollar also noticed the anesthetic effects of cocaine, and Kollar later described its utility as a topical ocular anesthetic. Later in 1884, William Halsted published a description of the injection of cocaine into a sensory nerve to provide surgical anesthesia.

Local Anesthetic Mechanism of Action
The cell membrane of a nerve axon contains sodium and potassium channels that control the flow of ions between the extracellular fluid and the interior of the cell. Local anesthetics exert their effects by inhibition of sodium channels.
When nerve cells are at rest, these sodium channels are in a resting, nonconducting state, and the cell has a resting membrane potential of about $-70 \text{ mV}$. During membrane depolarization, the sodium channels open briefly, allowing sodium ions to flow into the cell and the transmembrane potential to rise to $+35 \text{ mV}$. After a depolarization, the sodium channels get rapidly inactivated and the resting membrane potential is reestablished. This series of events is collectively referred to as the action potential.

Local anesthetics preferentially bind to sodium channels in the open or inactivated state and prevent ion conduction. When local anesthetic molecules have bound to a sufficient number of sodium channels, the membrane is unable to depolarize sufficiently to reach the threshold potential, and generation of an action potential is prevented.

Factors Affecting Local Anesthetic Action

- **Fiber size and type:** Peripheral nerves contain myelinated A and B fibers and unmyelinated C fibers, as outlined in Table 6.1. In general, smaller nerve fibers of the same type are more readily blocked than larger fibers, yet the smaller unmyelinated fibers are less easily blocked than the larger myelinated ones. The “size principle” leads to the commonly observed phenomenon of **differential conduction blockade**, in which sympathetic fibers are more easily blocked than pain and temperature fibers, which are more easily blocked than motor, pressure, and proprioceptive fibers. Clinically, this phenomenon is seen in patients who may have incomplete blockade of motor fibers and pressure sensations despite sympathectomy and blockade of pain sensations. There is considerable overlap of local anesthetic sensitivity among nerve fiber types.

- **pH:** Most local anesthetics are weak bases that exist as an equilibrium of a more lipid soluble, neutral form and a less lipid soluble, charged form. The local anesthetics typically have $pK_a$’s greater than 7.4, so less than 50% of the drug exists in the lipid soluble form in normal extracellular fluid. Additionally, commercial preparations of local anesthetics typically have

<table>
<thead>
<tr>
<th>Fiber type</th>
<th>Local anesthetic sensitivity</th>
<th>Size</th>
<th>Myelination</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>+</td>
<td>Large</td>
<td>Yes</td>
</tr>
<tr>
<td>B</td>
<td>++</td>
<td>Medium</td>
<td>Yes</td>
</tr>
<tr>
<td>C</td>
<td>+++</td>
<td>Small</td>
<td>No</td>
</tr>
</tbody>
</table>
pH’s between 6 and 7, further increasing the proportion of the drug in the protonated form. The action of local anesthetics requires that their molecules permeate lipid-rich neural membranes to reach their site of action. Clinical implications of these factors are:

- Addition of sodium bicarbonate to the local anesthetic solution (typically 1 ml of sodium bicarbonate solution to 10 ml of local anesthetic) increases pH and the fraction of local anesthetic in neutral form, speeding onset of action.
- Tissues with local acidosis (e.g., infected or ischemic tissues) will be relatively resistant to local anesthetic action.

- **Use-dependent blockade:** Access to sodium channels is enhanced by repeated membrane depolarization because depolarization increases the time that the channels spend in the open or inactivated forms. Frequent action potentials in the presence of local anesthetic speeds onset of neural blockade.

- **Epinephrine:** Epinephrine affects local anesthetic action in two ways:
  - Epinephrine-containing local anesthetic solutions are formulated at lower pH’s (4–5) than plain local solutions because of epinephrine’s instability in alkaline environments. Low pH slows onset of local anesthetic action as described above.
  - Epinephrine causes local vasoconstriction and slows absorption of the local anesthetic from its site of deposition, prolonging local anesthetic action. This effect is prominent with the shorter-acting local anesthetics (e.g., lidocaine blockade can be increased 50% by addition of epinephrine). The longer-acting local anesthetics (bupivacaine and ropivacaine) are released so slowly from neural tissue that epinephrine does not significantly increase their durations of blockade, but does decrease their peak blood concentrations after injection.

## Local Anesthetic Metabolism

The action of local anesthetics is terminated by absorption of the drug from the site of action into the circulation. Following absorption, the drug is metabolized and excreted.

Local anesthetics fall into two structural categories, **amides** and **esters**. A schematic representation of local anesthetic structure is shown in Fig. 6.1.

- **Amides** are metabolized by microsomal enzymes (cytochromes) in the liver
- **Esters** are primarily metabolized by pseudocholinesterase in the plasma (the exception is cocaine, which is partially metabolized by the liver and partially excreted unchanged by the kidneys)
Peak blood levels of local anesthetics are related to the dose administered and the rate of absorption of the drug from its site of action. Injection into a highly vascular area leads to higher blood levels of the drug than placing a similar amount of drug into a less vascular area. The rank order of peak blood concentrations of local anesthetic after administration of the same dose of drug at different sites is shown below:

<table>
<thead>
<tr>
<th>Agent</th>
<th>Onset of action</th>
<th>$pK_a$ (36°C)</th>
<th>Max dose (mg/kg)*</th>
<th>Duration of action (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amides</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Rapid</td>
<td>7.8</td>
<td>4.5 (7 with epi)</td>
<td>1–2</td>
</tr>
<tr>
<td>Mepivacaine</td>
<td>Moderate</td>
<td>7.7</td>
<td>5 (7 with epi)</td>
<td>1.5–3</td>
</tr>
<tr>
<td>Prilocaine</td>
<td>Slow</td>
<td>8.0</td>
<td>6 (9 with epi)</td>
<td>1–2</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>Slow</td>
<td>8.1</td>
<td>2.5 (3 with epi)</td>
<td>4–8</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>Slow</td>
<td>8.1</td>
<td>2.5 (3 with epi)</td>
<td>4–8</td>
</tr>
<tr>
<td>Esters</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-Chloroprocaine</td>
<td>Very Rapid</td>
<td>9.1</td>
<td>9 (15 with epi)</td>
<td>0.5–1</td>
</tr>
<tr>
<td>Procaaine</td>
<td>Rapid</td>
<td>8.9</td>
<td>7 (10 with epi)</td>
<td>0.75–1</td>
</tr>
<tr>
<td>Tetracaine</td>
<td>Slow</td>
<td>8.4</td>
<td>1.5 (2.5 with epi)</td>
<td>3</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Rapid</td>
<td>8.7</td>
<td>1.5</td>
<td>0.5</td>
</tr>
</tbody>
</table>

*Maximum dose for a single subcutaneous injection.

Epinephrine and other vasoconstrictors slow the rate of absorption. Strongly protein-bound local anesthetics (e.g. bupivacaine and ropivacaine) tend to be more lipid soluble, more potent, and have longer times to onset, longer durations of action, and slower absorption from neural tissue (Table 6.2).
Uses of Local Anesthetics in Anesthesia Practice

In addition to its use by the surgeon to infiltrate the incision site, local anesthetics are used by anesthesiologists in a variety of settings. For example, local anesthetics are used for IV placement, epidural placement, spinal or other regional block placement, pain management practice (patch), or lido for propofol pain.

Local Anesthetic Side Effects and Toxicity

- **Central nervous system effects:** Dysfunction of the central nervous system is often the first sign of local anesthetic toxicity. Signs and symptoms of local anesthetic toxicity tend to follow a stereotypical sequence. Early symptoms may include lightheadedness, perioral or tongue numbness, or a metallic taste. Higher levels may lead to tinnitus, visual dysfunction, agitation, and anxiety. Central nervous system depression can follow, with unconsciousness, respiratory arrest, and seizure activity. Local anesthetic-induced seizures can be treated with hyperventilation, benzodiazepines, or small doses of thiopental or propofol.

- **Cardiovascular effects:** If blood concentrations rise high enough, the local anesthetics can bind to sodium channels present on myocardial cells. This reduces myocardial automaticity and shortens the refractory period. Cardiac arrhythmias, depressed contractility, and cardiac arrest can ensue. In general, the high-potency agents such as bupivacaine and ropivacaine have greater cardiotoxicity than the lower-potency agents. Successful resuscitation of a patient with local anesthetic-induced cardiotoxicity can require prolonged efforts and may prove to be difficult (or impossible). Of note, cardiotoxic effects of bupivacaine and ropivacaine have been observed to occur without promontory central nervous system effects.

- **Neurotoxicity:**
  - **Lidocaine:** Permanent neurologic injury (cauda equina syndrome) has been associated with infusion of 5% lidocaine through spinal microcatheters. It has rarely been observed after single-dose spinal injections. It is thought that pooling of this concentrated local anesthetic solution around nerve fibers may cause neurotoxic effects.
  - **2-Chloroprocaine:** 2-chloroprocaine was used for spinal anesthesia in the 1950s, and is still commonly used for epidural anesthesia (particularly in obstetrics). In the early 1980s, multiple cases of neurological injury were associated with accidental intrathecal injections of large doses of chloroprocaine. Investigations showed that a likely cause of injury was
the low pH and metabisulfite preservative in the solutions used. Plain, preservative-free 2-chloroprocaine in appropriate intrathecal doses appears to be no more neurotoxic than other commonly used spinal anesthetic solutions, and it may carry a reduced risk of TNS (see below).

- **Transient neurologic symptoms (TNS):** Patients receiving spinal anesthesia may have transient hypesthesias, paresthesias, and motor weakness in the legs or buttocks. TNS is significantly more common with lidocaine than with bupivacaine or tetracaine (and likely 2-chloroprocaine). TNS symptoms typically resolve within 3 days, but occasionally may persist for as long as 6 months.

- **Methemoglobinemia:** Larger doses of prilocaine and benzocaine (a common ingredient in local anesthetic sprays) can convert hemoglobin to methemoglobin. Infusion of 1–2 mg/kg of methylene blue reverses this reaction.

- **Hypersensitivity/Allergy:** While an adverse reaction to a local anesthetic is not uncommon, a true allergy is exceedingly rare. Allergic reactions are most often associated with esters because of sensitivity to their metabolite, para-aminobenzoic acid (PABA). Should this occur, consider switching to an amide anesthetic.

### Treatment of Local Anesthetic Toxicity

Infusion of 20% lipid emulsion solution (such as Intralipid) has been reported to be effective in reversing the symptoms of local anesthetic toxicity. The presumed mechanism of action is that the lipid-soluble fraction of the local anesthetic is sequestered in the lipid emulsion and effectively removed from the plasma. Although this treatment is still being investigated, the following treatment protocol has been proposed (see www.lipidrescue.org):

- **Bolus** 1.5 mL/kg of 20% lipid emulsion, then run 0.25 mL/kg/min for 30–60 min.
- **Repeat** the bolus dose for persistent asystole.
- **Increase** the infusion rate for hypotension.

### Case Study

A 70 kg otherwise healthy male patient is undergoing bilateral inguinal herniorrhaphy under local anesthesia administered by the surgeon and intravenous sedation you are giving. The surgeon is planning to infiltrate the skin with lidocaine prior to skin incision.
The patient reports a history of an “allergic reaction” to Novocain (procaine) which she received during a dental procedure. Is it safe to administer the planned local anesthetics?

Most dental reactions are not true allergies, but either unpleasant sensations from the intended local anesthetic effect (numb tongue and lips that feel swollen), or tachycardia from absorbed epinephrine. Even if the patient were truly allergic to procaine, it is exceedingly unlikely that he would also be allergic to lidocaine or bupivacaine, which are amide type local anesthetics, whereas procaine is an ester type drug.

The surgeon is planning to use 2% lidocaine with epinephrine for initial infiltration, followed by bupivacaine, 0.5% for longer lasting analgesia. How can she enhance the onset of the block?

Lidocaine with epinephrine is prepared with very low pH in order to stabilize the local anesthetic and the epinephrine, which is unstable at neutral or basic pH. At pH 4–5, that of commercial epinephrine containing local anesthetic solutions, only a tiny fraction will be in the uncharged, base form, which can permeate nerve cell membranes. The addition of bicarbonate, 1 mL per 10 mL of local anesthetic solution, will raise the pH and the unionized fraction, speeding the onset. This treatment also significantly reduces the pain of injection, an additional benefit.

After infiltration with lidocaine, the surgeon is prepared to infiltrate further with bupivacaine and perform some deep nerve blocks to enhance analgesia. She asks you how much of a 0.5% solution she can safely use. How will you respond?

The limit for a single subcutaneous infiltration is approximately 2.5 mg/kg. A 0.5% solution of bupivacaine contains 5 mg/mL, so the surgeon can use 175 mg, or 35 mL. This is an estimate based on average rates of absorption, and in practice, actual toxicity often does not occur even at doses higher than this. Conversely, this limit assumes no drug is injected intravascularly.

The surgeon begins infiltration with bupivacaine. After about 15 mL have been injected, the patient complains of lightheadedness, and then his eyes roll back and he loses consciousness. The patient develops tonic-clonic movements of his extremities. How will you respond?
Seizures associated with local anesthetic toxicity are treated symptomatically. Tell the surgeon to immediately stop injecting to limit further toxicity. You should administer supplemental oxygen and maintain the airway. If the patient is not breathing, you should administer positive pressure ventilation by mask. Intubation is not always necessary, as seizures associated with local anesthetic are often short lived. A small dose of midazolam (a benzodiazepine) or thiopental will help terminate the seizure.

Despite your initial efforts, the patient remains unresponsive. The electrocardiogram shows ventricular tachycardia. You cannot palpate a pulse. How will you proceed?

Your patient has developed a much more severe form of toxicity, cardiovascular compromise. This syndrome is associated with potent lipophilic anesthetics such as bupivacaine (had the surgeon only been using lidocaine, this complication would have been less likely). Immediate treatment is supportive: administer CPR and begin ACLS treatment for ventricular fibrillation (epinephrine or vasopressin, defibrillation). Unfortunately, bupivacaine-associated cardiovascular toxicity is often very difficult to reverse. Supportive treatment may require cardiopulmonary bypass until the local anesthetic can be cleared. A still experimental but very promising treatment is infusion of a lipid emulsion solution like that used in total parenteral nutrition (Intralipid). Current recommendation is to use 1.5–2 mL/kg of a 20% solution given as IV bolus, followed by an infusion if successful. In animals and a few human case reports, this treatment has proven dramatically successful. Importantly, even though propofol is packaged in a lipid emulsion, it should not be substituted because the vasodilation and cardiac depression associated with a large dose of propofol may counteract the effects of the lipid.

Suggested Further Reading


For maximum impact, it is recommended that the case study and questions found on page xx are reviewed before reading this chapter.

Key Learning Objectives

- Understand the clinical properties and uses of direct and indirect-acting sympathomimetic drugs
- Learn the mechanism of action of antiemetic drugs
- Review the properties of nonsteroidal anti-inflammatory drugs

Sympathomimetics

Sympathomimetics (vasopressors) are drugs that are used to support the cardiovascular system, particularly the blood pressure. They work by individually or collectively affecting arterial vasoconstriction, heart rate (chronotropism), and contractility (inotropism). Many patients who require surgery are dehydrated, have a significant systemic illness, or an underlying cardiovascular disease. As most anesthetic agents are cardio-depressants, the temporary use of vasopressors is frequently required so that patients can tolerate anesthesia. The two most common vasopressors used in the administration of anesthesia to adults are ephedrine and phenylephrine (neosynephrine) (Table 7.1).

Ephedrine is an indirect acting vasopressor that has both alpha (vasoconstriction) and beta (increased heart rate) receptor effects. Ephedrine’s principal mechanism of action is to cause the release of norepinephrine from neuronal storage vesicles at the nerve terminus. This additional norepinephrine in the synaptic space then binds to and activates adrenergic receptors. Ephedrine is
usually administered to patients who have both low blood pressure and low heart rate. In addition to having vasopressor effects, ephedrine is also a bronchodilator, and it can be administered to patients who are in bronchospasm.

**Phenylephrine** acts on alpha receptors causing increased vascular resistance and blood pressure. It has no beta agonist effects and frequently causes a reflex bradycardia (i.e. high blood pressure stimulates baroreceptors thereby decreasing heart rate). Phenylephrine is usually administered to patients who have low blood pressure and high heart rates. It must be used with caution in patients with ischemic heart disease, as it can actually decrease cardiac output.

**Norepinephrine** acts on both alpha and beta receptors, with alpha activity predominating. Norepinephrine leads to increases in blood pressure primarily by causing increased systemic vascular resistance. Because of baroreceptor mediated reflex bradycardia, cardiac output may actually decrease with the administration of norepinephrine – in spite of an increase in blood pressure.

**Dopamine** acts on alpha, beta, and dopamine receptors, depending on the dose administered. At low doses (< 3 mcg/kg/min), dopamine will redistribute blood flow to the kidneys and may increase urine output. At higher doses, alpha and beta receptor actions predominate, leading to increased cardiac contractility and systemic vascular resistance.

Table 7.2 depicts commonly used vasopressors and their sites of action, and Table 7.3 depicts dosing regimens for these drugs.

**Antiemetics**
Postoperative nausea and vomiting (PONV) is one of the leading reasons for patient complaints, delayed postoperative discharge, and patient dissatisfaction with their anesthesia experience. There are various mechanisms by which
surgery and anesthesia can cause nausea and vomiting, and consequently there are many different drugs available for prevention and treatment.

**Serotonin antagonists** are the mainstay of antiemetic prophylaxis and PONV treatment. These drugs work by blocking 5HT3 receptor binding. Ondansetron is by far the most commonly used serotonin antagonist, but dolasetron and granisetron are also available. Patients at moderate to high risk for PONV can be given a prophylactic serotonin antagonist prior to surgery, but current guidelines for low risk patients are to treat only those who exhibit nausea and vomiting postoperatively. Side effects sometimes include headache, lightheadedness or drowsiness.

**Promethazine** (Phenergan) is a common second line agent for treatment of nausea and vomiting that has not responded to a serotonin antagonist. Promethazine is a nonselective antihistamine that may lead to drowsiness, and should be used with caution in patients for whom excessive sedation could be detrimental (e.g. those with sleep apnea or those receiving narcotics or other respiratory depressants).

**Dexamethasone** is recommended as part of a prophylactic regimen (typically in concert with a serotonin antagonist) for patients at moderate or high risk for postoperative nausea and vomiting. The exact mechanism by which dexamethasone decreases PONV is still unknown. Side effects are minimal at the recommended dose ranges.

### Table 7.2 Receptor actions of commonly used vasopressors.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Direct</th>
<th>Indirect</th>
<th>Site of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ephedrine</td>
<td>+</td>
<td>++</td>
<td>α, β</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>+</td>
<td></td>
<td>α</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>+</td>
<td></td>
<td>α, β</td>
</tr>
<tr>
<td>Dopamine</td>
<td>++</td>
<td>+</td>
<td>α, β, D (dopamine receptor)</td>
</tr>
</tbody>
</table>

### Table 7.3 Vasopressor dosing.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ephedrine</td>
<td>2.5–10 mg IV bolus</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>40–100 mcg IV bolus or 20–150 mcg/min infusion</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>0.01–0.1 mcg/kg/min infusion</td>
</tr>
<tr>
<td>Dopamine</td>
<td>2–20 mcg/kg/min infusion</td>
</tr>
</tbody>
</table>
Droperidol blocks the transmission of dopamine, serotonin, and GABA. Though an extremely effective antiemetic, it is typically reserved for refractory nausea and vomiting due to concerns about QT prolongation, and the consequent need to monitor the cardiac rhythm of patients after treatment. Droperidol also possesses sedative properties, and was once popular as a premedication prior to surgery.

Scopolamine is an anticholinergic drug which is often administered preoperatively via a transdermal patch (lasts up to 3 days). Patients should be counseled to wash their hands after the removal of a scopolamine patch, as inadvertent rubbing of the eyes may lead to prolonged pupillary dilation.

Commonly used antiemetics and their dosages are outlined in Table 7.4.

**Antihypertensives**
A full discussion of all antihypertensive agents is beyond the scope of this text, but it is worth noting that many patients require blood pressure reduction perioperatively. As with many anesthetic agents, favored antihypertensives tend to be available intravenously and have short (or at least consistent) durations of action.

Beta blockers such as metoprolol or labetelol are easy to dose, and have been shown in studies to positively affect outcomes in patients with preexisting coronary artery disease. Esmolol is a pure β1 receptor antagonist that is commonly used intraoperatively, because of its extremely quick onset and short duration of action. Calcium channel blockers can be administered as boluses or precisely titrated as drips, and are frequently used for tight control of blood pressure.
pressure (nicardipine), or for control of arrhythmias (diltiazem). Hydralazine, a direct-acting smooth muscle relaxant which preferentially vasodilates the arterial system, is frequently used in the recovery room for refractory hypertension, due to its potency and longer duration of action.

**Dexmedetomidine**

Dexmedetomidine is an $\alpha_2$ agonist that can be used for sedation, analgesia, or balanced anesthesia. It is popular as a sedative because it provides minimal respiratory depression, and patients can be aroused from the sedation to follow commands. This is especially useful for sedation and weaning of mechanically ventilated patients prior to extubation in the ICU. Dexmedetomidine has also gained popularity for procedures such as awake fiberoptic intubations, TEEs, awake craniotomies, and other neurosurgical cases that require frequent intraoperative assessment. In addition to sedation, positive side effects include analgesia, amnesia, and activity as an antisialagogue. Possible negative side effects include a reduction in serum catecholamines and consequent potential for a drop in blood pressure and heart rate. Dexmedetomidine has a slow onset of action (10–20 min), and is typically loaded as a bolus of 1 mcg/kg over 10 min, followed by titration to effect in the dose range of 0.2–0.7 mcg/kg/h.

**NSAIDS (Nonsteroidal Anti-Inflammatory Drugs)**

NSAIDS are nonopioid medications that have analgesic, anti-inflammatory, and anti-fever properties. They act by inhibiting the enzyme cyclooxygenase (COX), preventing the conversion of arachadonic acid into prostaglandins.

- **Ketorolac** (Toradol) is the only commercially available IV NSAID in the U.S. It is a nonselective COX inhibitor, which can lessen or eliminate the need for opioids in surgical patients. Debatable concerns exist over delayed bone healing, excessive bleeding, and worsening of renal problems (use half the dose in patients with mild renal failure, avoid in patients with severe renal failure), but these are typically not a concern with short-term perioperative dosing. The typical dose is 30 mg, or 0.5 mg/kg up to 60 mg.

- **Celecoxib** (Celebrex) is an oral selective COX-2 inhibitor, which has the theoretical advantage of a reduced risk of peptic ulcer formation. However, because of concerns related to increased cardiovascular risk, the use of COX-2 inhibitors has diminished greatly.
Case Study

You are asked to provide anesthesia for a woman undergoing needle-directed breast biopsy. She has had several past anesthetics and has not had good experiences. She explains that she has had severe nausea after all her general anesthetics, and that she has been very somnolent after general anesthesia as well as after monitored anesthesia care (local anesthetic plus sedation). Review of her medical record shows that she received reasonably ordinary general anesthesia, with a potent inhaled agent, nitrous oxide, and fentanyl. For her MAC case, she received intravenous boluses of midazolam and fentanyl. After both anesthetics, she recalls experiencing significant pain but could not tolerate oral opioids prescribed for her. She is motivated to avoid general anesthesia and would like you to develop an anesthetic plan that reduces her risk of excessive somnolence and nausea. She is otherwise healthy, exercises regularly, does not smoke or drink, and takes no medication regularly. She has fasted overnight.

The surgeon believes that she can perform the procedure under local anesthesia plus intravenous sedation (MAC). What drugs will you select for sedation? Her principal problem with sedation in the past has been excessive somnolence. Midazolam and fentanyl are both thought of as short acting drugs, although their effects are quite variable among individuals and are dose-related. A review of her previous record may reveal whether she is very sensitive to the effects or if large doses were used. Alternatives include intravenous infusions of drugs with rapid elimination or termination of effect (see the discussion of context sensitive half time, Chap. 4). You could consider propofol by infusion at doses less than that used for TIVA, 25–100 mcg/kg/min. This drug has the additional advantage that it is associated with a low incidence of nausea. A newer alternative is dexmedetomidine (Precedex), which has been used successfully for sedation during even complex and painful procedures such as awake fiber-optic intubation or awake craniotomy. It has little “hangover” sedation, and patients can generally be alert only minutes after discontinuing an infusion. It can cause bradycardia and hypotension, but in this healthy patient, these are likely to be well tolerated. Finally, it is possible to perform this case with only analgesia and sedation for the local anesthetic infiltration, and then no other sedatives during the case, if the patient is motivated, as she states she is. A popular approach is to use a bolus of a very
short-acting analgesic just prior to infiltration by the surgeon that will provide 3–5 min of sedation and analgesia. Remifentanil, 1 mcg/kg, given 75 s before the painful stimulus, offers such an effect and is very rapidly eliminated by ester hydrolysis shortly thereafter. Alfentanil, 1,000–1,500 mcg, is an alternative with a similar but slightly slower elimination.

What strategy will you follow to control her pain?
This procedure should not cause much postoperative pain, so there is no need for large doses of opioids, which could contribute to both nausea and somnolence. A multimodal approach is therefore indicated, including careful use of local anesthetic by the surgeon both before incision (which may reduce postoperative pain) and at the end of the procedure with a long-acting local anesthetic such as bupivacaine. If bleeding risk is not high, as it should not be in this case, a dose of an NSAID such as ketorolac, will help postoperatively and has an additional benefit of being anti-inflammatory, which may reduce pain even after its immediate analgesic effect has dissipated. Finally, some drugs considered for sedation, notably dexmedetomidine, have some analgesic properties themselves. In selected cases, patients have been discharged with a mechanical, nonelectronic pump that slowly infuses local anesthetic under the skin via a multihole “soaker hose” catheter placed during the operation. An example is the On-Q Painbuster system. This case should feature a very small incision, so this may not be feasible, but it could be considered in consultation with the surgeon. Finally, long-lasting pain control nerve blocks can be offered. In breast surgery, a popular option is a paravertebral block, usually performed preoperatively at several levels covering the breast (upper thoracic dermatomes). In this limited operation, this may be overly aggressive, but consultation with the surgeon, regarding the extent of the resection, and with the patient, regarding her expectations and experiences with postoperative pain, are needed to decide.

What strategy will you follow to avoid postoperative nausea?
This healthy, nonsmoking woman, with a history of PONV, is at high risk of recurrent symptoms. By one popular risk assessment scale, she would be expected to have a 60% chance of PONV after outpatient general anesthesia. The use of the MAC technique should reduce her risk somewhat, particularly if opioids are avoided. Should she need general anesthesia,
elimination of nitrous oxide, use of propofol for induction and possibly for maintenance, and avoidance of neuromuscular blockade, to avoid the emetogenic effects of NMB reversal agents, are all prudent choices. In any case, prophylactic antiemetics are indicated for this high risk situation. A popular combination is dexamethasone and ondansetron. Another good choice for outpatient surgery is a scopolamine patch, placed preoperatively. This patch elutes low dose scopolamine for up to 3 days, a distinct advantage over other drugs available on the day of surgery. It can be added as a third drug or substituted for dexamethasone. Patients should be counseled about its side effects of dry mouth and blurry vision, and instructed to wash their hands carefully after removing the patch, to avoid papillary dilation should they get the drug in their eyes. Another option is a new class of agents, the neurokinin-1 antagonists. Aprepitant (Emend) is the first such drug on the market, and it has the advantage of once daily dosing. It is usually combined with ondansetron and a steroid. It is unfortunately quite expensive.

Suggested Further Readings


Section III

Preoperative Considerations
The Preoperative Patient Evaluation

Amit Gupta and Elifce O. Cosar

For maximum impact, it is recommended that the case study and questions found on page xx are reviewed before reading this chapter.

Key Learning Objectives
- Understand the key aspects of a preoperative anesthetic evaluation
- Know how cardiovascular risk factors relate to intraoperative morbidity
- Learn the ASA physical status classification system

Introduction
Preoperative evaluation is a critical part of the perioperative care we provide for patients and must be approached in a carefully planned, systematic way. Generally, the goals of the preoperative anesthesia evaluation include:

- establishing a good physician–patient relationship
- obtaining a medical history
- performing a physical exam
- ordering and reviewing pertinent tests and consultations
- reviewing the patient’s medical records
- ordering appropriate preoperative medications
- obtaining informed consent

The anesthetic plan can then be built upon the collected and assessed preanesthetic evaluation data.
The Interview

Among the most vital parts of the preoperative evaluation is the development of strong rapport with the patient. Fear, anxiety, uncertainty, loss of control, and/or vulnerability are common emotions experienced by surgical patients. A high state of anxiety can have a detrimental effect on the recovery process. Studies have shown that well-informed patients experience less anxiety, are more easily mobilized, tend to be more satisfied with the care they receive, and report better overall postoperative well-being. Therefore, the development of a confident doctor–patient relationship can provide a strong foundation for good patient care and outcomes.

History and Physical

History

Obtaining a pertinent history and physical is an essential part of tailoring the anesthetic plan. A systematic approach to collecting patient history is often used to make sure that all relevant topics are covered.

Airway

The majority of anesthetic complications are due to respiratory injuries. Among the causes of the respiratory injury are inadequate ventilation, esophageal intubation, and difficult tracheal intubation. Since 17% of respiratory related injuries are due to difficult intubation, and up to 28% of all anesthesia-related deaths are due to the inability to mask-ventilate or intubate, recognizing a potential difficult airway in the preoperative evaluation holds great importance. It is important to question the patient about any prior anesthetics and any history of difficult intubation or mask ventilation.

Physical factors affecting mask ventilation can be determined from age and physical history as shown in the list below. If the likelihood of a difficult mask

<table>
<thead>
<tr>
<th>Factors Affecting Mask Ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Presence of a beard</td>
</tr>
<tr>
<td>• BMI &gt; 26 kg/m²</td>
</tr>
<tr>
<td>• Missing teeth</td>
</tr>
<tr>
<td>• Age &gt; 55</td>
</tr>
<tr>
<td>• History of snoring</td>
</tr>
</tbody>
</table>

Adapted from Langeron, O. Prediction of difficult mask ventilation. Anesthesiology 200; 92:1229.
ventilation is judged to be high, careful anesthetic planning (e.g., ensuring that a difficult airway cart is available) is needed. The second most common airway complication involves patient dentition. It is imperative to discuss with the patient if he/she has any dentures, loose teeth, caps, crowns or anything else that can put the patient at risk for tooth injury or aspiration of a dislodged piece.

**Cardiovascular**

In evaluating the cardiovascular system, the main objective should be to decide whether a patient needs further cardiac testing (stress test) or intervention (cardiac catheterization) prior to elective surgery.

Patients should be asked about any history of heart attacks, hypertension, shortness of breath, dyspnea, use of anticoagulants, diuretics, heart surgery, edema, chest pain, chest tightness, use of antibiotics before dental work, blood pressure medication, last echocardiogram, or stress test.

One should then determine a patient’s functional capacity (see table below). Studies have correlated better perioperative outcome with patients whose metabolic equivalent (MET) activity was greater than or equal to 4 METs (see Table 8.1).

The revised ACC/AHA guidelines (2007) recommend the following stepwise approach to evaluating a patient’s cardiac status for patients undergoing noncardiac surgery:

<table>
<thead>
<tr>
<th>Table 8.1 Energy requirements for various activities.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 MET</td>
</tr>
<tr>
<td>2 METS</td>
</tr>
<tr>
<td>3 METS</td>
</tr>
<tr>
<td>4 METS</td>
</tr>
<tr>
<td>5 METS</td>
</tr>
<tr>
<td>6 METS</td>
</tr>
<tr>
<td>7 METS</td>
</tr>
<tr>
<td>8 METS</td>
</tr>
<tr>
<td>9 METS</td>
</tr>
<tr>
<td>10 METS</td>
</tr>
<tr>
<td>11 METS</td>
</tr>
<tr>
<td>12 METS</td>
</tr>
</tbody>
</table>

Adapted with permission from Brigham and Women’s Hospital Preoperative Assessment Form.
Step 1: Determine the urgency of the planned surgery
If the patient requires emergent surgery, then further cardiac assessment should not delay treatment and the patient should go directly to the operating room.
If not emergent surgery, proceed to Step 2.

Step 2: Does the patient have an active cardiac condition or clinical risk factors?

- Unstable or severe angina
- Recent myocardial infarction
- Decompensated heart failure
- Significant arrhythmias (high-grade AV block, symptomatic ventricular arrhythmias, atrial fibrillation with uncontrolled ventricular rate, symptomatic bradycardia)
- Severe valvular disease (severe aortic stenosis: mean pressure gradient >40 mm Hg, aortic valve area <1.0 cm², or symptomatic)

If the patient has one or more of the above conditions, then the cardiac problem should be evaluated, clarified, and treated appropriately. This often means the postponement of surgery.
If the patient does not have any of the above conditions, then proceed to Step 3.

Step 3: Is the patient undergoing low-risk surgery?

- Low-risk surgeries (reported cardiac risk <1%) include endoscopic procedures, superficial procedures, cataract surgeries, breast surgeries, and most ambulatory surgeries

Since interventions based on cardiovascular testing rarely result in a change in management, these patients may proceed with the planned surgery.
If the patient is undergoing intermediate (intraperitoneal, intrathoracic, carotid endarterectomy, head/neck, orthopedic surgery) or high-risk surgery (aortic, major vascular, peripheral vascular surgery), then proceed to Step 4.

Step 4: Does the patient have good functional capacity without symptoms?
If that patient has good functional capacity (≥4METs without symptoms, see Table 8.1), then it is appropriate to proceed with the planned surgery.
If the patient has poor functional capacity, then proceed to Step 5.
Step 5: Patients with poor/unknown functional capacity

In these patients, the presence or absence of active clinical risk factors determines the need for further evaluation.

### Clinical Risk Factors for Increased Perioperative Cardiac Risk

<table>
<thead>
<tr>
<th># Clinical risk factors</th>
<th>Surgical Risk</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Any</td>
<td>Proceed with surgery</td>
</tr>
<tr>
<td>1–2</td>
<td>Intermediate risk surgery</td>
<td>Proceed with surgery with heart rate control or</td>
</tr>
<tr>
<td></td>
<td>Vascular surgery</td>
<td></td>
</tr>
<tr>
<td>3+</td>
<td>Intermediate risk surgery</td>
<td>Consider cardiac testing if it will change management</td>
</tr>
<tr>
<td></td>
<td>Vascular surgery</td>
<td>Consider cardiac testing if it will change management</td>
</tr>
</tbody>
</table>


**Pulmonary**

Postoperative pulmonary complications can prolong the hospital stay by an average of 1–2 weeks. Therefore, it is important to review patient and procedure-related risk factors, perform a clinical evaluation, and recommend risk-reduction strategies to improve patient care and outcome.

Potential *patient-related* risk factors for perioperative pulmonary complications include:

- Smoking
- Poor general health status (ASA > 2)
- Old age (>70)
- Obesity
- Chronic obstructive pulmonary disease
- Reactive Airway Disease (Asthma)

Potential *procedure-related* risk factors include:

- Surgery > 3 h
- General anesthesia
The type of surgery
Use of pancuronium

Clinical evaluation should encompass a thorough history (i.e., questioning about shortness of breath, wheezing, chest pain, bronchitis, asthma, emphysema, recent fever/chills, history of pneumonia or lung surgery, use of steroids) and a physical exam (i.e., auscultation for decreased breath sounds, wheezes, rhonchi, prolonged expiratory phase). Once all of the information is gathered, risk-reduction strategies (Table 8.2) can be applied to optimize patient care.

Hepatic and Gastrointestinal Disease
Hepatic disease can contribute to end-organ dysfunction (endocrine system, pulmonary edema, pulmonary hypertension, renal failure, and cardiomyopathy) and increase the risk during certain surgeries. Hepatic disease can also cause abnormal coagulation and altered drug pharmacokinetics.

<table>
<thead>
<tr>
<th>Table 8.2 Risk reduction strategies for pulmonary complications.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preoperative</strong></td>
</tr>
<tr>
<td>• Smoking cessation (for at least 8 weeks)</td>
</tr>
<tr>
<td>• Treat airflow obstruction (patients with COPD or asthma)</td>
</tr>
<tr>
<td>• Give antibiotics / postpone surgery in presence of respiratory infection</td>
</tr>
<tr>
<td>• Educate patients about lung-expansion maneuvers</td>
</tr>
<tr>
<td><strong>Intraoperative</strong></td>
</tr>
<tr>
<td>• Limit surgical duration &lt;3 h</td>
</tr>
<tr>
<td>• Avoid pancuronium</td>
</tr>
<tr>
<td>• Consider laparoscopic surgical approach</td>
</tr>
<tr>
<td><strong>Postoperative</strong></td>
</tr>
<tr>
<td>• Encourage incentive spirometry or deep breathing exercises</td>
</tr>
<tr>
<td>• Initiate CPAP (continuous positive airway pressure)</td>
</tr>
<tr>
<td>• Consider epidural analgesia / intercostal nerve blocks</td>
</tr>
</tbody>
</table>


¹A prospective study of 691 patients revealed three times as many pulmonary complications among the patients receiving pancuronium from residual paralysis (Metana, GW. Preoperative Pulmonary Evaluation. NEJM. Vol 340 (12);1999)
Gastrointestinal diseases may increase the potential for aspiration, dehydration, electrolyte disturbances, and anemia. While screening for gastrointestinal disease, it is important to inquire about history of hiatal hernia, diarrhea, bloody stools, heartburn, food regurgitation, gastric ulcers, nausea, vomiting, viral hepatitis, and alcoholism.

**Bleeding Disorders**
A history of a bleeding disorder can increase a risk of perioperative complications, and necessitate further preoperative evaluation and planning. Possible causes of bleeding may be due to disorders of coagulation factors (e.g., hemophilia, Von Willebrand’s disease), cancer, thrombocytopenia, leukemia, certain medications (e.g., warfarin, heparin, clopidrogel), platelet disorders (e.g., Bernard–Soulier syndrome), and liver disease.

**Endocrine**
Endocrinopathies can carry a high risk for morbidity and mortality. Patients should be assessed for any history of risk factors for diabetes mellitus.

Diabetic patients should be evaluated with regard to the type, duration, and severity of disease. The patient’s current therapy (diet, oral hypoglycemic drug, or insulin regimen) should be assessed, along with a morning blood sugar and HbA1c to determine degree of control. All diabetics should be evaluated for the presence of coronary artery disease and hypertension. Additionally, a serum creatinine level may be drawn to assess a patient’s degree of nephropathy. Most providers will avoid regional anesthesia techniques in diabetics with severe peripheral neuropathy. Typically, patients on insulin are instructed to take half their morning dose of insulin on the day of surgery. Diabetics should also be scheduled for elective surgery early in the day, to minimize the impact of prolonged fasting on their blood sugar management.

Perioperative mortality associated with pheochromocytoma and the carcinoid syndrome can reach 50% if undiagnosed. Thus, screening patients for any history of thyroid, parathyroid, carcinoid syndrome, adrenal and pituitary diseases can help decrease potential perioperative risks.

**Renal**
History of any kidney disorder holds importance during the preoperative evaluation, since derangement in kidney function can contribute to secondary physiologic imbalances, deficits in platelet function (impaired aggregation),
anemia, electrolyte imbalances, peripheral neuropathies, and abnormal drug metabolism and excretion. Investigation of a patient’s history of renal insufficiency, renal failure, and dialysis dependence should therefore be undertaken.

**Neurologic**

When screening a patient for neurologic disease, the anesthesiologist should elicit a history of seizures, convulsions, tremors, headaches, nerve injuries, multiple sclerosis, tingling, or numbness of an extremity. The use of a neuraxial blockade or a regional nerve block requires knowledge of any previous nerve injuries or deficits.

**Musculoskeletal**

One should ascertain any history of low back pain, radicular pain, herniated disks and chronic pain managed with opioids. Patients should also be assessed for any history or signs of myopathies – as they may portend postoperative muscle weakness.

**Physical Exam**

A preoperative physical exam starts with noting patient’s **baseline vital signs**. During the **airway evaluation**, first document the Mallampati score (see Chap. 9, Airway Evaluation and Management). Then be sure to note any gross external features such as facial trauma, large incisors, a beard or moustache, a large tongue, neck masses, tracheal deviation, or if patient is edentulous – all factors which could contribute to difficult mask ventilation or intubation. Note any possible airway obstruction (i.e., peritonsillar abscess, trauma) and neck mobility. The **cardiopulmonary exam** includes assessment of rate and rhythm, murmurs, wheezing, rhonchi, stridor (inspiratory versus expiratory), peripheral pulses and baseline pulse oximetry saturation. **Gastrointestinal exam** includes looking for signs of ascites, abdominal distension, and guarding. **Musculoskeletal exam** may include neck range of motion, scoliosis, and assessment of pectus excavatum/carinatum. Finally, a **neurologic exam** may include an assessment of baseline muscle strength, mental status and any signs of preexisting nerve injury.

**Medications/Allergies**

The generic name of all medications with the route and dosage should be noted. In some cases, it is helpful to include the period of time the patient has
been on a given medication – particularly opioids as chronic use may lead to higher opioid requirements during the surgery and postoperatively. Additionally, long-term use of steroids can lead to adrenal insufficiency and may require steroid supplementation during surgery.

A medication history should also encompass any over-the-counter or alternative medicines (i.e., herbal medications). This is important because many supplements have important side effects that may manifest during anesthesia (e.g., ginko and garlic both potentiate anticoagulant medications, St. John’s Wort can prolong anesthesia, and Ephedra may cause arrhythmias).

Patients often state allergies to medications, foods, and environmental agents. It is important to note what medications cause what allergic reaction, and the severity of that reaction. For example, does penicillin cause a mild rash or an anaphylactic reaction? Also, it is important to differentiate allergic reactions from mere side effects (e.g., nausea and vomiting induced by morphine is a side effect and not a true allergy). Allergies to latex (some OR supplies contain latex), iodine, and shellfish are essential to elicit as well.

**Medical Records/Family History**

Medical records often contain a substantial part of patient’s medical history – which the patient may or may not be aware of. They may include information that could change your anesthesia plan. For example, a history of a difficult airway may lead to a decision to perform an awake fiberoptic intubation to maximize the patient’s safety. A history of severe postoperative nausea and vomiting or hemodynamic problems during previous surgery may also help the anesthesia provider make adjustments to the planned anesthetic.

Reviewing and screening for anesthetic complications within the patient’s family may also alert the anesthesiologist to potential problems (e.g., history of pseudocholinesterase deficiency or malignant hyperthermia) and should therefore be elicited.

**Laboratory Data**

Up to 3 billion dollars are spent annually in the United States on preoperative laboratory and diagnostic studies. Therefore, these studies should be used in conjunction with patient’s medical history and surgical procedure. Unnecessary testing can result in OR delays or even cancellations due to false positive results. Table 8.3 illustrates the diagnostic and laboratory tests that correspond to specific medical conditions and procedures.
Table 8.3  Suggested indications for preoperative studies.

<table>
<thead>
<tr>
<th>Disease-based indications</th>
<th>CBC</th>
<th>T/S and Albumin</th>
<th>β-hCG</th>
<th>PT/PTT</th>
<th>Electrolytes</th>
<th>BUN/Cr</th>
<th>Glucose</th>
<th>AST/Alk phos</th>
<th>ECG</th>
<th>CXR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol abuse</td>
<td>x</td>
<td></td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Adrenal cortical disease</td>
<td>x</td>
<td></td>
<td></td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anemia</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>x</td>
</tr>
<tr>
<td>Hematologic abnormalities</td>
<td>x</td>
<td>x</td>
<td></td>
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<tr>
<td>Hepatic disease</td>
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<td></td>
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<td>x</td>
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<tr>
<td>Malignancy with chemotherapy</td>
<td>x</td>
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<td></td>
<td></td>
<td></td>
<td>±</td>
</tr>
<tr>
<td>Malnutrition</td>
<td>x</td>
<td>x</td>
<td>±</td>
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<tr>
<td>Morbid obesity</td>
<td></td>
<td></td>
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<td>x</td>
<td></td>
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<tr>
<td>Peripheral vascular disease or stroke</td>
<td>x</td>
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<tr>
<td>Personal or family history of bleeding</td>
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<td>x</td>
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<td>±</td>
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<tr>
<td>Possibly pregnant</td>
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<tr>
<td>Pulmonary disease</td>
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<td>±</td>
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<tr>
<td>Renal disease</td>
<td>x</td>
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<td>x</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>x</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Sleep apnea</td>
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<td></td>
<td></td>
<td>x</td>
</tr>
<tr>
<td>Smoking &gt; 40 pk-yr</td>
<td>x</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>x</td>
<td></td>
</tr>
</tbody>
</table>

*Note: X indicates the test is recommended, ± indicates the test may or may not be recommended, and x indicates the test is not recommended.*
**Therapy-based indications**

<table>
<thead>
<tr>
<th>Indication</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Radiation therapy</td>
<td>x</td>
<td></td>
<td>x</td>
</tr>
<tr>
<td>Use of anticoagulants</td>
<td>x</td>
<td></td>
<td>x</td>
</tr>
<tr>
<td>Use of digoxin and diuretics</td>
<td></td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Use of statins</td>
<td></td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Use of steroids</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>

**Procedure-based indications**

<table>
<thead>
<tr>
<th>Indication</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedure with significant blood loss</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>Procedure with radiographic dye</td>
<td></td>
<td>x</td>
</tr>
</tbody>
</table>

*Adapted from Miller’s Anesthesia, 6th ed. 2005 Churchill Livingstone, An Imprint of Elsevier.*
Anesthetic Plan

Upon gathering information from a patient’s history, exam, physical and laboratory results, the patient is then assigned an ASA (American Society of Anesthesiologists) physical status classification (see Table 8.4 below). The ASA status is a standardized way to communicate with other clinicians about the patient’s overall medical condition.

The information obtained from a history, physical exam, and discussion with the patient can help generate a reasonable and safe anesthetic plan. The purpose of the plan is to provide an anesthetic that is tailored to each individual patient and the anticipated procedure. For example, deciding on general anesthesia versus regional depends on the patient’s co-morbidities and the nature of the operation. Planning to perform an awake intubation versus an intubation after the induction of general anesthesia would be indicated for patients with a difficult airway and/or mask ventilation. The plan also includes possible preoperative invasive catheter placement (arterial and/or central venous catheters) for close monitoring of high-risk patients, and a preliminary plan for the place of recovery after surgery (i.e., PACU vs. ICU). Anesthesia is a proactive specialty rather than reactive because the pre anesthetic evaluation can prompt change in patient management (see Table 8.5), leading to optimum patient care and outcome.

Anesthesia Consent Form

The purpose of the anesthesia consent form (also see Chap. 31, on Ethical Issues) is to discuss with the patient or their representative the types of anesthetic options available for the planned procedure and explain possible risks and benefits that encompass the anesthetic plan.

<table>
<thead>
<tr>
<th>Table 8.4</th>
<th>ASA physical status classifications.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA Class I</td>
<td>A normal healthy patient</td>
</tr>
<tr>
<td>ASA Class II</td>
<td>A patient with mild systemic disease</td>
</tr>
<tr>
<td>ASA Class III</td>
<td>A patient with severe systemic disease that limits activity, but is a constant threat to life</td>
</tr>
<tr>
<td>ASA Class IV</td>
<td>A patient with incapacitating system disease that is a constant threat to life</td>
</tr>
<tr>
<td>ASA Class V</td>
<td>A moribund patient not expected to survive 24 h with or without surgery</td>
</tr>
<tr>
<td>E</td>
<td>Designates an emergency surgical procedure (i.e., Class IE)</td>
</tr>
</tbody>
</table>

Table 8.5 Formulation an anesthetic plan based on patient history.

<table>
<thead>
<tr>
<th>Patient history</th>
<th>Area to evaluate</th>
<th>Anesthetic considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airway perceived as difficult to intubate or ventilate</td>
<td>Head, eyes, ears, nose, throat: airway; pulmonary disease</td>
<td>Obtain fiberoptic equipment; obtain skilled help</td>
</tr>
<tr>
<td>Asthma</td>
<td>Head, eyes, ears, nose, throat: airway; pulmonary disease</td>
<td>Optimize therapy; use bronchodilators; consider extubating during deep anesthesia</td>
</tr>
<tr>
<td>Diabetes, insulin-dependent</td>
<td>Endocrine, metabolic, diabetes</td>
<td>Discuss insulin management with patient and primary care doctor; monitor blood glucose intraoperatively; determine presence of autonomic neuropathy and plan management appropriately, such as administration of metoclopramide and PACU or ICU stay</td>
</tr>
<tr>
<td>Drug abuse</td>
<td>Social history</td>
<td>Consider HIV and Hepatitis testing; prescribe medications to avoid withdrawal symptoms in perioperative period</td>
</tr>
<tr>
<td>Gastroesophageal reflux or hiatus hernia</td>
<td>Gastrointestinal disease: hiatus hernia</td>
<td>Administer H2 antagonists or oral antacids and use rapid-sequence induction of anesthesia; or use awake intubation techniques and obtain appropriate equipment</td>
</tr>
<tr>
<td>Heart disease: valve disease, risk of subacute bacterial endocarditis</td>
<td>Cardiac history and exam, imaging studies</td>
<td>Consider antibiotic prophylaxis. Arrange for antibiotic administration 1 h prior to surgery</td>
</tr>
<tr>
<td>Personal malignant hyperthermia history, family history, or suspected potential history</td>
<td>Prior anesthetic/surgical history</td>
<td>Obtain clean anesthesia machine; use appropriate technique and precautions; have agents to treat malignant hyperthermia available</td>
</tr>
<tr>
<td>Monoamine oxidase inhibitors</td>
<td>CNS: psychiatric/medication</td>
<td>Discontinue therapy preoperatively if patient is not suicidal; plan for perioperative pain therapy</td>
</tr>
<tr>
<td>Pacemaker or automatic implantable cardiac defibrillator</td>
<td>Cardiovascular disease: electrocardiogram</td>
<td>Evaluate cause of pacemaker implementation; obtain repolarizing equipment or magnet; use electrocautery with altered position; use bipolar electrocautery</td>
</tr>
</tbody>
</table>

(continued)
### Table 8.5 (continued)

<table>
<thead>
<tr>
<th>Patient history</th>
<th>Area to evaluate</th>
<th>Anesthetic considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral motor neuropathy</td>
<td>CNS disease: neurologic deficit</td>
<td>Avoid depolarizing muscle relaxants</td>
</tr>
<tr>
<td>Pregnancy or uncertain pregnancy status</td>
<td>Genitourinary: pregnancy</td>
<td>Monitor fetal heart rate; use oral antacids; adjust induction of anesthesia; determine status of pregnancy</td>
</tr>
<tr>
<td>Pulmonary tuberculosis</td>
<td>Pulmonary disease: tuberculosis</td>
<td>Use disposable breathing circuit or clean equipment; ensure adequate treatment of patient prior to surgery</td>
</tr>
<tr>
<td>Renal insufficiency</td>
<td>Genitourinary disease</td>
<td>Monitor fluid status intraoperatively</td>
</tr>
</tbody>
</table>

*CNS* central nervous system, *HIV* human immunodeficiency virus, *ICU* intensive care unit, *PACU* postanesthetic care unit

Adapted from Fischer et al. Cost-effective preoperative evaluation and testing. Chest 1999; 115; 96–100.
Types of anesthesia usually discussed include general, monitored anesthesia care (MAC), regional, and local. While patients may exhibit a preference for one type of anesthetic over another – the final decision should be made with the participation of all parties (patient, anesthesiologist, surgeon) involved.

Some of the more common risks of anesthesia that need to be mentioned include infection and bleeding if a regional block is to be placed, nerve injury from positioning or regional blocks, postoperative nausea and vomiting, dental damage, risk of viral hepatitis and HIV from blood transfusions, awareness under anesthesia and a need for postoperative mechanical ventilation (patient fails extubation after surgery ends). The aim of the consent process is to provide the patient with all available anesthetic options and possible risks due to anesthesia and the surgical procedure. The process of informed consent culminates in the signing of a legal document (the consent form) and must be signed by both the patient and the anesthesia provider. It is important to note that informed consent is a process of ensuring a patient understands the risks, benefits, and available options. It is not merely the signing of a consent form.

### Case Study

**You are seeing a 64-year-old man in the preop clinic. He is to undergo an open suprapubic prostatectomy a week from today. His past medical history is notable for an inferior non-q wave MI 2 years ago. He was managed at that time by placement of a bare-metal stent. He has smoked a pack of cigarettes a day for 35 years and sometimes gets shortness of breath during exertion, in cold weather, and when he has a URI. He has had hypertension for many years. Five years ago he was diagnosed with type 2 diabetes mellitus. He works as a carpenter, carrying boards around the job site, and he does his own yard work. His medications at present are aspirin 81 mg once per day, atenolol 100 mg daily, metformin, exenatide (Byetta), as well as an albuterol inhaler and sublingual nitroglycerin as needed.**

**What ASA physical status class is this patient?**

This patient has some significant comorbidities, making him at least ASA class II. Whether he is class II, III, or IV depends on your assessment of the severity of the diseases. He is likely not class IV, which requires
systemic disease that is a constant threat to life. The distinction between class II and III is based on your judgment as to whether he has “mild” or “severe” disease. Given that he has had a myocardial infarction and not just stable angina, it would be reasonable to classify him as III. However, if after you assess his cardiovascular, pulmonary, and endocrine disease more fully, you believe him to be robust and generally fit, a classification of II would be appropriate.

How would you assess his risk and prepare him for surgery from a cardiovascular standpoint?
The patient’s MI was not particularly recent (in the last 3–6 months) so the immediate post-MI danger period has passed. He was stented 2 years ago and is not taking clopidogrel (Plavix), so the major danger related to stent occlusion and/or anticoagulation has passed. However, we can assume that he remains at risk for myocardial ischemia. The best way to assess this risk is also the simplest: assess his exercise tolerance. He does moderately heavy exertion at work and at home, so we can conclude that his exercise tolerance is good. He is taking a beta-blocker and aspirin, both recommended for patients at elevated cardiovascular risk. He should continue both through the morning of surgery. Current data is inconclusive regarding the intensity of beta blockade required to minimize cardiovascular risk. There is no evidence to support imaging or further testing prior to surgery for this patient.

How would you assess his risk and prepare him for surgery from a pulmonary standpoint?
Pulmonary complications (prolonged postoperative ventilation, unexpected reintubation, pneumonia) are as common as cardiovascular complications, and actually are more costly to manage. There are risk stratification systems for pulmonary complications, though many include unmodifiable risk factors. This patient is at elevated risk due to advanced age, COPD, ASA class > II, and cigarette smoking (see Smetana GW, Ann Intern Med. 2006;144:581–595). In general, neither a chest X-ray nor pulmonary function testing (spirometry) are indicated. If he feels that he is at his baseline with regard to symptoms (shortness of breath on exertion, use of albuterol), then he should only plan to bring his inhaler on the day of surgery and use it
prior to going to the OR. However, if he feels that he is not at his personal best, it is reasonable to have him intensify his pulmonary regimen before going to the OR, since his surgery is not emergent. He might benefit in such cases by increased use of his inhaler, use of inhaled or oral corticosteroids, and in some cases antibiotics. Recent data suggest incentive spirometry performed preoperatively may also improve pulmonary outcomes.

He asks you if he should quit smoking before the surgery. How would you respond?

While every physician should encourage smokers to quit, the immediate preoperative period may not be the optimal time. Studies suggest smokers who abstain for eight weeks or more can lower their risk of pulmonary complications nearly to that of nonsmokers, in the absence of severe irreversible COPD. However, quitting a shorter period of time prior to surgery may actually be counterproductive, because cough and sputum production may worsen temporarily. The time following surgery may be a good time to quit, since hospitals generally disallow smoking anyway, and thus his admission may be an opportune time.

How should his diabetes be managed for surgery? Would your recommendation be different if he were taking insulin?

Metformin and other oral hypoglycemic agents should not be taken on the day of surgery and are generally stopped the evening before surgery. Some anesthesiologists stop metformin for 24–48 h. There is a very small risk of severe lactic acidosis when taking metformin (3–8 cases/100,000 patient-years), which may be increased in conditions, such as hypovolemia and hypoxia, as may occur in the perioperative period. Although rare, the mortality is very high (50%) and thus guidelines recommend at least not taking it on the day of surgery. Glucose should be checked on admission to the preop unit, and there is some evidence that avoiding hyperglycemia intraoperatively with intravenous insulin can improve outcomes such as wound infection. If the patient were taking insulin, the recommendations are different. He should not take any short-acting or prandial insulin on the day of surgery, such as aspart (NovoLog) or lispro (Humalog). However, basal insulin, such as glargine (Lantus), should be continued at the usual dose, typically taken in the evening. NPH is intermediate between
these two types and one approach is to take half of the usual morning dose. As with patients managed on oral agents, it is prudent to monitor blood glucose to avoid hypo- or extreme hyperglycemia during surgery.

**What other information would you like to obtain to complete your preoperative evaluation?**

All patients should have an airway examination such as determination of the Mallampati class (oropharyngeal structures visualized) and the thyromental distance and flexibility of the cervical spine. It is useful to inquire about previous experiences with anesthesia, with particular attention to complications, but also to help characterize the patient’s risk for postoperative nausea and vomiting and approach to pain management. A physical examination, especially directed at the cardiopulmonary systems, presence of vascular access sites, and possibly suitability for regional analgesia (e.g., an epidural catheter for postoperative pain management) is needed. Dentition, facial features predictive of difficult mask ventilation, and difficulty with expected positioning (this case may be performed in low lithotomy position or supine and flexed with head down) should also be sought on physical exam. You should make certain that the patient and family members have voiced any concerns about the surgery or the anesthetic, and you should try to address them. Finally, anesthesia consent should be obtained, and presence of surgical consent should be verified before proceeding to the OR.

**Acknowledgment**

We would like to thank Dr. Stephen O. Heard for his guidance and assistance with the preparation of this chapter.

**Suggested Further Reading**

Basic Standards for Preanesthesia Care (Approved by the House of Delegates on October 14, 1987, and amended October 25, 2005)


Airway Evaluation and Management

Shawn T. Beaman, Patrick J. Forte, and David G. Metro

For maximum impact, it is recommended that the case study and questions found on page xxi are reviewed before reading this chapter.

Key Learning Objectives
- Review the anatomy relevant to airway management
- Understand the components of an airway examination
- Learn the principles of mask ventilation and intubation

Introduction
The link between the practice of anesthesia and airway management is not entirely intuitive. How could anesthetizing a patient for a lower extremity procedure possibly impact that patient’s airway or respiratory status? The answer lies largely in the profound respiratory side effects of most anesthetic medications. Despite the site of surgery or the anesthetic technique chosen, every patient receiving anesthetic care is exposed to a varying degree of risk of airway compromise. That is, all levels of sedation, general anesthesia, and regional anesthesia carry with them at least a small risk of airway obstruction and apnea. Therefore, every anesthesia provider must examine each patient in anticipation of a need to mechanically ventilate and intubate, regardless of whether or not such interventions were part of the primary anesthetic plan. A thorough airway examination and history, combined with expert airway management, guard against the life-threatening risks of airway obstruction and apnea.

It is during the provision of general anesthesia that airway management is most commonly employed. General anesthesia renders patients insensate...
to noxious stimuli throughout their bodies and is therefore employed during a wide variety of surgical procedures from craniotomy and tonsillectomy to liver resection and prostatectomy. The intravenous induction of general anesthesia and apnea are most often synonymous. Expert airway management is the cornerstone of safety for any general anesthetic.

Airway management is not routinely employed during regional anesthesia. However, airway management could become necessary should the patient suffer an intravascular injection of local anesthetic that precipitates seizure or cardiovascular collapse. The same risks of apnea during sedation also apply, should the patient receive sedation either for the regional anesthetic itself, or during the ensuing surgical procedure.

Airway Anatomy
The human airway is a dynamic structure that extends from the nares to the alveoli. Obstruction can occur at any point because of anatomic collapse or a foreign body which includes liquids such as mucous, blood, and gastric contents.

Figure 9.1 demonstrates relevant upper airway anatomy. Figure 9.2 illustrates the glottis (the vocal cords and space between them) and epiglottis.

Airway Evaluation
In addition to the inherent risks of apnea with all anesthetic techniques, management of the difficult airway continues to be a clinically important source of liability. The goal of airway evaluation is to avoid failed airway management by implementing alternative strategies in patients who are predicted to be difficult to ventilate and/or intubate. Difficult mask ventilation occurs when there is an inadequate seal between the patient’s face and the mask, there is a leak of oxygen from the face mask, or there is excessive resistance to the inflow or outflow of oxygen. Difficult laryngoscopy occurs when no portion of the glottis is visualized after multiple laryngoscopic attempts. A patient is defined as having a difficult airway if a conventionally trained anesthesiologist experiences difficulty with facemask ventilation of the upper airway, difficulty with tracheal intubation, or both.

In order to predict difficult mask ventilation or difficult endotracheal intubation, each patient receiving anesthetic care should have a comprehensive airway history and physical examination performed (also see Chap. 8, Preoperative Evaluation). Patients should be queried about airway complications that occurred during past anesthetics. A history of trauma during previous airway management to the patient’s lips, teeth, gums, or mouth may indicate the presence of a difficult airway. Similarly, if the patient reports that many attempts
were made to “insert the breathing tube” or that he or she was “awake” during previous intubations, a difficult airway should be considered. Medical conditions that classically may portend a difficult airway include a recent or remote history of facial trauma or surgery, rheumatoid arthritis, pregnancy, epiglottitis, previous cervical fusion, neck masses, Down's syndrome, and other genetic syndromes such as Treacher-Collins and Pierre-Robin that have associated facial abnormalities. With a positive history, documentation regarding previous airway management should be reviewed.

Multiple physical examination features have been correlated with a difficult airway (see Table 9.1).
### Table 9.1 Components of the preoperative airway physical examination.

<table>
<thead>
<tr>
<th>Component</th>
<th>Nonreassuring finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of upper incisors</td>
<td>Relatively long</td>
</tr>
<tr>
<td>Relation of maxillary and mandibular incisors during normal jaw closure</td>
<td>Prominent “overbite” (maxillary incisors anterior to mandibular incisors)</td>
</tr>
<tr>
<td>Relation of maxillary and mandibular incisors during voluntary protrusion of the jaw</td>
<td>Patient’s mandibular incisors anterior to (in front of) maxillary incisors</td>
</tr>
<tr>
<td>Inter-incisor distance (mouth opening)</td>
<td>&lt;3 cm</td>
</tr>
<tr>
<td>Visibility of uvula</td>
<td>Not visible when tongue is protruded with patient in sitting position (e.g., Mallampati class II)</td>
</tr>
<tr>
<td>Shape of palate</td>
<td>Highly arched or narrow</td>
</tr>
<tr>
<td>Compliance of submandibular space</td>
<td>Stiff, indurated, occupied by mass, or non-resilient</td>
</tr>
<tr>
<td>Thyromental distance</td>
<td>&lt;3 finger breadths or 6–7 cm</td>
</tr>
<tr>
<td>Length of neck</td>
<td>Short</td>
</tr>
<tr>
<td>Thickness of neck</td>
<td>Thick (neck size &gt; 17 inches)</td>
</tr>
<tr>
<td>Range of motion of head and neck</td>
<td>Patient cannot touch tip of chin to chest or cannot extend neck</td>
</tr>
</tbody>
</table>

Figure 9.2 The glottis and epiglottis (Reproduced with permission from *Principles of Airway Management, 3rd ed.*, Brendan Finucane and Albert Santora, Springer 2003).

Every patient receiving anesthetic care should be thoroughly examined for the presence of these features. An adequate exam is difficult to accomplish without active participation and cooperation of the patient. That is, examinations performed solely by inspection may not only be incomplete, but may also be inaccurate. The most common examination performed to evaluate patients for the presence of a difficult airway is determination of what is known as the Mallampati Class. This classification system, first developed in 1985, seeks to predict difficult intubation by functionally assessing the ratio of the size of one’s tongue to the size of one’s oral cavity (see Fig. 9.3).

Increasing difficulty with direct laryngoscopy has been correlated with Mallampati Class III and IV examinations. Although a single worrisome predictor of difficult airway management may be clinically important, a richer and more predictive exam is obtained by screening for multiple predictors in every patient.

**Mask Ventilation**

Face mask ventilation is the most basic airway management intervention and is the first skill any student of anesthesia should seek to develop. Three goals need to be achieved for optimal face mask ventilation:

1. An optimal seal must be made between the mask and the patient’s face
2. The patient’s oropharynx must be opened by anterior displacement of the mandible into the face mask and extension of the head as seen in Fig. 9.4. Placement of an oral or nasal airway during facemask venti-
lation may assist in opening the oropharynx by creating an artificial passage for gases between the tongue and the posterior pharyngeal wall as seen in Fig. 9.1.

3. Sufficient positive pressure must be generated to overcome the resistance of the patient’s upper airway, chest wall, and diaphragm to effect efficient gas exchange at the alveoli.

Mask ventilation can be employed to augment patient’s spontaneous tidal volumes as a temporizing measure before definitive airway management occurs via endotracheal intubation—as in the case of an intensive care unit patient slowly succumbing to respiratory failure from pneumonia. In the operating room, mask ventilation is most commonly employed to oxygenate and ventilate patients who are apneic from general anesthetic induction agents.
The laryngeal mask airway (LMA) was first introduced in the United States in 1988 and FDA approved in 1991. The soft plastic device, seen in Fig. 9.5, has revolutionized the care of patients receiving general anesthesia who do not require endotracheal intubation. The device largely supplanted the delivery of face mask anesthesia and has also reduced the rate of endotracheal intubation. The most recent version of the American Society of Anesthesiologists Difficult Airway Algorithm (see Appendix A) places special significance on the use of the LMA in situations where mask ventilation is difficult.

Figure 9.5  Laryngeal mask airway (Photo courtesy J. Ehrenfeld).
The lubricated device is inserted blindly into a patient’s mouth following the hard palate, past the tongue, and seated with the tip in the hypopharynx. The cuff is inflated isolating the gastrointestinal tract from the respiratory tract above the glottis. Because the LMA does not enter the glottis, patients tolerate the device with less anesthetic than an endotracheal tube. However, being supraglottic, the LMA does not protect against pulmonary aspiration to the same degree as an endotracheal tube. Other than for emergency ventilation, relative contraindications to the use of the LMA include:

- patients at increased risk for pulmonary aspiration
- patients or procedures requiring positive pressure ventilation
- lengthy procedures
- procedures in any position other than supine

Direct Laryngoscopy and Tracheal Intubation

Direct laryngoscopy is the most common means of accomplishing endotracheal intubation. It is the process of visualizing a patient’s glottis through his/her mouth by aligning the axes of the oral cavity, the pharynx, and the larynx as seen in Fig. 9.6. Common errors in direct laryngoscopy include inserting the laryngoscope blade too deeply exposing the patient’s esophagus and improperly sweeping the tongue from the line of sight.

Using direct laryngoscopy, endotracheal tubes are most commonly passed through the patient’s mouth and into the glottis using a laryngoscope. A laryngoscope consists of a handle and an interchangeable blade with a light bulb on the end. The blades come in a variety of shapes and sizes, but the most commonly used are the Macintosh 3 (curved) and Miller 2 (straight) (see Fig. 9.7). Once the endotracheal tube passes through the glottis, a seal is formed between the endotracheal tube and the tracheal wall. In adults and older children, this seal is formed by inflating a cuff near the distal end of the tube with air. Because of an anatomical narrowing that exists at the level of the cricoid cartilage in children, uncuffed tubes are used and a seal forms directly between the tube and the trachea. For intraoral procedures (such as the excision of a tongue lesion), endotracheal tubes can be placed into the glottis via a nasal approach utilizing either direct or fiber-optic laryngoscopy.

The placement of an endotracheal tube is considered the “gold standard” – the definitive airway management for two principal reasons. First, particularly with the placement of a cuffed endotracheal tube, the possibility of aspiration of gastric contents into the airways is greatly reduced. Second, it is via an
endotracheal tube that greatest positive airway pressure can be achieved with mechanical ventilation.

**Rapid Sequence Induction**

The reflux of gastric contents from the stomach into the distal airways via the glottis is a universal concern at all stages of anesthetic care. Fasting prior to elective surgery is the main intervention that guards against pulmonary aspiration. Risk factors for pulmonary aspiration may include:

- trauma patients
- patients undergoing emergency surgeries (fasting guidelines do not apply)
- pregnant patients in labor

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**Figure 9.6**  Relationship of the oral, pharyngeal, and laryngeal axes for intubation. (Reproduced with permission from *Principles of Airway Management, 3rd ed.*, Brendan Finucane and Albert Santora, Springer 2003).
patients with severe, positional gastroesophageal reflux disease
- diabetics (decreased gastric emptying) or obese patients
- patients with neurological impairment

In order to decrease the interval between when a patient is awake with intact laryngeal muscles protecting their airway from aspiration and when the endotracheal tube is in place guarding against aspiration, a rapid sequence induction (RSI) may be performed. An RSI differs from a standard induction after the induction of general anesthesia in three ways:

1. During an RSI, face mask ventilation is **not used** to ventilate the patient. This is to avoid distension of the stomach that can occur with face mask ventilation.
2. Cricoid pressure is maintained from before the time the patient receives induction agents until the endotracheal tube placement in the trachea is
confirmed. The cricoid cartilage is the only tracheal cartilage that surrounds the entire trachea. Applying pressure to the anterior aspect of the cricoid cartilage occludes the esophagus by closing its lumen between the posterior aspect of the cricoid cartilage and the anterior aspect of the body of the cervical vertebrae.

3. Succinylcholine is classically used as the muscle relaxant of choice to facilitate intubation due to its short onset time. Rocuronium is another choice for patients who might have detrimental side effects from succinylcholine use (e.g. burn and spinal cord injury patients).

**Fiber-optic Intubation**

Endotracheal intubation can be accomplished via fiber-optic guidance. This is accomplished by passing the distal end of a bronchoscope through the glottis and then sliding an endotracheal tube off of the scope into the trachea under direct vision. Fiber-optic intubation can be accomplished in awake as well as anesthetized patients. Awake patients only tolerate the procedure with sufficient local anesthesia delivered to their airway beforehand via topicalization and/or nerve blockade. Sedation may be given to awake patients having fiber-optic intubation. Patients with anticipated difficult airways are often intubated using an awake fiber-optic technique.

**Video Assisted Endotracheal Intubation (Glidescope, C-Trach)**

Recently, there have been several airway management tools introduced that combine traditional laryngoscopy with fiber-optic technology such as the Glidescope or C-Trach. One of the benefits of these instruments is that they may allow intubation under conditions such as limited mouth opening that might have been impossible with direct laryngoscopy.

**Evaluation and Management of the Difficult Airway**

The ASA Difficult Airway Algorithm is a step-wise approach to managing a challenging airway (also see, Appendix A, ASA Difficult Airway Algorithm). The algorithm is designed to present a rational approach to utilizing a number of different management techniques for securing the airway. These include various types of equipment such as the intubating LMA, Lightwand, Combitube, and fiber-optic laryngoscope. Ultimately, if noninvasive attempts at airway management fail, options include waking the patient up or performing a cricothyroidotomy or a tracheostomy.
Case Study

You are preparing to anesthetize a 50-year-old man for abdominal hernia repair with mesh. He is 68 in. tall and weighs 260 pounds. He has a full beard and mustache. He has no other major comorbidities. He underwent general anesthesia 20-years-ago for arthroscopy of his knee and is not aware of any problems with the anesthetic. You are planning general endotracheal anesthesia.

What factors in this patient worry or reassure you regarding his airway management?

The patient is obese (BMI = 39.5). In itself, this is likely a risk factor for both difficult mask ventilation and difficult laryngoscopy. He also has a full beard, which can interfere with mask fit and make mask ventilation difficult. Conversely, he appears to have had an uncomplicated general anesthetic in the past. While reassuring, there are some caveats: his lack of awareness of problems does not mean that some did not occur but were not reported to the patient or recalled, and his physique may have been quite different 20 years ago.

How will you further assess his airway?

You will perform basic airway examinations on the patient. No one test is definitive, but most anesthesiologists use the Mallampati test, the thyromental distance, and a subjective assessment of neck mobility. Some use other tests as well, such as neck circumference (cut off > 17 in. or 43 cm), ability to protrude the lower incisors anterior to the upper incisors, mouth opening, or sternomental distance. Each correlates somewhat with difficult intubation, but ultimately the judgment is likely more subjective and reflects the clinical gestalt of the experienced anesthesiologist.

You decide to proceed with induction of anesthesia. After administering propofol you attempt mask ventilation. You find it difficult to obtain a good mask fit and mask ventilation is difficult. How will you proceed?

You anticipated this problem preoperatively, so you have backup plans already in place. You can try an oral or nasal airway, which may reduce the pressure required to ventilate the patient by helping hold the upper airway patent. In some cases, using both may be helpful. You can also perform two-person ventilation, with one person holding the mask fit with both
hands and the other ventilating by squeezing the reservoir bag. Finally, you can consider placement of an LMA to assist ventilation, or proceed directly with intubation.

**You are now successfully ventilating the patient. You administer rocuronium to facilitate intubation. After ventilating the patient for 3 min, you perform direct laryngoscopy with a Macintosh 3 blade. You can only visualize the tip of the epiglottis. How will you proceed?**

As before, you have anticipated the possibility of this situation and have alternative plans in place for intubation, but you will not simply try again with the same technique: Plan B is not more of Plan A! A common initial step is to apply external laryngeal pressure either yourself, watching the laryngoscopic view as you do, or with a skilled assistant. In any difficult situation, consider calling for help early; it is better to ask for help and not need it than it is to be in trouble and unable to get it. Next, change the head position, laryngoscope blade, or operator. In obese patients, ramping the head of the bed, by putting several blankets under the shoulders, and more under the head (or using a specialized pillow such as the Troop elevation device), can markedly improve the view. A straight blade (Miller) can sometimes lift the epiglottis more efficiently than the curved (Macintosh) blade. Always ensure you have a good mask airway between efforts. No one ever died from lack of intubation per se, but lack of ventilation can kill! Use of the LMA, even if you have not done so yet, can be lifesaving if mask ventilation becomes impossible. This technique is now a standard part of the ASA Difficult Airway algorithm (see Appendix A).

Your initial efforts are still yielding only a view of the epiglottis. You decide to use an alternative airway device to assist you. What are some of your options? In cases such as this, you can often successfully intubate the patient even without a view of the vocal cords. Some experienced anesthesiologists will attempt a blind pass of the stylet-angled endotracheal tube under the epiglottis. More frequently, an alternative device, such as the Bougie, is passed under the epiglottis first. One can often feel a clicking sensation as the tip brushes along the cartilage rings of the trachea. Then, an endotracheal tube can be passed over the Bougie into the trachea. Other options are to improve the view with different laryngoscopes. A video enhanced device, such as the GlideScope, Bullard laryngoscope, or C-Mac can display a better view
than a conventional laryngoscope because of the integration of a camera or fiber-optic port on the distal aspect of the laryngoscope blade. Still another option is to use a flexible fiber-optic bronchoscope with an endotracheal tube threaded over it to locate the glottis. The endotracheal tube is then threaded off the bronchoscope into the trachea. Still other options include use of the LMA for the case, intubation through the LMA with a fiber-optic technique or with the intubating LMA (which is specially adapted for passage of an endotracheal tube without the need for a fiber-optic scope), or even to awaken the patient and cancel the case. In this case, you have administered a nondepolarizing muscle relaxant, so you will need to continue to manage the airway until this drug can be reversed, many minutes from now. Most anesthesiologists have a favorite approach to situations such as these and it is generally prudent to use a technique that you are comfortable with, rather than attempting something unfamiliar in an emergency situation. For this reason, trainees should gain experience in elective situations with as many different devices and techniques as possible.

**Suggested Further Reading**


The Anesthesia Machine

Alvaro A. Macias

For maximum impact, it is recommended that the case study and questions found on page xxi are reviewed before reading this chapter.

Key Learning Objectives

- Understand the flow of gas from the central hospital supply to the patient
- Learn the key components of an anesthesia machine (vaporizers, flowmeters, breathing circuits, scavenger system, alarms)
- Understand the safety mechanisms incorporated into the anesthesia machine

The anesthesia machine is designed to receive gases from the hospital central supply, control their flow, vaporize volatile agents, and deliver a measured amount of gas to the breathing circuit. Modern machines utilize advanced electronics and integrated components to achieve these goals, while incorporating a number of important safety features that have been progressively engineered over the last several decades.

The Anesthesia Machine Components

The anesthesia machine can be divided into several subsystems, each with its own function and characteristics. The basic machine components are listed in Table 10.1. One way to look at the machine is in terms of the pressure of the gases inside each of its parts. By using this approach, one can divide the machine into a high pressure and a low pressure system (Table 10.2).

The high pressure system includes the components needed to take the gases from the wall to the flow control valve. The low pressure system takes the gases
from the flow control valve to the patient. This prevents high pressures (that could induce barotrauma, or lung damage) from being delivered to the patient. A modern day anesthesia machine and its major components is shown in Figure 10.1). Gases flow from the pipeline (or cylinder) into the machine, where they are directed through the fail-safe valve and into the flow control valve. From there, gases flow into the flowmeters, then into the vaporizers, and finally the anesthesia circuit and patient via the common gas outlet (see Fig. 10.2).

### Pipeline Inlets

Gases arrive from a central hospital supply via a pipeline system that connects to the anesthesia machine. The pipeline and the hoses are both color coded: green for oxygen, yellow for air, and blue for nitrous oxide. The hoses connect to the machine using a diameter-index safety system (DISS). The DISS (Fig. 10.3) is a noninterchangeable threaded connection that makes it physically impossible to attach an oxygen hose to any port other than an oxygen outlet, because the size and diameter of the wall connections and hose adapters are gas specific.
Cylinder Inlets
Gas cylinders use the pin index safety system (PISS, Figure 10.4) to prevent connection errors. On the back of the anesthesia machine, one can find places for at least one back-up gas cylinder (oxygen). As with the pipeline and the hoses, every cylinder is color coded to prevent errors. These cylinders are generally reserved for back-up use in case of a pipeline or central gas supply failure.
Figure 10.2 Flow arrangement of a basic two-gas anesthesia machine. (From “Check-out: A guide for Preoperative Inspection of an Anesthesia Machine,” 1987, American Society of Anesthesiologists. Used with Permission).

Figure 10.3 Diameter index safety system (Image Courtesy J. Ehrenfeld).
Cylinders come in a variety of standard sizes. The most commonly used in the operating room are E-cylinders. By understanding the physical properties of the gases stored in a cylinder (see Table 10.3), one can calculate the amount of gas left in a cylinder and how long the supply will last.

For example: if the pressure gauge on an oxygen cylinder reads 1,100 psi and you plan to deliver oxygen at a rate of 6 L/min → you could estimate that

<table>
<thead>
<tr>
<th></th>
<th>Oxygen</th>
<th>Air</th>
<th>Nitrous oxide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color of cylinder</td>
<td>Green</td>
<td>Yellow</td>
<td>Blue</td>
</tr>
<tr>
<td>Capacity had full</td>
<td>625 L</td>
<td>625 L</td>
<td>1,590 L</td>
</tr>
<tr>
<td>Pressure when full</td>
<td>2,200 psi</td>
<td>750 psi</td>
<td>1,800 psi</td>
</tr>
<tr>
<td>Physical state</td>
<td>Gas</td>
<td>Gas</td>
<td>Liquid and gas</td>
</tr>
</tbody>
</table>
there are 312 L remaining in the tank (1,100 psi/2,200 psi × 625 L). At 6 L/min, you could deliver oxygen for approximately 52 min (Table 10.3).

**Pressure Regulation**
Gases coming from the pipeline or central hospital supply have a wall pressure of 50–60 psi. A full oxygen tank delivers gas at 2,200 psi and a full nitrous oxide tank delivers gas at 745 psi. In order to ensure that a consistent and acceptable pressure is delivered to the patient, machines have pressure regulators incorporated into the gas flow. These regulators will drop cylinder gas pressures to 45–47 psi and pipeline pressures to no more than 50–60 psi. This allows gas to be preferentially taken from the central pipeline supply, rather than the cylinders, to ensure that cylinders are not unnecessarily drained. Finally, there is also a high-pressure relief valve for each individual gas that opens when pressure in the machine exceeds 95–110 psi.

**Fail-Safe System**
In the event of a failure in the oxygen supply, if the oxygen pressure (not flow) drops below a critical point, the supply of other gases will be interrupted and an alarm will sound. This is known as the *fail-safe system*. This system does not prevent against delivering hypoxic gas mixtures because if the oxygen pressure is normal, other gases can still be delivered. This is why inclusion of a working oxygen analyzer in the inspiratory limb of the breathing circuit and a proportioning system in the machine are critical.

**Flowmeters**
Flowmeters are the division line between the high pressure and the low pressure systems. The pipeline, cylinders, and gas lines – which all occur before the flowmeters – are considered part of the high-pressure circuit. Everything after the flowmeters – the vaporizers, breathing circuit, and common gas outlet – are considered part of the low pressure system. There are three types of flowmeters: variable-orifice, electronic, and constant-pressure.

Gas flow increases when the flow valve control is turned counterclockwise – delivering the amount of gas desired. It is worth mentioning that flowmeters are calibrated for the gas they deliver and are not interchangeable. The oxygen flowmeter is always downstream from all other flowmeters (far right in the US) to reduce the chances of delivering a hypoxic mixture should a leak occur within a flowmeter.
In order to prevent delivery of a hypoxic mixture of oxygen and nitrous oxide, all machines include an oxygen/nitrous oxide ratio controller that links the nitrous oxide flow to the oxygen gas flow. This guarantees a minimum oxygen concentration of 21–25%.

**Vaporizers**

The main function of the vaporizer is to vaporize the liquid volatile anesthetics before they reach the patient. All vaporizers are agent-specific and have concentration-calibrated dials that tightly control the amount of anesthetic gas delivered to the patient.

There are two types of vaporizers currently in use: variable-bypass (see Fig. 10.5) and electronic vaporizers. The variable-bypass vaporizer divides the fresh gas flow into two streams. One stream contacts the volatile agent and picks it up, whereas the other stream leaves the vaporizer unchanged. The two streams then merge as they leave the vaporizer and enter into the breathing circuit.

![Figure 10.5  Schematic of a variable-bypass vaporizer. (Reproduced with permission from Biomedical Engineering Handbook. Bronzino, J. Springer, 2000).](image-url)
Electronically controlled vaporizers are most commonly used for desflurane (because its low boiling point of 23.5°C (or 73.4°F) is very close to room temperature). They work by heating desflurane to a temperature of 39°C in order to create a constant vapor pressure. This particular vaporizer does not have fresh gas flow through it. Instead the vaporizer simply releases the amount desired and then mixes it with fresh gas.

All modern vaporizers (except for desflurane vaporizers) compensate for temperature and ambient pressure changes. This ensures that the same amount of agent will be delivered to the patient at all times. Note that these vaporizers do not compensate for changes in elevation or atmospheric pressure.

Because vaporizers are agent specific, it is critically important to fill up the vaporizer with the correct agent – or else an unanticipated concentration of agent may be delivered. In order to prevent misfilling, vaporizers are color-coded and have agent-specific keyed filling ports that only accept the correct key or straw.

Gas Outlet
The gas outlet transports fresh gas carrying volatile agent to the breathing circuit. In all anesthesia machines, there is an oxygen flush valve that provides a high flow of oxygen (40–55 L/min) and bypasses both the flowmeters and vaporizers. This high flow of oxygen “flushes” or fills the circuit with fresh oxygen that is free of volatile agent. Due to the fact that this valve bypasses all the machine’s regulatory systems, it has the potential to reach the patient with inappropriately high pressure. It is important to remember this, since if used improperly it can cause significant patient barotrauma (lung injury).

Alarms
Anesthesia machines include a number of alarms. Each will have a low pressure alarm that goes off when a set airway pressure is not reached in the circuit during positive pressure ventilation. It is the first alarm to go off when a disconnection occurs in the circuit.

The oxygen fail-safe monitor checks for the presence of low oxygen pressure within the system. If the pressure drops below a certain limit, the monitor sounds an alarm and shuts off the inflow of other gases – until the oxygen pressure is reestablished.

The oxygen sensor in the inspiratory limb of the breathing circuit checks the concentration of oxygen delivered to the patient and will alarm if the delivered FiO₂ drops below a set threshold. This is probably one of the most important monitors in the entire machine.
Waste-Gas Scavengers
Waste-gas scavengers prevent the operating room personnel from unnecessary exposure to volatile agents. The National Institute of Occupational Safety and Health (NIOSH) recommends limiting the room concentration of nitrous oxide to 25 parts per million (ppm) and halogenated volatile agents to 2 ppm. Each anesthesia machine has a port that connects to the hospital central suction and a vacuum control valve that should be adjusted to permit evacuation of 10–15 L of waste gas per minute. As with many systems there are some inherent hazards. If the system becomes occluded, it may deliver excessive positive pressure to the patient, increasing the risk of barotrauma. Conversely, if the system generates too much negative pressure, it may inadvertently suction out fresh gas from the patient and increase the amount of volatile agent needed.

Oxygen Flush Valve
The oxygen flush valve serves as safety device by allowing the anesthesiologist to deliver 100% oxygen directly to the breathing circuit at any given time. Looking carefully at Fig. 10.2, one can see that there is a direct communication between the oxygen source (pipeline or cylinder) to the common gas outlet, which bypasses the pressure regulator. When the valve is activated (by pressing the button located on the front of the machine) it allows oxygen to flow at the pressure delivered from the source, directly into the circuit and the patient's lungs. This action has the potential to cause barotrauma due to the high transmitted pressures.

Anesthesia Machine Checkout
As anesthesiologists, we rely heavily on our machine as a way of delivering fresh gases and anesthetic agents to our patients. In fact, machine failures can be potentially catastrophic or fatal. Because of this, it is critically important to perform a machine checkout at the start of each case. While many of the new machines do this checkout automatically, it is important to understand how each machine works and the potential steps one would take to troubleshoot a given problem should any kind of failure arise. For a set of sample machine specific checkout procedures, refer to the ASA web page http://www.asahq.org/clinical/checklist.htm.

Anesthesia Breathing Systems
It is important to realize that the breathing system is not a part of the anesthesia machine. The anesthesia machine ends at common gas outlet. In order to
deliver gases from the machine to the patient, we need a breathing system. Multiple designs have been developed and they are classified as closed, semi-closed, open, and semiopen. This classification is based on the presence or absence of unidirectional valves, a gas reservoir bag, rebreathing of exhaled gases, and ways to chemically neutralize the exhaled carbon dioxide coming from the patient.

The most common system in use these days is the **circle system**. The circle system (see Fig. 10.6) is composed of an inhalation check valve, inspiratory limb, Y-piece, expiratory limb, exhalation check valve and adjustable pressure limit (APL, or “popoff”) valve, reservoir bag, bag/vent selector switch, and a $\text{CO}_2$ absorber. This system itself can be classified as closed, semiclosed, and semiopen depending on how much fresh gas flow is used.

The circle system takes the gas delivered from the anesthesia machine via the common gas outlet and delivers it to the patient. Depending on the volume of the

![Figure 10.6 Anesthesia machine circle system flow schematic. (1) Fresh gas enters the circle system; (2) Inspiratory limb one-way valve; (3) Inspiratory tubing; (4) Patient y-piece; (5) Expiratory tubing; (6) Expiratory limb one-way valve; (7) Reservoir bag; (8) Adjustable pressure limiting (APL) Valve; (9) $\text{CO}_2$ absorbent (Image Courtesy J. Ehrenfeld).](image-url)
gas flow delivered to the patient, rebreathing of exhaled gases may occur. In order to prevent rebreathing of CO₂, unidirectional valves and a CO₂ neutralizing system are in place. These devices prevent any CO₂ rebreathing and in many cases reduce the total amount of inhaled anesthetic that must be put into the system.

**Humidifiers**

Because fresh gas delivered to a patient by an anesthesia machine bypasses all of the natural mechanisms which humidify inspired gases (i.e., nasal cilia and oral secretions), steps must be taken to prevent desiccation of the respiratory mucosa. This is accomplished by the use of humidifiers. These devices may be active or passive.

Passive humidifiers (the most commonly used) trap water released on exhalation such that some water will be added back to the fresh gas flow during the next inspired cycle. Active humidifiers contain a water chamber and add vaporized water to the inspiratory gas flow. While active humidifiers do not add any dead space to the circuit, passive humidifiers do.

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**Case Study**

You are working with your attending on a busy day. She tells you to go set up the room for your first case. You are familiar with the preparation of the airway equipment and have previously discussed the drugs you will be using. As you walk towards the OR, your attending calls out to you to “remember to check the anesthesia machine.” You walk into the OR and discover to your dismay that the machine is an older model that does not feature an automatic machine checkout like the more modern ones that you have been using.

(Note that this case will be easier if you have read the supplemental Internet material referenced in the chapter).

You begin by inspecting the hoses attached to the machine from the gas outlets on the wall. How can you tell if they are properly connected and functional? The gas lines are color coded, green for oxygen, yellow for air, and blue for nitrous oxide. You can check to make sure they are connected to the proper outlets on the wall and the machine, but it is extremely unlikely that
they could be misconnected. This is because they are diameter indexed and cannot be attached to the wrong outlet or inlet. You can inspect the pressure gauges on the anesthesia machine to ensure that there is adequate pressure in the lines (indicated by a green band).

**How can you tell if you have adequate backup gas supplies should the hospital supply fail?**

All anesthesia machines also have tanks of oxygen and usually nitrous oxide attached directly to the back of the machine to be used as backup supplies. You can open the valve on one of the oxygen tanks and inspect the pressure valve for tank pressure to ensure that you have an adequate supply. It is usually recommended that you have at least half a tank, which should register as 1,100 PSI, compared to a full tank at 2,200 PSI. This corresponds to a little more than 300 L of oxygen, enough for over an hour at 5 L per minute and much longer at low fresh gas flows.

**How can you test to make sure the machine will prevent administration of a hypoxic gas mixture?**

There are several safety mechanisms in the machine to ensure administration of adequate oxygen to prevent hypoxia. You can test the oxygen monitor’s accuracy by running 100% oxygen through the circuit and ensuring that it reads 100%, and you can place the sensor outside the circuit exposed to room air and make sure it reads 21%. This monitor should alarm if the mixture is hypoxic. There is also an interlock on the gas flow controls that should prevent you from setting a flow of nitrous oxide that is too high relative to the oxygen flow. You can turn on both gases and then decrease the oxygen flow; at some point the nitrous oxide flow should automatically decrease. Finally, there is a “fail-safe valve” that senses oxygen and nitrous oxide pressures and should turn off all other gases should oxygen pressure drop. You can test this valve by disconnecting the wall supply of oxygen while administering nitrous oxide (and with the oxygen tank turned off). The nitrous oxide flow should be turned off and an alarm should sound.

**Later you are doing the case, which began uneventfully. The patient is intubated and being mechanically ventilated. You note on the capnograph that there appears to be inspired CO₂. Given your understanding of the anesthesia machine, why might this be occurring (see Fig. 10.2)? Which of the causes should you have been able to pick up during the machine checkout?**
Inspired CO₂ implies that the circuit has either not successfully separated inspired and expired gas flows, or that the CO₂ absorber is not functioning properly. In particular, if the expiratory one-way valve is incompetent, then gas in the expiratory limb, which contains exhaled CO₂, could be inspired. Depending on fresh gas flow, the same phenomenon can occur if the inspiratory valve is incompetent. In this case, some of the exhaled gas can travel down the inspired limb rather than the expired limb, and if fresh gas does not “wash it out” before the next inspiration, the patient could breathe in some of this CO₂-containing gas. You should be able to detect malfunctioning valves on machine checkout. Different methods are suggested, but in essence one observes the functioning of the valves through their transparent covers during inspiration or expiration.

The other possibility is malfunctioning CO₂ absorbent. This canister contains granules that react chemically with exhaled CO₂ and remove it from the gas stream. The reaction turns a chemical indicator from white to purple, to indicate when the granules have become exhausted. Unfortunately, the granules will turn back to white after they dry out, so if an exhausted absorbent were left in place after the last case, you would not have detected this during checkout. You are off the hook!

Suggested Further Reading

ASA Machine Checklist.
http://www.asahq.org/clinical/checklist.htm


Chapter 11

Anesthesia Equipment and Monitors

Basem Abdelmalak and D. John Doyle

For maximum impact, it is recommended that the case study and questions found on page xxii are reviewed before reading this chapter.

Key Learning Objectives
- Learn the indications for applying the most common intraoperative monitors
- Understand the principles behind each of the key physiologic monitors
- Know the limitations associated with various types of monitoring

Introduction
Patient monitoring and the equipment to support it are vital to caring for patients in operating rooms, intensive care units, emergency departments, and in acute care settings. The process can be as simple as the periodic measurement of routine vital signs (blood pressure, heart rate, respiratory rate, temperature) or may involve techniques as advanced as placement of a pulmonary artery catheter. Patient monitoring also entails the interpretation of available clinical information to help identify or predict problems with a patient. Patient monitoring, thus, not only involves quantitative physiological measurements (such as respiratory rate), but also involves qualitative observations (e.g., observation of signs of patient distress such as agitation or diaphoresis). The process also involves inferring diagnoses. For example, unilateral chest rise may imply endobronchial intubation or a pneumothorax.

Visual and auditory surveillance is central to anesthesia monitoring, and involves many dimensions:
- Observing the patient’s color, respiratory pattern, accessory muscle use, and looking for movements, grimaces or unsafe patient positioning
- Observing the patient’s clinical data on intraoperative monitors
- Observing bleeding and coagulation at the surgical site (e.g., are the surgeons using many sponges or are they doing a lot of suctioning?)
- Monitoring the functioning of all lines to ensure that IV catheters have not infiltrated
- Conducting an anesthesia machine and workspace checkout

The importance of patient monitoring during anesthesia has been emphasized by the following policy statements from the American Society of Anesthesiologists (ASA):

**Table 11.1 ASA monitoring standards.**

| Standard 1: | Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics and monitored anesthesia care. Standard 2: During all anesthetics, the patient’s oxygenation, ventilation, circulation, and temperature shall be continually evaluated (where “continuous” means without interruption and “continually” means repeated regularly and frequently). |

Based on these principles, patients are monitored both by clinical observation (“look, listen, feel”) as well as by using specialized monitoring equipment (see Table 11.2). Most importantly, monitoring information of this kind can be useful in detecting various clinical problems. Some monitors (e.g., airway pressure) are usually built into the anesthesia machine. In addition, one should visually monitor the patient’s breathing pattern and color, look for signs of distress, etc.

**Table 11.2 Monitoring equipment typically employed in general anesthesia cases.**

<table>
<thead>
<tr>
<th>Equipment</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrocardiogram</td>
<td>Provides information about rate, rhythm, ischemia (ST Segments)</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Manual, automatic, arterial catheter</td>
</tr>
<tr>
<td>Pulse oximeter</td>
<td>Usually on fingertip or ear lobe</td>
</tr>
<tr>
<td>Capnograph</td>
<td>Especially in patients with an LMA or ETT</td>
</tr>
<tr>
<td>Oxygen analyzer</td>
<td>Part of anesthesia machine</td>
</tr>
<tr>
<td>Anesthetic agent concentration analyzer</td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>Usually esophageal or axillary</td>
</tr>
<tr>
<td>Precordial or esophageal stethoscope</td>
<td>Listen to heart sounds, breath sounds</td>
</tr>
<tr>
<td>Gas flows/spirometry</td>
<td>Part of anesthesia machine</td>
</tr>
<tr>
<td>Airway pressure monitor</td>
<td>Part of anesthesia machine</td>
</tr>
<tr>
<td>Airway disconnect alarm</td>
<td>Part of anesthesia machine</td>
</tr>
<tr>
<td>Peripheral nerve stimulator</td>
<td>Where appropriate</td>
</tr>
<tr>
<td>Urometer</td>
<td>Measure urine output where appropriate</td>
</tr>
</tbody>
</table>
**Blood Pressure Monitoring**

Manual blood pressure monitoring is easily achieved via auscultation of Korotkoff sounds as learned by every medical student. However, automatic blood pressure monitoring is more practical and is generally achieved via a technique known as *oscillometry*. Here, the cuff is inflated to a high pressure, then deflated slowly. Oscillations in the cuff pressure begin to be detected when the cuff pressure first falls below systolic pressure. As deflation continues, the mean blood pressure is identified as the cuff pressure at which the amplitude of the oscillations is the greatest. The oscillations then vanish as the diastolic pressure is approached (see Fig. 11.1).

![Recording of Cuff Pressure and Oscillations in Pressure](image)

**Figure 11.1** Sample blood pressure measurement using oscillometry. The peak oscillation corresponds to the Mean Arterial Pressure. (Reproduced with permission from *The Biomedical Engineering Handbook*, 2nd ed., Joseph D. Bronzino, Springer, 2000).
In most short cases, automatic blood pressure monitoring is done at least every 5 min. Many automatic blood pressure machines also have a “stat” mode where measurements are done immediately one after the other for a short period of time. Many anesthesiologists also put a manually operated blood pressure cuff on a second arm, in case the automatic blood pressure monitor fails to provide reasonable numbers. This may occur when the patient’s arms are tucked and the surgeon is leaning against the pressure cuff. This second cuff is usually placed on the arm with the pulse oximeter. When this cuff is inflated to the point that the pulse oximeter waveform is extinguished, the dial will indicate the patient’s systolic pressure. Alternatively, a second blood pressure cuff may be placed on a patient’s lower extremity.

In longer, complex cases or in very sick patients, invasive blood pressure monitoring via an arterial line is frequently utilized. Arterial lines are most often inserted into one of the radial arteries, although it may less commonly be placed in a brachial, femoral, ulnar, or dorsalis pedis artery. This method involves connecting an arterial catheter (usually a 20 gauge for adults) to an electronically amplified pressure transducer via a narrow fluid-filled tube (Fig. 11.2). This arrangement provides beat-to-beat pressure information that is helpful, for instance, in patients with poor ventricular function. In addition, since arterial blood gases are easily drawn from an arterial line, they can be particularly useful in patients with pulmonary disease or patients with acid–base disorders. Also, the ease with which blood samples can be sent for hemoglobin or glucose levels makes arterial lines useful in patients whose condition is changing rapidly over time or in which large blood losses are anticipated.

**Electrocardiographic Monitoring**

All anesthetized patients undergo electrocardiographic monitoring. This provides the clinician with three types of information: (1) heart rate, (2) cardiac rhythm, and (3) information about possible myocardial ischemia (via ST segment analysis). In addition, ECG monitoring can help assess the function of a cardiac pacemaker.

The most common electrocardiographic system used during anesthesia is a 5-electrode lead system. This arrangement (Fig. 11.3) allows for the recording of any of the six limb leads plus a single precordial (V) lead.
Pulse Oximetry

Pulse oximetry is a simple noninvasive method of monitoring arterial oxygen saturation (the percentage of hemoglobin (Hb) with oxygen molecules bound). The arterial saturation obtained in this manner is usually designated as \( \text{SpO}_2 \). A pulse oximeter consists of a probe attached to the patient’s finger, toe, or ear lobe, which is in turn attached to the main unit (Fig. 11.4). It measures the red (e.g., 660-nm wavelength) and infrared light (e.g., 940-nm wavelength) transmitted through and/or reflected by a given tissue. In most units, an audible tone occurs with each heart beat which changes pitch with the saturation reading.

A patient is generally said to be hypoxemic when the \( \text{SpO}_2 \) falls below 90%, a point usually corresponding to an arterial \( \text{PO}_2 \) of 60 mmHg. An important advantage of using a pulse oximeter is that it can detect hypoxemia well before the patient becomes clinically cyanotic. Note that pulse oximetry is required in all patients undergoing anesthesia. However, it is important to realize that pulse oximeters give no information about the level of arterial \( \text{CO}_2 \) and are therefore useless in assessing adequacy of ventilation in patients at risk of developing hypercarbic respiratory failure.
Pulse oximeter units are now available for well under $1,000. One newly developed pulse oximetry monitor is even capable of providing information regarding blood hemoglobin content, a feature expected to be very useful in many surgical patients.

Figure 11.3 Location of the five electrodes used in a typical intraoperative electrocardiographic monitoring setup. (Reproduced with permission from American Heart Association. Drew, B. J. et al. Practice Standards for Electrocardiographic Monitoring in Hospital Settings. Circulation 2004;110:2721–2746).
Capnography and Ventilation Monitoring

Capnography (see Fig. 11.5a–e) is the continuous analysis and recording of carbon dioxide (CO$_2$) concentrations in respiratory gases. A capnograph uses one of two types of analyzers: mainstream or sidestream. Mainstream units insert a sampling window into the breathing circuit for gas measurement, while the much more common sidestream units aspirate gas from the circuit and the analysis occurs away from the circuit. Capnographs may utilize infrared technology (most commonly), or other techniques such as mass spectroscopy, Raman scattering, or photoacoustic technology.

It is useful to monitor capnography for a number of important clinical situations:

- Detecting when an anesthetic breathing circuit disconnects
- Verification of endotracheal intubation (a sustained normal capnogram is not obtained when the endotracheal tube ends up in the esophagus)
- Assisting in the detection of hypoventilation (raised end-tidal CO$_2$ is often present) and hyperventilation (low end-tidal CO$_2$ is often present)
Figure 11.5  (a) Normal Capnogram. The CO₂ level at point D (end-tidal CO₂) is normally around 40 mmHg. As shown above, the capnogram has four segments that correspond to phases of the respiratory cycle. The first phase (AB) is a flat part due to exhalation of dead space. It should always fall to zero; otherwise rebreathing is occurring. The second phase (BC) is the ascending segment from exhalation of mixed dead space and alveolar air. The third phase (CD) is the plateau portion that represents exhaled CO₂ from the alveoli. The fourth phase (DE) represents the beginning of inspiration. (Used with permission from Oridion Medical, Inc. www.oridion.com).

(b) Hyperventilation. The end-tidal CO₂ here is substantially less than 40 mmHg. Hyperventilation is sometimes used as a means to reduce intracranial pressure in head-injured patients. (Used with permission from Oridion Medical, Inc.).

(c) Hypoventilation. The end-tidal CO₂ here is substantially greater than 40 mmHg. In this instance, this is due to a low respiratory rate, as might occur in a patient breathing spontaneously with opiate analgesics in use. (Used with permission from Oridion Medical, Inc.).

(d) Rebreathing. In this instance, the CO₂ concentration never falls to zero. A common cause of this is exhausted CO₂ absorbent (e.g., soda lime) in the anesthesia machine patient breathing circuit. This may be corrected by increasing the fresh-gas flow rate. (Used with permission from Oridion Medical, Inc.).

(e) A notch in the plateau ("a curare cleft") is an indication of a spontaneous respiratory effort during mechanical ventilation.
Detecting rebreathing of CO₂ (in which case the inspiratory CO₂ level is nonzero)

Detecting capnograph tracings suggestive of COPD (where no plateau is present in the capnogram)

Monitoring CO₂ elimination during cardiac arrest and CPR (the capnogram “improves” as pulmonary blood flow improves with adequate circulation)

Capnography is also important in monitoring ventilation in sedated or anesthetized patients, because mere observation of chest movement and (especially) skin color are often deceiving. Reliably estimating the degree of chest excursion by visual means is often difficult, and observation of cyanosis (dusky, bluish skin coloration) provides only a late warning of the presence of hypoxemia. By contrast, the use of capnography usually provides clinicians with **reliable respiratory rate data** and helps with the early detection of obstructed ventilation, hypoventilation, or apnea. Another point to remember is that since capnography measures ventilation, it will alert the caregiver to adverse ventilatory events well before a pulse oximeter signals an alarm. This is particularly true since patients receiving oxygen can have arterial saturation levels that are completely acceptable despite the patient having extreme hypercarbia.

Note that a sudden, severe decrease in end-tidal CO₂ is sometimes due to a catastrophic cardiorespiratory event such as circulatory arrest, a large pulmonary embolus, or severe hypotension (possibly from extreme blood loss or compression of an inferior vena cava).

In addition to capnography, mechanical ventilation can also be monitored through vigilant monitoring of tidal volumes (Vₜ) and airway pressures. Tidal volumes may change as the patient’s condition changes – for example, when pressure-controlled ventilation mode is used, the Vₜ is inversely related to total chest compliance (lung and chest wall). Nonpatient related factors, such as circuit leaks and partially inflated tube cuff, can cause a decrease in Vₜ being delivered.

Airway pressures (peak and plateau) can also change secondary to patient factors. For example, when volume controlled ventilation is used, the airway pressures are inversely related to the total chest compliance. Nonpatient factors such as tube kinks and mucous plugs can cause an increase in airway pressures.

**Monitoring Muscle Relaxation**

Muscle relaxation, or paralysis using neuromuscular blocking agents such as vecuronium is often required during surgery. For instance, muscle relaxation may be needed to facilitate tracheal intubation, to allow abdominal closure, or
to ensure that no movement occurs during neurosurgery. In such settings, neuromuscular blockade monitoring or “twitch monitoring” is employed. This usually involves electrode placement at the ulnar or facial nerve, with use of a peripheral nerve stimulator (see Fig. 11.6). The nerve stimulators are usually used in one of two modes: A “train of four” (TOF) high-voltage stimulation pulses spread over 2 s (the more commonly used mode) or rapid tetanic stimulation at 50 (or sometimes 100) stimulation pulses per second. The observed “twitch” response to either stimulus sequence allows the anesthesiologist to determine the degree of muscle paralysis. In addition, neuromuscular blockade

![Figure 11.6](image-url)  
monitoring is usually used toward the end of the surgical procedure to assess both the suitability of the patient for reversal of muscle relaxation utilizing reversal agents such as neostigmine, as well as a few minutes later to assess the degree to which reversal has been successful.

**Monitoring the Depth of Anesthesia**

While monitoring muscle relaxation is easy using “twitch” monitors, measuring the degree of unconsciousness during general anesthesia is not. Some clinical techniques that anesthesiologists use to help gauge the depth of anesthesia include noting patient movements or grimacing, measuring the end-tidal anesthetic gas concentration, and following the blood pressure and heart rate trends. In addition to these classical methods, several electronic indices of brain function are available. Among these are the Bispectral Index, EEG Entropy, Patient State Index, and others. Among these, the Bispectral Index (BIS) (Fig. 11.7) is by far the best validated and most commonly used technique to monitor patient unconsciousness.

Figure 11.7  Bispectral Index (BIS) monitoring system. Left: electrode assembly. Right: monitor showing a BIS score of 52 with the raw electroencephalogram shown on the upper right and the processed signal shown on the bottom.
BIS is a processed EEG (electroencephalography) parameter, a measure of electrical activity in the brain. BIS provides a quantifiable measure of the effects of anesthetic agents on the central nervous system, and can be related to the hypnotic component of the anesthetic state. A dimensionless number is used, ranging from 0 to 100, with 0 being complete brain electrical silence and 100 representing a fully awake EEG. A BIS index between 40 and 60 is usually regarded as corresponding to adequate surgical anesthesia. Recent studies have shown that BIS may not prevent intraoperative awareness and that monitoring end-tidal anesthetic gas concentrations may be just as effective.

**Temperature Monitoring**

Normal core temperature in humans usually varies between 36.5 and 37.5°C and typically decreases 0.5–1.5°C following the induction of general anesthesia. Heat loss is due to impairment of thermoregulatory control by anesthetic agents combined with exposure to the cold operating room environment. When the temperature drop is large, hypothermia may occur. Hypothermia is defined as a core body temperature of less than 35°C and may be classified as mild (32–35°C), moderate (28–32°C), or severe (<28°C). Although mild hypothermia is sometimes desirable in head-injured patients, under other conditions the adverse effects of hypothermia (e.g., impaired cardiac contractility, impaired cardiac conduction, impaired blood clotting, increased postoperative infection) may present undesirable clinical problems. Reductions in core temperature are particularly likely in patients undergoing abdominal or thoracic surgery, unless special precautions such as forced-air warming systems are used. Finally, malignant hyperthermia (see Appendix B) remains a theoretical risk in all patients undergoing general anesthesia. While a rise in core temperature is not typically the first sign, it occurs due to a hypermetabolic state associated with malignant hyperthermia.

Core temperature can be measured with sensors in the nasopharynx, esophagus, pulmonary artery, tympanic membrane, or even in the rectum or urinary bladder. Skin-surface temperature tends to run much lower than core temperature, but follows core temperature trends fairly well. The ASA standards for patient monitoring require that every patient receiving anesthesia have temperature monitoring “when clinically significant changes in body temperature are intended, anticipated, or suspected.”
Central Venous Pressure Monitoring

Central venous catheters are commonly placed percutaneously into the right internal jugular vein as well as via a number of other sites that lead to the superior vena cava and right atrium. These catheters are generally inserted for one of two reasons: (1) to establish vascular access for cases likely to involve a high degree of blood loss, and (2) to allow the determination of central venous pressure (right-sided cardiac preload). These catheters can also be useful to suction out air from the heart in a case of air embolus. In addition to providing an overall measure of central venous pressure, the pressure waveforms provided by a central venous catheter yield a great deal of information and are shown in Fig. 11.8.

![Central Venous Pressure Waveform](image)

Figure 11.8  The central venous pressure waveform. +a wave: This wave is due to the increased atrial pressure during right atrial contraction. It correlates with the P wave on an ECG. +c wave: This wave is caused by a slight elevation of the tricuspid valve into the right atrium during early ventricular contraction. It correlates with the end of the QRS segment on an ECG. −x descent: This wave is probably caused by the downward movement of the ventricle during systolic contraction. It occurs before the T wave on an ECG. +v wave: This wave arises from the pressure produced when the blood filling the right atrium comes up against a closed tricuspid valve. It occurs as the T wave is ending on an ECG. −y descent: This wave is produced by the tricuspid valve opening in diastole with blood flowing into the right ventricle. It occurs before the P wave on an ECG. (Used with permission. From Surgery: Basic Science and Clinical Evidence. Jeffrey A. Norton, et al. Springer, 2008.).
Pulmonary Artery Pressure Monitoring

Pulmonary artery catheters (see Fig. 11.9) are passed into the pulmonary artery via the right atrium, right ventricle and pulmonary valve. Often called a “Swan-Ganz” catheter after the device’s inventors, it is equipped with an inflatable balloon at the tip which “floats” along with the catheter as it ultimately “wedges” into position in a small pulmonary vessel. The device has at least two lumens: one for CVP measurements and one for PA pressure measurements. In addition, while all have a means to measure cardiac output via thermodilution, some can also be used for cardiac pacing, for mixed venous oximetry or for other specialized purposes. Table 11.3 shows the data typically obtainable using a PA catheter. Table 11.4 shows hemodynamic profiles of common clinical diagnoses depending mainly on data obtained from the PA catheter.

![A typical pulmonary artery catheter.](image)
Other Special Patient Monitors
In addition to the patient monitors discussed above, special clinical situations often dictate that specialized monitors be employed. Examples include spinal cord function monitoring during spinal surgery (both sensory and motor evoked potential types), specialized coagulation monitoring during cardiac surgery or liver transplant surgery (e.g., via thromboelastography), transesophageal echocardiography to assess heart function, and so on.

Other Anesthesia Equipment
While a good deal of anesthesia equipment is related to patient monitoring, some is used for other purposes. This includes the anesthesia machine (discussed in Chap. 10), equipment for airway management (discussed in Chap. 9), and equipment used to warm patients (e.g., forced air warmers and fluid warmers).

<table>
<thead>
<tr>
<th>Table 11.3 Data typically obtained using a PA catheter.</th>
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<tbody>
<tr>
<td>Formula</td>
</tr>
<tr>
<td>CO</td>
</tr>
<tr>
<td>CI</td>
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<tr>
<td>SV</td>
</tr>
<tr>
<td>SI</td>
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<td>LVSW</td>
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<tr>
<td>RVSW</td>
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<tr>
<td>SVR</td>
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<tr>
<td>PVR</td>
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</table>

CO is the cardiac output, the blood ejected per minute from the heart. BSA is the body surface area, typically around 2 m² for adults. CI is the cardiac index. Systemic vascular resistance (SVR) is the vascular resistance (afterload) that the left ventricle works against. Pulmonary vascular resistance (PVR) is the resistance that the right ventricle works against. Left ventricular stroke work (LVSW) is the amount of work that the left ventricle does with each beat, and is a rough indicator of left ventricular contractility. Right ventricular stroke work (RVSW) is the amount of work that the right ventricle does with each beat. Stroke volume (SV) is the amount of blood ejected with each heart beat.
Standards for Basic Anesthetic Monitoring

Committee of Origin: Standards and Practice Parameters
(Standards for Basic Anesthetic Monitoring, approved by the ASA House of Delegates on October 21, 1986, and last amended on October 25, 2005, is reprinted with permission of the American Society of Anesthesiologists, 520N. Northwest Highway, Park Ridge, IL 60068-2573).

These standards apply to all anesthesia care although, in emergency circumstances, appropriate life support measures take precedence. These standards may be exceeded at any time based on the judgment of the responsible anesthesiologist. They are intended to encourage quality patient care, but observing them cannot guarantee any specific patient outcome. They are subject to revision from time to time, as warranted by the evolution of technology and practice. They apply to all general anesthetics, regional anesthetics, and monitored anesthesia care. This set of standards addresses only the issue of basic anesthetic monitoring, which is one component of anesthesia care. In certain rare or unusual circumstances, (1) some of these methods of monitoring may be clinically impractical and (2) appropriate use of the described monitoring methods may fail to detect untoward clinical developments. Brief interruptions of continual† monitoring may be unavoidable. These standards are not intended for application to the care of the obstetrical patient in labor or in the conduct of pain management.

Standard I

Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics, and monitored anesthesia care.

Objective

Because of the rapid changes in patient status during anesthesia, qualified anesthesia personnel shall be continuously present to monitor the patient and provide anesthesia care. In the event there is a direct known hazard, e.g., radiation, to the anesthesia personnel, which might require intermittent remote observation of the patient, some provision for monitoring the patient must be made. In the event that an emergency requires the temporary absence of the person primarily responsible for the anesthetic, the best judgment of the anesthesiologist will be exercised in comparing the emergency with the anesthetized patient’s condition and in the selection of the person left responsible for the anesthetic during the temporary absence.
Standard II
During all anesthetics, the patient’s oxygenation, ventilation, circulation, and temperature shall be continually evaluated.

Oxygenation

Objective
To ensure adequate oxygen concentration in the inspired gas and the blood during all anesthetics.

Methods
1. Inspired gas: During every administration of general anesthesia using an anesthesia machine, the concentration of oxygen in the patient breathing system shall be measured by an oxygen analyzer with a low oxygen concentration limit alarm in use.*
2. Blood oxygenation: During all anesthetics, a quantitative method of assessing oxygenation such as pulse oximetry shall be employed.* When the pulse oximeter is utilized, the variable pitch pulse tone and the low threshold alarm shall be audible to the anesthesiologist or the anesthesia care team personnel.* Adequate illumination and exposure of the patient are necessary to assess color.*

Ventilation

Objective
To ensure adequate ventilation of the patient during all anesthetics.

Methods
1. Every patient receiving general anesthesia shall have the adequacy of ventilation continually evaluated. Qualitative clinical signs, such as chest excursion, observation of the reservoir breathing bag, and auscultation of breath sounds, are useful. Continual monitoring for the presence of expired carbon dioxide shall be performed unless invalidated by the nature of the patient, procedure, or equipment. Quantitative monitoring of the volume of expired gas is strongly encouraged.*
2. When an endotracheal tube or laryngeal mask is inserted, its correct positioning must be verified by clinical assessment and by identification of
carbon dioxide in the expired gas. Continual end-tidal carbon dioxide analysis, in use from the time of endotracheal tube/laryngeal mask placement, until extubation/removal or initiating transfer to a postoperative care location, shall be performed using a quantitative method such as capnography, capnometry or mass spectroscopy.* When capnography or capnometry is utilized, the end tidal CO₂ alarm shall be audible to the anesthesiologist or the anesthesia care team personnel.*

3. When ventilation is controlled by a mechanical ventilator, there shall be in continuous use a device that is capable of detecting disconnection of components of the breathing system. The device must give an audible signal when its alarm threshold is exceeded.

4. During regional anesthesia and monitored anesthesia care, the adequacy of ventilation shall be evaluated by continual observation of qualitative clinical signs and/or monitoring for the presence of exhaled carbon dioxide.

### Circulation

#### Objective

To ensure the adequacy of the patient’s circulatory function during all anesthetics.

#### Methods

1. Every patient receiving anesthesia shall have the electrocardiogram continuously displayed from the beginning of anesthesia until preparing to leave the anesthetizing location.*

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Blood pressure</th>
<th>CVP</th>
<th>CO</th>
<th>CI</th>
<th>PCWP</th>
<th>Pulmonary artery diastolic pressure</th>
<th>SVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemia</td>
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<td>⇓</td>
<td>⇓</td>
<td>⇓</td>
<td>⇓</td>
<td>⇓</td>
<td>⇑</td>
</tr>
<tr>
<td>Cardiogenic Shock</td>
<td>⇓</td>
<td>⇑</td>
<td>⇓</td>
<td>⇓</td>
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<td>⇑</td>
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<tr>
<td>Septic shock</td>
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<td>⇑</td>
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<td>⇓</td>
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<tr>
<td>Neurogenic shock</td>
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<td>⇓</td>
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<tr>
<td>Tamponade</td>
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<td>⇓</td>
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</tr>
</tbody>
</table>

β = Low; Ý = High; Þ = No change

*CVP central venous pressure, CO cardiac output, CI cardiac index, PCWP pulmonary capillary wedge pressure, SVR systemic vascular resistance.
2. Every patient receiving anesthesia shall have arterial blood pressure and heart rate determined and evaluated at least every 5 min.*

3. Every patient receiving general anesthesia shall have, in addition to the above, circulatory function continually evaluated by at least one of the following: palpation of a pulse, auscultation of heart sounds, monitoring of a tracing of intra-arterial pressure, ultrasound peripheral pulse monitoring, or pulse plethysmography or oximetry.

**Body Temperature**

**Objective**

To aid in the maintenance of appropriate body temperature during all anesthetics.

**Methods**

Every patient receiving anesthesia shall have temperature monitored when clinically significant changes in body temperature are intended, anticipated, or suspected.

* Under extenuating circumstances, the responsible anesthesiologist may waive the requirements marked with an asterisk (*); it is recommended that when this is done, it should be so stated (including the reasons) in a note in the patient’s medical record.

† Note that “continual” is defined as “repeated regularly and frequently in steady rapid succession,” whereas “continuous” means “prolonged without any interruption at any time.”

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**Case Study**

(Editor’s note: this case is primarily about monitoring, though figuring out the entire scenario will require your knowledge from other chapters).

You are providing anesthesia for a healthy young woman having a laparoscopic tubal ligation, your last case of a busy day of short gynecology cases. You induced anesthesia with propofol and succinylcholine and artfully intubated the woman’s trachea. You have maintained anesthesia with sevoflurane and fentanyl. The case is now over and you are preparing to wake the patient up. You have discontinued sevoflurane, increased oxygen flows, and have expected to see the patient open her eyes by now.
She remains apneic (ventilator dependent, no spontaneous respirations), unresponsive to verbal stimuli, and does not react when you suction her mouth. Your attending asks why we are not already on our way to the PACU.

How do you know she is apneic? Which monitors can verify this for you?
Several monitors and physical examination techniques are helpful in assessing ventilation. First, do not forget good old-fashioned auscultation! A stethoscope, placed either over the lung fields externally, or with a weighted bell precordially, or in the esophagus, can detect breath sounds, among other things. Second, you can turn off the ventilator, turn the selector to the reservoir bag, and observe the bag for motion. Third, you can check the capnogram during this same time, watching for exhaled carbon dioxide indicative of spontaneous respiration. Fourth, you can check the expired tidal volume monitor. This device uses one of a variety of physical principles to measure bulk flow of gas (such as a spinning propeller, a hot wire cooled by airflow, or the pressure drop across a mesh resistor). Finally, the airway pressure monitor can detect changes in the circuit pressure indicative of respiratory movements. If all of these demonstrate no flow, you can be certain that the patient is apneic. You should still observe the chest to make certain that the patient is not making respiratory efforts against an obstructed airway!

You conclude that the patient is indeed apneic. Two minutes into your examination, the pulse oximeter shows the saturation to be 99%. How is this possible? Do you suspect a malfunction?
The pulse oximeter is not a ventilation monitor! Desaturation during apnea takes some time, particularly if the patient has been breathing 100% oxygen for some time. In fact, this is exactly the principle behind “preoxygenation” or “denitrogenation” prior to induction of anesthesia. A well-oxygenated patient will remain saturated for 4 or more minutes in the absence of cardiopulmonary disease or other physiologic abnormalities affecting oxygen consumption or functional residual capacity (pregnancy, obesity). It is likely that the monitor is not malfunctioning. You can verify that it is picking up an arterial pulse signal by inspecting the display and comparing the pulse rate to the ECG rate.
How can you tell if you have allowed enough time for the anesthetics to be eliminated?
You can check the end-tidal agent monitor. Most modern operating rooms have such a monitor, most commonly one based on infrared absorption of light by inhaled anesthetics. Other technologies in less frequent use at the present time are mass spectroscopy and Raman spectroscopy. If the concentration of expired sevoflurane has decreased to 0.1–0.3 MAC (the “MAC awake,” about 0.2–0.5% for sevoflurane), it is likely that you have washed most of this anesthetic out. It is more difficult to assess the presence or absence of fentanyl. In spontaneously breathing patients, you can assess opioid effect by measuring respiratory rate, which will be slow in a “narco-tized” patient. You can inspect the pupils, who will generally be pinpoint in a patient with substantial opioid concentrations, but this sign can be unreliable in the presence of inhaled agents.

Although you believe that enough time has indeed elapsed, you would like to confirm whether or not she is “asleep.” What other monitors can help you?
First, do not forget to use your own eyes! **Look at the patient** for signs of arousal: grimacing, tearing, patient movement, rapid shallow breathing. Next, you can interpret basic hemodynamic data in comparison to the patient's preoperative and intraoperative vital signs. A deeply anesthetized patient should have blood pressure and heart rate similar to the period during the operation at times of light or no surgical stimulation. A “light” patient will often show increasing heart rate and blood pressure, signs of sympathetic activation. Of course, patients taking beta blockers or who have received heavy doses of opioids may not demonstrate these signs. Finally, you can use a consciousness monitor analyzing the processed EEG, such as the bispectral index (BIS) or patient state index (Sedline), to measure the degree of brain sedation.

On the basis of these investigations, you are convinced that the patient’s anesthetics have been eliminated, and that she is not anesthetized. What else might explain her failure to awaken? What monitor could help you verify the diagnosis?
The remaining drug class that you have not explored is the neuromuscular blocking agents. You intubated this patient using succinylcholine and did not use other relaxants. Normally this drug is eliminated by plasma
cholinesterase in 5–8 min, but in rare individuals with an atypical enzyme, the effect can be vastly prolonged. In this case, the patient would exhibit signs of lightness (hemodynamic stimulation, tearing, absence of end-tidal anesthetic, brain activity compatible with consciousness on EEG) but not move. You can verify the diagnosis by placing a neuromuscular blockade (“twitch”) monitor and demonstrating absence of twitch in response to train-of-four stimulation. Be cautious about using tetanic stimulation, which is painful, in this potentially “awake” patient. If you find her to be paralyzed but potentially conscious, you should immediately reassure her and explain that she will need to stay intubated until the drug wears off. You should sedate her with a short acting drug, such as midazolam or propofol, to keep her comfortable until the succinylcholine wears off, which may take several hours.

**Suggested Further Reading**


Intraoperative Considerations
Chapter 12

Anesthetic Techniques: General, Sedation, MAC

Brian C. McLean and Anthony R. Plunkett

For maximum impact, it is recommended that the case study and questions found on page xxiii are reviewed before reading this chapter.

Key Learning Objectives

- Learn how to prepare for the different phases of an anesthetic
- Understand the continuum of sedation
- Discuss the advantages and disadvantages of different anesthetic techniques

The Anesthesiologist and the Airline Pilot

A common analogy compares the job of an anesthesiologist to that of an airline pilot. This analogy is fitting in that each professional is charged with peoples’ lives – failure to perform the job appropriately and consistently can result in death or injury of those in their care. This analogy is also fitting because in doing their job, each professional must faithfully perform a set of key steps.

Preflight Check

The preflight check is performed prior to the pilot allowing any passengers onto the plane. This preflight check is analogous to the preanesthetic setup and machine check. Both the pilot and anesthesiologist must ensure that their equipment is ready and in optimal operating conditions – before patients or passengers are allowed to board or enter into the operating room.
Our preflight check starts at the beginning of the day with our initial room setup. In preparing a room, most anesthesiologists will use the mnemonic **M.S.M.A.I.D.S.** (Table 12.1) just as a pilot will use a written checklist to make sure that nothing is missed. The seven individual components of the mnemonic are outlined in the discussion below.

The first “M” stands for the anesthesia **Machine**. In performing a machine check, one should use a written check list in order to ensure that nothing is overlooked. A typical machine check will include:

1. Assure an adequate source of gases is coming from the wall
2. Ensure an alternative source of oxygen (E-cylinder) is attached to the back of the anesthesia machine and that it is full
3. Calibrate the oxygen sensor
4. Make sure fail-safe alarms are working
5. Check the level of volatile agent in the machine vaporizers
6. Perform a high pressure test
7. Perform a low pressure test
8. Make sure ventilator bellows are working

Suction is a vital part of any room setup. It is imperative that suction be present and powerful enough to quickly evacuate any secretions in the oropharynx if they are present on induction – as this can improve the anesthesia provider’s view of the airway structures and help avoid aspiration of gastric contents. Prior to bringing a patient into the operating room, the anesthesiologist should ensure that there is an adequate source of suction available and that it will reach the patient.

The second “M” of the mnemonic reminds an anesthesia provider to prepare the standard American Society of Anesthesiologists recommended monitors as well as to consider if additional or invasive monitoring is necessary. Minimum monitoring requirements (see Chap. 11) include pulse oximetry, blood pressure, ECG, and capnography.
The Airway part of the mnemonic is vital to ensure that the necessary airway equipment is present and in good working order. If there is a possibility that the patient may have a difficult airway, emergency airway equipment or a difficult airway cart should be readily available. The minimum airway set up should include a working laryngoscope with at least two types and sizes of blades. An endotracheal tube of appropriate size should also be available and the endotracheal cuff should be tested to ensure that it is patent.

The "IV" portion of the mnemonic is a cue to consider how much intravenous access will be necessary for a given case. The degree of access required is determined by the expected blood loss and intraoperative fluid requirements. For patients, you may also need fluid warmers, pressure bags, rapid infusers, or even central venous access. Again, ideally these considerations should be made before the case begins.

The anesthesiologist must have an adequate supply of Drugs. This includes medications necessary to induce and maintain anesthesia, as well as emergency medications should the patient require vasoactive, inotropic, or chronotropic support. Typically, succinycholine, atropine, ephedrine, and phenylephrine are drawn up and available in addition to standard induction drugs (propofol, fentanyl).

The final “S” of the mnemonic encompasses all other considerations about the case such as padding, positioning, or other Special equipment.

As a part of this “pre-flight checklist,” the anesthesia provider should also carefully consider the preoperative assessment of the patient and administer any preoperative medications that might be appropriate given the patient’s comorbidities. Typical preoperative medications might include antibiotics, sedatives for anxiolysis, antiemetics for patients at risk of post-operative nausea, and antacids for patients at high risk of gastric aspiration.

**Takeoff**

The two most difficult and dangerous times for a pilot come during takeoff and landing – this corresponds to induction and emergence during anesthesia. Both the pilot and the anesthesiologist work hard to ensure a safe and smooth takeoff and landing.

Prior to induction, the anesthesiologist will apply monitors to the patient. After confirming that the patient is appropriate for anesthesia and that all of the monitors are working, the anesthesiologist will preoxygenate the patient by having them inhale 100% oxygen through a sealed mask. The purpose of preoxygenation is to replace the nitrogen that is in the patient’s lungs with oxygen – as well as to maximally oxygenate all of the patient’s vital organs prior to induction. This essential step is a safety measure, which will help ensure that
the patient is best able to tolerate any period of apnea from the time of anesthetic induction to the time when the airway is secured.

After the patient is maximally oxygenated, the anesthesiologist will induce anesthesia in the patient, usually with a combination of sedative hypnotics and analgesic drugs. After medications are given, the anesthesiologist will check for a lid-lash reflex by brushing a finger gently across the eye lashes. If no blink reflex is elicited, a mask airway will then be established by applying gentle positive pressure to the breathing circuit. Only after a mask airway has been established will paralytic agents then be administered to allow further manipulation of the airway. With the airway secured, the patient can then be properly positioned for surgery, prepped, and draped. Prior to surgical incision, a “time-out” or “hard stop” should be performed to verify that the correct procedure is about to be undertaken on the correct patient.

Cruising Altitude
Once a plane has reached altitude, many people think that the pilot can just turn on the auto-pilot and take a nap – but this is simply not true. The pilot and co-pilot must remain vigilant, constantly check the instruments, and communicate with the air traffic controllers to avoid a mishap. Similarly, during the maintenance phase of anesthesia, although on the surface it may appear that nothing is happening, the anesthesiologist must remain as vigilant as ever. The needs of a patient during the maintenance portion of an anesthetic may include fluid resuscitation, adjustment of the anesthetic and analgesic agents, monitoring of the patient’s blood pressure, heart rate and temperature, and paying attention to what is going on in the surgical field.

Landing
Landing a plane safely is the goal of every pilot just as a safe wake up and extubation is the goal of every anesthesiologist. Occasionally passengers on a plane will clap after a successful touchdown; similarly, our patients expect us to land them safely and comfortably. Depending on the patient, the anesthesiologist can choose to remove the endotracheal tube while the patient is still deeply asleep (Stage 3) or fully awake (Stage 1). Patients who have their airways manipulated during the intermediate Stage 2 of anesthesia are much more likely to suffer from laryngospasm and agitation than patients in either Stage 1 or Stage 3. There are multiple numerical endpoints that anesthesia providers
use to ensure that a patient is ready for extubation. If a patient is going to be extubated awake, he/she should be following commands, able to oxygenate and ventilate without assistance, and able to protect his/her airway. The four stages of general anesthesia are outlined in the Table 12.2.

**Table 12.2  Stages of general anesthesia.**

<table>
<thead>
<tr>
<th>Stage 1 – Amnesia</th>
<th>Patients should follow commands; Respiration pattern typically regular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 2 – Delirium</td>
<td>Period of uninhibited excitation; Patients at risk for laryngospasm; Pupils often divergent; Respirations often irregular</td>
</tr>
<tr>
<td>Stage 3 – Surgical anesthesia</td>
<td>Target depth for anesthesia during surgery; Respiration pattern typically regular</td>
</tr>
<tr>
<td>Stage 4 – Overdosage</td>
<td>Patients at risk for hypotension and cardiovascular collapse</td>
</tr>
</tbody>
</table>

**Taxi to the Terminal**
The taxi to the terminal and the post flight check list is analogous to the trip from the operating room to the post anesthesia recovery area (PACU). The anesthesia provider should be at the head of the bed continuously evaluating the patient and ready to support the airway if necessary. Once in the PACU, the anesthesiologist will give a report to the PACU nurse and turn the care of the patient over to the PACU staff. Orders should be written to prepare for potential postoperative problems, such as pain, post operative nausea and vomiting, hypoxia, and blood pressure and heart rate perturbations (see Chap. 27, Postoperative Care Unit and Common Postoperative Problems) (Table 12.3).

**Anesthetic Techniques**
Having outlined the basic sequence of a general anesthetic (Table 12.3), we will now turn to the different types of anesthetic techniques available to take a patient safely through surgery (also see Chap. 13, Regional Anesthesia). Keep in mind that there is no absolutely correct technique for any given procedure. The type of anesthesia administered will depend on the anesthesia provider, surgeon, and patient’s preferences and may be dictated by the type of surgery and/or patient co-morbidities. Some surgeries are minimally invasive and cause the patients little pain or psychological discomfort. In such cases, a surgeon may request to have an anesthesia provider present to monitor the patient and administer sedation while the procedure is being performed. This is called Monitored Anesthetic Care (MAC).
Table 12.3 Action sequence of a general anesthetic.

<table>
<thead>
<tr>
<th>Airplane analogy</th>
<th>Anesthesia tasks</th>
<th>Important points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preflight check</td>
<td>− Operating room setup</td>
<td>− M.S.M.A.I.D.S</td>
</tr>
<tr>
<td></td>
<td>− Preoperative patient evaluation</td>
<td>− Assessment of medical history</td>
</tr>
<tr>
<td></td>
<td>− Preoperative medications</td>
<td>− Confirm NPO status</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Obtain informed consent</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Obtain I.V. access</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Administer appropriate preoperative medications and/or anxiolysis</td>
</tr>
<tr>
<td>Takeoff</td>
<td>− Patient monitoring</td>
<td>− Place and confirm appropriate monitors</td>
</tr>
<tr>
<td></td>
<td>− Induction of anesthesia</td>
<td>− Position patient and pad pressure points</td>
</tr>
<tr>
<td></td>
<td>− Airway management</td>
<td>− Preoxygenate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Administer induction agent</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Place endotracheal tube or other advanced airway device</td>
</tr>
<tr>
<td>Cruising altitude</td>
<td>− Maintenance of anesthesia</td>
<td>− Protect patient eyes</td>
</tr>
<tr>
<td></td>
<td>− Maintenance of homeostasis</td>
<td>− Monitor vital signs and maintain appropriate blood pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Ensure amnesia and anesthesia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Monitor blood loss and administer appropriate fluids</td>
</tr>
<tr>
<td>Landing</td>
<td>− Antagonism of neuromuscular blockade</td>
<td>− “Reversal” of neuromuscular blockade</td>
</tr>
<tr>
<td></td>
<td>− Emergence/extubation</td>
<td>− Turn off anesthetic agents</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Ensure patient is awake, following commands, protecting airway and can ventilate and oxygenate adequately prior to extubation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Confirm stable vital signs</td>
</tr>
<tr>
<td>Taxi to the terminal</td>
<td>− Safe transfer to PACU</td>
<td>− Monitor airway</td>
</tr>
<tr>
<td></td>
<td>− PACU orders and discharge</td>
<td>− Maintain oxygenation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Confirm stable vital signs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Write appropriate order to treat pain, nausea, vomiting and hyper or hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>− Give report to PACU staff</td>
</tr>
</tbody>
</table>

Monitored Anesthesia Care (MAC)/Anesthesia Sedation

Monitored Anesthesia Care or MAC is not a technique of anesthesia but rather a descriptive term for an anesthetic service in which an anesthesiologist is requested to be present at a surgical or diagnostic procedure to monitor the patient and administer medications for anxiolysis, analgesia, or sedation. It may or may not involve sedation of the patient. It is appropriate here to discuss
the continuum of depth of sedation from minimal sedation to general anesthesia, as outlined in Table 12.4. The main point here is that the depth of sedation is a continuum, and sometimes it is difficult to categorize exactly what type of anesthesia the patient is getting. During the course of the procedure, the patient can easily slip from one type to the other.

All anesthetic techniques fall on a continuum and many are combined. On one side of the continuum is “sedation” (which progresses from minimal to deep) that is delivered during a typical MAC case. On the other end is “general anesthesia” during which patients are completely unarousable and are often, but not always, intubated.

Different anesthetic techniques can be combined and the anesthetic technique can be changed during the case. For example, an anesthesiologist may provide IV sedatives and hypnotics during a MAC case if the patient begins to have discomfort or pain. In addition, the anesthesia provider must always be prepared to convert to a general anesthetic if the patient cannot tolerate sedation alone – or becomes oversedated and requires ventilatory support. Also, some patients can have a regional anesthetic alone, while others may need a regional anesthetic (epidural, regional block) as well as a general anesthetic.

Anesthetic agents used to sedate patients are rapid-acting and can affect different patients in profoundly different ways based on the patient’s

<table>
<thead>
<tr>
<th>Table 12.4 ASA continuum of depth of sedation.</th>
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<tr>
<td><strong>Minimal sedation (anxiolysis and analgesia)</strong></td>
</tr>
<tr>
<td><strong>Responsiveness</strong></td>
</tr>
<tr>
<td><strong>Airway</strong></td>
</tr>
<tr>
<td><strong>Spontaneous ventilation</strong></td>
</tr>
<tr>
<td><strong>Cardiovascular function</strong></td>
</tr>
</tbody>
</table>

pharmacogenetics, age, sex, co-morbidities, and home medication regimen. An anesthesiologist must be prepared to rescue a patient who was intended to have minimal sedation, but ends up being deeply sedated. Similarly, the anesthesiologist must be able to convert from sedation to general anesthesia. Drugs commonly used during anesthesia sedation may include midazolam, propofol (sedation dose: 30–100 mcg/kg/min), ketamine, fentanyl, remifentanil, and dexmedetomidine (see Chap. 4, Table 4.7).

Choice of Anesthetic Technique

In choosing an appropriate level of sedation and anesthetic technique, the anesthesiologist evaluates:

1. the type of procedure
2. patient comorbidities/health status
3. the preference of the surgeon
4. the preference of the patient

The primary concerns when considering whether or not a patient can tolerate deep sedation and general anesthesia are the airway and cardiovascular status. For a patient with severely depressed cardiovascular function who is scheduled to undergo a procedure on a distal extremity, it may be wiser to choose an anesthetic technique other than general anesthesia which could further depress their cardiac function. Similarly, an anesthesiologist may choose general anesthesia for a healthy patient who is undergoing a procedure that normally only requires conscious sedation but has a full stomach or severe gastrointestinal reflux, in which case protection from aspiration of gastric contents is important.

The goal of an anesthetic is to allow a patient to tolerate a procedure with the least degree of discomfort and the greatest degree of safety. For minor procedures, this may mean injecting local anesthetic to block the transmission of pain and administering a benzodiazepine for anxiolysis. However, for major procedures that require patients to be completely immobile, their level of consciousness deeply depressed, and their muscles paralyzed, it will usually mean inducing general anesthesia.

General Anesthesia

General anesthesia implies the loss of consciousness and protective airway reflexes. A patient under general anesthesia will not respond purposefully to noxious stimuli. The main goals of general anesthesia are to provide adequate hypnosis, relaxation, amnesia, immobility, and analgesia. General anesthesia can be
induced and maintained with either intravenous medications or the inhalation of volatile anesthetics. Table 12.5 depicts the major components of a typical general anesthetic. Table 12.6 lists the common drug classes employed to achieve these components.

**Physiology of General Anesthetics**

**Sedative-hypnotic medications** such as propofol, etomidate, barbituates, and benzodiazepines all appear to have similar mechanisms of actions. These medications can be used for light sedation if given slowly and in small doses or can be used to induce general anesthesia if given in large bolus doses. Sedative-hypnotic agents act by binding to and activating GABA\textsubscript{A} receptor chloride channels in neuron transmembrane proteins. Activation of these receptors causes an influx of ions, results in cell hyperpolarization, and prevents depolarization. If a neuronal cell cannot depolarize, it is said to be inhibited and cannot send information. This is the neurobiological basis for the effect of these drugs. Sedative-hypnotics can cause sedation, loss of consciousness and amnesia, but in general are not effective at providing analgesia or inhibiting movement.

In contrast to sedative-hypnotic medications, **volatile anesthetics** can produce both loss of consciousness and inhibit movement. We still do not have a complete understanding of the mechanism of action of volatile anesthetics (also see Chap. 5, Pharmacology of Inhalational Anesthetics). There is no unified theory to explain how and why all volatile anesthetics work, but it is felt that they must act on the central nervous system as well as at the level of the spinal cord in order to produce amnesia, sedation, and inhibition of movement to noxious stimuli. Unlike neuromuscular blocking agents which bind to receptors at the neuromuscular endplates to prevent movement, volatile anesthetics are thought to work at the level of the spinal cord to inhibit purposeful and reflexive movement.

<table>
<thead>
<tr>
<th>Table 12.5 Important components of a general anesthetic.</th>
</tr>
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<tbody>
<tr>
<td>Hypnosis</td>
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<tr>
<td>Analgesia</td>
</tr>
<tr>
<td>Amnesia</td>
</tr>
<tr>
<td>Paralysis</td>
</tr>
<tr>
<td>Reflex blunting</td>
</tr>
<tr>
<td>Time of administration</td>
</tr>
<tr>
<td>-------------------------</td>
</tr>
</tbody>
</table>
| **Preoperative medications** | Anxiolysis | Benzodiazepines  
- Midazolam (Versed)  
- Diazepam (Valium) |
| Antacid | Nonparticulate |  
- Sodium citrate (Bicitra)  
Histamine blockers  
- Ranitidine (Zantac) |
| Beta blockade | Beta blockers |  
- Metoprolol |
| Analgesia | Opioids |  
- Fentanyl  
- Morphine, hydromorphone |
| **Induction** | Induction of anesthesia | GABA receptor agonists  
- Propofol (Diprivan)  
- Etomidate (Amidate)  
- Thiopental (Pentothal)  
NMDA receptor antagonists  
- Ketamine |
| Neuromuscular blockade | Neuromuscular blockers |  
- Succinylcholine  
- Vecuronium, cisatracurium  
- Rocuronium, pancurium |
| **Maintenance of anesthesia** | Volatile anesthetics | Volatile anesthetics  
- Sevoflurane  
- Desflurane  
- Isoflurane  
- Nitrous oxide |
| Intravenous anesthetics | IV Anesthetics |  
- Propofol  
- Ketamine |
| Antihypotensives | Sympathomimetics |  
- Ephedrine  
- Phenytolephrine |
| Analgesics | Opioids |  
- Morphine, hydromorphone  
- fentanyl, remifentanil,  
- sufentanil, alfentanil  
Other  
- Ketamine |
A common misconception is that general anesthesia requires a patient to have an endotracheal tube and artificial respiration. Patients that are not at risk for gastroesophageal reflux (GERD) and can maintain adequate oxygenation and ventilation while under anesthesia can be allowed to spontaneously breathe during an anesthetic, even while rendered unconscious by anesthetic drugs. Another common misconception is that general anesthesia is always maintained with a volatile gas anesthetic. General anesthesia can be induced and maintained with a variety of different medications. **Total Intravenous Anesthesia** (TIVA) has become increasingly popular as a general anesthetic technique. TIVA avoids the use of inhalational agents by utilizing i.v. agents to induce and maintain anesthesia. The main advantage of this technique is avoidance of the side effects of the inhalational agents such as nausea and vomiting. Additionally, this technique is an important option for patients who may be susceptible to malignant hyperthermia (see Appendix B) as the inhalational agents are known triggering agents for this condition. Medication infusions commonly used to provide a TIVA anesthetic include propofol, remifentanil, sufentanil, and dexmedetomidine.
Case Study
A 78-year-old ASA III male with a Mallampati class III airway presents for a cerebral angiogram due to a recent episode of severe headache and transient neurological deficit. He has a history of stable coronary artery disease, poorly controlled hypertension, hyperlipidemia, and type II diabetes mellitus. He is a former heavy drinker and smoker but quit both last year. He has no known drug allergies and takes atorvastatin, lisinopril, metoprolol, and roziglitazone (Avandia). You plan monitored anesthesia care (MAC).

The case will be done in the angiography suite, not the OR, and you plan MAC, not general anesthesia. How will this alter your anesthetic equipment set up?

The short answer is, it won't! In any anesthetizing location, you should have all of your usual tools, drugs, and equipment. Any case planned for monitored anesthesia care could potentially require advanced airway management or conversion to general anesthesia. The remote location of an increasing fraction of anesthesia cases poses a challenge and requires flexibility, since the geometry of the radiology, endoscopy, and cardiac catheterization laboratory suites will differ from the operating room. But the basic elements should always be present.

What drugs will you select for the case?
Midazolam and fentanyl are often used for light sedation, but they can produce respiratory depression and may have a greater effect in the elderly or those with cardiopulmonary disease. You might consider instead the use of shorter acting drugs with predictably short offset, such as a low-dose propofol infusion (25–75 mcg/kg/min) or a dexmedetomidine infusion (0.2–0.5 mcg/kg/h).

After imaging the patient, the radiologist discovers an aneurism and small intracerebral hemorrhage and wishes to coil embolize it to prevent further bleeding. She requests that you alter conditions to completely immobilize the patient for the procedure. What are your options?
You could deepen the sedation but given his comorbidities and age you might prefer to induce general anesthesia instead. This also lets you use neuromuscular blocking drugs to provide immobility without the fear that
oversedation would lead to apnea. Moreover, in some neuroradiology procedures, immobility also includes periods of deliberate apnea, so in this case, general anesthesia with a controlled airway is the only option.

**Suppose you select general anesthesia. How will you induce and maintain anesthesia? Do you need to intubate the patient and control ventilation?**

This case does not involve much surgical stimulation. In fact, the case will not be any more painful than it has already been. Therefore, you do not need a particularly deep anesthetic plane. You may wish, therefore, to use NMB drugs with light general anesthesia, to avoid the use of deep general anesthesia with its attendant cardiovascular depression. This will also allow you to provide the immobility and periods of apnea that may be required. You will also generally choose short acting drugs, to allow for a neurological examination shortly after emergence from anesthesia. A reasonable combination would be propofol for induction, a nondepolarizing neuromuscular blocking drug such as vecuronium, and maintenance with a low dose volatile anesthetic such as sevoflurane. If you had been using propofol or dexmedetomidine for sedation, you could consider continuing these drugs with a TIVA technique, but higher doses will be required to keep the patient comfortable for endotracheal intubation and controlled ventilation, as well as to prevent awareness under anesthesia when paralyzed.

**How will you monitor the patient after you induce general anesthesia? Will your plan change, relative to the monitored anesthesia care phase of the case?**

You will already have been using ASA standard monitors as you do for any anesthetic. You may consider adding an arterial line, as careful control of blood pressure may be needed in this neurovascular case. You may be asked to raise or lower blood pressure with intravenous agents. Although there will be a femoral arterial sheath in place for access to the cerebral vasculature, the catheters threaded in the sheath may not allow high fidelity recording of pressure, so some radiologists will ask you to have your own arterial catheter. You will be using a light general anesthetic and may be concerned about awareness. However, it may not be possible to use a consciousness monitor like BIS because the electrodes may obscure the cerebral images. You will probably use an end-tidal gas monitor to assess the concentration of inhaled agent in the patient’s brain. You will also add a neuromuscular blockade (twitch)
monitor, continuous capnography, tidal volume, and airway pressure monitors, and may consider continuous temperature monitoring.

_How do your recovery (PACU) plans change with the decision to change to general anesthesia?_

They do not change markedly. All patients recovering from anesthesia, be it regional, general, or monitored anesthesia care, require postoperative observation in an area with careful nursing care and availability of cardiovascular monitoring and resuscitation. However, the nature of the anesthetic does influence the intensity of care, the length of stay in recovery, and the particular details to be monitored. You may choose to take the patient to the main PACU rather than the recovery area used for conscious sedation or MAC cases, which may be part of the radiology suite. Since you have administered a general anesthetic with paralysis, you will make this known to the PACU or post-procedure recovery area. Because this is a neurological case, you will assess the patient's neurological exam immediately after emergence. This is often done cooperatively with the radiologist.

**Suggested Further Reading**


Anesthetic Techniques: Regional

Anthony R. Plunkett and Brian C. McLean

For maximum impact, it is recommended that the case study and questions found on page xxiii are reviewed before reading this chapter.

Key Learning Objectives
- Understand the different types of regional anesthetics (neuraxial, peripheral, intravenous)
- Learn indications, techniques, and potential complications associated with regional anesthesia
- Review the relevant anatomy for regional anesthesia

Introduction
Regional anesthesia includes a variety of anesthetic approaches such as neuraxial (epidural and spinal anesthesia), peripheral, and intravenous techniques. Regional anesthesia plays an important role both inside and outside of the operating room. In addition to its use for surgical anesthesia, it is also gaining widespread use for postoperative pain control. In this chapter, we will review the basic tenets of neuraxial, peripheral, and intravenous regional anesthesia.

Neuraxial Anatomy
The vertebral column extends from the foramen magnum to the sacral hiatus. The spinal cord is contained within this bony framework. There are 24 vertebrae (7 cervical, 12 thoracic, 5 lumbar, and 5 fused vertebrae forming the sacrum). Each vertebrae is composed of a lateral transverse process and a posterior spinous process (which is what we feel when we palpate a patient’s back).
The spinous process and transverse process are connected via bilateral lamina, while the transverse process is connected to the vertebral body via the pedicles (see Fig. 13.1).

The spinal cord is contained within the spinal canal and covered by three layers called the meninges. The pia mater is closely adherent to the spinal cord, while the arachnoid mater is more closely adherent to the outer dura mater. Cerebral spinal fluid (CSF) is contained within the space between the pia mater and arachnoid mater, called the subarachnoid space. This is the site of injection when performing spinal anesthesia. The spinal cord normally extends from the foramen magnum to the level of L1 in adults and L3 in children. As a result, performing a spinal (subarachnoid block) below the level of L3 avoids potential trauma to the spinal cord. An important surface landmark when performing neuraxial anesthesia is the level of the iliac crest, which most commonly corresponds to the level of L4–L5 (Fig. 13.2).

The spinal cord has a rich vascular supply from a single anterior spinal artery and paired posterior spinal arteries. The anterior spinal artery supplies approximately 2/3 of the spinal cord, while the paired posterior spinal arteries provide the remaining 1/3. There is a prominent feeder artery called the artery of Adamkiewicz or Radicularis Magna that provides blood supply to the anterior, lower 2/3 of the spinal cord. Trauma or ischemia of this artery can
lead to **anterior spinal artery syndrome**, resulting in bilateral lower extremity paralysis with preservation of proprioception and vibration.

The spinal nerve roots exit the spinal canal via intervertebral foramen. The nerves arise above their respective vertebrae, but starting at T1, they exit below their vertebrae. As a result, there are eight cervical nerve roots, but only seven cervical vertebrae. Each spinal nerve innervates an area of skin referred to as a dermatome (see Fig. 13.3).

**Indications and Contraindications**

As with any anesthetic procedure, the risks and benefits of neuraxial regional anesthesia must be discussed with the patient. Potential risks are shown in Table 13.1.

Spinal anesthesia is primarily indicated for lower abdominal surgery, the perineum, and lower extremities. Epidural anesthesia is primarily indicated for lower abdominal surgery, thoracic surgery, surgery on the lower extremities, and labor. Epidurals can have sacral nerve root “sparing” and may not be optimal for surgery involving this area. Contraindications to neuraxial anesthesia are listed in Table 13.2.

**Mechanism of Action**

The most common medication given for regional anesthesia is a local anesthetic. Local anesthetic that has been injected directly into the subarachnoid space (spinal) or that has diffused into the subarachnoid space from the epidural space (epidural) bathes the nerve root and inhibits synaptic transmission of action potentials. The effect of local anesthetics on nerve fibers varies
Figure 13.3  Dermatomes. (Reproduced with permission from Stewart, O. *Functional Neuroscience*. Springer Press. 2000).
Anesthetic techniques: regional

According to the size of the nerve fiber, myelination and the concentration of the local anesthetic (also see Chap. 6, Pharmacology of Local Anesthetics). Differential blockade (the order of effects among the different nerve types) typically results in sympathetic blockade (often accompanied by change in temperature sensitivity), followed by sensory blockade (pain, light touch), and finally motor blockade (paralysis). A well-placed neuraxial anesthetic can provide total anesthesia for a variety of surgical procedures.

There are a number of other medications that can be used for both spinal and epidural anesthesia. Opioids, alpha-2-receptor agonists (e.g., clonidine), and vasoconstrictors (e.g., epinephrine, phenylephrine) have all been given with the effect of enhancing the quality or the duration of the block. Epinephrine can prolong the duration of spinal anesthesia by decreasing the rate of absorption of the local anesthetic.

Epidural Anesthesia

Epidural anesthesia allows the delivery of medication either continuously or intermittently into the epidural space for up to several days after the surgical procedure. Sitting is the most common position in which an epidural is performed. Benefits of the sitting position include better identification of the midline and more flexion of the vertebral column. As the spine is flexed, it helps to

<table>
<thead>
<tr>
<th>Table 13.1 Risks of neuraxial anesthesia.</th>
</tr>
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<tbody>
<tr>
<td>Bleeding</td>
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<tr>
<td>Infection</td>
</tr>
<tr>
<td>Nerve injury</td>
</tr>
<tr>
<td>Post-dural puncture headache</td>
</tr>
<tr>
<td>Failure of block to provide adequate anesthesia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 13.2 Contraindications to neuraxial anesthesia.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Absolute contraindications</strong></td>
</tr>
<tr>
<td>Patient refusal</td>
</tr>
<tr>
<td>Infection in the area of needle puncture</td>
</tr>
<tr>
<td>Elevated intracranial pressure</td>
</tr>
<tr>
<td>Uncontrolled bleeding</td>
</tr>
<tr>
<td><strong>Relative contraindications</strong></td>
</tr>
<tr>
<td>Bacteremia</td>
</tr>
<tr>
<td>Pre-existing neurologic disease (e.g., multiple sclerosis)</td>
</tr>
<tr>
<td>Cardiac disease</td>
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<td>Abnormal coagulation studies</td>
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open the space between spinous processes, allowing more room for the epidural needle to enter. An epidural may also be performed with the patient in the lateral position. This increases patient comfort, especially pregnant patients in active labor. However, the midline may be more difficult to identify.

The risks and benefits must be discussed with the patient and informed consent obtained. Standard monitors should be applied including blood pressure, ECG, and pulse oximetry. The patient may be sedated with an intravenous opioid or benzodiazepine. The desired interspace is identified and the patient’s skin is prepared with antiseptic solution. An epidural kit is typically used, which includes a 17- or 18-G Tuohy needle and a 19- or 20-G catheter.

**Technique**

A midline or paramedian approach can be used. After infiltration of skin with local anesthetic, the epidural needle is advanced through the skin, subcutaneous tissue, the supraspinous ligament, the interspinous ligament, and finally into the ligamentum flavum. Identification of the epidural space may be found with a loss of resistance technique or a hanging-drop technique.

With the loss of resistance technique, a syringe containing saline or air is attached to the epidural needle. As the needle is slowly advanced, the anesthesiologist places pressure on the syringe. The positive pressure encountered in the supraspinous ligament, interspinous ligament and ligamentum flavum prevents the plunger of the syringe from depressing (see Fig. 13.4). As the needle

![Figure 13.4 Trajectory of epidural anesthesia. (Image Courtesy J. Ehrenfeld, M.D.)](image-url)
advances past the ligamentum flavum, a distinct loss of positive pressure is felt, as the plunger gives way and the saline or air is injected into the epidural space. A small catheter can then be threaded into the epidural space, usually 3–5 cm past the needle tip. Once the catheter is placed, a syringe containing a “test dose” of lidocaine with epinephrine 1:200,000 is attached. The catheter is aspirated first, to ensure no blood or cerebrospinal fluid (CSF) can be withdrawn. The test dose, typically 3 mL, is injected rapidly through the epidural catheter. The epinephrine serves as a surrogate marker to ensure the catheter has not threaded into a blood vessel (if positive, one would expect to see an increase in heart rate). The test dose also helps to determine if the catheter is in the subarachnoid space (spinal). If there are no sensory or motor changes within 3 min, the catheter is most likely not in the subarachnoid space.

With the hanging-drop technique, a small drop of saline is placed at the hub of the needle. As the needle passes through the positive pressure structures stated above, the drop of saline will remain at the hub of the needle. Once the needle contacts and passes through the ligamentum flavum, the drop of saline is retracted back into the needle as the negative pressure of the epidural space is encountered.

**Pharmacology of Epidural Anesthesia**

Similar local anesthetics can be used for both epidural and spinal anesthesia. Chloroprocaine and lidocaine are fast onset medications with a short duration of action, while bupivacaine and ropivacaine have a slower onset and longer duration. Unlike spinal anesthesia, the level of anesthesia in an epidural is not influenced by baricity or position of the patient immediately after injection (see below).

The amount of local anesthetic required to produce surgical anesthesia with an epidural is significantly more than with a spinal, as the local anesthetic must traverse more layers to act on the nerve roots. The addition of epinephrine can prolong the effect of local anesthetic by decreasing vascular uptake, allowing more time for the medication to act on the nerve roots. Opioids, such as morphine or fentanyl, can also be added to an epidural. They help to enhance the quality of the epidural as well as provide postoperative pain control.

**Spinal Anesthesia**

As with general anesthesia, prior to starting a spinal patient monitors should be applied (blood pressure, pulse oximeter, and ECG). Supplemental oxygen is often administered. Intravenous access also must be established. In most situations,
the patient may be sedated with an intravenous opioid such as fentanyl and/or a benzodiazepine such as midazolam. Patient comfort will help in both positioning and anxiolysis while performing the spinal. As with an epidural, a spinal may be placed in either the sitting or lateral position.

As stated above, the spinal cord typically ends at the level of L1 in adults and L3 in children. Placing the spinal needle below the level of L3 provides an additional margin of safety, by decreasing the likelihood of any spinal cord penetration. The iliac crest has been traditionally used as an anatomic landmark corresponding with an L4–L5 interspace (see Fig. 13.2).

**Technique**

There are two main techniques for performing a spinal anesthetic: midline and paramedian. With each technique, the patient is positioned optimally for both physician and patient, the desired interspace is identified, and the skin is cleaned and prepared with antiseptic solution. Local anesthesia is infiltrated in the skin and subcutaneous tissues to improve patient comfort. With the **midline approach**, the spinal needle is first introduced into the skin between the upper and lower spinous processes at the desired interspace. After passing through the skin, the needle continues to pass through subcutaneous tissue, the supraspinous ligament, the interspinous ligament, the ligamentum flavum, and finally advancing through the epidural space into the subarachnoid space (Fig. 13.4). Often a distinct “pop” is felt by the anesthesiologist as the needle penetrates the ligamentum flavum. Correct identification of the subarachnoid space is confirmed by free flow of CSF out of the hub of the needle.

The **paramedian approach** is used in patients where the midline may be difficult to identify (e.g., scoliosis) or the interspace may be challenging to pass a needle through (e.g., thoracic level for epidural placement, elderly patients with calcified ligaments or loss of disc space). Needle insertion is typically 1 cm from the midline. After the transverse process is contacted, and the needle is redirected cephalad and medial to pass through the interlaminar space. One of the main differences between the paramedian and midline approach is that the ligamentum flavum is the first resistance encountered with the paramedian approach. Again, correct identification of the subarachnoid space is confirmed by free flow of CSF out of the hub of the needle.

Assuming there is no blood exiting the needle and the patient has not experienced a paresthesia, administration of the local anesthetic can proceed.
Common local anesthetics include lidocaine, chloroprocaine, and bupivacaine. Each local anesthetic has slightly different properties, which affect onset, duration, and potential for toxicity (see Chap. 6, Pharmacology of Local Anesthetics). When the syringe containing the local anesthetic is attached to the spinal needle, care must be taken to avoid moving the needle. The anesthesiologist’s hands are usually braced against the patient’s back while holding the spinal needle steady. Before injection of the local anesthetic, one should aspirate first and allow a small volume of CSF to enter the syringe. This can be confirmed by visualizing a CSF “swirl” when mixing with the local anesthetic in the syringe. The local anesthetic is injected slowly over 3–5 s. CSF can be aspirated at the end of the injection as well to confirm the needle has not moved from the spinal space while injecting. The onset of anesthesia will be rapid (within 60 s) with a spinal anesthetic.

Factors Effecting Level and Duration of Local Anesthesia

Two of the most important factors determining the distribution of local anesthetic in the subarachnoid space are the baricity of the solution (density compared to CSF) and the position of the patient immediately after injection of the solution. Addition of a vasoconstrictor (e.g., epinephrine) and the type of local anesthetic selected influence the duration of the spinal block. Local anesthetic solutions are classified as hypobaric, isobaric, or hyperbaric based on their density relative to the density of CSF. Knowledge of the local anesthetic baricity can help the anesthesiologist control both the direction and extent of local anesthetic spread within the subarachnoid space.

Hyperbaric solutions usually contain glucose/dextrose. They allow for a greater cephalad spread of the local anesthetic. If a higher dermatomal level is needed, the patient may be placed in a head-down (Trendelenburg) position, allowing the hyperbaric solution to migrate cephalad. Likewise, if the surgery requires dense anesthesia for a perirectal procedure, the patient may be left in a sitting position for several minutes after completion of the spinal.

Hypobaric solutions are used less commonly in clinical practice. A patient undergoing hip arthroplasty may benefit from having the hypobaric solution “float up” to the operative side. Hypobaric solutions can be made by mixing the local anesthetic with sterile water, or normal saline.

Isobaric solutions tend to have limited spread within the subarachnoid space and are thought to produce a more profound motor block and longer
duration of action. Isobaric solutions can be prepared by mixing the local anesthetic with normal saline or the patient’s CSF.

Addition of epinephrine (0.1–0.2 mg) or phenylephrine (2–5 mg) to the local anesthetic solution increases the duration of the spinal block. The resultant decrease in spinal cord blood flow and uptake of the local anesthetic prolongs the exposure to the nerve roots of the local anesthetic.

**Caudal Anesthesia**

This type of regional anesthetic is most commonly performed in pediatric patients. After induction of general anesthesia the child is placed in the lateral position. The sacral cornu are identified as well as the sacral hiatus. The skin is prepared in sterile fashion. A needle is introduced perpendicular to the skin through the sacrococcygeal ligament (beneath the sacral hiatus), advanced slightly, then the angle is dropped and the needle is advanced slightly further into the epidural caudal canal. Confirmation of proper needle position can be obtained by rapidly injecting 3–5 mL of air or saline while the anesthesiologist’s fingers are palpating the skin directly over the needle. Skin swelling or crepitus indicates the needle has not penetrated the epidural space. Once proper position is confirmed, a syringe is connected to the end of the needle and aspirated to ensure no blood or CSF is obtained. Local anesthetic is then injected in slow 3–5 mL aliquots.

**Combined Spinal–Epidural**

The last technique for neuraxial anesthesia combines the advantageous qualities of both a spinal (fast, dense onset of anesthesia) and an epidural (placement of a catheter for continuous medication infusion). A special combined spinal–epidural kit is often used that contains an epidural needle with a small hole at the tip to allow passage of a spinal needle. An epidural technique is performed. Once the needle has reached the epidural space, the spinal needle is then introduced through the epidural needle and pierces the dura, allowing free flow of CSF back through the needle. Local anesthetic is injected into the spinal space, the spinal needle is withdrawn, and the epidural catheter is then threaded through the epidural needle. While this technique combines advantages of both spinal and epidural anesthesia, it also exposes a patient to the risks of both. Combined spinal–epidural anesthesia is often used in obstetrics.
Complications and Side Effects: Spinal and Epidural Anesthesia

Cauda Equina Syndrome (CES)

There have been some reports of permanent neurologic injury when using lidocaine for spinal anesthesia. This was first associated with high doses of medication being administered through a continuous spinal catheter, but has also been reported with single-dose injections. The patient develops bowel and bladder dysfunction as well as lower extremity paralysis.

Transient Neurologic Symptoms

This phenomenon has also been linked to the use of lidocaine. It results in pain in the back, buttocks, and lower extremities without motor or sensory deficit. It is usually self-limiting and resolves in a few days. The incidence is increased when patients are placed in the lithotomy position.

Cardiovascular Changes

As a result of sympathetic nervous system blockade, spinal anesthesia and epidural anesthesia can cause hypotension. Treatment centers around volume replacement to restore adequate venous return and cardiac output. The anesthesiologist may also need to administer vasoconstrictor medications (e.g., ephedrine, phenylephrine) to raise blood pressure.

As the level of blockade rises, there is an increased risk of bradycardia. The cardioaccelerator fibers originate at the T1–T4 level and may be blocked by neuraxial anesthesia approaching this level. Again, treatment centers around volume replacement to restore preload, but may also require atropine or ephedrine.

Post-dural Puncture Headache (PDPH)

When the dura mater is violated (as with spinal anesthesia and unintentionally during epidural anesthesia), CSF is allowed to leak through the hole faster than it is being produced. This causes downward displacement on sensitive brain structures and may result in a headache. Obviously, a larger hole will lead to a higher incidence of PDPH, thus inadvertent dural puncture with a larger needle when placing an epidural leads to higher rates of PDPH. The pathognomonic feature of PDPH is a headache that worsens with sitting or standing and
is relieved by lying flat (postural component). Patients may also experience nausea, vomiting, and vision changes. Children and elderly patients have the lowest risk of PDPH. Initial treatment focuses on bed rest and fluid replacement. Pain medications such as opioids may also help. Caffeine administered orally or intravenously can also be given. However, one of the most definitive treatments is an epidural blood patch. Approximately 15–20 mL of the patient’s blood is withdrawn in sterile fashion and then injected into the epidural space at the same level of the previous regional anesthetic. The patient should experience almost immediate relief.

**High/Total Spinal Anesthesia**

Total spinal anesthesia refers to excessive sensory and motor anesthesia associated with loss of consciousness. Loss of consciousness is thought to be due to ischemia of medullary ventilator centers due to profound hypotension. Treatment focuses on the “ABCs” (Airway, Breathing, Circulation) and tracheal intubation is often necessary.

**Urinary Retention**

Blockade of S2–S4 nerve roots can decrease bladder tone and inhibit the voiding reflex. Most patients that have neuroaxial anesthesia require a catheter in the bladder to avoid bladder distention.

**Intravascular Injection**

Since the total dosage of drug administered in a spinal is relatively small, complications resulting from intravascular injection typically occur with epidural anesthesia. Local anesthetic may be injected via the needle or a catheter that has been inadvertently threaded into a vessel. Frequent aspiration, administration of a “test dose” (addition of epinephrine), and slow, incremental injections of local anesthetic all help to minimize the chance of intravascular injection.

**Spinal/Epidural Hematoma**

The incidence of hematoma after an epidural is approximately 1/150,000 and 1/200,000 after a spinal. Most cases occurred in patients that had abnormal coagulation profiles. The mass effect of the evolving hematoma causes injury via direct pressure and ischemia. Immediate recognition is paramount to avoid permanent neurologic insult. Symptoms usually include sharp back pain with
progression to sensory and motor deficit. An imaging study such as an MRI and a neurosurgical consult should be obtained as soon as possible. Emergent surgical decompression of the spine is required and can prevent permanent neurologic damage if performed early.

**Epidural Abscess**
Abscess formation is a potentially devastating complication of an epidural. The average time frame for the development of symptoms is 5–14 days after catheter placement. There is a progression of symptoms that typically result in back pain exacerbated by percussion over the epidural insertion site, followed by the development of radicular pain, then motor or sensory deficit, and finally paraplegia. As with spinal hematoma, an imaging study and a neurosurgical consultation should be obtained as soon as possible.

**Peripheral Nerve Blocks**
Peripheral nerve blocks (PNB) and peripheral nerve catheters are gaining increasing popularity in today’s surgical environment. As ambulatory surgeries grow in number, the ability to provide quick, safe, and effective anesthesia with minimal residual effects takes on a greater importance. PNBs are also very effective for postoperative analgesia and can allow earlier, more intense participation in rehabilitation. As with neuraxial anesthesia, the patient must be made aware of the risks and benefits of PNB. Patient refusal and infection at the insertion site are contraindications. The patient’s coagulation status and medication history must be carefully reviewed to ensure safe performance of the block. Standard monitors should be applied, as well as supplemental oxygen. The patient may be sedated with an intravenous opioid and/or benzodiazepine and the skin is prepared in sterile fashion. If the block is taking place in a separate “block room,” all the monitors, equipment, and medications should be close by in the event of a complication (e.g., seizure from local anesthetic toxicity). While there are many types of PNBs, we will focus on a few of the most commonly performed for both upper and lower extremity surgery.

**Identification of the Target Nerve**
There are three major techniques used to identify the desired neural structure: **paresthesias**, **nerve stimulation**, and **ultrasound**. Paresthesias are radiating electric shock-like sensations that can occur as a needle contacts or comes very
close to a nerve. When a paresthesia does occur, the block needle should be withdrawn slightly and only then should the local anesthetic be administered. Injection of local anesthetic on the paresthesia itself may result in pain and permanent nerve injury. Nerve stimulation (Fig. 13.5) elicits a motor response from a peripheral nerve as the stimulating needle approaches closer to the nerve. A motor response maintained at a current of less than 0.5 mA is thought to indicate close enough proximity to the target nerve to produce anesthesia. Motor response at a current of 0.2 mA or less may indicate needle placement directly in the nerve and should not be sought. Finally, ultrasound is a relatively new technology for visualizing peripheral nerve and adjacent structures.

**Cervical Plexus Blockade**

The cervical plexus is formed by the first four cervical nerves. The superficial cervical plexus can be blocked by infiltrating local anesthetic along the posterior border of the sternocleidomastoid muscle (see Fig. 13.6). This block can be used for patients undergoing awake carotid endarterectomy.
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Brachial Plexus and Upper Extremity Blocks

The brachial plexus is formed from the anterior rami of cervical nerves C5–C8 and T1 (Fig. 13.7). The brachial plexus runs through the groove formed by the middle and anterior scalene muscles. The plexus initially emerges as the cervical roots, then forms three trunks, six divisions, three cords, and finally the terminal branches that innervate almost all of the upper extremity. A mnemonic sometimes used is “Randy Travis Drinks Cold Beer” with the first letters of each word standing for roots, trunks, divisions, cords, and terminal branches.

Interscalene Block

A line is drawn laterally from the cricoid cartilage (the level of the transverse process of C6). The interscalene groove (between the anterior and middle scalene muscle) is palpated (see Fig. 13.8). The brachial plexus is superficial at this
level and a nerve block needle is typically inserted only 1–2 cm. Approximately 25–40 mL of local anesthetic can be administered for successful anesthesia of shoulder and upper arm surgery.

An interscalene PNB will often miss the inferior trunk (C8 and T1) and is thus not appropriate for lower arm and hand surgery. Hemidiaphragmatic paralysis via blockade of the ipsilateral phrenic nerve is a side effect in nearly 100% of patients. In a patient with normal respiratory function, this hemidiaphragmatic paralysis is not a concern. Blockade of sympathetic nerves can also produce an ipsilateral Horner’s syndrome (ptosis, anhidrosis, miosis, enophthamos, and nasal congestion).

Figure 13.7  Brachial plexus anatomy. (Reproduced with permission from Tsui, B. Atlas of Ultrasound and Nerve Stimulation-Guided Regional Anesthesia, Springer, 2007).
Supraclavicular Block
A supraclavicular PNB is an excellent choice for surgery of the arm or hand. Once the interscalene groove is palpated, the groove is followed down the neck to the clavicle. Approximately 1 cm above the clavicle is the insertion point for the block needle. Under ultrasound guidance, the brachial plexus appears as a “cluster of grapes” lateral to the subclavian artery. Again, 25–40 mL of local anesthetic may be administered. The most common serious complication is pneumothorax, which can occur in 1% of cases.

Infraclavicular Block
An infraclavicular PNB is a good block for surgery of the lower arm and hand. As the brachial plexus passes under the clavicle, the plexus forms three cords surrounding the axillary artery. The nerve block needle is further removed from the pleura and the neuraxis and the risk of pneumothorax or neuraxial anesthesia is low. There are several approaches to the infraclavicular PNB. The most common approach is to identify the midpoint of the clavicle and a line is drawn 2–3 cm caudad from this point. The nerve block needle is then directed
at a 45 angle towards the axilla. A motor response with nerve stimulation is usually sought in the hand with a current <0.5 mA.

**Axillary Block**

Axillary PNBs are used for surgery involving the lower arm and hand. It offers the advantage of being far removed from the lung and neuraxis. As the brachial plexus enters the axilla, the three cords become the terminal branches surrounding the axillary artery. The patient is placed supine with the elbow flexed 90°. The pulse of the axillary artery is then palpated as high in the axilla as possible. The needle is purposely advanced into the axillary artery and after blood has been seen coming back into the hub of the needle, the needle is advanced further until the blood disappears with aspiration. At this point, local anesthetic can be administered posterior to the artery, as well as anterior to the artery. Approximately 40 mL of local anesthetic is administered. The

musculocutaneous nerve is a terminal branch that exits very proximal from the brachial plexus and must be blocked separately by injection of local anesthetic into the substance of the coracobrachialis muscle. As the brachial plexus runs more distal from the roots, the time to onset increases. The axillary PNB takes the longest time to set up of all the upper extremity blocks.

Table 13.3 provides a summary of upper extremity peripheral nerve blocks.

**Lower Extremity Peripheral Nerve Block**

**Femoral Nerve Block**

To perform a femoral nerve block, the patient is placed in the supine position. A line drawn from the anterior superior iliac spine to the pubic tubercle represents the inguinal ligament. The femoral artery is then palpated along this
Anesthesiology Student Survival Guide

The femoral nerve PNB can be used for surgery involving the knee, anterior thigh, and medial portion of the lower leg. Since the femoral nerve is located in close proximity to the femoral artery, careful aspiration is important to avoid intravascular injection of local anesthetic (Figure 13.9).

### Sciatic Nerve Block

The sacral plexus is formed from the ventral rami of L4–S3 nerve roots. The patient is placed in the lateral position with the operative side up. The operative leg is flexed at the knee, while the nonoperative leg remains straight. A line is drawn between the greater trochanter and the posterior superior iliac spine. A second line can be drawn from the greater trochanter to the sacral hiatus. A third line is drawn from the mid-point of the first line, intersecting the second line. This is the point of needle entry. The needle is inserted perpendicular to all planes with the desired motor response of plantar or dorsiflexion of the foot. A sciatic nerve block can be used for surgery below the knee (with the exception of the medial portion of the lower leg innervated by a

<table>
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<tr>
<th>Table 13.3 Summary of upper extremity nerve blocks.</th>
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<tr>
<td><strong>Type of nerve block</strong></td>
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<tr>
<td>-------------------------</td>
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<tr>
<td><strong>Interscalene</strong></td>
</tr>
<tr>
<td><strong>Supraclavicular</strong></td>
</tr>
<tr>
<td><strong>Infraclavicular</strong></td>
</tr>
<tr>
<td><strong>Axillary</strong></td>
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</table>
branch of the femoral nerve). When combined with a lumbar plexus block, it can provide complete anesthesia to the entire leg.

**Ankle Block**

Five nerves supply sensation to the foot (Figure 13.10). Four of the five nerves are branches of the sciatic nerve, while one is a branch of the femoral nerve. The *saphenous nerve* (branch of the femoral nerve) provides sensation to the anteromedial aspect of the foot. It can be blocked by infiltrating local anesthetic just anterior to the medial malleolus. The *deep peroneal nerve* provides sensation to the dorsal medial aspect of the foot and the web space between the first two digits. It can be blocked by infiltrating local anesthetic lateral to the dorsalis pedis artery. The *superficial peroneal nerve* provides sensation to the dorsum of the foot and all five digits. It can be blocked by administering a subcutaneous wheal of local anesthetic from the anterior border of the tibia to the lateral malleolus. The *sural nerve* provides sensation to the lateral aspect of the foot. It can be blocked by injection of local anesthetic just lateral to the Achilles tendon, toward the lateral malleolus. Finally, the *posterior tibial nerve* provides sensation to the heel. The nerve can be blocked by injection of local anesthetic posterior to the medial malleolus. Approximately 5–8 mL of local anesthetic is injected for each nerve.

Table 13.4 provides a summary of lower extremity peripheral nerve blocks.

**Intravenous Regional Anesthesia (Bier Block)**

A Bier block is a fairly simple block to perform and can produce profound anesthesia and analgesia. It is often used for short surgical procedures of the hand or forearm (e.g., carpal tunnel). A peripheral intravenous line is started and a double pneumatic tourniquet is placed on the arm. The arm is exsanguinated and the proximal cuff on the double tourniquet is inflated. Approximately 25–50 mL of 0.5% lidocaine is injected into the IV and the IV is removed.

If the patient begins to complain about tourniquet pain, the distal cuff can be inflated and the proximal cuff deflated. If the surgical procedure is extremely short, the tourniquet must still be left in place for at least 20 min to avoid rapid systemic absorption of a high concentration of local anesthetic. Due to concern of inadvertent early tourniquet deflation and systemic absorption, long-acting local anesthetics, such as bupivacaine, are not recommended for intravenous regional blocks.
The use of ultrasound in regional anesthesia has increased in popularity over the past few years. As more research is done, ultrasound may ultimately prove to be safer, faster, and more effective than the paresthesia or neurostimulation techniques. Ultrasound emits high-frequency sound waves, which are reflected back when they encounter different types of tissue. Different tissues have different degrees of echogenicity and thus reflect the sound waves at different speeds. The resulting image provides varying shades that helps distinguish the tissue types.

Nerves can be seen as round, oval, or triangular shaped structures and can be hyperechoic (light) or hypoechoic (dark). For example, nerves visualized above the diaphragm tend to be hypoechoic, while those below the diaphragm...
tend to be hyperechoic. Color flow Doppler can be applied to distinguish blood vessels from other structures.

Another advantage of ultrasound is the ability to view the nerve block needle in its entirety as it approaches the target nerve, and then see the local anesthetic spread around the nerve. As the cost, portability, and image resolution improve, ultrasound will most likely become an integral part of regional anesthesia.

**Case Study**

A 58-year-old man is to undergo right total knee replacement (TKR). After a thorough H&P and consultation, he elects to have the procedure under regional anesthesia. He is otherwise healthy, though he smokes a pack of cigarettes a day and does not exercise regularly due to his arthritic knee. He takes an NSAID daily for pain and lately has been taking oxycodone and acetaminophen for worsening pain.

Which dermatomes or nerves will you need to block to perform a total knee replacement comfortably?

The anterior portion of the thigh and leg are innervated by the L3, L4, and L5 dermatomes. The back of the knee, though not in the incision, is stimulated nonetheless in TKR, and is innervated by S2. In addition, a thigh tourniquet is usually employed to prevent blood loss, so L2 and possibly L1 should be blocked. In practice, the femoral, lateral femoral cutaneous, obturator, and portions of the sciatic nerve need to be blocked.

Which regional anesthetic techniques are suitable for total knee replacement? Which will you choose?

In theory, several techniques are possible. Spinal anesthesia will reliably block all the involved nerve roots, whether a plain solution or hyperbaric solution containing glucose is used. Hyperbaric solutions produce higher levels than are necessary, so plain solutions may be favored for the lower incidence of hypotension. Epidural anesthesia is commonly used for TKR and allows titration of local anesthetic to the desired level. Disadvantages include a 5–10% incidence of failed or inadequate block (asymmetric anesthesia or incomplete sacral nerve blockade). An additional advantage is the ability to extend the block for either prolonged surgery or for postoperative
analgesia. Peripheral nerve blocks may also be used. Individual nerve blocks can provide surgical anesthesia. It is more practical to perform a lumbar plexus or three-in-one block (which will cover the femoral, lateral femoral cutaneous, and obturator nerves with a single injection or catheter). A separate sciatic block, or a spinal or general anesthetic is then added to complete the anesthetic.

If you choose epidural analgesia, how will you locate the epidural space? What precautions will you take to avoid toxicity?

Standard monitors are placed and an IV is inserted. The patient can be seated or lying on his side; many find the sitting position easier to locate the midline. The back is steriley prepped and draped and local anesthetic is infiltrated in a lumbar interspace, typically L3–L4 or L2–L3. The epidural needle is advanced until it is seated in ligament. Then a loss-of-resistance syringe is attached, containing either air or saline. The epidural needle is advanced in slow increments, checking for resistance to injection, indicating the tip is still in ligament. When the needle enters the epidural space, a loss of resistance to injection will be felt. The epidural catheter is then inserted 3–5 cm and the needle withdrawn. To avoid toxicity, it is important to exclude intravascular or intrathecal (spinal) placement. A test dose of lidocaine with epinephrine is given (typically 3–5 mL of a 2% concentration) and signs and symptoms of intravascular injection are sought. The heart rate will increase if epinephrine is injected IV, and the patient may experience symptoms such as tinnitus, perioral numbness, or metallic taste. If 60–100 mg of lidocaine were injected intrathecally, an immediate spinal anesthetic would be obtained.

After verifying proper position of the epidural catheter, what drugs will you use?

Assuming neither intravascular nor intrathecal placement is detected, an additional 10–15 mL of lidocaine can be injected in divided doses to obtain a low thoracic dermatomal level and motor blockade of the legs. Care should be taken not to inject too much drug without ensuring that the block is symmetrical (or at least that the operative site is numb). The case can be continued with lidocaine, or a longer-acting local anesthetic, such as bupivacaine (0.5 or 0.75%) or ropivicaine (1%), can be given to ensure a dense block for surgery.
Will you continue to use your epidural after the procedure?
Although no one technique has been shown to be better than others, use of regional analgesia in the immediate postoperative period and for 1–3 days following surgery can help facilitate active rehabilitation efforts and improve joint mobility. If you use the epidural postop, you will reduce the concentration of local anesthetic, so that you are providing analgesia rather than surgical anesthesia. Bupivacaine or ropivacaine, 0.125–0.2%, often with an opioid such as fentanyl or hydromorphone are common choices.

Suggested Further Readings


Chapter 14

Electrolytes, Fluids, Acid-Base Analysis, and Transfusion Therapy

Francis X. Dillon

For maximum impact, it is recommended that the case study and questions found on page xxiv are reviewed before reading this chapter.

Key Learning Objectives
- Understand the risks and benefits of fluid replacement therapy options
- Know how to calculate a patient’s fluid requirements and allowable blood loss
- Learn the types of blood transfusion therapy available and their indications

Electrolytes and Fluid Compartments
The body is about 60% water by weight. Water is partitioned in various named compartments in the body (see Table 14.1), including the intracellular and extracellular spaces. Many of the problems patients develop in the perioperative period are a direct result of fluid shifts within the extracellular (intravascular $\rightarrow$ interstitial) spaces. These range from peripheral edema, to intravascular hypovolemia and shock, to cellulitis and decubitus ulcers, to pericardial and pleural effusions, to cerebral edema, to the Adult Respiratory Distress Syndrome (ARDS). Fluid shifts predispose patients to serious infections and increased mortality via a number of mechanisms.

Abnormal fluid shifts from the intracellular (40 L) to the extracellular (15 L) compartment and vice versa cause even more dramatic illnesses, some fatal. These include lysis of various cells ranging in function from erythrocytes to neurons, swelling of the brain and spinal cord, and renal failure.
<table>
<thead>
<tr>
<th>Fluid compartment</th>
<th>Intracellular</th>
<th>(Extracellular)-intravascular</th>
<th>(Extracellular)-interstitial</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synonym</td>
<td>Cytosol</td>
<td>Plasma</td>
<td>Interstitial fluid</td>
</tr>
<tr>
<td>Routinely assessed during anesthesia?</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Can infuse into with iv catheter and fluids?</td>
<td>No</td>
<td>Yes (usual route to replace blood/fluid)</td>
<td>No</td>
</tr>
<tr>
<td>Compartment volume (l)</td>
<td>36 L</td>
<td>2.4 L</td>
<td>9.6 L</td>
</tr>
<tr>
<td>pH</td>
<td>7.3–7.5</td>
<td>7.4</td>
<td>7.4</td>
</tr>
<tr>
<td>Protein (mosm/l)</td>
<td>4</td>
<td>1.2</td>
<td>.2</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Na (meq/l)</td>
<td>14</td>
<td>142</td>
<td>139</td>
</tr>
<tr>
<td>K (meq/l)</td>
<td>140</td>
<td>4.2</td>
<td>4.0</td>
</tr>
<tr>
<td>Ca (meq/l)</td>
<td>&lt; 0.0002</td>
<td>1.3</td>
<td>1.2</td>
</tr>
<tr>
<td>Mg (mosm/l)</td>
<td>0.02</td>
<td>0.8</td>
<td>0.7</td>
</tr>
<tr>
<td>Cl (meq/l)</td>
<td>4</td>
<td>108</td>
<td>108</td>
</tr>
<tr>
<td>HCO₃ (meq/l)</td>
<td>10</td>
<td>24</td>
<td>28.3</td>
</tr>
<tr>
<td>Lactate (mosm/l)</td>
<td>1.5</td>
<td>1.2</td>
<td>1.2</td>
</tr>
<tr>
<td>Total mosm/l</td>
<td>301.2</td>
<td>301.8</td>
<td>300.8</td>
</tr>
<tr>
<td>Corrected mosm/l&lt;sup&gt;b&lt;/sup&gt;</td>
<td>281.0</td>
<td>282.0</td>
<td>281.0</td>
</tr>
<tr>
<td>Total osmotic pressure, mmHg (37°C)</td>
<td>5,423</td>
<td>5,443</td>
<td>5,423</td>
</tr>
</tbody>
</table>


<sup>b</sup>Corrected for reduced osmotic activity of ions in solution.
Oncotic vs. Osmotic Pressures
Fluid in the bloodstream stays in the bloodstream in part because its electrolyte and non-electrolyte solute composition is different from fluid in the interstitial spaces surrounding the vessels. There are two kinds of pressure in body fluids:

- **osmotic** pressure: caused by dissolved salts or nonionic small solute molecules
- **oncotic** pressure: form of osmotic pressure exerted by proteins in blood plasma; typically pulls water into the circulatory system

Overall, the oncotic plus osmotic pressure gradients tend to favor free water coming back into the intravascular space from the extravascular space. Hydrostatic pressure and the intact semipermeable membranes of the capillaries provide a counterbalancing pressure gradient in the opposite direction. Between these two forces an equilibrium forms.

Blood Volume and the Fluid Compartments
Blood is made up of parts of two different compartments: both the intracellular compartment (the inner volume of all the circulating blood cells or red blood cell volume (RBCV) whose total is 2 L); and the plasma (the extracellular – intravascular compartment whose total volume is 2.8 L). These two volumes added together make up the blood volume which is 2 + 3 = 5 L (Fig. 14.1).

![Fluid compartments](image.png)
The entire blood volume circulates in the closed circulatory tree (aorta → arteries → arterioles → capillaries → venules → veins → vena cava) in about one minute (60 s). Therefore on average, the cardiac output is 5 L per minute. Because the circulatory system, the heart, and the pulmonary circulation are closed and blood is incompressible, the sum total flow of all the bloodstream fluid going around the circulatory tree exactly equals the amount going through the heart.

Anesthesiologists are able to exert some control over the solute components and sizes of the fluid compartments by infusing fluids intravenously (into the extracellular – intravascular space). The goal is to maintain the compositions, pressures, and volumes of all the various fluid compartments by the proper choice of IV fluids.

Anesthesiologists also transfuse blood products intravenously to replace cells and fluids lost during procedures or as a result of trauma or illness. Transfusing blood products adds volume to both the intracellular space (i.e., the interior volume of red blood cells and platelets) and the extracellular– intravascular space (the non-cellular volume of water, electrolytes, and plasma proteins in plasma). Transfusion will be covered in more detail later in this chapter.

Physicians try to replace intravascular fluids with solutions that have the right tonicity, osmolality, oncotic pressure, viscosity, and cellular composition (among other characteristics) so that they tend to stay intravascular. In doing so, we are trying to accomplish several things:

1. Support preload of the heart, and therefore blood pressure and body perfusion
2. Avoid excessive expansion of interstitial space (edema) and the problems it causes
3. Allow some interstitial fluid to be transported back into the intravascular space (by osmotic or oncotic forces)
4. Avoid perturbing the intracellular space, in particular neurons and other cells for which swelling can be catastrophic

**Patient Evaluation: Fluid Management**

The first step in evaluating a patient in need of fluid management is to look at several clinical indicators of intravascular volume status (see Table 14.2). Evaluation and replacement of fluid status is an ongoing process. It is safe to say that after management of the anesthetic depth and control of oxygenation and ventilation, fluid management is the next most important task the anesthesiologist has.
Table 14.2 Clinical variables used to assess intravascular volume status during anesthesia.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Skin turgor</th>
<th>Neck veins</th>
<th>Systolic Blood Pressure</th>
<th>Variability of blood pressure with respiration</th>
<th>Central venous pressure (CVP)</th>
<th>Urine output (UO)</th>
<th>Heart rate (HR)</th>
<th>Hypotension with anesthesia esp. volatile</th>
<th>Orthostasis</th>
<th>Base excess (BE) or (HCO₃⁻) meq/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td>Loose</td>
<td>Flat</td>
<td>Low</td>
<td>High</td>
<td>Less than 8</td>
<td>Low</td>
<td>High</td>
<td>Likely</td>
<td>High</td>
<td>Less than -2 (or less than 22)</td>
</tr>
<tr>
<td>Euvolemic</td>
<td>Normal</td>
<td>Pulsatile</td>
<td>Normal</td>
<td>Normal</td>
<td>8–12 (0.5–1 ml/kg/h)</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>[-2 to +2] (22–30)</td>
</tr>
<tr>
<td>Hypervolemic</td>
<td>Puffy</td>
<td>Bulging, distended</td>
<td>Normal to High</td>
<td>Low</td>
<td>Greater than 12</td>
<td>High</td>
<td>Low</td>
<td>Unlikely</td>
<td>Low</td>
<td>[-2 to +2] (22–30)</td>
</tr>
</tbody>
</table>

aPatients with low oncotic pressure (from low serum albumin, etc.) or patients who have been given considerable crystalloid, will be puffy yet may still be hypovolemic in the intravascular space and thus prone to hypotension.

bMany patients do not show higher blood pressures if hypervolemic. Also, hypertensive patients may still be hypovolemic.

cRequires central venous line access. Values noted are approximate; follow trends in CVP rather than absolute values.

dThis is affected by many other factors besides volume: ADH secretion, diuretics, intrinsic renal function, etc.

eHR variability with volume status is best seen in young healthy awake patients. It is not well seen in elderly, deeply anesthetized or beta-blocked or calcium-channel blocked patients. Nor is it seen in patients with intrinsic nodal or conduction system disease.

fHypovolemic patients become hypotensive with even small amounts or concentrations of anesthetics.

gTilting the patient’s body or trunk “head up” when initially supine will result in hypotension if hypovolemic; less so if hypervolemic.

hBase excess or bicarbonate (HCO₃⁻) measurements if acidotic (less than -2 (BE) or less than 22 (HCO₃⁻)) suggest hypovolemia and hypoperfusion leading to lactate accumulation in the blood. This rule presupposes no other cause of acidosis besides hypovolemia.

iRequires an arterial line for instantaneous pressure (variability with respiration) or sampling of arterial blood (base excess or HCO₃⁻).

How to use Table 14.2: Each clinical variable in the top row is easy to assess. The table describes signs of hypo-, euvoema, and hypervolemia. Exceptions exist for each of the above rules; they are for approximate assessment of volume status. More than one variable typically follows during anesthesia, and they usually confirm each other.
After assessment of a patient’s volume status, the essential question: *Is the patient: hypovolemic, euvoolemic, or hypervolemic?*

Armed with the answer to this question, the decision is next to either give fluid or not give fluid, depending on the hemodynamic goals of the moment. There are patients who are kept deliberately hypovolemic, or “dry”, for example, patients with elevated pulmonary artery pressures, COPD, or after certain surgeries, particularly thoracic surgeries. There are also patients who are best kept hypervolemic or “full”; although this is less common. But in general, most caretakers are trying to find euvoolema and maintain it in their patients.

**Calculating Fluid Requirements**

One can calculate a patient’s fluid requirements using a set of rules. These are summarized in Table 14.3 and an example follows in Table 14.4.

**Fluid Replacement Options**

When choosing a fluid replacement option, it is important to differentiate between the various kinds of intravenous fluid used during anesthesia and surgery and in critical care. There are two traditional classes of fluids, crystalloids & colloids (see Tables 14.5 and 14.6):

- **Crystalloids** are the fluids of choice for most minor procedures. They are sterile aqueous solutions which may contain glucose, various electrolytes, organic salts, and nonionic compounds. Some examples of these solutes are sodium chloride, potassium chloride, sodium bicarbonate, calcium carbonate, sodium acetate, sodium lactate, and sodium gluconate. The fluids themselves are known colloquially as normal saline, Ringer’s Lactate, Normosol-R®, etc. Table 14.5 lists the ingredients and characteristics of some commonly used IV fluids; Table 14.6 lists typical practical applications of these fluids in routine anesthetic care.

- **Colloids** are aqueous solutions of derivatized human serum protein macromolecules (albumin 5% or Plasmanate); or carbohydrate macromolecules (Hetastarch). They are prepared so as to be nonimmunogenic and non-infectious. Because of their component solute sodium chloride, they have tonicity and osmolality like crystalloid solutions. But additionally, their macromolecule solute components give them oncotic pressure similar to serum. *The result is that these solutions remain in the intravascular space longer (hours to days) than do crystalloids (minutes to hours).*

Colloids are therefore thought to improve the patient’s intravascular volume and perfusion and minimize weight gain and edema, as compared with crystalloids.
Table 14.3  Calculating perioperative fluid requirements sections 1 + 2 + 3 + 4 = Total fluid needed; *give as directed in italics.*

1. **Basal fluid requirement** based on weight of the patient in kg. 10 kg infant 40 ml/h; 80 kg adult 120 ml/h. *Give continuously.*

<table>
<thead>
<tr>
<th>Wt (kg)</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hourly maintenance ml/h</td>
<td>40</td>
<td>60</td>
<td>70</td>
<td>80</td>
<td>90</td>
<td>100</td>
<td>110</td>
<td>120</td>
<td>130</td>
<td>140</td>
</tr>
</tbody>
</table>

2. **The "NPO" deficit:** basal requirement times hours since fasting started: (8 h × 1.) *Replace in the first hour or two.*

| NPO deficit after 8 h (ml) | 40 × 8 = 320 | 60 × 8 = 480 | 70 × 8 = 560 | 80 × 8 = 640 | 90 × 8 = 720 | 100 × 8 = 800 | 110 × 8 = 880 | 120 × 8 = 960 | 130 × 8 = 1,040 | 140 × 8 = 1,120 |

3. **The replacement for surgical blood loss** is three (3) times the estimated blood loss: *Give as the loss occurs.*

<table>
<thead>
<tr>
<th>Blood loss (ml)</th>
<th>25</th>
<th>50</th>
<th>75</th>
<th>100</th>
<th>150</th>
<th>200</th>
<th>300</th>
<th>400</th>
<th>500</th>
<th>750</th>
</tr>
</thead>
<tbody>
<tr>
<td>Replacement for blood loss (ml crystalloid)</td>
<td>75</td>
<td>150</td>
<td>225</td>
<td>300</td>
<td>450</td>
<td>600</td>
<td>900</td>
<td>1,200</td>
<td>1,500</td>
<td>2,250</td>
</tr>
</tbody>
</table>

4. **The replacement** for “third-space losses” is related to the type of surgery: *Give as needed to support blood pressure, CVP, and urine output.*

| Type of surgery | Minor or peripheral surgery such as ankle fracture, ENT surgery. | Intermediate such as hip surgery, healthy laparoscopy. | Heavy losses such as intraabdominal sepsis, radical neck dissection, large flaps. |
|-----------------|---------------------------------------------------------------|------------------------------------------------------|
| Replacement for third space loss (ml/kg/h) | 1–3 ml/kg/h | 3–6 ml/kg/h | 6–10 ml/kg/h or more |

**How to use Table 14.3:** There are four (4) separate components to be calculated to replace losses with intravenous fluids: (1) Maintenance fluid requirement (in ml/h); (2) NPO deficit from fasting before surgery (in ml); (3) Blood loss to be replaced (in ml); and (4) The so-called “third-space losses” which occur by expansion of the interstitial space after trauma or illness (in ml/h). This table’s four sections show how to calculate each component. Add them up and then administer fluid as indicated by the italics.

*aNotes: (a) Using crystalloid the rule is: administer roughly three times the EBL. (b) If colloid is used to replace EBL, the ratio is about 1 to 1.*

*bNotes: (a) Use crystalloid to replace third space losses. (b) If colloid is used, less is needed.*
Colloids may even under some circumstances draw interstitial fluid back into the intravascular space.

*Albumin 5%* is the colloid most commonly used as a volume replacement. If diluted from 25% albumin it must be diluted with NS, not with hypotonic solutions like water or \( \frac{1}{2} \) NS. Improperly diluted albumin can cause fatal hemolysis after infusion into a patient.

*Plasmanate*® (*purified protein fraction 5%*), contains mostly albumin (88%) but also alpha- and beta- (12%) and some gamma-globulins (1%). Plasmanate is heat-treated to be nonreactive immunologically. But, Plasmanate®, like albumin, is considered to be a “blood product” and therefore unacceptable to many individuals on religious or other grounds. It has 145 meq/l NaCl and is isotonic to plasma.

*Hetastarch*® (ethoxylated amylopectin 6%), is a solution of derivatized macromolecular complex carbohydrates. It has the same tonic, osmotic, and oncotic properties as the protein solutions, but is derived from vegetable matter, and therefore is not a “blood product” and is acceptable to many otherwise opposed to receiving derivatized plasma, such as Jehovah’s Witnesses. Hetastarch® and other similar products are also much less expensive than protein derivative solutions.

### Table 14.4 Example fluid replacement calculation.

<table>
<thead>
<tr>
<th>Patient and procedure:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>An 80 kg male patient undergoes a 1-h tonsillectomy at 8:00 am after being made NPO at midnight.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood Loss:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimated blood loss is ultimately 250 ml</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Crystalloid vs. colloid choice:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Crystalloid is adequate, no colloid needed for this small volume blood loss. Lactated Ringers is optimal though saline could be used.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Replacement:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>(Calculated from the four parts of Table 14.3.)</td>
<td></td>
</tr>
<tr>
<td>Total crystalloid administered is:</td>
<td></td>
</tr>
<tr>
<td>120 ml (maintenance for the 1 h duration)</td>
<td></td>
</tr>
<tr>
<td>+960 ml (for the NPO deficit)</td>
<td></td>
</tr>
<tr>
<td>+750 ml (for the blood loss)</td>
<td></td>
</tr>
<tr>
<td>±250 ml (for the third space loss, estimated at 2 ml/kg/h)</td>
<td></td>
</tr>
<tr>
<td>=2080 of NS or LR over the 2 h perioperative period.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Postoperative maintenance:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>May be 120 ml per hour with adjustments made based on vital signs and urine output.</td>
<td></td>
</tr>
</tbody>
</table>
Table 14.5 Ingredients and characteristics of commonly used crystalloid and colloid fluids.

<table>
<thead>
<tr>
<th>IV fluid</th>
<th>H₂O</th>
<th>D5W</th>
<th>NS</th>
<th>D5½ NS + KCl 20 meq/l</th>
<th>LR</th>
<th>D5LR</th>
<th>Normosol-R®</th>
<th>Plasmalyte-148®</th>
<th>Hetastarch</th>
<th>Albumin 5%</th>
<th>Plasmanate 5%</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.0</td>
<td>4.0</td>
<td>5.5</td>
<td>4.5</td>
<td>6.5</td>
<td>4.9</td>
<td>7.4</td>
<td>5.5</td>
<td>5.5</td>
<td>7.4</td>
<td>7.4</td>
</tr>
<tr>
<td>Mosm/l</td>
<td>0</td>
<td>252</td>
<td>308</td>
<td>446</td>
<td>279</td>
<td>525</td>
<td>296</td>
<td>294</td>
<td>310</td>
<td>309</td>
<td>280–285</td>
</tr>
<tr>
<td>Na meq/l</td>
<td>0</td>
<td>0</td>
<td>154</td>
<td>77</td>
<td>130</td>
<td>140</td>
<td>140</td>
<td>140</td>
<td>154</td>
<td>160</td>
<td>145</td>
</tr>
<tr>
<td>K meq/l</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>4</td>
<td>5</td>
<td>5</td>
<td>0</td>
<td>&lt;2</td>
<td>0.25</td>
</tr>
<tr>
<td>Cl meq/l</td>
<td>0</td>
<td>0</td>
<td>154</td>
<td>77</td>
<td>109</td>
<td>98</td>
<td>98</td>
<td>98</td>
<td>154</td>
<td>130</td>
<td>100</td>
</tr>
<tr>
<td>Ca meq/l</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mg meq/l</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>HCO₃ meq/l</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lactate mmol/l</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>28</td>
<td>28</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Acetate mmol/l</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>27</td>
<td>27</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Gluconate mmol/l</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>23</td>
<td>23</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Glucose mg/dl</td>
<td>5000</td>
<td>0</td>
<td>5,000</td>
<td>0</td>
<td>5,000</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Colloid</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>60 g/L starch</td>
<td>40–50 g/L human albumin</td>
<td>50 g protein: (88% albumin; 12% α,β; 1% γ)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kcal/l</td>
<td>0</td>
<td>170</td>
<td>0</td>
<td>170</td>
<td>9</td>
<td>179</td>
<td>15</td>
<td>21</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Serum osmolality \( \text{sosm} \) is 275–295 and may be calculated by \( 2(Na+K) + \text{glu}/18 + \text{BUN}/2.8 \).
<table>
<thead>
<tr>
<th>Fluid →</th>
<th>H2O</th>
<th>D5W</th>
<th>D5 ½NS+20 meq KCl/l</th>
<th>NS</th>
<th>Lactated Ringer's</th>
<th>Normosol-R® or Plasmalyte-148®</th>
<th>Hetastarch®</th>
<th>Albumin 5 % or Plasmanate® 5%</th>
<th>Plasmalyte-148®</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical use</td>
<td>Diluent for small volumes of medication – hypotonic – may not be infused intravascularly because it will lyse RBCs</td>
<td>Keep open fluid used just to give medicines</td>
<td>Classic maintenance fluid for medicine patients on the ward having nothing but insensible and obligatory losses.</td>
<td>Classic replacement fluid for initial resuscitation for dehydration and blood loss</td>
<td>Classic replacement fluid for perioperative surgical losses</td>
<td>Used in cardiac, renal, hepatic, especially transplantation surgeries because it produces no lactate load</td>
<td>Volume expansion in cases where losses have exceeded 2 L.</td>
<td>Used in cardiac, renal, hepatic, especially transplantation surgeries</td>
<td></td>
</tr>
<tr>
<td>Advantages</td>
<td>NaCl free, so good to dilute antibiotics and other salts.</td>
<td>NaCl free, will keep withdrawing alcoholic patients from becoming hypoglycemic if fasted.</td>
<td>Correct amount of NaCl &amp; free water for insensible losses</td>
<td>Cheap, may be used to administer blood</td>
<td>This is used to prevent the metabolic acidosis found after NS used to replace blood losses</td>
<td>Calcium free so it may be used to administer blood just as saline is.</td>
<td>Inexpensive, contains fixed amount of sodium; not derived from blood</td>
<td>May be given in large quantities without concern for coagulopathy other than dilutional.</td>
<td></td>
</tr>
<tr>
<td>Disadvantages</td>
<td>Special properties</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypotonic, dangerous to infuse; causes H₂O intoxication if given enterally in excess</td>
<td>Hurts veins and causes hemolysis. Don't use to dilute 25% albumin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May lower Na, contains too much glucose for many patients if infused rapidly</td>
<td>Does not hurt veins; gives 20 kcal per 100 ml, prevents hypoglycemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not ideal to bolus in hypovolemic or oliguric patients: NS or LR better</td>
<td>This is sold with the added KCl because 3 liters will replace exactly the obligate daily K⁺ loss in adults</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>May cause mild metabolic acidosis if used to replace moderate blood loss; contains too much sodium for some patients</td>
<td>May be given intraoperatively to mildly hyponatremic patients to help normalize serum sodium levels very gently.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium in it makes it incompatible with citrated blood products. Patients with liver disease may not tolerate lactate due to impaired gluconeogenesis.</td>
<td>Avoid in anephric patients because of the small amount of K⁺ in it.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not a source of calories as a maintenance fluid; more expensive than NS</td>
<td>Does not have glucose so alcoholic patients may need to have glucose monitored intraoperatively to avoid hypoglycemia.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Given in quantities greater than 2L it may induce coagulopathy</td>
<td>Inhibits von Willebrand factor (vWF) function on platelets.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Derived from blood, which is unacceptable to some patients. Expensive. (Plasmanate 5%: non-albumin proteins may be immunogenic: avoid Plasmanate in transplant patients.)</td>
<td>Albumin: Has a variable amount of sodium chloride in it.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Introduction to Acid-Base Analysis**

Acid-base equilibrium is important because almost all cellular biochemical reactions take place in the aqueous phase. The concentration of hydrogen ions (the pH) in the various fluid compartments controls, among other things, the conformation of proteins and the feasibility and speed of all the reactions. The pH is highly regulated; cellular death will occur quickly if the normal ranges are exceeded by being too basic (high pH) or too acidic (low pH). So diagnosis and treatment of acid-base disturbances must be accurate, often immediate.

The arterial blood gas panel consists of four values: pH, PaCO₂, PaO₂, and HCO₃⁻ (or a related derivative calculation of HCO₃⁻ called the Base Excess, BE). It is important to know the inspired oxygen concentration (FiO₂) paired with each individual blood gas to determine the quality of oxygen delivery to the blood.

It is also important to know the Anion gap (AG) drawn from arterial or venous blood. Anion gap is a derived quantity that is obtained by subtracting the values for serum chloride and HCO₃⁻ from serum sodium. The normal AG is 12–20 meq/L. To check this, here we substitute the following normal serum electrolyte values into the equation: (Na⁺–HCO₃⁻ – Cl⁻ = AG); [Normals: 140 – 24 – 101 = 15 meq/L, with a range of 12–20 meq/L].

The first step in blood gas analysis is to decide whether the patient has a normal pH (7.35–7.45), is acidemic (low pH, less than 7.35), or is alkalemic (high pH, greater than 7.45).

1. If the blood gas shows the patient is acidemic (pH < 7.35), then:
   (a) Look at the PaCO₂. If it is greater than 40 mm Hg, then the patient has respiratory acidosis. Respiratory acidosis is caused by excess dissolved CO₂ in the blood, due to either inadequate ventilation of CO₂ out of the lungs or excess production of CO₂ in the body. There are several possible underlying causes: hypoventilation, which is decreased minute ventilation (decreased respiratory rate or decreased tidal volume); obstruction of the small airways (COPD, asthma); overdosage of alcohol, sedatives, opioid medications; or neuromuscular disease (like myasthenia gravis). Or, overproduction of CO₂ may be from hyperthermia or overfeeding.
   (b) If the PaCO₂ is normal or slightly decreased, then the patient has a metabolic acidosis. This is caused by one of several dissolved “acids” or acidic substances in the blood (either endogenous, such as lactic acid, or exogenous, such as ethanol) that are lowering the pH. In response, the body may encourage hyperventilation to counterbalance this to some extent.
There are two kinds of metabolic acidosis: Anion gap acidosis (AG > 20 meq/L) and Non-anion gap or Normal anion gap acidosis (AG < 20).

Anion gap acidosis is summarized by the classic mnemonic MUDPILES which is used to recall its most likely causes: Methanol, Uremia, Diabetic ketoacidosis, Propylene glycol, Isoniazid (INH), Lactate, Ethylene glycol, and Salicylates. The mnemonic is useful but almost quaint in that it recalls a number of toxins or drug side effects rarely seen today clinically.

Non-anion gap acidosis (also known as hyperchloremic acidosis) is caused by either diarrhea, administration of NaCl solutions (normal saline) especially during surgery or after traumatic blood loss, acetazolamide use, or renal tubular acidosis. All four have the common etiology of bicarbonate loss. Therefore, the treatment of metabolic acidosis is the administration of intravenous bicarbonate solutions or a precursor: lactate, citrate, or acetate.

2. If the pH is greater than 7.45 (recall that normal pH is 7.35–7.45) then the patient has alkalemia:
   (a) Look at the PaCO₂ as before. If it is less than 40 mm Hg, then the patient has respiratory alkalosis. Respiratory alkalosis is caused by decreased levels of dissolved CO₂ in the blood, due to either hyperventilation of CO₂ out of the lungs or decreased production of CO₂ in the body. There are several possible underlying causes: central or CNS-induced hyperventilation, which is increased minute ventilation (increased respiratory rate and/or increased tidal volume), usually from anxiety or a CNS lesion; the respiratory stimulus of altitude; pregnancy; or too much mechanical ventilation. Alternately, underproduction of CO₂ may be from hypothermia or muscle relaxation from nondepolarizing muscle relaxant drugs. One can correct respiratory alkalosis by adjusting ventilation or treating anxiety with sedatives.
   (b) If the PaCO₂ is normal or slightly increased, then the patient has a metabolic alkalosis. This is caused by one of several causes: vomiting or loss of protons in gastric secretions owing to nasogastric or orogastric suction (classically, in the face of gastric outlet obstruction); diuretic use (classically after heavy furosemide diuresis in the postoperative period – especially after cardiac surgery); antacid use; or hyperaldosteronism. Metabolic alkalosis needs to be corrected because the condition predisposes to dysrhythmias, cerebral vasoconstriction, and coronary vasoconstriction. Also, in mechanically ventilated patients, the condition leads to a vexing
secondary effect: the retention of CO$_2$ in the blood, which makes weaning from mechanical ventilation more troublesome in some patients. One corrects metabolic alkalosis by simply infusing normal saline, potassium chloride, or both, intravenously, or, in severe cases, dilute hydrochloric acid is carefully infused centrally. Acetazolamide may also be used if the patient can't tolerate the increased volume load of intravenous solutions.

In summary, rules for the analysis of blood gases and acid-base status are found in the two tables below. Table 14.7 summarizes the four primary acid-base disorders. Table 14.8 quantifies the degree of pH, PaCO$_2$, PaO$_2$, and HCO$_3^-$ secondary compensation expected for the purest examples of the various acid-base disturbances. In actual clinical practice, a patient may manifest one, two, or three combined acid-base disturbance, all of which ultimately contribute to the pH. Therefore the clinicians overall goal is to restore the pH to the normal range, or near to it, as quickly as possible.

<table>
<thead>
<tr>
<th>Disorder</th>
<th>pH</th>
<th>PaCO$_2$</th>
<th>HCO$_3^-$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory alkalosis</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>

**Table 14.8 Expected compensatory responses in primary acid–base disorders.**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Equation (PaCO$_2$ – 40)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute respiratory acidosis</td>
<td>No change in base deficit or excess</td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>No change in base deficit or excess</td>
</tr>
<tr>
<td>Chronic respiratory acidosis</td>
<td>Base deficit or excess = 0.4 × (PaCO$_2$ – 40)</td>
</tr>
<tr>
<td>Chronic respiratory alkalosis</td>
<td>Base deficit or excess = 0.4 × (PaCO$_2$ – 40)</td>
</tr>
<tr>
<td>Acute metabolic acidosis</td>
<td>PaCO$_2$ = 40 + base deficit or excess</td>
</tr>
<tr>
<td>Acute metabolic alkalosis</td>
<td>PaCO$_2$ = 40 + (0.6 × base deficit or excess)</td>
</tr>
</tbody>
</table>

Adapted from Acute Heart Failure By Alexandre Mebazaa, Mihai Gheorghiade, Faiez M. Zannad; Published by Springer, 2008 ISBN 1846287812, 9781846287817 page 464.
Transfusion of Blood Products: Goals and Indications

The goals of transfusion are several; one or more may apply to any given patient. Transfusion may be done prior to surgery to replace RBC volume in acutely or chronically anemic patients. It is used during and after surgery to replace traumatic, intraoperative, or postoperative losses of red blood cells. In cases of coagulopathy, it is done to replace coagulation factors and thereby, restore hemostasis. In autoimmune or dilutional thrombocytopenia, transfusion of platelets may at least partly correct these conditions and allow thrombosis to occur normally. In cases of platelet inactivity due to disease or medications (e.g., NSAIDs), a small amount of platelets (one unit rather than 6 pooled units) can serve as a catalyst and initiate platelet thrombus formation and achieve the first steps of hemostasis. Finally, long after surgery, or in protracted illnesses or recuperation, it is often necessary to give RBC when a critical anemic threshold is met.

As the length of procedure and blood loss increase, replacement of blood products may be needed. Besides the clinical volume criteria listed above in Table 14.3, the hematocrit (HCT) is another clinical datum used for assessing red blood cell volume (RBCV) and anemia indirectly. HCT is really a surrogate measurement for RBCV, which is impossible to measure practically.

As a case begins, one can calculate a patient’s allowable blood loss (ABL) by using the formula below in Fig. 14.2. This gives the anesthesiologist a guide to know how much blood loss can occur prior to starting a blood transfusion (Fig. 14.3).

Serial HCT readings (plus the clinical criteria used to assess volume status, see Table 14.3) are the basis for choosing to transfuse blood cells. By practical convention, one gram of Hb is equivalent to 3 HCT percentage points. For example, if a patient has a Hb of 10 g/dl, the HCT will be approximately 30. Furthermore, each unit of PRBC in an adult is expected to raise the HCT by

\[
\text{Allowable Blood Loss (ABL) Formula}
\]

\[
\text{EBV} \times \left( \frac{\text{HCT}_{\text{initial}} - \text{HCT}_{\text{final}}}{\text{HCT}_{\text{initial}}} \right)
\]

\[
\text{where } \text{HCT}_{\text{final}} = \text{lowest acceptable hematocrit}
\]

Figure 14.2 Allowable Blood Loss (ABL) Formula.
3 points. If such a predicted increase does not occur, one should be concerned about ongoing blood loss, hemolysis, or hemodilution with excess IV fluids.

HCT is drawn from venipuncture, peripheral or central venous line, or arterial line. One must interpret HCT carefully, because dilution from crystalloid or colloid may cause significant variation in HCT even without any significant blood loss.

Decisions about giving platelets and plasma or plasma derivatives are based, in a similar way, on both clinical criteria and lab values. Diagnostic lab studies such as coagulation panels (PT, PTT, INR, platelet counts), and more specific studies such as specific factor levels, may be used in more challenging cases such as hepatic transplantation (high volume fluid turnover), or in the setting of end-stage liver disease (because of the confounding factor of pre-existing coagulopathy). Patients with known hemophilia or platelet abnormalities may also warrant more specialized studies of coagulation in the perioperative period.

Using CVP and PA Catheters for Volume Assessment

If surgical blood loss is expected to exceed one liter, placement of a CVP should be a consideration. The insertion of these lines is discussed in Chap. 15. Insertion of a CVP line allows convenient monitoring of right atrial (RA) pressure, central venous oxygen saturation (CvO₂), and serial HCT—all of which are useful to assess volume status and RBCV (see Table 14.2).

The use of pulmonary artery (PA) catheters is much less common than the use of CVP catheters. They nevertheless are useful at assessing volume status more precisely than a CVP can. PA catheters also allow sampling of mixed venous (SvO₂) blood, which is a more accurate means of assessing total-body oxygen delivery than is CvO₂. PA catheters also allow one to manage fluids

**Estimated Blood Volume (EBV) Formula**

\[
EBV = \text{weight (kg)} \times \text{average blood volume}
\]

*Note: Avg. blood volume in adult male = 75 ml/kg
Avg. blood volume in adult female = 65 ml/kg*
meticulously in the setting of CHF, COPD, and pulmonary hypertension. One typically measures the PA occlusion pressure (PAOP or wedge pressure) intermittently, in order to reduce the hazard of PA perforation. Equally valid but safer is to serially follow the PA diastolic pressure trends. Armed with this information about volume status, one can replace fluid accordingly, but usually with smaller doses of fluid (100 or 200 ml at a time). The advantage of this is that less excess fluid will be administered over time to a vulnerable patient.

**Transfusion of Blood Products: Practical Aspects**

There are some general considerations to keep in mind when transfusing blood products. Transfusion is much more hazardous, expensive, and controversial than infusing crystalloid or non-blood-product derived colloids. Fortunately, most anesthetics are accomplished with crystalloid administration only, or crystalloid plus colloid. When transfusion is indicated because of coagulopathy, anemia, or massive blood loss, it should be given promptly to prevent end-organ damage and death from life-threatening anemia, tissue hypoxia, and acidosis.

Blood products available for transfusion include:

- **Red blood cells** (RBCs) given for anemia or ongoing blood loss
- **Fresh-frozen plasma** (FFP) given for mild coagulopathy (PT or INR elevation or severe fibrinogen deficiency)
- **Platelets** given for immune or dilutional thrombocytopenia
- **Cryoprecipitate** given for severe coagulopathy and Factor VIII deficiency
- **Whole blood** is rarely used, since blood is typically separated into components (RBC, plasma, and platelets) in order to allow more efficient use
- **Other more specific coagulation factors** (human or recombinant) may be used to treat coagulopathy

More specialized factors used to treat coagulopathy include activated Factor VII (FVIIa), a newer agent given for severe diffuse postsurgical coagulopathy when there is no discrete source of bleeding. FVIIa has also been used to treat intracranial hemorrhage. Another specialized factor product is known as Factor IX concentrate. This is a combination of Factor IX (i.e., Christmas factor, antihemophilic factor B), Factor II (prothrombin), Factor X (Stuart-Prover Factor), and low non-therapeutic levels of Factor VII (proconvertin), all derived from human pooled plasma. The indication for giving them is for severe coagulopathy. These products are also used before surgery if a specific Factor IX deficiency (i.e. hemophilia) is demonstrated with lab studies.
Hazards of Transfusion

Some of the hazards of transfusion are very well known and quantified, and others not so well known. These hazards are discussed also in Chap. 16, Common Intraoperative Problems. Here we will emphasize the major risks of transfusion and how they relate to the decision to transfuse. These include infection, immunosuppression, long-term morbidity, and transfusion reactions.

The public is most concerned about the risks of transfusion-associated infection, especially viral infection, from HBV, HCV, CMV, and HIV. There are other infectious hazards as well, listed in Table 14.9.

There are some emerging data on long-term immunosuppression and other increased morbidity and mortality following transfusion. This is not well described in the literature. The difficulty in all transfusion-related outcomes research is separating true causes of bad outcomes from mere epiphenomena or anecdotal evidence.

Transfusion Reactions (Also see Chap. 16, Common Intraoperative Problems)

Transfusion reactions come in varying kinds and degrees of severity. Table 14.10 lists the various kinds of immunologically-mediated transfusion reactions.

The most common type of serious transfusion reaction is the major acute hemolytic reaction (from ABO or Rh- incompatibility): The usual cause is clerical error prior to transfusion. The problem is that the transfusion recipient has antibodies against donor RBC membrane ABO or Rh- antigens. The antibodies bind to the donor RBC membrane antigens and activate complement, inducing hemolysis. The free Hb goes into the bloodstream and can damage the kidneys. There are many other sequelae to the hemolysis. The treatment for such a reaction is first to immediately stop transfusion, resend patient and unit blood for re-crossmatch (clerical or crossmatching errors are most likely), use mannitol and furosemide for diuresis, monitor urine volume and hemoglobin, check serum haptoglobin to monitor hemolysis, and support hypotension with volume, pressors, inotropes. Major acute hemolytic reactions are often fatal.

Compatibility and anticipating reactions is therefore the greatest concern when transfusing blood. Table 14.12 shows recipient versus donor compatibility, for various blood products. It allows one to identify the recipient, choose...
Table 14.9 Infectious risks of transfusion and estimates of occurrence.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Microorganism</th>
<th>Transmitted in this blood product</th>
<th>Incidence per unit transfused</th>
<th>Transmissible by “needle stick” or blood exposure</th>
<th>Prophylaxis or treatment</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDS or HIV disease</td>
<td>HIV 1 virus</td>
<td>All blood products, not in albumin</td>
<td>1:500,000</td>
<td>Yes</td>
<td>HAART</td>
<td>Consider post exposure prophylaxis, consult ID specialist</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>HCV virus</td>
<td>All blood products, not in albumin</td>
<td>1:100,000</td>
<td>Yes</td>
<td>Interferon alpha 2a plus ribavirin</td>
<td>Liver transplantation not contraindicated in some pts who have liver failure from HCV</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>HBV virus</td>
<td>All blood products, not in albumin</td>
<td>1:70,000</td>
<td>Yes</td>
<td>HBIG plus Lamivudine</td>
<td>More common in Asia and complicates posttransplant liver function</td>
</tr>
<tr>
<td>CMV infection</td>
<td>Cytomegalovirus</td>
<td>RBC, platelets</td>
<td>1:50</td>
<td>Yes</td>
<td>WBC filters; Frozen deglycerolized RBC, screen donors</td>
<td>CMV-free blood now only indicated for immunosuppressed pts</td>
</tr>
<tr>
<td>Malaria</td>
<td><em>Plasmodium falciparum</em></td>
<td>RBC</td>
<td>1:3,000,000</td>
<td>Yes</td>
<td>Antimalarial therapy</td>
<td>Not common in nonendemic regions</td>
</tr>
<tr>
<td>Bacterial infection</td>
<td><em>Staphylococcus</em> spp; <em>Salmonella</em> spp; <em>Enterobacter</em> spp; <em>Serratia marcescens</em></td>
<td>Platelets</td>
<td>1:15,000</td>
<td>Yes</td>
<td>Broad then narrow antibiotic coverage according to cultures</td>
<td>Platelets pooled and administered at room temperature</td>
</tr>
<tr>
<td>Bacterial infection</td>
<td><em>Staphylococcus</em> spp; <em>Salmonella</em> spp; <em>Enterobacter</em> spp; <em>Serratia marcescens</em></td>
<td>RBC</td>
<td>1:1,000,000</td>
<td>Yes</td>
<td>Broad then narrow antibiotic coverage according to culture results</td>
<td>Bacterial sepsis from blood products has a high mortality of 25% according to some authors, so it should be treated aggressively</td>
</tr>
</tbody>
</table>

Bacterial sepsis from blood products has a high mortality of 25% according to some authors, so it should be treated aggressively.
<table>
<thead>
<tr>
<th>Type</th>
<th>Incidence</th>
<th>Commonest after administration of</th>
<th>Symptoms</th>
<th>Treatment or prophylaxis</th>
<th>Immune Mechanism</th>
<th>Time course</th>
<th>Fatality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-hemolytic febrile reaction</td>
<td>Common</td>
<td>Platelets, RBC</td>
<td>Chills, fever</td>
<td>Acetaminophen, ibuprofen, diphenhydramine, leukocyte reduction of transfused blood.</td>
<td>Mediated by inflammatory cytokines in the recipient</td>
<td>Onset 16 h after transfusion</td>
<td>Not fatal</td>
</tr>
<tr>
<td>Acute hemolytic transfusion reaction</td>
<td>1:10,000 occurring with resulting 20 fatalities per year in USA</td>
<td>Clerically mismatched blood. Worst is from Type A donor given to Type O recipient</td>
<td>Flank pain if awake, bloody or dark urine, shock. This is the classic severe transfusion reaction</td>
<td>Careful crossmatching and checking of blood by caretakers before administration. To treat, see text</td>
<td>Hemolysis of the donor red blood cells by host IgM antibodies usually related to ABO blood group incompatibility. Complement is activated</td>
<td>May begin minutes after transfusion begun.</td>
<td>May be fatal, may cause renal failure</td>
</tr>
<tr>
<td>Delayed hemolytic transfusion reaction</td>
<td>Rare except in patient receiving many transfusions such as SCD patients</td>
<td>Multiple RBC transfusions as for SCD patients</td>
<td>Fever, lower than expected blood hemoglobin, jaundice, urobilinogenuria</td>
<td>Supportive therapy.</td>
<td>Delayed hemolysis of blood from alloimmunization developing in recipient. IgM antibodies and complement are involved</td>
<td>Onset one to several weeks</td>
<td>May range from subclinical to fatal</td>
</tr>
<tr>
<td>Condition</td>
<td>Frequency</td>
<td>Description</td>
<td>Treatment</td>
<td>Duration</td>
<td>Outcome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>-----------</td>
<td>------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------</td>
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<td>--------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaphylactic reaction</td>
<td>1:20,000</td>
<td>Most common in recipients with selective IgA deficiency</td>
<td>Shock, breathing difficulties, wheezing, etc</td>
<td>Supportive</td>
<td>IgE and IgA mediate,</td>
<td>Within minutes of</td>
<td>May be fatal</td>
</tr>
<tr>
<td>Transfusion-related acute lung injury (TRALI)</td>
<td>1:2,000</td>
<td>Large amounts of whole blood or plasma</td>
<td>May be mild to life threatening: Respiratory distress, fever, non-cardiogenic pulmonary edema, hypotension</td>
<td>Supportive,</td>
<td>Antibodies in donor</td>
<td>Transfusion.</td>
<td>Mortality is less than 10%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>including</td>
<td>blood product against HLA (A, B, C, DR) and other antigens in the recipient. Pulmonary capillary alveolar leak</td>
<td>Onset hours, patients recover fully within 96 h</td>
<td></td>
</tr>
</tbody>
</table>

*SCD Sickle cell disease.*
### Table 14.11 Transfusion of blood products: description, indications, contraindications.

<table>
<thead>
<tr>
<th>Productyna</th>
<th>Packed red blood cells (PRBC)</th>
<th>Fresh frozen plasma</th>
<th>Platelets</th>
<th>Cryoprecipitated Antihemophilic Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Synonyms</td>
<td>Packed Cells, Red Cells, Packed Red Blood Cells, RBCs, PRBCs</td>
<td>FFP, FFP24</td>
<td>Random donor platelets, RDPs. Plateletspheres are single donor platelets, or SDPs</td>
<td>Cryoprecipitate, cryo, pooled cryo</td>
</tr>
<tr>
<td>Description</td>
<td>RBC are prepared from whole blood with plasma &amp; platelets removed. HCT of RBC is 70%. Citrate anticoagulant added</td>
<td>Noncellular portion of blood that is separated &amp; frozen after donation. It may be prepared from whole blood or collected by apheresis. Citrate anticoagulant added</td>
<td>4-10 RDPs are pooled by blood bank. SDPs are ready for transfusion. Citrate anticoagulant added</td>
<td>A cryoprecipitate unit is prepared by thawing one unit of FFP between 1-6 °C &amp; recovering the cold insoluble precipitate. Cryoprecipitate contains fibrinogen, Factor VIII:C, Factor VIII: vWF, Factor XIII, and fibronectin. Citrate anticoagulant added</td>
</tr>
<tr>
<td>Indications</td>
<td>Not bleeding &amp; stable: (a) Patients without cardiovascular dz &amp; esp. younger pts, keep Hb range 7 – 9 g/dl (b) Patients with cardiovascular disease: Keep Hb in the range ≥ 10 g/dl</td>
<td>1. Active bleeding due to deficiency of multiple coagulation factors, or risk of bleeding due to deficiency of multiple coagulation factors. 2. Severe bleeding due to warfarin therapy, or urgent reversal of warfarin effect.</td>
<td>1. Use platelets prophylactically to prevent bleeding at pre-specified low platelet counts. 2. In general, maintain platelet count &gt;10,000/mm³ in stable, non-bleeding patients, &gt;20,000/mm³ in unstable non-bleeding patients and &gt;50,000/mm³ in patients undergoing invasive procedures or actively bleeding</td>
<td>1. Bleeding associated with fibrinogen deficiency (&lt; 100 mg/dl) 2. Bleeding associated with Factor XIII deficiency. 3. Prophylactic treatment for head trauma associated with DIC</td>
</tr>
</tbody>
</table>
### Bleeding:

(a) 1500-2000 ml (30%) blood loss: transfusion of RBC likely
(b) > 2000 ml blood loss: RBC transfusion needed

*In all cases: Use clinical judgment & check HCT before transfusing.*

<table>
<thead>
<tr>
<th>3. Massive transfusion with coagulopathic bleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>4. Bleeding or prophylaxis of bleeding for a known single coagulation factor deficiency for which no concentrate is available</td>
</tr>
<tr>
<td>5. Thrombotic thrombocytopenic purpura</td>
</tr>
<tr>
<td>6. Rare specific plasma protein deficiencies, such as C1-esterase inhibitor</td>
</tr>
<tr>
<td>3. Intraoperative cardiovascular, thoracic, or neurosurgical patients, maintain platelets above 100,000/mm³</td>
</tr>
</tbody>
</table>

### General Information

<table>
<thead>
<tr>
<th>Contraindications</th>
<th>1. Do not give if patients are not bleeding, healthy, BP and HR normal, and HCT is greater than 21</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2. Do not transfuse in other patients if hemoglobin is greater than 10</td>
</tr>
</tbody>
</table>

| 1. Not to be used for increasing blood volume or albumin concentration |
| 2. Do not use for treating coagulopathy that can be corrected with Vitamin K |
| 3. Do not use to normalizing abnormal coagulation screen results, in the absence of bleeding |
| Do not use in patients with autoimmune thrombocytopenia or thrombotic thrombocytopenic purpura except for life-threatening hemorrhage |

| Do not transfuse cryoprecipitate unless laboratory studies confirm deficiency of a specific clotting protein for which this component is indicated (e.g., fibrinogen) |
AnesthesiA student survivAl Guide

The blood product to be given, and then determine which donors would be compatible with the recipient. Even if donor and recipient are compatible by crossmatching, there may still be immune reactions to blood transfusion. The most common benign transfusion reaction is the minor febrile non-hemolytic transfusion reaction (mild immunoglobulin incompatibility or cytokine reaction). This is more common than an acute hemolytic transfusion reaction and is much less problematic. Treatment involves administration of diphenhydramine 25 mg IV, acetaminophen 500 mg, or ibuprofen 400 mg enterally, and monitoring vital signs along with urine output. Often, the transfusion may continue if the patient is stable.

Another problem in crossmatch-compatible blood transfusion is known by the acronym TRALI (transfusion-related acute lung injury). It has an estimated incidence of 1:2000 and is thought to be mediated by leucoagglutinating...
antibodies in the donor plasma directed against HLA antigens in the recipient. It manifests as non-cardiogenic pulmonary edema, and has a mortality rate of less than 10 percent.

Other Problems Associated with Transfusion

**Hypothermia** is a common problem associated with transfusion. As with any infusion, use an inline IV fluid warmer and don’t warm blood products or fluids in a microwave or non-FDA-approved device.

**Hyperkalemia** may occur because PRBCs, especially those close to expiration, have a significant K⁺ load. Be sure to monitor potassium in patients with renal insufficiency who receive PRBCs.

**Hypocalcemia** is also common because the citrate anticoagulant used to store blood products is a calcium binder. If given in enough quantity (8–10 units of blood), citrate may cause transient hypocalcemia manifested as vasodilatation and hypotension. In order to treat, one should obtain an ionized (not standard) calcium level, and administer 1–2 g of calcium chloride or calcium gluconate through a central catheter or large IV. Do not give calcium with bicarbonate or it will precipitate and cause catastrophic tissue necrosis.

Transfusion: Legal and Ethical Issues

There are legal, professional, religious, and economic issues related to transfusion. Physicians have a legal duty to give blood when indicated (and permitted by the patient) to prevent organ damage from hypotension, tissue hypoxia, and acidosis. A competent patient, however, also has the absolute right to refuse transfusion or any therapy. Informed consent applies to blood transfusion and some institutions have a dedicated form for obtaining it.

**Religious or philosophical issues:** Jehovah’s Witnesses and others are doctrinally opposed to transfusion of blood products and should be queried regarding their wishes during anesthesia and postoperative care. Remember that besides PRBCs many other products (albumin, Plasmanate®, platelets, cryoprecipitate, as well as factor IX concentrates) are derived from human blood. However, patients’ specific beliefs about these products vary, and a detailed conversation and written documentation of a patient’s wishes will avoid confusion.

**Professional issues:** It is wise to include other physicians and caretakers in discussions about transfusion prior to initiation. It is also a good practice to use evidence based professional guidelines for transfusion therapy (see Tables 14.11 and 14.12). Patients and families are very worried about
transfusion and will want to know the indications (Table 14.10 clarifies these) and give informed consent. Transfusion and the use of coagulation factors is fraught with complications and therefore it is wise to achieve consensus among the caretakers, patient, and family before transfusing. The standard guideline thresholds and dosages for transfusion of various blood products are listed in Table 14.11.

**Economic issues:** Transfusion is very expensive (compared with infusing crystalloid or colloid) as are the recombinant-derived blood proteins. Usually it’s wise to confer with others about cost-effectiveness before prescribing.

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**Case Study**

A 25-year-old otherwise healthy woman is to undergo radical resection of a pelvic sarcoma with prosthetic reconstruction to attempt to salvage the hip joint and thigh. The surgeon estimates blood loss will be 2–5 liters, depending on the findings at operation and extent of major vascular involvement. The estimated surgical time is 6 h. She has a peripheral 14 G IV, a three-lumen central venous catheter in the right internal jugular vein, and a 20G right radial arterial line. She has 4 units of packed red cells available. She weighs 60 kg. Her preoperative hemoglobin and hematocrit are 12 and 36 respectively. She has fasted overnight and is scheduled for the first case in the morning.

**How will you estimate her basic fluid requirements for the case?**

You can estimate her hourly maintenance fluid needs with the “4-2-1” rule, calculating 4 ml/kg/h for the first 10 kg of body weight, 2 ml/kg/h for the next 10 kg, and 1 ml/kg/h for each additional 10 kg. This results in 40+20+4(10)=100 ml/h. Assuming an 8 h overnight fast, her deficit preop is 800 ml. Her ongoing maintenance fluid requirement for 6 h of surgery will be 600 ml. Her estimated blood loss is likely extreme, and will be replaced initially at three times EBL, or some 6–15 L. Clearly, some of this will be replaced with blood or colloid solutions, not merely crystalloid. Her “third space” or interstitial fluid losses will be moderate to severe, depending on whether the peritoneum is exposed by the dissection or not. We can estimate these losses at 6 ml/kg/h or more, totaling 360 ml/h or approximately 2.5 L for the case.
How low will you let her hemoglobin drop?
The overwhelming preponderance of the evidence suggests that the optimal Hb target for most patients is 7–9 g/dl. This is true even in the case of stable coronary artery disease, and it is certainly the case for this otherwise healthy young woman. In fact, in volunteers, isovolemic hemodilution to at least 5 g/dl is well tolerated.

What is her acceptable blood loss?
ABL is often calculated with a formula based on the assumption that blood loss occurs at a constant rate throughout the case, and that the patient’s blood volume remains constant by replacement with blood-free solutions. In this young woman, her estimated blood volume is 65 ml/kg × 60 kg = 4 L. Her ABL, given a starting hematocrit of 36 and an acceptable nadir of 21 (equivalent to a hemoglobin of 7 g/dl), is ABL = 4 L*(36–21)/36 = 1.7 L. In practice, anesthesiologists will check hemoglobin/hematocrit periodically as well as make judgments regarding the rate of ongoing blood loss and the adequacy of volume repletion and thus begin transfusion either earlier or later than when this amount has been lost.

How will you assess and correct other blood product requirements?
In sudden blood loss situations such as massive trauma, some authorities recommend empirical administration of packed red cells, plasma, and platelets. In the case of operative losses, it is generally prudent to replace factors by monitoring PT and PTT and platelets by monitoring the platelet count. Keeping the PT less than 1.5 times control and the platelet count above 50,000 is generally recommended, although in the setting of ongoing blood loss, more aggressive replacement is often performed. Fibrin is the ultimate substrate for blood clot, so fibrinogen should also be monitored and kept over 100 mg/dl.

What options do you have for reducing transfusion requirements?
There are at least three possibilities. First, controlled hypotension is a strategy to reduce blood loss by reducing the hydrostatic pressure causing blood to leave traumatized blood vessels. Reducing the blood pressure to a mean of approximately 50–60 mm Hg is considered safe in healthy patients and reduces blood loss in a variety of types of surgery. This can be
achieved with short acting beta blockers (e.g., esmolol), high concentration of inhaled agents, or direct acting vasodilators (e.g., nitroprusside). Second, normovolemic hemodilution is a technique, which “pre-dilutes” the blood of the patient to a lower hematocrit prior to surgery, so that surgical blood loss contains fewer red cells. Blood is removed from the patient and stored in the same containers used in the blood bank; it is replaced with crystalloid or colloid solutions in a normovolemic fashion (typically 3:1 or 1:1, respectively, or as guided by a CVP catheter). Later in the case, the patient’s own blood is returned by transfusion. Finally, intraoperative cell salvage has been successfully employed in a variety of clinical situations. Blood is aspirated from the surgical field into a reservoir where it is periodically washed and filtered to yield a high hematocrit blood product from the patient’s own blood. It is controversial in cases of malignancy, because theoretically tumor cells can be aspirated and reinfused intravenously. Recently, however, leukocyte depletion filters (which do not allow cells much larger than RBC’s to remain in the product to be infused) have been shown to efficiently remove all tumor cells from the aspirated blood. Moreover, it is not at all clear that infusion of tumor cells is actually a risk for metastasis, which requires numerous other cellular steps.

Suggested Further Reading


Chapter 15

IV, Gastric Tube, Arterial & Central Line Placement Techniques

Francis X. Dillon

For maximum impact, it is recommended that the case study and questions found on page xxiv are reviewed before reading this chapter.

Key Learning Objectives

- Learn anatomy, indications, and techniques associated with line placement
- Understand the complications associated with invasive lines
- Know how to select a site for cannulation

Invasive line placement procedures are common to many anesthetics, and are also an integral part of inpatient care outside of the operating room. However, any procedure, from a simple IV to a pulmonary artery catheter carries with it a small but real risk of patient complications.

Mastery of common line placements performed by an anesthesiologist takes cumulative experience and dexterity. Most students and trainees often make their first successful attempts at these procedures while on an anesthesia rotation; because patients are anesthetized, there is excellent lighting and room for placement, one has expert supervision, and patients are optimally positioned. Knowing how to perform these procedures efficiently and safely will pay dividends throughout one's career, although it takes years to become truly expert in them.
Intravenous (IV) Lines

Relevant Anatomy

The extremity veins are very thin-walled. They have an endothelium, a thin muscle layer capable of contracting and constricting the lumen of the vein, and are supported by an adventitia of thin connective tissue. The latter makes them sometimes difficult to puncture because they are hard to fix in space by the penetrating catheter stylet tip. In some body areas, they are valveless, but in others, like the extremities, they have valves, bends, angles, and dead ends. These factors may make catheter threading difficult. Veins usually run anatomically in neurovascular bundles with the accompanying arteries and nerves, making nerve injury and arterial puncture potential complications of venipuncture.

A useful mnemonic for remembering the position of these structures is NAVAL – denoting from lateral to medial: Nerve, Artery, Vein, Empty space, Lymphatic. This is important to visualize when cannulating the femoral artery or vein.

Physiology of Veins

Veins have smooth muscle in their walls, which may contract after the stimulus of puncture, making it harder to cannulate in subsequent attempts. The pressure of veins is low (27–31 mm Hg) but that of surrounding tissues is even lower, making hematomas likely after unsuccessful puncture – unless firm pressure is applied at the site for 30–45 s. Veins have sensory and motor (sympathetic) innervation and may cause pain if the infused fluids are cold, hypotonic, or contain certain irritating electrolytes, such as potassium chloride, or emulsions, such as intralipid, for TPN. The repair mechanism of the punctured vein first involves thrombus formation on denuded or injured endothelium. Therefore, the way to keep a catheter patent is to keep a slow flow of intravenous fluid entering the vein.

Technique of Peripheral Cannulation

One should begin by taking time to look and feel by gentle palpation for a vein at a favorable site for insertion. If you do not find a promising vessel in the first site you examine, consider moving to a different extremity. Frequently, the patient's skin and subcutaneous tissues will not allow one to see the veins beneath the surface but they may often be felt. The vein in an extremity with a tourniquet applied feels bouncy or rubbery to the finger tip, distinct from the surrounding soft tissue. Injecting lidocaine around the vein will anesthetize the overlying skin as well as inhibit venospasm. The intracatheter (14–24 g)
is straight PTFE (Teflon®) or other plastic tubing that is tapered at the end. There is a steel stylet introducer fitting tightly within it. The stylet has a clear chamber attached to its hub, which allows visualization of blood immediately upon venipuncture. The bevel of the stylet tip is kept upwardly oriented, and the tourniqueted vein is entered at a 30° angle by tensioning the skin distally. This is done gently and not so much that the vein is flattened.

One frequent error is trying to thread off the catheter when the flashback of blood is first seen, which often threads the catheter into the subcutaneous tissues. To avoid this error, make sure the catheter with stylet is inserted an additional 2–3 mm after the first flash of blood is seen. This will ensure that the catheter, not merely the stylet tip, is securely placed within the vein lumen before the catheter is threaded off.

Do not insist on 14 g or 16 g catheters for all cases. Catheters of size 18 g and 20 g are adequate in most cases, and blood may be transfused through catheters larger to or equal to 22 g, albeit more slowly. Of course, cases in which rapid blood loss is anticipated are best accommodated with multiple large bore (14 g, 16 g, and 18 g) catheters. These give more rapid volume support than the longer 16 g triple lumen catheter usually inserted during emergencies, owing to the length of the triple lumen catheter and the Hagen–Poiseuille hydrodynamic law – where the flow is proportional to the fourth power of the radius (thus, small increases in intraluminal diameter confer large increases in flow).

Complications
Complications associated with intravenous cannulation are infrequent but may include hematoma, thrombosis, phlebitis, leakage or infiltration, nerve contact or injury with the stylet, and infection in the chronic indwelling catheter. Pain on injection may be from a misplaced or infiltrating catheter. Should pain occur, one may troubleshoot the problem by infusing fluid with a syringe to determine how well test bolus infuses without swelling. But pain in an IV may be just due to the cold IV fluid or an additive (e.g., potassium). Adding lidocaine to the fluid or injecting lidocaine around the vein may thus salvage an otherwise good IV. One should have a low threshold for replacing any IV causing symptoms. When treating an infiltration or inflammation, elevate the extremity, take out the catheter, and passively warm the insertion site (put a blanket over and let the body). Hot pads or warm packs are sometimes used but are an occasional cause of serious patient burns.
Arterial Line Placement

Anatomy

The radial arterial line is the most commonly used, although other potential arterial sites include ulnar, brachial, axillary, femoral and dorsalis pedis arteries. The relevant anatomy for arterial line placement is shown in Fig. 15.1.

Although no longer recommended (because a positive test does not correlate with impaired blood flow to the hand), an Allen's test is sometimes performed prior to insertion of the arterial line. First, press down firmly on both the radial and ulnar arteries and occlude flow while the patient exsanguinates the hand by making a tight fist repeatedly. Next, release the ulnar artery from pressure: The hand should become pink within 6 s if the result is normal. If it is abnormal, there is a theoretically higher risk of incomplete collateral circulation and complications from arterial line placement.

Figure 15.1  Radial artery cannulation. The arm is immobilized in the supine position with an armboard. The wrist is partially extended by placing a gauze roll underneath. The operator locates the pulse with the index finger of the nondominant hand and follows the trajectory of the artery with the third finger. The catheter is held like a pencil, with the needle bevel up. The catheter is inserted 0.5–1 in. proximal to the wrist and advanced at a 30° angle. (With permission from Criner, et al. Critical Care Study Guide: Text and Review, Springer, 2002).
Physiology
The artery has innervation and is tender when punctured. Using lidocaine liberally around the vessel ensures patient comfort and keeps the vessel from spasming if it is contacted by the needle tip but not entered. This is crucial because it will preserve the lumen of the vessel for subsequent attempts at cannulation.

Technique
There should be a sterile skin preparation and drape, and many advocate gowning, wearing a hair cover and mask. The use of audio Doppler or ultrasound may help localize the vessel. Palpation with gloves is necessary to maintain aseptic technique, but it makes it harder to palpate the maximal pulsation.

Generally, the artery is entered with a 20 g catheter. The angle of incidence is approximately 30°. The bevel should be up when inserted. After a flash is seen, the needle should be advanced 3-5 millimeters to transfix the vessel. The needle is removed, and the catheter should be withdrawn until brisk bleeding is evident. A guidewire is then inserted and the catheter advanced over the wire. A line is then attached and the catheter is secured. Care is taken to exclude air from the line, catheter hub, and catheter itself to prevent distal embolization into the fingers. Problems, complications, and artifacts of arterial monitoring will be discussed in Chap. 10.

Central Venous Line (CVL)
Anatomy
Knowledge of the relevant anatomy is critical to avoid serious complications during CVL placement. The sites of insertion commonly used are internal jugular, subclavian, and femoral veins.

Internal Jugular Vein
Figure 15.2 shows the anterior approach of cannulation of the internal jugular vein (IJ). The internal carotid artery is posterior and medial to the jugular vein on either right or left side, although there is considerable variation. The carotid may lie either directly posterior to, or, in a small percentage of cases, posterior and lateral to the vein. When inserting IJ catheters, there is always a risk of carotid puncture.
Figure 15.2  Right internal jugular cannulation via anterior approach. The head is rotated 30° to the contralateral side. Landmarks are the cricoid cartilage, the sternocleidomastoid muscle, and the carotid artery. One palpates the cricoid cartilage with the left index finger and then palpates 2 cm lateral to the midline of the neck. Care is taken to palpate and localize the carotid artery between the midline and the site of insertion. The carotid artery is identified so that the seeker needle tip will not enter it. Some operators retract the carotid artery medially (toward the midline). This must be done gently so that the internal jugular vein is not flattened, thereby making it difficult to enter. One inserts the seeker needle (22 g) 2 cm lateral to the midline, and just lateral to where the carotid artery pulse was palpated. The needle is inserted through the belly of the sternocleidomastoid muscle, with gentle suction applied to the syringe plunger. Once the seeker needle “finds” the vein, an 18 g intracath or needle may make a single pass at the same location.
Subclavian Vein
Subclavian veins are better sites for longer-term insertion, but they carry the risks of pneumo- or hemothorax and thoracic duct injury (left subclavian) during insertion.

Key Points in IJ Cannulation (Figs. 15.2 and 15.3)
1. The simplest IJ approach is the anterior one (Fig. 15.2) in which the catheter is inserted at the level of the cricoid cartilage, 2 cm lateral to the midline, through the sternocleidomastoid muscle. The patient’s head is turned to the contralateral side 30°. The right side of insertion is less complication-prone because the path of catheter insertion is straight.
2. The relation of the subclavian vein is immediately below the clavicle in the medial half of the bone. The subclavian artery changes its relative position in the lateral half of the bone and may be easier to puncture if the attempt is made more lateral rather than more medial (Fig. 15.3)
3. Sterile skin preparation and surgical gowning and draping of the patient have been shown to decrease line infections.
4. Ultrasonographic visualization is now being routinely used to identify vessels for catheter insertion.
5. A “seeker needle” of gauge 22 or smaller, attached to a small syringe, may be used to locate the vein. Head-down (Trendelenberg) positioning helps dilate the IJ and the subclavian veins and increases the success rate.

6. Often the vein is entered on the way in, or, the needle may enter the lumen on the way back out of the vessel if traversed. Gentle suction on the syringe plunger keeps the lumen from collapsing, and nicking the skin with a larger needle will allow the seeker to penetrate the delicate vein wall without carrying down the skin and soft tissues and thus compressing the vein.

**Femoral Vein**

Inserting a femoral venous catheter in the area of the groin may be problematic with regard to infection and thrombosis, and once placed may make it difficult for the patient to ambulate. Nevertheless, if venous access is necessary and the other sites impractical at times, the femoral vein and artery are cannulated. Figure 15.4 shows the landmarks for insertion. A sterile prep and meticulous dressing of the region is important, and the use of ultrasound is becoming common during femoral cannulation.

The mnemonic, **NAVEL** recalls the structures encountered going from lateral to medial (i.e., going toward the navel): Nerve, Artery, Vein, Empty space, and Lymphatics (see Fig. 15.4).

![Femoral anatomy. The left femoral triangle, anterior aspect. The landmarks are the anterior superior iliac spine, the pubic tubercle, between which runs the inguinal ligament. The femoral nerve, artery, and vein may be shown to run closely together in the femoral canal, beneath the femoral sheath. The artery is plainly palpable and pulsatile. Therefore the vein may be entered by inserting a needle medial to the pulsating femoral artery, inferior to the inguinal ligament.](image-url)
Choosing a Central Line Insertion Site

Table 15.1 shows the relative risk of complications depending on site of insertion for the internal jugular, subclavian, and femoral venous catheters.

Thus, when choosing a cannulation site, consider the following:

- **How rapidly and reliably must fluids and medications be infused?**
  Smaller bore catheters (22 and 20 g) are suboptimal for rapid fluid resuscitation; the fluid goes in too slowly. Larger bore catheters are essential if a large blood-loss case is planned such as a liver transplant.

- **What kind of fluids or medications will be administered?**
  If vasopressors are to be used, necrosis may occur if the peripheral vein infiltrates. Therefore, inotropic and vasopressor drugs typically require central venous access.

- **Will the patient be ambulatory?**
  In ambulatory patients, one should avoid cannulating the femoral vein.

- **Will the patient need hemodialysis?**
  If the patient will need hemodialysis, it is wise to consult vascular surgery regarding optimal catheter placement site. Placing catheters predisposes to thrombosis and stenosis of large vessels, which may be needed in the future if the patient goes on to require hemodialysis or a permanent catheter for chemotherapy or plasmapheresis.

<table>
<thead>
<tr>
<th>Complication</th>
<th>Risk of complication at catheterization site</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Subclavian vein</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>1.5–3.1%</td>
</tr>
<tr>
<td>Hemothorax</td>
<td>0.4–0.6%</td>
</tr>
<tr>
<td>Infection (rate/1,000 catheter day)</td>
<td>4</td>
</tr>
<tr>
<td>Thrombosis (rate/1,000 catheter day)</td>
<td>0–13</td>
</tr>
<tr>
<td>Arterial puncture</td>
<td>0.5%</td>
</tr>
<tr>
<td>Malposition</td>
<td>Low risk</td>
</tr>
</tbody>
</table>

Nasogastric (and Orogastric) Tube Placement

Patients who have large or small bowel obstructions, obstipation (absent flatus), or abdominal distension may need to have a nasogastric tube placed while awake, prior to induction of anesthesia. Aspiration of enteric contents under conditions of an undecompressed GI tract may result in pulmonary aspiration, lung injury, and death. It is not possible to prevent such pulmonary aspiration by using cricoid pressure alone during intubation.

Orogastric tubes are more easily placed and better tolerated in anesthetized or heavily sedated patients. In awake patients, orogastric tubes may stimulate intense gagging. Conversely, nasogastric tubes are better tolerated in awake patients and they may be fixed by taping them against the upper lip. They are usually placed in awake patients with their cooperation and after topical anesthesia (lidocaine spray) to the nares, lubrication, and warming of the nasogastric tube with warm water. These measures may help prevent epistaxis (nosebleeds).

Technique

Orogastric tube: After induction of anesthesia, have the patient supine and flat on the OR table, with the operator standing at the head of the bed overlooking the patient’s head and neck. Place the left index finger into the patient’s mouth and reach down into the pharynx and hypopharynx, sweeping the base of the tongue upward with the extended index finger. Then, with the right hand, while keeping the curved tip of the orogastric tube oriented so that it naturally curves downward, insert the tube into the right side of the pharynx and gently wiggle it until you feel it pass into the esophagus. Using a gloved finger is very important to detect and avoid submucosal placement of a nasogastric tube, which can lead to serious complications, including infections. After the tube has been placed deeply enough, apply suction or auscultate injected air to confirm tube placement. After surgery, consider obtaining a chest film to ensure that the tube is in the GI tract, not the bronchus.

Nasogastric tube. Good preparation of the nares, vasoconstriction, warming of the nasogastric tube, and gentle technique will help avoid nosebleeds. The key difference in placing a nasogastric tube is that the tube must be inserted straight back along the plane of the hard palate. Then, it must make a 90° turn at which point it may be caught on the structures of the nasopharynx. The key move is to first insert the left index finger through the mouth into the hypopharynx, and as in orogastric intubation, use the index finger to hold up the
base of the tongue by extending the index finger anteriorly in the hypopharynx. The index finger will also palpate, guide, and direct the tip of the nasogastric tube into the esophagus. Again, it is important to use a gloved finger to avoid submucosal placement. It is easy to palpate the tip as it makes the 90° turn. Some operators put 10 ml of water-based lidocaine gel in the naris. This provides good anesthesia and lubrication and prevents nosebleeds. One may choose to use a nasal spray for vasoconstriction prior to inserting the tube, such as phenylephrine 0.25% or oxymetazoline 0.05% spray.

Case Study

A 35-year-old woman comes to the OR for emergency laparoscopic resection of a ruptured ectopic pregnancy. She was admitted to the emergency department with abdominal pain and was found to have a positive beta-HCG, a mass on abdominal ultrasound in her right Fallopian tube, and an empty uterus. Her last menstrual period was approximately 8 weeks ago. She states that she is otherwise healthy. She ate dinner approximately 4 h ago but had little appetite at the time so states that it was “just a little.” She has a 20 G antecubital IV in place, which is slowly infusing lactated Ringer’s.

Is the existing IV sufficient for this case? How will you decide whether or not you need better IV access?

You can open up the IV fluids and assess how well this 20 G catheter flows. In a large vein, even an IV of this relatively small size will often run briskly. Check the tubing set and make certain it is a high-flow set; you may choose to change it to the standard set you use in the OR, which generally is optimized for rapid flow and injection of drugs. You can inspect the IV site itself and see if there are signs of swelling or redness indicative of infiltration (i.e., migration out of the vein). You can ask the patient if the IV is comfortable or painful. Properly situated IVs are generally painless. You should also discuss the prospect of blood loss and other fluid shifts with the surgeon, including the possibility of requiring an open procedure and the expected duration of the operation. The latter will influence the degree of third space fluid that will need to be replaced.
Exhaustive search for other veins yields no obvious prospects for additional access. The patient states that she has always been “a tough stick.” How will you proceed?

You can certainly induce anesthesia with this IV and then attempt to locate a second site after induction. General anesthesia often leads to vasodilation and easier location of veins due to direct effects of anesthetics as well as relief of anxiety, which may cause sympathetic activation and vasoconstriction. This presumes that you believe that the present IV is indeed intravascular! You should not proceed with induction if you are not sure. Some anesthesiologists will inject a dose of a rapid-acting sedative or opioid to assess whether the drug has entered the bloodstream and reached the brain, but this may not be definitive due to variation in individual patient responses to the drugs.

You plan a rapid sequence induction with propofol and succinylcholine. 60 s after injecting propofol, the patient has not lost consciousness. You have not yet injected succinylcholine. How will you proceed?

At this point, you should suspect that the IV might not be intravascular. You can determine if this is a pharmacodynamic or kinetic problem (i.e., the patient has just not yet fallen asleep but the drug is IV) by assessing whether your injection has had any effect at all on the patient’s level of consciousness. Although the rapid sequence technique generally implies quick sequential injection of a hypnotic and a paralytic, you should not inject succinylcholine at this point. This is because even if not IV, succinylcholine will eventually be absorbed and will produce weakness or paralysis in an unsedated patient. Extravascular propofol and lactated Ringer’s are probably benign (unlike thiopental, which can be irritating). However, you should monitor the limb for signs of edema, or compartment syndrome by observation and palpation of the distal pulse. If possible, elevate the arm somewhat over the level of the chest.

Can you induce anesthesia by inhalation instead?

This technique is commonly performed in children but is rarely employed in adults in modern practice. In this case, however, it is contraindicated because the emergency nature of the surgery, the fact that the patient has consumed food in the last few hours, and the abdominal nature of the emergency, all of which relatively contraindicate mask ventilation.
You decide that you will need another IV to proceed. What options do you have to establish access?

There are many “tricks” anesthesiologists use to secure venous access in patients with difficult anatomy. The first is to look beyond the forearms: the upper arm, distal hand, and feet are sometimes options. Only a small IV is needed for induction, and then as you had previously planned, better veins may become visible after induction. The external jugular vein can be percutaneously cannulated in many patients. Gentle pressure applied to the distal neck just above the clavicle can help you visualize this valveless vein. The femoral vessels are sometimes used, and the femoral artery is a useful landmark for locating the femoral vein, about 1 cm medial to the pulse. A femoral line may be somewhat compromised during laparoscopy because the increased abdominal pressure will impede flow. A second option is to enhance visibility of veins. Warming the extremity or use of topical nitroglycerin ointment to promote vasodilation are sometimes successful (though the latter can cause a headache as a side effect). Inflating a blood pressure cuff on the arm to above the arterial pressure for a few minutes, then lowering it to approximately 30 mm Hg, which is above venous but below arterial pressure, may reveal veins by causing mild ischemia-induced vasodilation. Operators skilled in the technique have used ultrasound to locate veins too deep to see or feel. In particular, the deep brachial vein in the arm above the elbow can be localized in many patients. Finally, central venous access via the internal jugular or subclavian veins may be the only option. Again, ultrasound has been shown to reduce complications, particularly for the internal jugular approach.

Suggested Further Reading


Chapter 16

Common Intraoperative Problems

Francis X. Dillon

For maximum impact, it is recommended that the case study and questions found on page xxv are reviewed before reading this chapter.

Key Learning Objectives
- Learn to identify common intraoperative problems including hypoxia, hypotension, and hypercarbia
- Provide a differential diagnosis for each intraoperative problem based on available data
- Identify the most important management options once a diagnosis is made

General Concepts
Anesthesiologists must act independently and quickly to detect and correct problems. The anesthesiologist is charged with diagnosing, monitoring, debugging, and remedying intraoperative and postoperative problems outside of those dealt with by the surgical team. At the same time, the operating room environment should be collegial and relaxed enough such that all caretakers can work cooperatively and communicate quickly and constructively.

The ultimate goal is the rapid diagnosis and correction of a problem, before the patient is put at risk of injury. The anesthesiologist may need to ask the surgical team to halt or delay a procedure if a greater risk presents itself. Examples of such circumstances are hypotension, massive blood loss, or dysrhythmia. Table 16.1 lists the most common intraoperative problems that may occur during anesthesia.
<table>
<thead>
<tr>
<th>Problem</th>
<th>Differential diagnosis</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxemia</td>
<td>Low inspired oxygen (FiO₂)</td>
<td>Always start by increasing the FiO₂ and then continue to look for other causes.</td>
</tr>
<tr>
<td>Hypoventilation</td>
<td></td>
<td>May be from opioids, benzodiazepines, or muscle relaxants, which decrease respiratory drive and muscle strength. If a patient is being ventilated, consider increasing respiratory rate or tidal volumes.</td>
</tr>
<tr>
<td>Disconnection of breathing circuit</td>
<td></td>
<td>The most common cause of serious hypoxemic accidents.</td>
</tr>
<tr>
<td>Atelectasis</td>
<td></td>
<td>Often a result of positive pressure ventilation, intubation and/or hypoventilation. Consider alveolar recruitment maneuvers.</td>
</tr>
<tr>
<td>Bronchospasm</td>
<td></td>
<td>Consider administering albuterol</td>
</tr>
<tr>
<td>Mucus plugging</td>
<td></td>
<td>Perform suction and alveolar recruitment maneuvers.</td>
</tr>
<tr>
<td>Right-mainstem bronchial intubation</td>
<td></td>
<td>Maximum depth for tracheal tubes measured at teeth: females 21 cm; males 23 cm. May visualize with bronchoscope or auscultate with stethoscope.</td>
</tr>
<tr>
<td>Pulmonary thromboembolism (PE)</td>
<td></td>
<td>This may be diagnosed by a sudden drop in end-tidal CO₂ and hypoxemia that does not improve with 100% FiO₂. Often accompanied by tachycardia and hypotension.</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td></td>
<td>May be from fluid overload, diastolic or systolic dysfunction, or myocardial infarction.</td>
</tr>
<tr>
<td>Venous air embolism</td>
<td></td>
<td>Also causes drop in end-tidal CO₂ and an increase in end-tidal N₂.</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Hypovolemia</td>
<td>Common causes include blood loss or dehydration from preoperative fasting.</td>
</tr>
<tr>
<td>Relative anesthetic overdose</td>
<td></td>
<td>Minimal or a decrease in surgical stimulation can lead to relative anesthetic overdose and hypotension.</td>
</tr>
<tr>
<td>Vasodilatation from medication</td>
<td></td>
<td>Opioids, sedatives, and most anesthetics reduce central sympathetic outflow and cause vasodilatation. Virtually every anesthetic induction or heavy sedation will be accompanied by this finding. Usually treated with phenylephrine, 40–100 mcg.</td>
</tr>
<tr>
<td>Low cardiac output</td>
<td></td>
<td>Many anesthetics decrease cardiac output. Other causes include congestive heart failure, myocardial infarction, or tamponade.</td>
</tr>
<tr>
<td>Severe bradycardia</td>
<td></td>
<td>This may cause low cardiac output (CO=HR×SV), leading to hypotension.</td>
</tr>
<tr>
<td>Severe tachycardia or arrhythmias</td>
<td></td>
<td>If atrial fibrillation or flutter becomes too fast, hypotension may result.</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Pneumothorax</td>
<td>Uncommon, but may spontaneously arise during positive pressure ventilation.</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>--------------</td>
<td>-------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Anaphylactic reaction</td>
<td></td>
<td>Most commonly from reaction to muscle relaxants or antibiotics. Give epinephrine to treat.</td>
</tr>
<tr>
<td>Sepsis</td>
<td></td>
<td>Sudden decreases in blood pressure may result from sepsis.</td>
</tr>
<tr>
<td>Pain from surgical stimulus</td>
<td></td>
<td>Always consider “light” or insufficient anesthesia.</td>
</tr>
<tr>
<td>Essential hypertension</td>
<td></td>
<td>These patients may be adequately anesthetized, but still markedly hypertensive.</td>
</tr>
<tr>
<td>Tourniquet pain</td>
<td></td>
<td>A tourniquet can produce a hard, recalcitrant kind of hypertension called “cuff hypertension.”</td>
</tr>
<tr>
<td>Light anesthesia</td>
<td></td>
<td>Check for empty vaporizers or medication infusors</td>
</tr>
<tr>
<td>Hypervolemia</td>
<td></td>
<td>Fluid overload from intravenous fluid or blood products; may lead to pulmonary edema and congestive heart failure in patients with heart disease.</td>
</tr>
<tr>
<td>Failure to ventilate</td>
<td>Kinked endotracheal tube</td>
<td>Most likely during ENT or thoracic surgery.</td>
</tr>
<tr>
<td></td>
<td>Biting on endotracheal tube</td>
<td>May occur during “light” anesthesia or emergence; Can cause negative pressure pulmonary edema.</td>
</tr>
<tr>
<td></td>
<td>Disconnection of endotracheal tube from circuit or adapter</td>
<td>The most common cause.</td>
</tr>
<tr>
<td></td>
<td>Complete endotracheal tube obstruction from mucus or tissue</td>
<td>Can occur rapidly in infants/children whose ETT are narrow (especially when no humidification is used). Suction or replace tube.</td>
</tr>
<tr>
<td></td>
<td>Hole in endotracheal tube or a punctured cuff</td>
<td>Most often during laser airway surgery or tracheostomy. Both surgeries also have risk of airway fire!</td>
</tr>
<tr>
<td></td>
<td>Bronchospasm</td>
<td>(1) deepen anesthesia, (2) consider neuromuscular blockade, (3) administer beta-agonists, inhaled or intravenous corticosteroids, theophylline or epinephrine</td>
</tr>
<tr>
<td></td>
<td>Kinked endotracheal tube</td>
<td>May require the use of an armored (metal spring reinforced) tube to prevent kinking.</td>
</tr>
<tr>
<td></td>
<td>Biting on endotracheal tube</td>
<td>Consider placing a bite block or mouth gag.</td>
</tr>
<tr>
<td></td>
<td>Mucus plugging</td>
<td>Common in patients with COPD, asthma and cystic fibrosis. This may require replacement of the ETT.</td>
</tr>
<tr>
<td></td>
<td>Stacking or auto-PEEP of mechanical breaths</td>
<td>Occurs when the expiratory phase isn’t long enough to allow exhalation. Decrease the respiratory rate.</td>
</tr>
<tr>
<td></td>
<td>Dynamic airway obstruction</td>
<td>May be from an airway tumor or mediastinal mass, especially after change in patient position.</td>
</tr>
</tbody>
</table>
Table 16.1  (continued)

<table>
<thead>
<tr>
<th>Problem</th>
<th>Differential diagnosis</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity or chest wall rigidity</td>
<td>May be difficult to manage. High opioid dose can cause a “rigid chest syndrome.”</td>
<td></td>
</tr>
<tr>
<td>Acute Respiratory Distress Syndrome (ARDS)</td>
<td>A common cause of high mean pressures, especially in the ICU.</td>
<td></td>
</tr>
<tr>
<td>Hypocarbia</td>
<td>Hyperventilation</td>
<td>May see in anxious (awake) or mechanically hyperventilated (anesthetized) patients.</td>
</tr>
<tr>
<td></td>
<td>Leak of CO₂ in sampling tubing</td>
<td>This may also cause an abnormality in the capnograph waveform or envelope.</td>
</tr>
<tr>
<td></td>
<td>Massive pulmonary embolus</td>
<td>Can impair gas exchange, manifesting as a sudden drop in expired CO₂.</td>
</tr>
<tr>
<td></td>
<td>Hypothermia</td>
<td>Most evident in severe hypothermia as during cardiopulmonary bypass.</td>
</tr>
<tr>
<td></td>
<td>Cardiac Arrest</td>
<td>Impaired circulation and CO₂ elimination.</td>
</tr>
<tr>
<td>Hypercarbia</td>
<td>Hypoventilation</td>
<td>Often from opioids, residual neuromuscular blockade, or low respiratory rate/ventilator tidal volumes.</td>
</tr>
<tr>
<td></td>
<td>CO₂ insufflation during laparoscopy</td>
<td>May need to increase minute ventilation to overcome hypercarbia.</td>
</tr>
<tr>
<td></td>
<td>Malignant hyperthermia</td>
<td>Uncoupling of calcium metabolism in mitochondria from a rare (1:15,000) genetic defect in the ryanodine receptor of the calcium channel</td>
</tr>
<tr>
<td></td>
<td>Hyperthermia</td>
<td>Metabolic rate increases 15% for every degree centigrade.</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Convective, conductive, radiative, evaporative losses</td>
<td>Convective losses are the #1 cause of heat loss in the OR (skin to air). Heat loss also occurs from wet drapes and sheets, exposed skin or body cavities, non-heated breathing circuits.</td>
</tr>
<tr>
<td></td>
<td>Anesthetic effects on hypothalamus</td>
<td>Anesthetics cause impaired central thermoregulation due to effects on the hypothalamus.</td>
</tr>
<tr>
<td></td>
<td>Administration of unwarmed fluid or blood products</td>
<td>Fluids should be warmed by an FDA-approved device.</td>
</tr>
<tr>
<td></td>
<td>Massive blood loss</td>
<td>May be difficult to manage. High opioid dose can cause a “rigid chest syndrome.”</td>
</tr>
<tr>
<td>Hypercarbia</td>
<td>Excessive warming</td>
<td>Use the air-warming blanket at a room-temperature setting to cool the patient.</td>
</tr>
<tr>
<td></td>
<td>Fever from sepsis or transfusion reaction</td>
<td>Give acetaminophen or ibuprofen in addition to a cooling blanket.</td>
</tr>
<tr>
<td></td>
<td>Stroke</td>
<td>Sudden extreme hyperthermia (&gt;105°F) may be from a stroke to the hypothalamus.</td>
</tr>
<tr>
<td></td>
<td>Neuroleptic malignant syndrome</td>
<td>Uncommon side effect of antipsychotic medications (chlorpromazine, haloperidol, olanzepine).</td>
</tr>
<tr>
<td></td>
<td>Malignant hyperthermia</td>
<td>(1) stop anesthetic, (2) give iv dantrolene, (3) call for help (see Appendix B, Malignant Hyperthermia)</td>
</tr>
</tbody>
</table>
We will now discuss the most common of these problems in detail. The goal here is to be able to (1) formulate a differential diagnosis and (2) learn the most immediate corrective action. It is often the case in anesthesia that one must immediately act to correct a problem, while simultaneously contemplating the underlying cause of a particular issue. This is a skill that demands an ability to act under pressure, separating anesthesiologists from other specialists.

**Hypoxemia**

If *sudden hypoxemia* occurs during an anesthetic, one must first determine the severity of the hypoxemia. Severe hypoxemia may be defined as an $\text{SpO}_2$ of less than 90%, which corresponds to a $\text{PaO}_2$ of under 60 mm Hg. An arterial blood gas sample is more sensitive at revealing changes in oxygenation than is an oximeter. This is due to the physiologic characteristics of hemoglobin described by the hemoglobin-oxygen dissociation curve (see Fig. 16.1). Note that at the right side of the curve, the additional $\text{PaO}_2$ from 70 to 100 mm Hg and beyond (which represents a significant increase in concentration in the administered $\text{O}_2$) only increases the saturation of hemoglobin slightly.

In clinical practice, this means that a patient may have a significant problem in oxygenation for several minutes (as manifested by the decrease in $\text{PaO}_2$) before

![Hemoglobin–oxygen dissociation curve](image)

*Figure 16.1  Hemoglobin–oxygen dissociation curve (Image Courtesy J. Ehrenfeld).*
it is reflected on an oximeter reading by the decrease in \( \text{SaO}_2 \). This is important because most anesthetics are performed only with oximetery. Therefore, time is of the essence when responding to hypoxemia as detected by an oximeter because the problem or process causing the hypoxia may have been going on for quite some time before its severity reached the threshold of the pulse oximeter.

Immediate steps to take when addressing hypoxemia include those in Table 16.2.

Other causes of perioperative hypoxemia include V/Q mismatch as seen in pneumonia, pulmonary edema or pulmonary embolism, improper endotracheal tube placement, right-to-left intracardiac shunting, impaired transfer of oxygen from the alveolus across the capillary membrane as seen in interstitial lung disease, alveolar hypoventilation as seen in COPD, asthma, bronchospasm or neuromuscular disease, and decreased oxygen carrying capacity of the hemoglobin as seen in hypothermia or carbon monoxide poisoning.

### Table 16.2 Intraoperative hypoxemia: corrective actions.

1. Assure 100% \( \text{FiO}_2 \) delivery
2. Hand ventilate to assess compliance and chest excursion
3. Look for any disconnection: circuit, endotracheal tube, connectors
4. Check expired gases for \( \text{ETCO}_2 \)
5. Inform the surgical team and operating room nurse if severe
6. Examine the capnograph waveform
7. Inspect the endotracheal tube and circuit for kinking, biting, obstruction
8. Inspect the endotracheal tube for excessively deep insertion (should be 21 cm in females, 23 cm in males)
9. Suction the endotracheal tracheal tube to remove secretions and assess patency or obstruction
10. Perform direct laryngoscopy to verify tube placement in the trachea
11. Apply recruitment maneuvers to open up alveoli

**Hypotension**

Hypotension is common during anesthesia, especially during induction due to the vasodilating effects of intravenous and inhalational anesthetic agents. All drugs used for induction, sedation, and anxiolysis can cause a centrally-mediated depression in sympathetic tone. The arteries and veins controlled by the brain’s vasomotor center are all over the body. These vessels dilate and blood pressure decreases from both arterial and venous dilation. This is a predictable consequence of almost all anesthetic inductions and most sedation techniques. In addition, since patients usually fast for at least 8 h prior to surgery, they are
often dehydrated and hypovolemic. Thus, the hypotensive induction drug effect is amplified by preexisting low intravascular volume.

Hypotension at induction is usually treated with vasopressors, in addition to giving IV fluids. Commonly used medications include phenylephrine 40–100 mcg and ephedrine 5–10 mg iv. In addition to starting an infusion of a crystalloid fluid solution when treating hypotension and giving a small dose of a vasopressor, it is also acceptable to temporarily lighten the anesthetic depth. If a patient is severely hypovolemic, septic, experiencing an anaphylactic reaction, or manifesting shock from other causes, phenylephrine or ephedrine may not be potent enough to raise the blood pressure. In such cases, one might consider the use of epinephrine, norepinephrine, vasopressin, or dopamine.

While taking temporizing measures to control hypotension, volume status should be assessed and correct placement of the blood pressure cuff ascertained. Other possible causes of hypotension should be ruled out, including bleeding, error in measurement, medication error or overdose, spinal cord injury, myocardial ischemia, pneumothorax, pericardial tamponade, electrolyte disturbances, cardiac arrhythmias, sepsis, or an anaphylactic reaction. Invasive hemodynamic monitoring (i.e., arterial line) should be considered in hypotension that is refractory to treatments.

**Hypertension**

Hypertension is also a commonly encountered problem during anesthesia. However, brief episodes of hypertension are rarely harmful. Usually, the best option is simply deepening anesthesia with an inhalational agent or an intravenous bolus of propofol or thiopental. If pain is suspected, an opioid medication such as fentanyl or remifentanil can be given. Also, because sudden increases in blood pressure may connote intraoperative awareness, some anesthesiologists give 1 or 2 mg midazolam at such a time to reduce the likelihood of this potential complication. If anesthetic depth or pain are not the likely contributors to hypertension, practitioners generally consider the use of either esmolol (10–20 mg IV), or labetolol (5–10 mg IV). These are also used if deepening anesthesia is insufficient or impractical. The use of nitroglycerine (50 mcg IV) or nitroprusside (50 mcg IV) boluses or infusions in more severe episodes of hypertension is occasionally needed. Other common adjunctive drugs used for blood pressure control are hydralazine (5–10 mg IV) and nicardipine (0.2–0.5 mg IV).

It is important to ascertain correct placement of the blood pressure cuff to avoid spurious measurements. Other causes of intraoperative hypertension
may include baseline hypertension, hypercarbia, hypoxia, hyperthermia, increased intracranial pressure, hyperthyroidism, a patient who has not taken his usual antihypertensive medication, and bladder distension.

Another cause of hypertension is tourniquet pain. Upper or lower extremity tourniquets are typically used in orthopedic cases to decrease blood loss during surgery. Pain usually increases over a 1–2 h period, and may be difficult to treat. One might increase the volatile agent concentration, or use a short-acting agent such as propofol, esmolol, nitroglycerine, or nitroprusside. Pain manifested as hypertension will subside once tourniquet is released.

**Failure to Ventilate**

Failure to ventilate may occur from a disconnect or leak in the anesthesia circuit. It is important to inform the surgical team about any airway problem for two reasons. First, during many procedures the surgeons will have access to the patient’s airway (e.g., various ENT procedures such as neck dissections) and be able to help quickly. Second, the surgical team may ultimately be required to provide emergent access to the trachea (i.e., cricothyroidotomy).

The most common cause of failure to ventilate is still the disconnection of the anesthesia circuit. Be prepared to immediately palpate along the length of the circuit and tube to find the disconnection or hole in the circuit. Also check the machine connections to the inspiratory and expiratory limbs. Check the seals of any CO₂ absorbent-canister fittings because these often become dislodged when the canisters are replaced, and consider using a laryngoscope to verify tube placement through direct laryngoscopy.

Obstruction of the circuit, tracheal tube, or sudden increase in airway peak pressure requires rapid diagnosis and remedy. Dried mucus can completely obstruct a large adult-size tracheal tube over time. This phenomenon is even more common in smaller patients and endotracheal tubes. Humidifiers may reduce the risk of this happening, but cannot prevent it entirely. When it does happen, replacement of the tracheal tube with a new one may be the only way to restore tube patency. Other causes of elevated peak airway pressures may include a kinked tube or a patient who is biting on the tube. If mechanical measures such as separating the teeth with a bite block or inserting a mouth gag are not feasible, it may be necessary to administer a dose of a neuromuscular blocking drug. Other causes of sudden increase in peak airway pressure include chest wall rigidity, pneumothorax, or the patient coughing or bucking on the tube.
Hypocarbia
Hypocarbia is a reflection of decreased CO₂ levels as measured by either end-tidal monitoring or a blood gas. It can be due to either increased CO₂ elimination or decreased CO₂ production. Common causes include decreased metabolic rate as seen in hypothermia and hypothyroidism, hyperventilation, pulmonary embolism, cardiac arrest, ETT tube dislodgement, circuit disconnect, or a disconnected CO₂ sampling line.

The first step in investigating causes of hypocarbia is to check the breathing circuit, to rule out any loose connections or other mechanical problems. Next step should be to examine the patient’s blood pressure, heart rate, and SpO₂ to evaluate for signs of hemodynamic compromise. Finally, if the patient is being mechanically ventilated, one should scrutinize the ventilator settings to ensure they are appropriate.

Hypercarbia
Hypercarbia, as measured by end-tidal CO₂ or blood gas analysis, is a common occurrence during general anesthesia. The normal EtCO₂ value is 38–42 mm Hg. Hypercarbia may result from either increased CO₂ production or decreased CO₂ elimination. Causes of increased CO₂ production include fever, a hypermetabolic state as seen during burns or malignant hyperthermia, shivering, and thyrotoxicosis. Causes of decreased CO₂ elimination from the body include hypoventilation, airway obstruction, atelectasis, residual effects of paralytics or opioids, endobronchial intubation, and an exhausted CO₂ absorber. When thinking about the potential causes of hypercarbia, it is often useful to consider when the hypercarbia is occurring. Elevations in CO₂ at the beginning or a case are more likely to be from improper ETT placement, oversedation or inadequate ventilator settings, whereas elevated CO₂ at emergence is more likely to be from residual medication effects.

The first step in investigating causes of hypercarbia is to check the pulse oximeter, to ensure adequate oxygenation and evidence of circulation. One should also examine the ventilator settings and CO₂ absorber for signs of exhaustion. If the patient is spontaneously breathing, it is often helpful to gently assist the patient or lighten sedation to increase the overall minute ventilation.

Myocardial Ischemia
Myocardial ischemia may occur any time during surgery or in the postoperative period. It can occur despite a preoperative patient evaluation and assessment of the risk factors for developing myocardial ischemia in the perioperative period.
Myocardial ischemia can manifest as ECG changes, dysrrhythmias, hypotension, or heart wall motion abnormalities seen on the echocardiogram.

In treating myocardial ischemia, the goal is to maintain an appropriate balance between oxygen supply and demand. You should immediately notify the surgeon and ensure appropriate blood pressure to maintain coronary perfusion. Elective surgery may need to be cancelled or finished quickly. Management may include administration of β-blockade, nitroglycerin, and anticoagulation. A cardiology consult is also typically warranted.

Anaphylactic/Anaphylactoid Reactions
Anaphylactic and anaphylactoid reactions may occur due to administration of anesthetic agents, exposure to antibiotics, or contact with latex. Anaphylactic reactions are mediated by IgE, whereas anaphylactoid reactions are not IgE-mediated so that no prior sensitization to antigen is required. Clinical manifestations of anaphylaxis include tachycardia, hypotension, arrhythmias, bronchospasm, hypoxemia, pulmonary edema, and body swelling/rashes.

Treatment includes removal of the offending agent, hemodynamic support with fluids and vasopressors, administering steroids, epinephrine, antihistamines and intubating the patient if the airway has not already been secured.

Delayed Emergence
Delayed emergence is defined when a patient fails to wake up within 20–30 min of the conclusion of an anesthetic. This most commonly occurs in elderly or debilitated patients. The differential diagnosis includes residual drug effects such as paralytics, intravenous, or inhalational agents; metabolic derangements such as hypoglycemia or electrolyte abnormalities; respiratory impairment due to hypoxia or hypercarbia; neurologic problems such as a stroke or a seizure; cardiovascular collapse, hypothermia or sepsis.

Intraoperative Awareness
Intraoperative awareness is a relatively rare, but significant complication of general anesthesia. It occurs in approximately 0.1–0.2% of patients each year, and can cause long-term distress to the patient. It may occur when not enough anesthesia is given to the patient to render full unconsciousness during the operation. Constant vigilance by the anesthesiologist is the best way to prevent this complication from occurring – which includes paying close attention to the trends in vital signs, end-tidal anesthetic gas concentration, and watching for patient movement.
Recently, a number of brain function monitoring devices have been introduced which provide a read out of the patient’s processed EEG (e.g., BIS, SEDLine) – a measure of the electrical activity of the brain. The goal of these devices is to prevent awareness by providing information about the patient’s level of consciousness. However, the largest most recent randomized prospective study failed to demonstrate an ability of these devices to prevent awareness.

Failed Regional Anesthesia
Many patients may undergo surgery with regional, instead of general anesthesia. For example, a patient having shoulder surgery may receive an interscalene block with local anesthesia that may serve as that patient’s only anesthetic. Sometimes blocks cannot control surgical pain adequately. This can occur if an operation takes longer than expected or if a block is simply not effective in reaching the needed sensory distribution. If a patient begins to note pain during a procedure, the anesthesiologist should decide what the next step should be. Options include repeating the block (although there is a risk of local anesthetic toxicity), supplementing the block with intravenous analgesics and sedation, or converting to general anesthesia. Communication with both the surgeon and the patient is critical.

Patient Movement/Waking Up During Surgery
Occasionally, patients will move during surgical stimulation or at a point when the surgeon requires complete patient immobility to safely proceed with the procedure. Patient movement is usually due to inadequate anesthesia or incomplete paralysis.

Appropriate actions may include deepening the anesthetic by increasing the concentration of inhalational agent being delivered, giving a bolus of an intravenous agent such as propofol, or administering a muscle relaxant. It is important to realize that different patients require different amounts of anesthesia, and it is important to individualize the doses administered to each patient.

Venous Air Embolism
Venous air embolism occurs when air enters into the peripheral or central venous circulation. During surgery, this most commonly occurs during neurosurgical procedures in the sitting position or during cardiopulmonary bypass. Although small amounts of air typically do not cause significant problems,
entrapment of large amounts of air (≥50 cc) can lead to cardiovascular collapse, severe neurologic injury and in some cases death. The most common two signs of a venous air embolus include a drop in end-tidal CO₂ readings and hemodynamic collapse (i.e., hypotension).

Immediate treatments of a venous air embolus includes flooding the surgical field with water, lowering any exposed vessels below the level of the heart, and attempting to withdraw air from the central circulation if a central venous catheter is present. One underutilized modality is hyperbaric oxygen, which has been shown to have some beneficial neurologic effects even when applied 48 hours after the air embolism event.

**Gastric Acid Aspiration**

Gastric acid aspiration can occur during intubation, intraoperatively, or during emergence from anesthesia. Aspiration of gastric contents can have devastating effects on the patient, including aspiration pneumonitis and death. Early signs of aspiration may include coughing, hypoxia, wheezing, and cyanosis. Late signs can show lung infiltrates on the chest x-ray and fever. Prevention includes protecting the airway and using anti-acids and H2-blockers.

Management of aspiration includes placing the patient head-down to prevent aspirant from entering the lungs and administering 100% oxygen. The physician may also perform a bronchoscopy or obtain a chest X-ray 1–2 days after the event to assess the effect of aspiration. Antibiotics or saline lavage are not typically indicated.

**Intraoperative Dysrhythmias**

Intraoperative dysrhythmias are relatively common. Some are part of the patient’s preoperative condition, whereas others arise during surgery due to a variety of triggering conditions. There are standard protocols that have been established by the American Heart Association (AHA), and most anesthesia providers become certified in Advanced Cardiac Life Support (ACLS). Table 16.3 outlines some common dysrhythmias, differential diagnosis, and suggested management.

**Transfusion-Related Problems (also see Chap. 14, Fluids, Electrolytes and Transfusion Therapy)**

Nonhemolytic febrile transfusion reactions are the most common transfusion reactions and do not require stopping the transfusions. These are treated
Table 16.3  Common intraoperative dysrhythmias.

<table>
<thead>
<tr>
<th>Problem</th>
<th>Differential diagnosis</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradycardia</td>
<td>β-blockers</td>
<td>Probably the most common cause.</td>
</tr>
<tr>
<td></td>
<td>Hypoxia</td>
<td>Occurs with severe hypoxia.</td>
</tr>
<tr>
<td></td>
<td>Myocardial infarction</td>
<td>Likely if the right coronary artery and sinus node are involved in the infarction.</td>
</tr>
<tr>
<td></td>
<td>Increased vagal tone</td>
<td>Surgical stimulus on the gut, bladder, or other organs may increase vagal tone. <strong>Atropine</strong> and, later, <strong>deepening anesthesia may</strong> be indicated. May also be seen in athletes.</td>
</tr>
<tr>
<td></td>
<td>3rd degree heart block</td>
<td>The ECG rhythm strip will provide the diagnosis.</td>
</tr>
<tr>
<td></td>
<td>Calcium channel blockers</td>
<td>Especially caused by diltiazem (used for this purpose in atrial fibrillation and flutter).</td>
</tr>
<tr>
<td></td>
<td>Reversal of neuromuscular blockade with cholinesterase inhibitors such as edrophonium or neostigmine</td>
<td>Co-administration of an anticholinergic medication (atropine or glycopyrrolate) is standard practice, so this happens rarely. However, edrophonium may be used alone to try to convert SVT or a slow heart rate, during testing for myasthenia gravis.</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Increased pain or surgical stimulus</td>
<td>The most common cause at the start of the surgical procedure. May suggest insufficient anesthesia.</td>
</tr>
<tr>
<td></td>
<td>Vasopressors or inotropes</td>
<td>Ephedrine, epinephrine, norepinephrine, isoproterolol can all cause tachycardia</td>
</tr>
<tr>
<td></td>
<td>Myocardial infarction</td>
<td>The most common dysrhythmia associated with MI.</td>
</tr>
<tr>
<td></td>
<td>Arrhythmias</td>
<td>Atrial fibrillation, Ventricular Tachycardia.</td>
</tr>
<tr>
<td></td>
<td>Malignant hyperthermia</td>
<td>Tachycardia in MH follows an observed increased CO₂ production and precedes hyperthermia.</td>
</tr>
<tr>
<td></td>
<td>Atropine, scopolamine, or glycopyrrolate administration</td>
<td>These are commonly given as antisialogogues (dry secretions), vagolytics (increase heart rate) or antiemetics (nausea control).</td>
</tr>
<tr>
<td></td>
<td>β-adrenergic agonists</td>
<td>Bronchodilators, tocolytics, and decongestant medications may cause tachycardia.</td>
</tr>
<tr>
<td>Premature ventricular contractions</td>
<td>Hypoxia</td>
<td>Always consider hypoxia first.</td>
</tr>
<tr>
<td></td>
<td>Myocardial ischemia</td>
<td>Check a 12-lead when feasible.</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis/alkalosis</td>
<td>Should always be high in the differential. Consider checking a blood gas.</td>
</tr>
<tr>
<td></td>
<td>Hypokalemia</td>
<td>Patients on diuretic therapy, without potassium replacement, may have this. Patients on digoxin who are hypokalemic are at particular risk.</td>
</tr>
<tr>
<td></td>
<td>Digoxin</td>
<td>Commonly used for atrial dysrhythmia therapy.</td>
</tr>
</tbody>
</table>

(continued)
Table 16.3  *(continued)*

<table>
<thead>
<tr>
<th>Problem</th>
<th>Differential diagnosis</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sympathomimetic drugs</td>
<td>Ephedrine or pseudoephedrine (found in cold remedies).</td>
<td></td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>Occurs in alcoholism or after prolonged use of diuretics like furosemide.</td>
<td></td>
</tr>
<tr>
<td>Hypokalemia</td>
<td>Especially pronounced with digoxin.</td>
<td></td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Patients who are post cardiopulmonary bypass or post exposure injury may have this.</td>
<td></td>
</tr>
<tr>
<td>Hypercarbia</td>
<td>In postoperative patients with pre-existing PVCs, mild hypercarbia in the PACU may cause increases in rates of PVCs.</td>
<td></td>
</tr>
<tr>
<td>Hypocarbia</td>
<td>If severe enough to cause respiratory alkalosis, PVCs may occur.</td>
<td></td>
</tr>
<tr>
<td>Myocarditis</td>
<td>PVCs are common with viral myocarditis.</td>
<td></td>
</tr>
<tr>
<td>Toxic overdose of drug</td>
<td>Unlikely, but can occur from antidepressants.</td>
<td></td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>Same causes as PVCs</td>
<td>Ventricular tachycardia may be thought of as three or more PVCs in a row.</td>
</tr>
<tr>
<td>Hypoxia or ischemia</td>
<td>Most likely acute causes in the OR.</td>
<td></td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>Rapid diuresis and its effect on volume sensors in the atria may cause PACs and atrial fibrillation.</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>PACs commonly occur with concomitant hypertension.</td>
<td></td>
</tr>
<tr>
<td>Previous thoracic surgery</td>
<td>PACs occur in 25–30% of patients undergoing thoracic (lung, mediastinal, or esophageal) surgery.</td>
<td></td>
</tr>
<tr>
<td>Mediastinal infection</td>
<td>Consider anastomotic leak in recent esophagectomy.</td>
<td></td>
</tr>
<tr>
<td>Atrial Fibrillation/</td>
<td>Same causes as PACs</td>
<td>Approach includes:</td>
</tr>
<tr>
<td>Atrial Flutter</td>
<td></td>
<td>1. Treat underlying causes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Control the heart rate by slowing the ventricular response</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Convert back to sinus rhythm if possible</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Useful drugs include digoxin, diltiazem, metoprolol and amiodarone.</td>
</tr>
<tr>
<td>Asystole</td>
<td>Severe hypoxemia</td>
<td>Treat underlying causes.</td>
</tr>
<tr>
<td></td>
<td>Severe hypovolemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe electrolyte abnormality</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Myocardial infarction</td>
<td>May require transcutaneous or transvenous pacing.</td>
</tr>
<tr>
<td></td>
<td>Severe metabolic acidosis</td>
<td>Treat underlying causes.</td>
</tr>
<tr>
<td></td>
<td>Pneumothorax</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pericardial tamponade</td>
<td></td>
</tr>
</tbody>
</table>
with acetaminophen or ibuprofen and diphenhydramine, and usually do not result in any significant sequelae.

**Non-immunogenic problems** related to transfusion often occur with **massive transfusion** (>4 units of packed red blood cells). Complications include hypothermia, hyperkalemia, dilution of coagulation factors, thrombocytopenia, and citrate toxicity causing hypotension.

**Severe transfusion reactions** most commonly occur because of **clerical or transporting error** after typing and crossmatching blood. Careful checking of blood products with patient identification is the best single measure to prevent these types of reactions. Management of a severe transfusion reaction is found below in Table 16.4.

### Miscellaneous Intraoperative Problems

**Foley (bladder) catheters:** If the Foley catheter was working but suddenly stopped putting out urine, check to see that it is not disconnected, kinked, or obstructed. This should be done before giving fluids or diuretics. A malfunctioning Foley catheter may be the reason for low urine output.

**Nasogastric (NG) or orogastric (OG) tube related problems:** The nasogastric or orogastric tube will need ongoing care to maintain patency. This is how the GI tract remains decompressed during surgery and in the perioperative period. Keep both lumens open by occasionally injecting small amounts of air into each lumen while applying suction to the suction lumen. If the NG or OG is not working, it may be coiled or knotted. If possible, ask the surgeon to palpate it. If not, withdraw the NG/OG while maintaining suction. If the NG or OG does not come out with a little traction, do not force it: 1% of the time

<table>
<thead>
<tr>
<th>Problem</th>
<th>Differential diagnosis</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulseless electrical activity (PEA)</td>
<td>Severe electrolyte abnormality</td>
<td>Treat underlying causes.</td>
</tr>
<tr>
<td></td>
<td>Myocardial infarction</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe metabolic acidosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pneumothorax</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pericardial tamponade</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Problem</th>
<th>Differential diagnosis</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulseless electrical activity (PEA)</td>
<td>Severe electrolyte abnormality</td>
<td>Treat underlying causes.</td>
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<tr>
<td></td>
<td>Myocardial infarction</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe metabolic acidosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pneumothorax</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pericardial tamponade</td>
<td></td>
</tr>
</tbody>
</table>
## Table 16.4  Management of a suspected severe transfusion reaction.

<table>
<thead>
<tr>
<th>Action</th>
<th>Rationale</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stop transfusion immediately</td>
<td>Limit exposure</td>
<td>Mild fevers or urticarial reactions usually do not require stopping the transfusion; they can be treated with diphenhydramine, acetaminophen or ibuprofen, and famotidine + epinephrine if wheezing occurs. Monitor carefully</td>
</tr>
<tr>
<td>Replace infusion set with saline</td>
<td>Limit exposure</td>
<td></td>
</tr>
<tr>
<td>Give 100% oxygen</td>
<td>Hemolysis will limit the useful carrying of O₂ by Hb</td>
<td></td>
</tr>
<tr>
<td>If hypotension/bronchospasm, give epinephrine 50 mcg iv to 100 mcg</td>
<td>Supports blood pressure and bronchodilates</td>
<td>Give small doses as needed to support blood pressure; Use norepinephrine or dopamine to support blood pressure if needed.</td>
</tr>
<tr>
<td>Consider IV corticosteroids</td>
<td>Helps inhibit the immune response and anaphylactoid hypotension</td>
<td>Hydrocortisone 150–300 mg</td>
</tr>
<tr>
<td>Give bronchodilators through the breathing circuit or ETT</td>
<td>Helps bronchodilate more specifically and gently than epinephrine</td>
<td>Albuterol</td>
</tr>
<tr>
<td>Send the blood bag, infusion set, a fresh urine sample, a fresh T&amp;C to the blood bank.</td>
<td>The blood bank will initiate their protocol for finding the cause of the reaction.</td>
<td>Most serious transfusion reactions are a result of bookkeeping or other clerical errors in checking and matching blood</td>
</tr>
<tr>
<td>Consider central line placement</td>
<td>This may help diagnose hypovolemia in the face of oliguria</td>
<td></td>
</tr>
<tr>
<td>Place Foley catheter</td>
<td>Monitor urine out every hour and check for hemoglobinuria</td>
<td></td>
</tr>
<tr>
<td>Monitor serum K⁺, BUN, Cr</td>
<td>Follow renal failure by these electrolytes.</td>
<td></td>
</tr>
<tr>
<td>Send a Coombs’s test (direct and indirect) and serum haptoglobin</td>
<td>Indirect: used for typing and crossmatching</td>
<td>Direct: answers the question if hemolysis is immune-mediated</td>
</tr>
<tr>
<td>Check serum haptoglobin</td>
<td>Haptoglobin binds free Hb with high affinity</td>
<td>If haptoglobin is low, hemolytic anemia has occurred</td>
</tr>
<tr>
<td>If bacteremia suspected, give broad-spectrum antibiotics</td>
<td>Prevent fulminant sepsis</td>
<td></td>
</tr>
</tbody>
</table>
it will make an overhand knot around the ET tube. If this occurs, don’t remove the NG or OG until it is simultaneously removed with the endotracheal tube.

**Intravenous line infiltration:** Venous infiltration is usually not problematic as long as it is promptly recognized. One should avoid infusing any medication into a possibly infiltrated vein – as infusing certain medications such as norepinephrine, vasopressin, or dopamine may cause catastrophic tissue necrosis. After recognition of an infiltrated line, one should stop the infusion, elevate the extremity, and perform a baseline neurologic examination of the affected extremity. Use warm compresses or cutaneous warming devices with caution to avoid burns.

**Postoperative Complications**

**Corneal abrasions** often occur during intubation (before the eyes are protected) or during emergence (due to a patient scratching his or her eyes). These injuries are typically mild, and are usually not recognized until the patient is in the recovery room. One should consider the use of eye lubricant and optical guards or tape to reduce this possibility. These injuries usually heal spontaneously after several days.

**Ulnar** and other **neuropathies** are some of the most common anesthetic complications. There is little evidence that this is preventable or exclusively positioning-related. Nevertheless, some experts advocate having the arms in a supine position without tight constriction from soft restraints. Other measures include moving or changing positions of the extremities during lengthy procedures and elevating them on cushions or pillows. It is important not to hyperextend the elbow and to be cognizant of some patients’ flexion deformities, which may make it impossible to fully extend some joints. Femoral neuropathy most likely occurs from surgical retraction in the pelvic brim and not from positioning. Peroneal neuropathy may be attributed to external pressure exerted on this nerve at the fibular head when the high lithotomy position is used.

**Postoperative blindness** is a rare but devastating complication associated with specific patient risk factors such as lengthy surgery, prone position, anemia, edema of orbit, pressure on orbit, and hypotension. Postoperative blindness has also been particularly associated with prone spine surgery cases and cardiopulmonary bypass. The cause is either ischemic optic neuropathy (the vast majority) or central retinal artery occlusion. Should postoperative blindness occur, an ophthalmologic consultation should be obtained and a careful eye exam should be documented.
Case Study

A 52-year-old man is undergoing proctocolectomy for rectal cancer. He was admitted this morning for the operation after undergoing a bowel prep at home the day before. Anesthesia was induced with thiopental and vecuronium and intubation was uneventful. You have placed a peripheral IV, a right internal jugular central line, and a right radial arterial line. You are infusing cefazolin prior to incision.

Five minutes after induction, the blood pressure has decreased to 82/50. What is the differential diagnosis? What will your initial steps be to manage his blood pressure?

The patient is likely hypovolemic after his bowel prep the day before surgery and his overnight fast. Induction agents frequently lead to vasodilation and in some cases myocardial depression, both of which can cause hypotension even in normovolemic patients. The combination of induction agent (thiopental) and volume depletion is the most likely etiology. Other common causes of hypotension early in a case include relative anesthetic overdose, when the anesthetic dose exceeds that required for the amount of surgical stimulation. Incision has not taken place yet, and it has been several minutes after laryngoscopy, so stimulation is likely very light. In this case, you will most likely treat with intravenous fluids to counteract hypovolemia and a vasoconstrictor such as phenylephrine, 40–80 mcg.

The differential diagnosis also includes rarer but serious causes, including anaphylaxis from the antibiotic cefazolin or the neuromuscular blocking drug vecuronium, or pneumothorax from central line placement. One should also rule out artifact by comparing the tracing on the arterial line to a reading on the blood pressure cuff.

Your intervention is successful and the case begins. The patient develops tachycardia in the first few minutes. What is your differential diagnosis and initial response?

The first response is to determine whether it is sinus tachycardia or a dysrhythmia. Abnormal rhythms are more likely to be accompanied by normal or low blood pressure; sinus tachycardia is more likely to parallel hypertension. Initial incision is one of the more stimulating aspects of the procedure, and light anesthesia is a common cause of tachycardia, often preceding by a few seconds or minutes the development of hypertension.
If this is the case, then deepening of anesthesia by increasing the inspired concentration of volatile anesthetic or administration of a rapid acting opioid such as fentanyl, 50–100 mcg, would be prudent.

The patient’s hemodynamic status has stabilized and the case is proceeding. Fifteen minutes later the patient’s oxygen saturation begins to decrease and is now 90%. The patient is breathing 50% oxygen and 50% air by volume controlled ventilation. What is your differential diagnosis? What will be your response? Hypoxia demands a prompt response. The first step, while searching for the etiology, is to increase the FiO₂. You can then check for adequacy of ventilation by observing the CO₂ tracing on the capnograph, and the reading on the exhaled gas volume monitor. Most anesthesiologists will immediately listen to breath sounds to ensure that they are equal, bilateral, and free from wheezes or rhonchi. Common causes at this stage include migration of the endotracheal tube into the right mainstem bronchus (particularly if the patient’s position has changed, for example if the patient is now in Trendelenberg position or head down), and mucus plugging of the endotracheal tube or a bronchus. If unequal breath sounds are heard, checking insertion depth of the tube, possibly verifying proper position with a bronchoscope, or empirically pulling the tube back slightly are all reasonable interventions. Suctioning of the tube with a flexible suction catheter is a prudent maneuver, particularly if breath sounds are unequal or diminished, and/or if airway pressures are increased.

Your initial response to hypoxia has raised the saturation to 92% on 100% oxygen. Auscultation of the lungs reveals bilateral wheezes on exhalation. What steps will you take?

Wheezing can be due to reactive airways disease, or more rarely to anaphylaxis, aspiration of gastric contents, or cardiac failure. The initial steps in management are to ensure adequate oxygen delivery (by increasing FiO₂, checking the circuit, tube, and ventilator settings) and length of exhalation time (by decreasing the respiratory rate or the I:E ratio). Deepening the anesthetic with inhalation anesthetics, which are potent bronchodilators, may help. Inhaled beta-adrenergic agonists, administered via metered dose inhaler into the endotracheal tube, are often effective. Far more than the usual 1–2 puffs given to awake patients is needed, typically 5–10 puffs, as much drug is lost in the tubing and upper part of the trachea.
Wheezing resolves but the patient develops tachycardia and ST segment depressions. How will you respond?

There are many possible causes of myocardial ischemia, and the tachycardia from beta-adrenergic drugs is one possibility. However, it will not be possible to know for sure during the operation. The initial steps include ensuring or augmenting coronary perfusion pressure; if the patient is hypotensive or even significantly below her preoperative baseline, raising the blood pressure with phenylephrine is indicated. Tachycardia from albuterol or other inhaled beta agonists is usually short lived, but a short-acting beta-1 selective beta blocker (esmolol) will slow the heart rate without causing bronchospasm. Nitroglycerin may be administered intravenously if hemodynamic maneuvers fail to resolve the ST segment depressions. The patient should be evaluated postoperatively for ischemia and possibly for myocardial infarction.

Suggested Further Reading


Section V

Systems Physiology and Anesthetic Subspecialties
Chapter 17

Physiology and Anesthesia for Cardiac and Thoracic Surgery

Amanda J. Rhee and Linda Shore-Lesserson

For maximum impact, it is recommended that the case study and questions found on page xxv are reviewed before reading this chapter.

Key Learning Objectives
- Learn relevant cardiovascular physiology and common pathologic conditions
- Understand the anesthetic considerations for cardiac surgery
- Learn thoracic physiology and anesthetic considerations for thoracic surgery

Cardiac Anesthesia
Normal Cardiovascular Anatomy
The heart can be embryologically divided into three layers. The endocardium is a single cell layer on the surface of heart valves and all four chambers. The subendocardium contains the cardiac conduction system, nerves, veins, and structural fibers. The myocardium is the thickest layer, which contains bundles of cardiac muscle cells. The epicardium comprises the visceral pericardium which is a serosal layer adherent to the external wall of the heart. The visceral pericardium is contiguous with the parietal pericardium which completes the pericardial sac. This sac contains pericardial fluid which serves to minimize friction between tissues with each heartbeat.

There are two main coronary arteries that serve as blood supply to the heart (see Table 17.1). They are located at the aortic root where the aorta exits the heart. The left main coronary artery branches into the left anterior descending...
(LAD) artery and the circumflex artery. These provide blood supply to the left ventricle and the anterior 2/3 of the interventricular septum. The right main coronary artery gives rise to the posterior descending artery (PDA) and other branches which supply the right ventricle and posterior 1/3 of the interventricular septum. The origin of the PDA (right coronary versus circumflex artery) determines whether the coronary circulation is right or left dominant, respectively. Perfusion of the left sided coronary arteries occurs during diastole only.

The venous supply from the heart follows the arterial supply and the coronary veins drain into the coronary sinus which then drains into the right atrium. Thebesian veins, which connect directly to the left ventricular cavity also provide a route for venous drainage.

There are four heart valves that promote unidirectional flow (see Fig. 17.1). They open and close based on pressure changes that occur on either side of the valve. The heart's atrioventricular valve on the left side is the mitral valve (between the left atrium and ventricle), which has two leaflets: anterior and posterior. The atrioventricular valve that connects the right atrium and right ventricle is the tricuspid valve, which has three leaflets: anterior, posterior, and septal. The left ventricle pumps blood into the aorta via the aortic valve. The right ventricle pumps blood into the pulmonary artery via the pulmonary or pulmonic valve. Both of these valves are semilunar valves and have three cusps.

The cardiac conduction system is comprised of autorhythmic cells that initiate and conduct action potentials. In a normal heart, the sinoatrial (SA) node is the heart's pacemaker. After an impulse is generated here, it conducts to the atrioventricular (AV) node, where it splits into the Bundle of His down the left bundle branch and right bundle branch. Eventually, the ventricular muscle is innervated when the impulse reaches the Purkinje fibers.

---

**Table 17.1 Coronary artery supply.**

<table>
<thead>
<tr>
<th>Source</th>
<th>Branch #1</th>
<th>Branch #2</th>
<th>Supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left main</td>
<td>Circumflex</td>
<td>Obtuse marginal branches</td>
<td>Left ventricle lateral &amp; posterior walls</td>
</tr>
<tr>
<td></td>
<td>Left anterior descending (LAD)</td>
<td>Septal branches</td>
<td>Majority of interventricular septum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diagonal branches</td>
<td>Left ventricle surface</td>
</tr>
<tr>
<td>Right main</td>
<td>Acute marginal branches</td>
<td></td>
<td>Right ventricle</td>
</tr>
<tr>
<td></td>
<td>AV nodal &amp; SA nodal</td>
<td></td>
<td>AV &amp; SA nodes</td>
</tr>
<tr>
<td></td>
<td>Posterior descending</td>
<td></td>
<td>Inferior &amp; posterior ventricles</td>
</tr>
</tbody>
</table>
Figure 17.1 Anatomy of the heart. (Reproduced with permission from Allen, D.C. Histopathology specimens: clinical, pathological and laboratory aspects. Springer, 2004).
Sympathetic and parasympathetic nerves innervate the heart. \( \beta \)-adrenergic stimulation increases cyclic AMP levels and enhances \( \text{Ca}^{2+} \) influx which causes depolarization of conduction cells. Cholinergic signals, via parasympathetics (vagus nerve), oppose \( \beta \)-adrenergic stimulation and slow down the heart.

**The Cardiac Cycle**

The cardiac cycle is a highly coordinated series of events that requires the participation of the cardiac conduction system, valves, and muscle to orchestrate the movements of systole and diastole (see Fig. 17.2). Systole is isovolumic ventricular contraction and ejection of blood from the heart. Diastole is isovolumic ventricular relaxation and filling of the heart.

Blood returns from the systemic vasculature through the superior vena cava (SVC) and inferior vena cava (IVC) into the right atrium. Pulmonary veins drain into the left atrium. Filling of each atrium occurs continuously. Once atrial pressure exceeds ventricular diastolic pressure, the atrio-ventricular valves open and the ventricles fill (early ventricular filling). Contraction of the atrium comprises late ventricular filling and is often called the "atrial kick". As the ventricles begin to contract, the mitral and tricuspid valves close (S1 heart sound). **Isovolumetric ventricular contraction** occurs when atrio-ventricular valves are closed but pressure in the ventricles has not yet exceeded

![Cardiac Cycle Diagram](image-url)

*Figure 17.2 Cardiac cycle. (From Fung, Y.C. *Biomechanics: Circulation*. Springer, 1997. Used with permission).*
pulmonary artery or aortic pressure. Intraventricular pressure continues to rise, but volume remains constant. As ventricular pressure exceeds pressure in the respective great vessel, the semilunar valves open and blood is ejected. After ejection, ventricular pressure falls below aortic and pulmonary artery pressure, causing the aortic and pulmonic valves, respectively, to close (S2 heart sound). **Ventricular isovolumetric relaxation** occurs when aortic and pulmonic valves are closed and the volume in the ventricles remains constant while the ventricle relaxes. When the ventricular pressure falls below atrial pressures, early diastolic filling occurs and the cycle repeats.

Definitions for cardiac output, stroke volume, and several other important components of the cardiac cycle are listed in Table 17.2.

### Common Disease States Affecting the Heart

#### Ischemic Heart Disease
There are many manifestations of ischemic heart disease (see Table 17.3). Determinants of myocardial perfusion depend on the relationship of supply and demand. Myocardial supply is provided by coronary perfusion pressure, heart rate, $\text{PaO}_2$, and coronary diameter. Myocardial demand parameters are myocardial oxygen consumption, heart rate, left ventricular wall tension, contractility, conduction, and relaxation.

Percutaneous coronary interventions (i.e. coronary stenting) are indicated for persistent anginal episodes, significant stenosis of one to two coronary arteries, and lower risk patients with three vessel disease and favorable anatomy. It is

<table>
<thead>
<tr>
<th>Table 17.2 Other cardiac cycle definitions and equations.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac output (CO)</strong></td>
</tr>
<tr>
<td>$\text{CO} = \text{heart rate} \times \text{stroke volume}$</td>
</tr>
<tr>
<td><strong>Stroke volume</strong></td>
</tr>
<tr>
<td><strong>Preload</strong></td>
</tr>
<tr>
<td><strong>Afterload</strong></td>
</tr>
<tr>
<td><strong>Starling’s Law</strong></td>
</tr>
<tr>
<td><strong>Coronary perfusion pressure</strong></td>
</tr>
<tr>
<td><strong>Left ventricular wall tension</strong></td>
</tr>
<tr>
<td><strong>Fick equation</strong></td>
</tr>
</tbody>
</table>
also indicated in patients with unstable angina or Non ST Segment Elevation MI (NSTEMI) who are in shock, and in STEMI. Coronary artery bypass graft surgery is used for patients with >50% stenosis of the left main coronary artery and patients with two and three vessel disease who have either reduced left ventricular contractile function or diabetes.

**Valvular Disease**

Common causes for **mitral stenosis** (MS) are rheumatic fever and congenital stenosis. It can lead to pulmonary edema and left ventricular failure. Mild MS is a valve area of ≤2 cm² and critical MS is ≤1 cm². Treatment is medical therapy, balloon mitral valvuloplasty, open mitral commissurotomy, or mitral valve replacement. *During anesthesia it is important to maintain sinus rhythm (atrial kick provides 40% of ventricular filling), preload, stroke volume, and low/normal heart rate (to allow time for filling). Avoid drops in SVR and prevent increases in PVR by preventing hypoxia, hypercarbia, and acidosis.*

**Mitral regurgitation** (MR) can be caused by myxomatous mitral valve disease resulting in prolapse, ruptured chordae, or flail segments of the mitral valve. Other causes of mitral regurgitation include ischemic heart disease, which can result in left ventricular enlargement, mitral annulus abnormalities, or necrosis of papillary muscle structures. Other less common etiologies include endocarditis, rheumatic heart disease, and hypertrophic cardiomyopathy. Acute MR leads to high pulmonary pressures and pulmonary congestion, whereas chronic MR can

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**Table 17.3 Ischemic cardiac disease.**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease</td>
<td>Narrowing of coronary arteries from atherosclerosis</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>Myocardial O₂ demand not met by coronary blood flow</td>
</tr>
<tr>
<td>Acute coronary syndrome / myocardial ischemia</td>
<td>Term that includes any of the life threatening conditions below which represent acute myocardial ischemia</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>Myocardial ischemia with chest discomfort</td>
</tr>
<tr>
<td>Stable angina</td>
<td>Exercise induced chronic angina pectoris</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>Myocardial ischemia at rest or with minimal exertion</td>
</tr>
<tr>
<td>Variant angina</td>
<td>Discomfort from coronary artery vasospasm</td>
</tr>
<tr>
<td>Non-ST-segment elevation myocardial infarction (NSTEMI)</td>
<td>Myocardial ischemia from a partially occlusive coronary thrombus</td>
</tr>
<tr>
<td>ST-segment elevation myocardial infarction (STEMI)</td>
<td>Myocardial ischemia from a totally occlusive coronary thrombus</td>
</tr>
</tbody>
</table>
be more compensated with lower pulmonary artery pressures but a low cardiac output. Medical treatment includes inotropic agents and vasodilators. Surgical treatment is with mitral valve repair or replacement. During anesthesia, avoid myocardial depression and increases in SVR (will worsen regurgitation), while maintaining a normal/high heart rate (less time for regurgitation).

Aortic stenosis (AS) is caused by senile degenerative disease, congenital bicuspid aortic valve, or rheumatic heart disease. Male gender, hypercholesterolemia, and smoking are risk factors. Blood flow is obstructed during systole which results in concentric left ventricular hypertrophy. There is a fixed stroke volume and filling is 40% dependent on the atrial kick. Mild AS is a valve area <2.5 cm², moderate is 0.7–1.2 cm², and critical is <0.7 cm². Treatment is percutaneous balloon valvuloplasty or aortic valve replacement. Anesthetic management includes maintaining sinus rhythm (need atrial kick) and slow to normal heart rate (allows for filling time). Also, avoid decreases in SVR because stroke volume is fixed (and thus cardiac output without a rise in heart rate) and the coronary perfusion pressure will fall. Chest compressions during cardiopulmonary resuscitation are usually ineffective.

Aortic regurgitation (AR) is usually caused by leaflet abnormalities (rheumatic disease, endocarditis, and congenital bicuspid valve) or dilation of the aortic root (aortic aneurysm/dissection, Marfan’s syndrome, syphilis-cystic medial necrosis). Acute AR is a surgical emergency manifested by a sudden increase in LV diastolic pressure which causes acute pulmonary congestion, hypertension, and pulmonary edema. In chronic AR, the LV compensates with dilation and hypertrophy which leads to heart failure. Asymptomatic AR can be treated with medical management until the left ventricular dilation causes ventricular dysfunction. Asymptomatic disease in the presence of LV dysfunction or symptomatic AR should be treated with an aortic valve replacement. Anesthetic management includes maintaining sinus rhythm and normal to high heart rate. Avoid myocardial depression and increases in SVR which will worsen the regurgitant fraction. Consider using afterload reduction which decreases the regurgitant fraction.

Arrhythmia Management
Pacemakers are indicated in sick sinus syndrome, tachy-brady syndrome, advanced second degree or third degree heart block, and symptomatic bifascicular block. In general, pacemakers can be left as programmed during a surgical procedure, but electrocautery may inhibit their function. Thus, in patients who
are pacemaker-dependent, the pacemaker should be converted to an asynchronous mode of pacing with a magnet or programming. Exposure to an MRI can convert pacemakers to asynchronous mode. Consider interrogating a patient’s pacemaker at the end of the procedure to ensure proper functioning.

**Automatic Implantable Cardioverter-Defibrillators (AICDs)** are indicated for survival of sudden death episode, sustained VT, syncope from VT, low ejection fraction, or hypertrophic cardiomyopathy. Most AICDs have a pacemaker function. To avoid unnecessary shocks due to interference by electrocautery, AICD function should be turned off with a magnet or programming (preferred). Pacemaker function is maintained even after defibrillation capabilities are turned off. Lithotripsy should be avoided and MRI is contraindicated. An external defibrillator should be available in the operating room.

**Heart Failure**

Heart failure occurs when the heart is no longer able to provide adequate cardiac output to meet the body’s needs. Medical therapy includes diuretics, ACE-I, beta-blockers, inotropes, and vasodilators. Mechanical support can be provided by an aortic balloon pump which is placed via a femoral artery into the descending thoracic aorta such that the tip is positioned just below the left subclavian artery. The intra-aortic balloon pump inflates during diastole improving coronary perfusion, and deflates during systole which improves forward cardiac output. There are many different types of left ventricular assist devices (L.V.A.D.) that can be placed surgically or in the cardiac catheterization lab.

**Anesthetic Management of Cardiopulmonary Bypass (CPB)**

**Preoperative Evaluation**

In addition to the typical preoperative evaluation (see Chap. 8, Preoperative Evaluation), patients undergoing cardiac surgery should be questioned regarding their cardiac symptoms (duration, frequency, precipitating factors) in order to understand their degree of medical optimization. Any history of bleeding abnormalities or clotting disorders should be investigated because patients will be systemically anticoagulated during CPB. Given the association of CPB and postoperative neurological impairment, any preexisting neurologic disease (e.g., stroke, TIA) should also be assessed and documented.

Preoperative laboratory testing should include routine labs (CBC, basic metabolic panel), coagulation studies, and a blood bank sample should also be obtained. All patients undergoing cardiac surgery should also have a
preoperative ECG with a rhythm strip to assess for rhythm abnormalities and a chest X ray to assess for signs of heart failure (pulmonary edema) or other co-existing pulmonary disease. An echocardiogram will give a determination of a patient's left and right ventricular function as well as provide information about any valvular abnormalities. Finally, many patients who present for cardiac surgery will have either a stress test or cardiac catheterization performed – both of which will provide an understanding of areas of the myocardium which are at risk for ischemia during the perioperative period.

**Monitoring**

Monitoring should include a pre-induction arterial line and at least one large bore (16–18 gauge or greater) IV line. A CVP line, and sometimes a PA catheter will be placed before or after induction depending on IV access and severity of the patient's disease (See Chap. 11 on Patient Monitoring). After induction, a transesophageal echocardiography (TEE) probe is placed to evaluate heart anatomy and function. In addition to standard ASA monitors, the patient’s temperature and urine output are also monitored.

**Induction and Maintenance**

Induction is typically performed with a high dose opiate induction (fentanyl 5–50 mcg/kg) and either etomidate, propofol, or thiopental, depending on the patient's underlying disease state. Sevoflurane and isoflurane are acceptable, provided that hemodynamics are well controlled. Pancuronium, cisatracurium, and vecuronium are good choices for paralytics, although pancuronium may cause tachycardia which is undesirable in coronary artery disease. Succinylcholine can be used carefully when indicated with a precurarizing dose of non-depolarizing neuromuscular junction blocker. Avoid using ketamine, which can increase the risk of myocardial ischemia, and can cause cardiac dysfunction in patients who are already catecholamine-depleted. During CPB procedures, nitrous oxide is avoided because of its ability to expand the size of gas emboli that can arise in the pump.

**Pre-Bypass Considerations**

During sternotomy, deflate the lungs to prevent injury during chest opening using the electric saw. Reoperation patients require a great degree of preparation and large bore IV placement, since the heart and large vessels can be adhered to the chest wall anteriorly and ruptured upon chest entry. Consider an antifibrinolytic agent such as epsilon-aminocaproic acid or tranexamic acid
to reduce bleeding. Administer heparin (which activates anti-thrombin III) at a dose of 300–400 U/kg with a goal ACT (activated clotting time) of >480 s before initiating cardiopulmonary bypass. Lower the SBP to 90–120 mm Hg before aortic cannulation in order to minimize the degree of aortic trauma and reduce the risk of aortic dissection and bleeding.

Management of Intraoperative Myocardial Ischemia

Should a patient exhibit signs of myocardial ischemia during surgery, steps must be taken to minimize the amount of damage that occurs. Diagnosis is often made based on ECG or echocardiogram findings. Therapy will be guided by the specific mechanism of ischemia as shown in Table 17.4:

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
<td>– phenylephrine</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>– increase the FiO₂</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>– β-blockers</td>
</tr>
<tr>
<td>Vasospasm</td>
<td>– nitroglycerin</td>
</tr>
<tr>
<td>Anemia</td>
<td>– transfusion</td>
</tr>
<tr>
<td>Thrombosis</td>
<td>– heparin</td>
</tr>
<tr>
<td>Hypovolemia</td>
<td>– fluid administration</td>
</tr>
</tbody>
</table>

Cardiopulmonary Bypass (CPB)

A basic cardiopulmonary bypass circuit (see Fig. 17.3) consists of venous drainage by gravity via a cannula in the right atrium. Certain operations require cannulation of the superior vena cava and the inferior vena cava separately in order to enhance surgical exposure to the heart. The venous cannula drains into a venous reservoir. The blood is then passed through an oxygenator (membrane or bubble), temperature regulator, and actively pumped (roller or centrifugal) back to the patient via an arterial filter into the aorta. The heart is cooled and arrested with a cardioplegia solution that is high in potassium concentration. Cardioplegia is given antegrade into the aortic root, or retrograde through the coronary sinus. Of note, the prime solution of the extracorporeal circuit often contains albumin, mannitol, and steroids, depending on surgeon preference.

During CPB, the ventilator is turned off. It is common to run an infusion of benzodiazepine, narcotic, and muscle relaxant, or to give these agents by

Isotflurane can be given to the patient via the perfusion pump. Hyperglycemia can result in adverse outcomes in cardiac surgery and should be treated appropriately with insulin as indicated.

Potential surgical or perfusion catastrophies during cardiopulmonary bypass include aortic dissection, inadvertent carotid or innominate artery cannulation, reversed cannulation, obstruction to venous return, and massive air embolism. Other medical disasters include drug administration errors such as protamine administration while still on bypass. Postoperative complications after CPB include pulmonary edema (“pump lung”/ARDS), stroke, global cerebral ischemia, fluid/electrolyte imbalances, coagulopathy, and renal dysfunction.

**Weaning From Cardiopulmonary Bypass**

The patient’s blood is warmed by the heat exchanger in the CPB circuit. A core body temperature of at least 36°C is optimal. Many providers administer benzodiazepines because recall is most common during this period of rewarming. Potassium, glucose, and hematocrit levels are all checked and corrected before weaning. The heart may temporarily require inotropes or pacing in order to wean from CPB. Positive pressure ventilation is often used to evacuate air from the heart, great vessels, and grafts. Hypotension upon weaning from CPB can be the result of hypovolemia, myocardial dysfunction, valve abnormalities, vasodilation, and pulmonary hypertension. Respiratory abnormalities (“pump lung”) can also prevent a successful wean from bypass and should be treated with aggressive respiratory therapy.
Once the patient is weaned from extracorporeal circulation, protamine (a basic compound that ionically binds to and deactivates the acidic heparin) can be administered at a dose of 1 mg IV for every 100 units of heparin administered. The goal is to restore a normal ACT of 120–130 s. Potential reactions to protamine are of three types: (1) hypotension (vasodilatory), (2) anaphylactic, anaphylactoid, and (3) catastrophic pulmonary hypertension. Type three reactions often necessitate reheparinization and a return to cardiopulmonary bypass.

**Post Operative Care**
Postoperative cardiac patients are transferred to the cardiac surgical ICU often with the trachea still intubated. Potential post operative complications include return to the operating room for bleeding (inadequate surgical hemostasis or coagulopathy), cardiac tamponade (keep patient “fast, full, tight”), and unexplained poor cardiac performance (occluded graft).

**Minimally Invasive Cardiac Procedures**
These are done off pump or through a smaller alternate incision. Alternate cannulae placement and one-lung ventilation may be necessary depending on the operation (see Table 17.4).

**Thoracic Anesthesia**

**Anatomy**
The trachea gives rise to the right and left main pulmonary bronchi. These do not participate in air exchange. The right mainstem bronchus takes off at a shallower angle than the left mainstem bronchus (see Fig. 17.4), thus, endobronchial intubation and aspiration are more likely to occur on the right side. The right upper lobe exits the right main bronchus posteriorly almost immediately after its take-off from the carina.

**Preoperative Evaluation**
Preoperative evaluation should include a thorough history focusing on dyspnea, cough, cigarette smoking, exercise tolerance, and risk factors for lung injury. Physical exam findings should involve investigation of cyanosis, clubbing, respiratory rate and pattern, and breath sounds. Patients with increased pulmonary vascular resistance should be identified because they require special attention.
Spirometry and pulmonary function testing (PFT) are important ways to determine lung function (see Fig. 17.5). Forced vital capacity (FVC) and forced expired volume in 1 s (FEV$_1$) are two of the most important parameters. An FVC/FEV$_1$ ratio ≥0.75 is normal. A reduced FEV$_1$/FVC ratio is indicative of obstructive lung disease. A normal or increased FEV$_1$/FVC ratio is seen in restrictive lung disease because both values are decreased. A post-resection calculated FEV$_1$ of less than 800 mL in a 70-kg male is a contraindication to lung resection as it is doubtful that the patient will be able to be weaned
from mechanical ventilation. A vital capacity of <50% of predicted or <2 L is associated with increased risk.

Smoking increases the risk of developing postoperative respiratory complications. *Cessation of smoking immediately before surgery is not recommended* because while cessation decreases carboxyhemoglobin levels, it is also associated with an increase of postoperative pulmonary complications. Cessation of smoking longer than 2–4 months is needed before there is a decrease in the incidence of postoperative complications.

**Anesthetic Management**

The patient should have at least one large bore IV (18 gauge). Monitoring should include an arterial line for blood pressure measurement and blood sampling. A CVP, or PA catheter should be considered based on need. Patients are commonly placed in the lateral decubitus position after induction. Be careful to pad exposed peripheral nerves and keep joints at neutral angles. Induction agents and maintenance agents are based upon an individual patient’s medical needs. Intrathecal preoperative injection, or thoracic epidural catheters can be placed for postoperative pain control. Surgical insertion of a local anesthesia pump into the intrapleural space can also help with postoperative pain control.
Often during surgery, one-lung ventilation (OLV) is required. During anesthesia, with the chest open, OLV creates an obligatory right-to-left transpulmonary shunt through the nonventilated, nondependent lung because the V/Q ratio of that lung is zero.

One-Lung Ventilation

Absolute indications for OLV include: (1) Isolation to prevent contamination of a healthy lung in abscess, infected cyst, or massive hemorrhage, (2) Control of distribution of ventilation to one lung as in bronchopleural fistula, bronchopleural cutaneous fistula, unilateral cyst or bullae, and major bronchial disruption of trauma, (3) Unilateral lung lavage, or (4) Video-assisted thoracoscopy surgery (VATS). Other indications are surgical exposure such as thoracic aortic aneurysm, pneumonectomy, upper lobectomy, esophageal surgery, middle and lower lobectomy, and thoracoscopy under general anesthesia.

OLV is accomplished by isolating one lung using a double-lumen endotracheal tube, or a bronchial blocker. The bronchial blocker can be used either in the form of a prefabricated tube with blocker attached (Univent tube®) or as a separate blocker inserted through a T-piece adapter at the top of the tube (Arndt blocker®, Cohen blocker®, Fogarty® catheter).

During OLV, the non-dependent lung is not ventilated, and thus becomes atelectatic. Without ventilation, hypoxic pulmonary vasoconstriction occurs in that lung and diverts blood flow to the ventilated lung. This results in an improvement in oxygenation by reducing the shunt fraction. OLV creates a physiological shunt where the non-dependent lung is perfused but not ventilated. The shunt fraction typically increases from 10% (in the two-lung ventilated anesthetized patient) to 27.5% (in the one lung ventilated patient).

During OLV, if the patient is hypoxic, first apply CPAP (continuous positive airway pressure) to the non-ventilated lung. If no improvement occurs, apply PEEP (positive end-expiratory pressure) to the ventilated lung. If the patient still cannot tolerate OLV, two-lung ventilation must be reinstituted.

Double Lumen Endotracheal Tube

Double lumen tubes (DLT) come in two varieties: left- and right-sided (see Fig. 17.6). Although many feel that left-sided tubes are easier to manage clinically, this has been recently refuted in the literature (Ehrenfeld et al.) and a tube should be selected based on the surgical site (typically placed contralateral to the surgical procedure). All double lumen tubes have both cuffed endobronchial portions and tracheal cuffs. The endobronchial portions are curved either
to the left or right. The bronchial lumen is positioned into the left mainstem bronchus for a left-DLT or the right mainstem bronchus for right-DLT. This lumen has a smaller cuff that usually takes 2–3 ml of air. When placing a right-sided DLT, one must be careful to not occlude the right upper lobe bronchus which takes off almost immediately posteriorly from the right mainstem bronchus (see Fig. 17.4).

A DLT is inserted by holding the tube such that the tip is pointed anteriorly. During laryngoscopy, after the tip of the tube is visualized passing the vocal cords, the tube is rotated 90 degrees, the stylet removed, and the tube is advanced into the left or right mainstem bronchus until slight resistance is felt. The tracheal opening should rest just proximal to the carina. Once the patient is connected to the ventilator, correct placement of the tube can be confirmed several ways. First, a flexible fiberoptic bronchoscope allows for
direct visualization of the bronchial tip within the left or right mainstem bronchus. Only a small portion of the bronchial cuff should be seen at the level of the carina. The cartilagenous rings of the trachea can be seen in the trachea along with the non-intubated mainstem bronchus. When both cuffs are inflated and the tracheal lumen is clamped, proper placement of a left-sided DLT will yield breath sounds and chest rise only on the left side. Conversely, if the bronchial lumen is clamped, breath sounds and chest rise should only occur on the right side.

**Case Study**

A 48-year-old woman presents for resection of extensive rectal hemorrhoids. She first developed the condition during pregnancies in her late 30s and now has had unremitting symptoms of pain, itching, and occasional bleeding. Her surgeon also plans to perform a “tension free vaginal tape” (TVT) procedure for moderate stress urinary incontinence. She has a history of rheumatic heart disease and has had progressively worsening mitral stenosis. She takes digoxin and a baby aspirin daily.

How will you assess the severity of her mitral valve disease?

The history and physical exam are important. One should evaluate for the presence and severity of symptoms of elevated left atrial and pulmonary pressures and systolic dysfunction (positional or exertional dyspnea, edema, weakness, exercise tolerance). An echocardiogram is usually done to follow the mitral valve disease, and you should obtain the most recent one (and older ones if possible, to evaluate progression of her disease). Even if these were done at an outside hospital, you should make the effort and even delay surgery, to obtain the reports prior to this elective procedure. On the echocardiogram report, you will focus on the valve area and estimated
LVEDP (left atrial pressure estimate), pulmonary artery pressures, presence of mitral regurgitation and/or other valve lesions, regional wall motion abnormalities, and systolic function.

You conclude that she has moderately severe mitral stenosis with moderately reduced systolic function. What are your hemodynamic goals for the perioperative period?

A rule of thumb for valve disease is that stenotic lesions are kept “slow and tight,” while regurgitant lesions are kept “fast and full.” This means that you will avoid excessive volume loading to avoid pulmonary edema, peripheral vasodilation to avoid hypotension and compensatory increases in heart rate, and tachycardia. Patients with mitral stenosis are not able to increase stroke volume markedly in response to decreased arterial tone, and they do not tolerate the usual physiologic response to hypotension, an increase in heart rate, because it does not allow adequate time for ventricular filling across the stenotic mitral valve. Sinus rhythm is very beneficial in these patients, as the atrial kick can significantly augment ventricular filling. However, many patients are in atrial fibrillation due to the enlarged left atrium. In this case, ventricular rate control is vitally important. If she is in sinus rhythm, you will try to avoid triggers of atrial fibrillation such as excessive sympathetic activation or atrial stretch, and you will have drugs and cardioversion capability available should she develop uncompensated atrial fibrillation in the perioperative period.

Her cousin had a very similar procedure performed recently and had spinal anesthesia. She had spinal anesthesia herself for a cesarean section and was very pleased with it. She asks you if she can have this form of anesthesia for her current procedure. How will you respond?

Spinal anesthesia is relatively contraindicated in significant mitral stenosis. This is because the decreases in preload due to venodilation and in “afterload” or peripheral arterial tone are poorly tolerated. Relatively high filling pressures are needed to fill the left ventricle across the stenosis, and as noted above, decreases in arterial tone cannot be compensated for by increasing stroke volume or heart rate. A carefully and slowly titrated epidural block has been successfully employed in cases of mitral stenosis, but this case will require dense sacral blockade, and epidural analgesia may spare or only partially block the sacral roots.
Does she need antibiotic prophylaxis?
Not necessarily. Previously, American Heart Association guidelines called for antibiotic prophylaxis for patients with valvular heart disease, including rheumatic heart disease, when undergoing dental, GI, or GU procedures. The most recent guidelines, published in 2007, now limit antibiotic recommendations to those with synthetic prosthetic valves, complex congenital heart disease, and patients with a previous history of infective endocarditis. The severity of the disease and likelihood of sustained bacteremia are potential factors that might elevate her risk and thus lead to recommended antibiotics, but routine GU and GI procedures are no longer considered indications for antibiotics solely on the basis of the risk of endocarditis. However, most surgical patients should receive prophylactic antibiotics to reduce the risk of surgical site infection, so you may choose to broaden your antibiotic coverage to include prophylaxis for her heart.

You decide to administer general anesthesia. What drugs will you avoid? Which will you choose?
You will avoid drugs with potent vasodilatory effects or tendency to produce tachycardia. Therefore, you may avoid propofol (vasodilation) and desflurane, pancuronium, ketamine, and anticholinergics (tachycardia). Nitrous oxide is controversial, since it can increase pulmonary artery pressure. Thiopental or etomidate would be reasonable choices for induction. Sevoflurane and short-acting opioids would be reasonable choices for maintenance.

What other special precautions will you take in the intra- and post-operative periods?
You will watch for bleeding, and treat volume loss aggressively, but avoid volume overload or accidentally infusing excess fluids without a specific indication. You wish to avoid shivering and other stimulants that could cause tachycardia. Therefore, good pain and nausea prophylaxis are important. You will monitor her heart rhythm carefully and be prepared for rate control and cardioversion should she develop atrial fibrillation. Finally, you will be wary of position changes, such as putting the patient's feet up into the lithotomy position, or down at the end of the case, which may cause hemodynamic challenges.
Suggested Further Reading


Chapter 18

Physiology and Anesthesia for Neurologic, ENT, and Ophthalmologic Surgery

Joshua H. Atkins

For maximum impact, it is recommended that the case study and questions found on page xxvi are reviewed before reading this chapter.

Key Learning Objectives
- Understand the relationship between cerebral blood flow, PaO₂, and PaCO₂
- Learn the effects of anesthetic agents on cerebral physiology
- Know the anesthetic approaches used for ENT and ophthalmologic surgery

Neuroanesthesia
The central tenets of neuroanesthesia are brain protection and optimization of surgical exposure. These are based on the physiology of cerebral autoregulation and associated reflex and iatrogenic modulation of brain volume, intracranial pressure, cerebral blood flow, and cerebral metabolic rate.

Intracranial Pressure (ICP)
The cranium is a closed space. ICP is determined by the combination of brain cellular volume (80%), cerebrospinal fluid (CSF) volume (10%), and blood volume (10%). Normal intracranial pressure is < 10 mm Hg. Cerebral blood flow (CBF) is a function of mean arterial blood pressure (MAP) and ICP or central venous pressure (CVP), and is defined as CPP = MAP - ICP (or CVP, whichever is greater). A cerebral perfusion pressure (CPP) of 55–70 mm Hg
is usually targeted, although in the presence of severe intracranial disease, the target must be individualized to patient physiology.

Increase in brain mass (tumor, edema, traumatic brain injury), overproduction of CSF, or obstruction to outflow (e.g., tumor, hemorrhage or clot) or increased blood volume (\(\downarrow\) venous drainage, \(\uparrow\) arterial blood flow) all increase ICP. The normal physiologic response to increased ICP, in the absence of severe pathology, is diversion of CSF to the spinal canal.

As ICP continues to increase, mental status decreases, focal neurologic signs (e.g., dilated pupils, cranial nerve defects) appear, and herniation of brain contents occurs. Eventually, manifestations of the Cushing’s response (Cushing Triad = hypertension, bradycardia, irregular respiration) are present due to brainstem compression. These signs herald a neurosurgical emergency.

Management of ICP/brain volume is a critical part of anesthetic management for the neurosurgical patient. ICP can be measured by direct catheter insertion into a CSF-containing space or via a surgically placed subarachnoid bolt. Interventions to reduce ICP include:

1. Head elevation to 30 degrees
2. Optimization of jugular venous drainage
3. Direct drainage via a lumbar drain or intraventricular catheter
4. Hyperventilation (\(P_a CO_2\) 25–30 mmHg) to decrease CBF
5. Osmotic diuresis (mannitol, hypertonic saline)

Cerebral Blood Flow
Under normal conditions cerebral blood flow is autoregulated in the range of MAP 50–150 mm Hg. As cerebral metabolic rate increases, blood flow increases proportionally. Autoregulation is assumed to be disrupted in patients with chronic hypertension or pathologic conditions including traumatic brain injury and stroke or by inhaled anesthetic agents. Figure 18.1 shows a relationship between CBF and arterial \(O_2\) content, \(CO_2\) content, as well as CPP.

Blood Brain Barrier (BBB)
Brain capillaries contain tight-junctions that limit the passive diffusion of many substances into the brain tissue. The physiology of the BBB facilitates reduction of brain volume by osmotic agents such as mannitol and hypertonic saline, which are not freely permeable. Many pathologic states, including trauma, sepsis, and hemorrhage disrupt the BBB.
Neuromonitoring

Neuroelectrophysiologic monitoring for surgical procedures on the brain and spine is increasing in scope and usage. The fundamental goal of these techniques is to avoid injury to functional pathways from either direct anatomic disruption or ischemia during surgical resection and manipulation.

Techniques include EEG (Electroencephalography) monitoring, monitoring of descending motor pathways and the corticospinal tract via MEP (motor evoked potentials), ascending sensory pathways and dorsal column system via SSEP (somato sensory evoked potentials), local neuromuscular pathways via EMG (electromyography), and cranial nerve function monitoring. The positive and negative predictive value of changes in the monitored parameters varies based on the type and location of signal monitored, the anesthetic agents, blood pressure, temperature, and preoperative neurologic deficits.

It is important to understand the use of neuromonitoring in selected cases, to know the basic tract(s) under surveillance, and to understand the general impact of anesthetic agents on these parameters (see Table 18.1).

Maintenance of intraoperative neuromuscular blockade is contraindicated in any cases in which motor response will be monitored. Cortically generated potentials of any kind are significantly depressed by inhaled potent agents, which should be avoided or used in low concentrations during cortical monitoring. Spinal potentials and deeper brain potentials (e.g., auditory) are substantially more resilient to the effects of anesthetic agents and are compatible...
with a wider range of anesthetics. Benzodiazepines in anxiolytic doses, and most opioid agents in typical analgesic doses have little impact on monitored potentials. A critical caveat is to avoid bolus delivery of anesthetic agents and provide a relatively stable depth of anesthesia throughout the monitoring period. Maintenance of steady blood pressure and core body temperature also fall under the purview of anesthetic management during neuromonitoring.

**Neurophysiology: Anesthetic Effects**

Anesthetic agents almost universally decrease brain activity, with the exceptions of ketamine and nitrous oxide when used alone (see Table 18.1). For this reason, ketamine and nitrous oxide are often omitted from anesthetic management in intracranial surgery. The decrease in brain activity with other agents (e.g., propofol) correlates with a decrease in global CMR (cerebral metabolic rate). However, inhaled potent agents such as isoflurane will vasodilate the major intracerebral arteries resulting in an overall increase in cerebral blood flow and intracranial volume, which reflects an uncoupling of CMR with CBF and excess perfusion. Inhaled agents are relatively contraindicated in situations of increased ICP or when increased brain volume impedes surgical access to the anatomy of interest. In contrast, the intravenous agents propofol and thiopental decrease both CMR and CBF (i.e., they maintain the normally coupled relationship). Infusions of

| Table 18.1 Effects of anesthetic agents on cerebral physiology. |
|---|---|---|---|---|
| Agent | CBF | CMR | EP | Comments |
| Halogenated Potent Agents | ↑ | ↓ | ↓↓ | <0.5MAC generally suitable “luxury perfusion" uncoupling of CBF/CMR relationship |
| N₂O 60% (alone) | ↑↑ | ↑ | ↔ | |
| N₂O + Potent Agent | ↑ | ↔ | ↓↓ | effects ↑ with ↑ MAC of the potent agent |
| N₂O + Propofol | ↔ (↓) | ↔(↓) | ↔ | |
| Propofol | ↓↓ | ↓↓ | ↔ | often used to ↓ intra-op brain volume |
| Etomidate | ↓ | ↓ | ↑ | may enhance MEP’s; ↑ risk of seizure |
| Ketamine (alone) | ↑ | ↑ | ↑ | generally contraindicated in neurosurgery |
| Ketamine + Propofol | ↔ | ↔ | ? | propofol modifies effects of ketamine |
| Fentanyl | ↔ | ↔ | ↔ | effects may occur in >10 mcg/kg bolus |
| Midazolam | ↔ | ↓ | ↔ | may ↓ EPs in >0.2 mg/kg bolus dose |

*EP Evoked potential, CBF Cerebral blood flow, CMR Cerebral metabolic rate.*
these agents may be beneficial in the management of patients with increased ICP or used to facilitate surgical exposure in the “tight,” swollen brain.

Other agents commonly used in balanced anesthesia include opioids and benzodiazepines. Generally speaking, these agents have minimal impact on CMR or CBF and are commonly used as part of a balanced anesthetic.

Neurosurgical Procedures: Anesthetic Management

General Goals

The goals for the management of a neurosurgical patient are similar across the spectrum of patient disease. Attainment of these goals relies on a thorough appreciation of basic neurophysiology, understanding of the effects of individual anesthetic agents on brain function, and clear perioperative communication with the neurosurgical team.

Key Features of a Neuroanesthetic

<table>
<thead>
<tr>
<th>(1) Neuroprotection</th>
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<tbody>
<tr>
<td>(a) optimization of CBF/CMR balance</td>
</tr>
<tr>
<td>(b) control of ICP</td>
</tr>
<tr>
<td>(c) temperature regulation (avoid hyperthermia)</td>
</tr>
<tr>
<td>(2) Provision of optimal operating conditions, including neuromonitoring and “relaxed” brain</td>
</tr>
<tr>
<td>(3) Maintenance of normal glucose and electrolyte balance</td>
</tr>
<tr>
<td>(4) Prompt emergence from anesthesia to facilitate neurologic assessment</td>
</tr>
</tbody>
</table>

Craniotomy

Preoperative Considerations

Questions to ask at the beginning of an evaluation include:

- **Why is the surgery being done?**
- **Is the targeted pathology related to tumor, neurovascular malformation (aneurysm/AVM), traumatic brain injury with intractable intracranial hypertension, or intracranial hemorrhage (epidural, subdural, intracerebral)?**
- **Will neuromonitoring be employed?**

A detailed neurologic exam must be performed with attention to recent signs and symptoms such as mental status, seizures, focal deficits, and signs of increased ICP. Available neuroimaging studies should be reviewed and any procedures noted (e.g., embolization of AVM or tumor, placement of intraventricular
catheter or tissue oxygen monitor). Current medications (especially blood pressure agents, anticonvulsants, steroids, and sedative-narcotics) should be reviewed and time of last dose noted. Blood products should be immediately available for most procedures.

**Intraoperative Considerations**

General endotracheal anesthesia is indicated for most intracranial procedures except for the “awake craniotomy” for epilepsy or resection of a lesion in the motor or speech cortex. Invasive monitoring is indicated for all but the most limited neurosurgical procedures (e.g., stereotactic biopsy or Burr hole drainage). An arterial line will facilitate close management of blood pressure, carbon dioxide, serum osmolality, hemoglobin, and oxygenation. Central venous access should be considered based on likelihood of high volume blood loss (e.g., invasive cancer, AVM resection) or air embolus (sitting position). Maintenance with intravenous or inhaled agents should be individualized to the patient and the proposed surgical approach. Opioids should be used judiciously; fentanyl and hydromorphone are most commonly employed. The most stimulating periods of surgery are head pinning, skin incision, and dural opening. Benzodiazepines should be used sparingly to facilitate rapid emergence and postoperative neurologic evaluation. Some anesthesiologists avoid Lactated Ringers because it is hyponatremic and hypo-osmolar. Large volumes of normal saline, however, may produce a non-anion gap metabolic acidosis, which must be considered in assessment of arterial blood gases.

Rapid emergence and extubation is feasible after most neurosurgical procedures. Exceptions include patients with profoundly decreased mental status prior to surgery, significant intraoperative complications, acute traumatic brain injury, marginal surgical hemostasis with high likelihood for re-exploration, and procedures involving critical neural structures of the posterior fossa.

**Neurovascular Surgery: Aneurysm Clipping/AVM Resection**

Arteriovenous malformations are abnormal collections of veins and arteries with convoluted vessel contributions that lack capillaries. These lesions may feed functional cortex, which can be studied prior to surgery by selective barbiturate injection in the awake patient. An AVM may be selectively embolized in the radiology suite preoperatively to reduce bleeding.

These procedures are technically challenging, high-risk interventions with unique considerations for anesthetic management. The complexity of the dissection, the risk of rupture, and the surgeon’s plan for CSF drainage, burst-suppression,
deliberate hypotension, deep hypothermic circulatory arrest, or temporary clipping should all be outlined in detail during the preoperative preparations.

Blood pressure control is of central importance. Acute hypertension prior to clipping can lead to catastrophic aneurysmal rupture. AVM’s, by nature of the anatomy involved, are generally much less prone to rupture than aneurysms. Intubation, pinning, and incision are times of high risk for this complication. A smooth induction to a deep plane of anesthesia with complete muscle relaxation, generous narcotic administration, glottic topicalization, and brief laryngoscopy is often desirable. Hypertension should be treated immediately with additional intravenous hypnotic agents, rapidly acting vasodilators (nitroprusside; nicardipine), and prompt cessation of stimulation. Aneurysm rupture is a catastrophic, albeit rare complication. Blood loss can be substantial and sudden.

**Neurosurgical Anesthesia Controversies**

For the advanced student these key questions (with no clear answers) serve as excellent starting points for reading on current topics and intraoperative discussion with both residents and faculty. References are provided for further reading and to stimulate discussion.

(a) *Is nitrous oxide contraindicated in neurosurgery?*


(b) *Are deliberate hypothermia or EEG burst suppression useful methods of neuroprotection during neurovascular surgery or after traumatic brain injury?*


(c) *Does neuromonitoring in aneurysm surgery reduce complications?*


(d) *Is hypertonic saline better than mannitol for ICP management?*

Neurologic Disease and Anesthesia: Special Considerations

Several neurological conditions deserve a special mention, since they have significant implications for the anesthesiologist. These include myasthenia gravis, multiple sclerosis, Guillain-Barré syndrome, neuroleptic malignant syndrome, and Parkinson’s disease and are listed in Table 18.2.

Otolaryngology (ENT): Anesthetic Considerations

ENT procedures have extraordinary variation from the relatively simple and straightforward (sinus surgery) to the technically complex and challenging (resection of a glottic lesion). A common theme is the notion of the “shared”

<table>
<thead>
<tr>
<th>Table 18.2</th>
<th>Anesthetic considerations in neurologic diseases.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Myasthenia Gravis (MG)</strong></td>
<td>Autoimmune antibodies against nicotinic cholinergic receptors</td>
</tr>
<tr>
<td><strong>Etiology</strong></td>
<td>Dysphagia, dysarthria, ptosis</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td>Anticholinesterases, steroids, plasmapheresis, thymectomy</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Assess degree of weakness &amp; duration of symptoms</td>
</tr>
<tr>
<td><strong>Preoperative Considerations</strong></td>
<td>Optimize patient prior to surgery; maintain home anticholinesterase therapy</td>
</tr>
<tr>
<td></td>
<td>Consider PFTs, ECG (can see myocardial changes), electrolytes</td>
</tr>
<tr>
<td></td>
<td>Anticholinesterase overdose can lead to cholinergic crisis</td>
</tr>
<tr>
<td></td>
<td>• Diagnosis = worsening of symptoms with edrophonium (10mg)</td>
</tr>
<tr>
<td></td>
<td>• Treatment = anticholinergic administration (i.e., atropine)</td>
</tr>
<tr>
<td><strong>Anesthetic Management</strong></td>
<td>Minimize sedatives/respiratory depressants; consider regional</td>
</tr>
<tr>
<td></td>
<td>Consider rapid sequence induction (patients at risk for aspiration)</td>
</tr>
<tr>
<td></td>
<td>Patients at risk for postoperative respiratory failure</td>
</tr>
<tr>
<td></td>
<td>Avoid muscle relaxants if possible</td>
</tr>
<tr>
<td></td>
<td>Use caution when using neostigmine (risk of cholinergic crisis)</td>
</tr>
</tbody>
</table>

| **Multiple Sclerosis** | CNS disorder leading to demyelinated nerve plaques |
| **Etiology** | Visual disturbances, limb weakness, paralysis, respiratory failure |
| **Symptoms** | Steroids, interferon, baclofen, dantrolene |
| **Treatment** | Increased risk of aspiration |
| **Anesthetic Management** | Increased risk for postoperative respiratory failure |
| | Spinal associated with worsening symptoms (epidurals are not) |

| **Guillain-Barre Syndrome** | Acute demyelinating polyneuropathy (often after minor infection) |
| **Etiology** | Limb weakness, decreased reflexes, autonomic instability |
| **Symptoms** | IVIG, plasmapharesis |

(continued)
Physiology and Anesthesia for Neurologic, ENT, and Ophthalmologic Surgery

Airway between the surgeon and the anesthesiologist. Detailed communication with the surgical team in the preoperative and intraoperative periods is imperative along with an appreciation for both the lack of access to the airway and the possibility of surgical disruption of the airway.

Specialized equipment

ENT surgery provides exposure to a variety of specialized airway and surgical equipment. This includes a variety of endotracheal tubes (nasal and oral RAE, reinforced, anode, red-rubber) that generally afford the operative team improved access, a more secure airway, or special monitoring capability. Procedures on the larynx or trachea may utilize high-frequency jet ventilation and laser technology for lesion ablation, whereas sinus surgery increasingly utilizes real time CT-image guidance. Early familiarity with the available anesthesia equipment will facilitate anesthetic planning.
Preoperative Planning
The goals and indications for the planned procedure should be clearly defined. An algorithm for operative planning for ENT surgery patient is presented in Table 18.3.

A feature relatively specific to ENT anesthesia is the increased likelihood of an anticipated difficult airway. This is especially true in patients who present with lesions of the oropharynx, trachea, or large thyroid mass. These patients require special consideration than other “routine” ENT procedures including neck dissection and sinus surgery.

A thorough preoperative assessment should include review of nasopharyngeal laryngoscopy reports and discussion of the type and location of the lesions with the surgeon. The patient should be queried regarding signs or symptoms of airway obstruction (positional dyspnea, cough, stridor, dysphagia, hoarseness, wheezing) or a diagnosis of obstructive sleep apnea. Radiologic studies, and particularly 3D multi-planer CT reconstructions of the airway may be

<table>
<thead>
<tr>
<th>Table 18.3</th>
<th>Perioperative considerations for ENT surgery.</th>
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<tr>
<td><strong>General Considerations</strong></td>
<td></td>
</tr>
<tr>
<td>Higher incidence of a difficult airway</td>
<td></td>
</tr>
<tr>
<td>Be prepared for an airway emergency</td>
<td></td>
</tr>
<tr>
<td>Airway is often shared with the surgeon: two-way communication is essential</td>
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<tr>
<td><strong>Procedure-Specific Considerations:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Neck Dissection</strong></td>
<td></td>
</tr>
<tr>
<td>Lengthy procedure with increased risk of air embolus</td>
<td></td>
</tr>
<tr>
<td>Usually performed under general endotracheal anesthesia with controlled ventilation</td>
<td></td>
</tr>
<tr>
<td>Brachial plexus nerve monitoring may preclude the use of paralytics</td>
<td></td>
</tr>
<tr>
<td>Higher incidence of anesthesia circuit disconnect and significant bleeding</td>
<td></td>
</tr>
<tr>
<td><strong>Endoscopic Sinus Surgery</strong></td>
<td></td>
</tr>
<tr>
<td>Short-duration procedure</td>
<td></td>
</tr>
<tr>
<td>Pituitary tumors may have associated conditions (acromegaly, diabetes insipidus)</td>
<td></td>
</tr>
<tr>
<td>Usually performed under general anesthesia</td>
<td></td>
</tr>
<tr>
<td>“Smooth” wakeup to minimize hypertension and bleeding is recommended</td>
<td></td>
</tr>
<tr>
<td><strong>Inner Ear Surgery</strong></td>
<td></td>
</tr>
<tr>
<td>Anesthetic techniques include general anesthesia or sedation (stapedectomy)</td>
<td></td>
</tr>
<tr>
<td>Generally minimal blood loss and postoperative pain</td>
<td></td>
</tr>
<tr>
<td>Facial nerve monitoring precludes the use of paralytics</td>
<td></td>
</tr>
<tr>
<td>Higher incidence of postoperative nausea and vomiting</td>
<td></td>
</tr>
<tr>
<td><strong>Airway Surgery</strong></td>
<td></td>
</tr>
<tr>
<td>Higher incidence of a difficult airway (awake intubation sometimes needed)</td>
<td></td>
</tr>
<tr>
<td>Increased risk of airway fire during the use of electrocautery</td>
<td></td>
</tr>
<tr>
<td>Postoperative intubation and ventilation may be needed</td>
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</tbody>
</table>
performed in some centers. Lung spirometry will commonly show evidence of obstruction. **Warning signs of impending severe obstruction include signs and symptoms such as inability to lie flat or produce a strong cough, and also stridor, dyspnea at rest, drooling, and baseline hypoxemia.** Awake fiberoptic intubation should always be considered in the management of a tenuous airway. In particularly difficult airways with impending obstruction, an awake tracheostomy under local anesthesia may be performed prior to anesthesia.

**Intraoperative Issues**

General anesthesia for ENT procedures can be maintained with a variety of techniques. Total intravenous anesthesia (TIVA) may be considered in procedures with delicate hemostasis (sinus surgery, tonsillectomy, inner ear surgery). TIVA may help reduce bleeding and coughing at emergence, and reduce postoperative nausea and vomiting. TIVA should also be considered in cases where periodic interruption of ventilation is likely to be required or jet ventilation employed. An infusion of propofol and an opioid (fentanyl, sufentanil, remifentanil) is the most common approach.

The surgical airway is often rotated away from the anesthesia team and may become inaccessible after surgical draping. Extreme neck extension, rotation, or flexion for surgical positioning can result in extubation or endobronchial intubation, respectively. Intrusion on the jugular vein during spontaneous ventilation could result in air embolus.

As in certain neurosurgical procedures, nerve monitoring has a role in ENT surgery when the facial, acoustic, and recurrent laryngeal nerves are at risk. The procedures include resection of acoustic neuroma, mastoidectomy, tympanoplasty, parotidectomy, and thyroidectomy. A specialized endotracheal tube with electrodes located at the glottis may be used to monitor vocal cord function. Nerve monitoring for these procedures precludes the use of intraoperative muscle relaxation, but due to the high fidelity of EMG signals there is rarely any need to further adjust the anesthetic management.

**Neck dissection**

Neck dissection is a common procedure performed to remove tumors and lymph nodes. The procedure is often lengthy, and spontaneous ventilation is relatively contraindicated due to possible air embolus with surgical trespass on neck veins. General endotracheal anesthesia is the standard approach.
Surgeons may desire to monitor the brachial plexus during fine dissection, yet gross dissection around large neck muscles often benefits from muscle relaxation. Therefore, coordination with regard to dosage and timing of neuromuscular blockade should occur. Frequent manipulation of the head during the procedure often leads to sudden circuit disconnect or tube malposition (mainstem intubation with head flexion or cuff herniation with head extension). These possibilities should be considered immediately if ventilator fault alarms sound or hypoxemia develops.

**Endoscopic Sinus Surgery**

This is a common procedure performed for chronic sinusitis, severe epistaxis, tumor resections of the anterior skull base, pituitary, and sinus cavities, and repair of CSF leaks. Most patients who present for these procedures have limited co-morbidities. One should be aware, however, of the physiologic consequence of pituitary tumors and their removal (acromegaly, diabetes insipidus, thyroid dysregulation). The anesthetic approach typically involves general endotracheal anesthesia with non-invasive monitoring and single intravenous access. Postoperative pain is usually limited and blood loss typically modest. In complex cases of tumor resection or epistaxis treatment, large-bore IV access and blood products should be available. A lumbar drain, to facilitate CSF drainage and fluorescein dye injection, may be requested. Thorough suctioning of the oropharynx prior to extubation is critical as large volumes of secretions may accumulate. Some elect to pass an orogastric tube to evacuate blood and secretions prior to extubation.

**Inner Ear Surgery**

Chronic mastoiditis, sensorinueral hearing loss, and otosclerosis are all common indications for inner-ear surgery. Procedures include tympanoplasty, mastoidectomy, stapedectomy, and cochlear implant. The procedures are routinely performed under general anesthesia with LMA or endotracheal tube, although stapedectomy can be safely performed with sedation in selected patients. There is rarely significant blood loss and postoperative pain is usually not significant. Intraoperative monitoring of the facial nerve is standard and requires avoidance of muscle relaxation during the intraoperative period. A major problem is postoperative nausea and vomiting which requires aggressive multi-modal prophylaxis: a serotonin 5HT-3 antagonist, dexamethasone, scopolamine patch, and promethazine are commonly employed.
Airway Surgery
Surgery to diagnose and treat airway disease (vocal cord polyp, oral cancer, laryngeal mass) is a mainstay of ENT practice. These patients tend to have multiple medical conditions, have a long history of smoking or heavy alcohol consumption, and the potential for a difficult airway. Airway fire is a potential intraoperative complication. Postoperative intubation and ventilation may be considered in patients with significant residual airway disease, or procedures in which significant surgery involving the airway may predispose to swelling, recurrent laryngeal nerve injury, or bleeding with concomitant airway compromise.

Ophthalmology
The majority of ophthalmologic procedures are done on an outpatient, elective basis. However, the patient population varies widely from healthy children having strabismus surgery to sick, elderly patients presenting for cataract surgery. Procedures generally require a cooperative patient and an immobile globe.

Intraocular pressure (IOP) is akin to ICP and is a primary physiologic consideration in ophthalmologic surgery. It is particularly important in direct injury to the globe and glaucoma, and IOP may be increased by severe hypertension, valsalva, coughing, hypercapnia, succinylcholine-induced fasciculations, and injection of fluid/anesthetic into the orbit.

Procedures such as Lasik® and cataract surgery are conducted with sedation accompanied by local infiltration or eye block. Others, including vitrectomy and strabismus repair usually require general anesthesia. Sometimes choice of anesthetic is influenced by co-existing conditions, such as inability to lay flat or remain still. For the sedation technique, a bolus of a hypnotic agent such as propofol, etomidate, or ketamine will facilitate block and injection of local anesthetic. Following the injection, anesthetic requirements are minimal. The head of the patient is usually fully covered and inaccessible once surgery has commenced under the operating microscope. A nasal cannula with capnographic monitoring capability is used.

Many ophthalmologists are accustomed to placing an eye block (retrobulbar, peribulbar, Sub-Tenon's injection).

For the retrobulbar block (Fig. 18.2), a 25G sharp needle (25 mm length) is used to inject several milliliters of a mixture of bupivacaine 0.5% with lidocaine 2% and hyaluronidase to facilitate diffusion and penetration.
Epinephrine is avoided in most patients on the presumption of exacerbation of cardiac disease or risk of optic ischemia.

Anesthesiologists must be able to recognize and treat potential complications associated with eye blocks. Complications include subarachnoid injection (causes apnea), intravascular injection (causes seizures), intraneural injection (causes severe pain, blindness), and globe rupture, bleeding, and increased IOP.

The **oculo-cardiac reflex** is an additional consideration. Mediated by the ciliary branches of cranial nerve V1 (trigeminal nerve) and the vagus nerve, the reflex causes a profound bradycardia, and occasionally arrhythmia or asystole in response to manipulation of the globe or pressure within the orbit. The resultant bradycardia can be treated with immediate cessation of stimulus, administration of atropine, deepening of general anesthesia, and in some cases infiltration of additional local anesthetic. The reflex is extremely common in pediatric strabismus surgery and less so during procedures conducted under local block.

Finally, procedures on the eye increase the risk of postoperative nausea and vomiting and aggressive prophylaxis is recommended.
Case Study

A 20-year-old male is attending a company picnic. After lunch, the attendees play softball. Your patient is struck in the head by a hit ball. He immediately loses consciousness and paramedics are called to the scene. He is transported to the hospital where a CT scan shows an acute subdural hematoma requiring surgical evacuation. He is awake but confused and sluggish and does not respond appropriately to verbal commands. He does withdraw purposefully to painful stimuli. He does not have any other injuries. His friends tell you he has “never been sick a day in his life.” He is 6 feet, 185 pounds. BP 185/90, HR 55, SpO₂ 96% on room air.

Do you believe his intracranial pressure (ICP) to be elevated? What signs, symptoms, or tests can help you decide? Does it matter when deciding how to induce anesthesia?

There are several possible reasons to suspect the patient’s ICP is elevated. First, there is the mechanism of injury itself and the nature of the CT finding of subdural hematoma. The presence of a mass lesion intracranially can certainly raise ICP. Also, the patient’s altered mental status is consistent with elevated ICP. His blood pressure and heart rate are suggestive of the Cushing reflex, a compensatory mechanism which attempts to maintain cerebral perfusion pressure in the setting of an elevated ICP. Other signs and symptoms might include papilledema, unequal or poorly reactive pupils, or CT findings of altered ventricular size or midline shift in brain contents. The presence of elevated ICP does indeed influence the choice of anesthetic drugs and technique for induction. The goal is to maintain cerebral perfusion pressure (CPP) by avoiding any further increase in ICP and a decrease in mean arterial pressure (MAP). Factors that might increase ICP include hypercapnia, light anesthesia or vigorous laryngoscopy, vasodilators, and controversially, succinylcholine. Any drug that lowers MAP could theoretically reduce CPP; however, propofol and thiopental also reduce cerebral blood flow and cerebral metabolic rate, and thus may lower ICP as well.

What determinants of ICP can you influence prior to induction? Will you lower his blood pressure prior to induction?

The three determinants of ICP are the volumes of the intracranial contents: brain matter, CSF, and blood. It is possible to reduce the volume of all three,
though in practice, brain water and blood volume are the most amenable to intervention. Ventriculostomy tubes are sometimes placed by neurosurgeons preoperatively to drain CSF. Blood volume can be reduced by elevating the head of the bed about 30 degrees. Hyperventilation to $\text{PaCO}_2=30$ mm Hg can reduce cerebral blood flow but this maneuver is controversial in the setting of head trauma and elevated ICP, because it may worsen ischemia in vulnerable areas. Similarly, lowering the blood pressure, though reducing the tendency to bleed and expand the hematoma, may compromise cerebral perfusion pressure in vulnerable brain regions. Reducing brain water by administration of mannitol is sometimes used as well, although more frequently after induction. However, in cases of vascular disruption, extravasation of mannitol may actually worsen ICP.

What other considerations are there in deciding how you will induce anesthesia?

The patient had eaten just before his injury and thus has a “full stomach” as well as a “tight head.” This usually indicates a rapid sequence induction of anesthesia and use of succinylcholine. Because one does not ventilate the patient prior to intubation, $\text{PaCO}_2$ may rise and CBF may increase, particularly if laryngoscopy is difficult. An airway examination, if possible in this obtunded patient, is important. Also, succinylcholine may transiently increase ICP; some have suggested avoiding it for this reason, though no data supports omitting it.

Given all of the above considerations, what drugs will you choose for induction of anesthesia?

There is likely no ideal induction sequence. A reasonable approach is careful preoxygenation and rapid sequence induction with thiopental and succinylcholine, followed by normocapnic ventilation and maintenance of blood pressure close to preoperative levels. Only if there are clinical indications that ICP is worsening would you consider hyperventilation, changing the blood pressure, or other maneuvers.

What will you do if you are unsuccessful in intubating him?

Your choices are to continue with apneic attempts at intubation with alternative airway devices or ventilation to preserve normocapnia.
Once you have successfully induced anesthesia and secured the airway, what anesthetic considerations do you have for the remainder of the case? Close communication with the surgeons will be necessary. Use of mannitol, placement of ventricular drains, and blood pressure management will be issues that will depend on the surgical findings. You should plan your anesthetic to avoid wide swings in blood pressure and you should have both pressors (e.g., phenylephrine) and pressure lowering drugs (beta blockers, nitroprusside, nicardipine) available. An arterial line is customary. Use of opioids to blunt surgical stress is prudent, but you should also plan for relatively rapid emergence to allow assessment of the patient’s neurologic status. (This may be modified if a decision to leave the patient intubated and sedated is made with the surgeons).

Suggested Further Reading


Chapter 19

Physiology and Anesthesia for Obstetrics

Stephen M. Howell and Mario Serafini

For maximum impact, it is recommended that the case study and questions found on page xxvi are reviewed before reading this chapter.

Key Learning Objectives

- Learn the physiologic changes associated with pregnancy
- Know the various methods of pain control available during labor
- Understand the anesthetic management of the obstetric patient

Obstetric challenges for the anesthesiologist include simultaneous care of the mother and fetus, dire emergencies, and complex disease. In the course of battling these challenges, physicians are immersed in their patient’s life-changing experiences. For this reason, obstetric anesthesia is considered by many to be one of the most rewarding anesthetic subspecialties.

Normal Physiologic Changes of Pregnancy

In order to provide safe and effective obstetric anesthesia, you must understand maternal physiology. Pregnancy represents a state of profound physiologic adaptation. Some adaptations become apparent in the first trimester and many persist well after delivery. Every organ system is affected. Table 19.1 summarizes some of the important changes.

Cardiovascular

During pregnancy, maternal oxygen requirements and metabolism steadily increase and the cardiovascular system must adapt to meet these increased demands.
Cardiac output escalates throughout pregnancy, due to increased stroke volume and elevated heart rate. Central venous and pulmonary artery occlusion pressures are unchanged. During labor, uterine contractions cause a cyclical increase in cardiac preload, further augmenting cardiac output. Systemic vascular resistance and mean arterial pressure decrease early in pregnancy and return to baseline at term.

In the supine position, the gravid uterus readily compresses the inferior vena cava. The aorta is affected to a lesser extent. This **aortocaval compression** impedes venous return and can lead to decreased cardiac output, hypotension, and decreased uterine perfusion. This syndrome, called the supine hypotensive syndrome, may occur as early as **twenty weeks gestation** and is exacerbated by conditions that increase uterine size – such as macrosomia (large fetus) and multiple gestation. The lateral decubitus, knee-chest, and left uterine displacement positions help to avoid the detrimental effects of aortocaval compression.

Some women may develop pregnancy-induced hypertension (systolic bp > 140 mm Hg or diastolic > 90 mm Hg), **pre-eclampsia** (hypertension + proteinuria + peripheral edema), or **eclampsia** (pre-eclampsia + seizures). The only effective therapy for pre-eclampsia is delivery of the fetus.

**Respiratory**

Tidal volume increases during pregnancy. Respiratory rate is also increased, but less profoundly. The increased minute ventilation leads to a **compensated respiratory alkalosis**, a fact that is especially important to remember when initiating mechanical ventilation.
A number of physiologic changes place the obstetric patient at increased risk for airway complications including failed endotracheal intubation and pulmonary aspiration. Increased oxygen consumption and decreased functional residual capacity (FRC) lead to rapid development of hypoxemia during periods of apnea. Parturients are at an increased risk for difficult and failed intubation because the airway becomes less favorable during pregnancy and even labor. At term, mucosal engorgement frequently afflicts the upper and lower airway, mandating gentle laryngoscopy, smaller endotracheal tubes, and avoidance of nasal airways. In the supine position, the enlarged breasts of pregnant females at term are upwardly displaced and may impede laryngoscopy. Laryngoscopes with short handles are more easily utilized in this setting.

Gastrointestinal anatomic and physiologic changes increase the risk of aspiration, demanding “full stomach” precautions in laboring women. If the parturient loses the ability to protect her airway (e.g., high spinal block, overzealous hypnotic administration), endotracheal intubation is advisable.

**Central Nervous System**

The parturient is more sensitive to both inhalational and local anesthetics, an effect that has been attributed to increased progesterone. Endogenous endorphins may also play a role in mediating this effect, especially during the peripartum period. The minimal alveolar concentration (MAC) for volatile anesthetics declines throughout pregnancy. Hormonally-mediated changes may also increase neuronal sensitivity to local anesthetic agents. In addition, the gravid uterus causes distention of epidural veins which is thought to decrease dose requirements for neuraxial blockade.

**Hematologic**

Total blood volume increases significantly (≈45%) during pregnancy. Dilutional anemia occurs because plasma volume increases more so than red cell mass. The blood loss associated with a typical vaginal delivery (500 cc) or cesarean section (1,000 cc) is usually well tolerated as a result of these changes. Other notable hematologic changes include leukocystosis, increased serum clotting factors, and an occasional mild decrease in platelet count. Parturients become relatively hypercoagulable, which is advantageous during acute obstetric blood loss. Unfortunately, the hypercoagulable state predisposes these patients to thromboembolic disease, deep venous thrombosis, and pulmonary emboli.
A small number of parturients (≈0.5%) may develop a worsening thrombocytopenia (i.e. low platelet count), liver dysfunction, hemolysis, and anemia – termed HELLP syndrome. This is a life-threatening obstetric complication which usually appears late in pregnancy or even after delivery. The treatment for HELLP is delivery of the fetus.

**Gastrointestinal**

The obstetric patient is at increased risk for aspiration of gastric contents because of:

- Impaired esophageal and intestinal motility
- Stomach conformation and position changes
- Decreased lower esophageal sphincter tone
- Delayed gastric emptying during labor

Prophylactic measures aimed at reducing the risk of aspiration pneumonitis are generally focused on modifying these risk factors. The most important prophylactic measure is the avoidance of solid food during labor. Other measures should be considered prior to surgery. Many routinely administer oral sodium citrate, a non-particulate antacid. Sodium citrate quickly buffers existing stomach acid, but at the expense of increasing gastric volume and possibly causing nausea. The buffering capacity of sodium citrate is time-limited, and it should therefore not be administered far in advance of surgery. H₂-receptor antagonists or proton-pump inhibitors can be used, but their beneficial effects are likely delayed. Metoclopramide increases gastric emptying and lower esophageal sphincter tone and is advocated by some practitioners. The possibility of extrapyramidal reactions is a major drawback to its routine use.

**Renal**

Renal blood flow and glomerular filtration rate increase markedly during pregnancy. As a result, the obstetric patient’s creatinine should be less than her non-pregnant value. Additionally, total body water increases by ≈30%. Increased glomerular permeability to proteins may lead to a mild proteinuria during pregnancy.

**Musculoskeletal**

As the gestation progresses, the lumbar spine becomes increasingly lordotic. Lordosis hampers the interlaminar approach for the lumbar spinals and epidurals. Although less feasible with advancing uterine size, good positioning
helps to offset the undesirable effects of lordosis. Ligaments tend to become more lax near term as the body prepares for vaginal delivery. Many operators have noted that the ligamentum flavum (see Chap. 13, Regional Anesthesia) has a more spongy texture at term when compared to the non-pregnant state.

**Utero-placental Blood Flow**

By the end of the third trimester, uterine blood flow may represent up to 12% of cardiac output. Perfusion of the uterus is adversely affected by decreased uterine arterial pressure (hypovolemia, aortic compression), increased uterine venous pressure (vena cava compression), and increased uterine vascular resistance (uterine contractions, severe preeclampsia). Derangement of these variables may adversely affect fetal oxygen delivery.

Exogenous vasoconstrictors can also adversely affect uterine perfusion. Animal data from several decades ago led many to avoid the use of $\alpha$-agonists (phenylephrine) because of supposed increases in uterine vascular resistance. However, more recent human studies have confirmed the safety of low dose $\alpha$-agonists, and many providers use either ephedrine or phenylephrine for treatment of maternal hypotension.

**Maternal Fetal Exchange**

Blood from the maternal uterine spiral arteries bathes fetal villi capillaries within the maternal intervillous spaces of the placenta. Since placental exchange occurs across a membrane, it is dependent on diffusion, bulk flow, and active mechanisms. Oxygen and carbon dioxide diffuse readily across the placenta. Unloading of maternal oxygen is facilitated by a rightward shift in the oxyhemoglobin dissociation curve. Fetal oxygen transfer is further bolstered by fetal hemoglobin’s high affinity for oxygen (leftward shift of the oxyhemoglobin dissociation curve compared to adult hemoglobin).

The maternal-to-fetal transfer of drugs is a complex topic that is beyond the scope of this text. In general, molecules that are small and lipophilic (e.g., most anesthetics) cross the placenta easily, while large, hydrophilic molecules that are protein-bound diffuse poorly (e.g., neuromuscular blocking drugs, insulin). Unfortunately, the situation is often more complicated. For example, local anesthetics may accumulate in the fetus through so-called ion-trapping. This occurs when local anesthetics (which are non-ionized weak bases) cross into the relatively acidic fetus and become ionized and “trapped”.
Intrapartum Fetal Evaluation

The goal of intrapartum fetal evaluation is to detect fetal hypoxia such that one can intervene (e.g., change positions, initiate tocolysis, or perform a cesarean section) before irreversible fetal harm occurs. Intrapartum fetal evaluation is an evolving, complex topic. Fetal heart rate, though nonspecific, may be a useful surrogate for fetal oxygen delivery. The baseline fetal heart rate (FHR) should be between 120 and 160 beats per minute (Fig. 19.1). Abnormalities may include:

- loss of variability – a nonspecific finding that sometimes indicates fetal distress
- tachycardia (FHR > 160 bpm) – often due to maternal fever or drugs
- bradycardia (FHR < 120 bpm) – ominous sign, may represent fetal hypoxia if severe and prolonged

Decelerations are a periodic slowing of FHR. Three principal deceleration patterns have been described according to their relationship to uterine contraction: early, late, and variable decelerations (Figs. 19.2–19.4).

Figure 19.1 Normal fetal heart rate pattern. The heart rate (140 beats/min) variability is normal. There are no periodic changes. (From Datta, S. Anesthetic and Obstetric Management of High-risk Pregnancy. Springer, 2004. Used with permission).
Increased vagal activity due to fetal head compression is believed to cause early decelerations. Early decelerations begin soon after uterine contraction, tend to have a uniform shape, and do not herald fetal hypoxia. Late decelerations...
represent uteroplacental insufficiency, that is, insufficient fetal oxygen delivery during uterine contraction. Variable decelerations are typically due to umbilical cord compression and have a variable relationship to uterine contraction.

**Neonatal Evaluation: The Apgar Score**

Once the fetus has been delivered, the Apgar Score (Table 19.2) can be used to evaluate its well-being. Named after Virginia Apgar (an anesthesiologist who developed the system in the 1950’s), the score is made up of five criteria each on a scale of 0–2. The five scores are then summed to provide a single total

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<tr>
<th>Appearance</th>
<th>completely blue</th>
<th>extremities blue</th>
<th>Pink</th>
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</thead>
<tbody>
<tr>
<td>Pulse</td>
<td>absent</td>
<td>&lt;100 bpm</td>
<td>&gt;100 bpm</td>
</tr>
<tr>
<td>Grimace</td>
<td>no response to stimulation</td>
<td>grimaces when stimulated</td>
<td>pulls away when stimulated</td>
</tr>
<tr>
<td>Activity</td>
<td>none</td>
<td>some flexion</td>
<td>moving actively</td>
</tr>
<tr>
<td>Respiration</td>
<td>none</td>
<td>weak</td>
<td>good</td>
</tr>
</tbody>
</table>

**Table 19.2 Apgar score.**
Apgar Score of the newborn. The score ranges from 0 to 10, with 7–10 generally considered normal.

**Anesthesia for Vaginal Delivery**

The coordinated uterine movements and cervical dilation cause significant discomfort commonly known as labor pain. Labor itself can be divided into three stages:

- the **first stage** of labor begins with contractions and ends with complete cervical dilatation
- the **second stage** of labor begins with full cervical dilation and ends when the fetus is delivered
- the **third stage** of labor begins with the delivery of the fetus and ends with delivery of the placenta

The majority of pain during the latent phase of labor is visceral in quality and uterine in origin. During the first stage of labor, pain is due to cervical dilatation and uterine contractions. The pain pathway involves visceral afferents that enter the spinal cord at T10-L1. As labor progresses to second stage, it is increasingly accompanied by somatic pain, which reaches the spinal cord via pudental afferents (S2–S4). Fig. 19.5 depicts pain pathways in the parturient.

Non-Pharmacologic Options for Labor Pain

The discomfort associated with vaginal delivery can be mitigated by a variety of techniques. Supraspinal modulation of pain may underlie the effectiveness of psycho-prophylactic techniques, such as the Lamaze technique of breathing and relaxation. Other non-pharmacologic pain management techniques include biofeedback, hypnosis, acupuncture, hydrotherapy, and massage.

Systemic Medications for Labor Pain

Systemic (intravenous) analgesia with opioids can cause undesirable fetal respiratory depression. That being said, opioids such as morphine, fentanyl, meperidine, hydromorphone, and remifentanil have been used, as well as mixed agonist-antagonist opioids (e.g., butorphanol, nalbuphine). Patient-controlled analgesia (PCA) has been employed utilizing some of the opioids mentioned above. Benzodiazepines (midazolam) have also been used for anxiolysis. The main disadvantage of systemic medications is that they can cause respiratory depression in the fetus and the mother.

Regional Anesthesia

Paracervical blockade controls pain during first stage of labor only, associated with cervical dilatation and uterine contractions. Unfortunately, the technique places the viable fetus at risk for bradycardia and death, and has been mostly abandoned. Though infrequently performed, a pudendal nerve block is safe and provides excellent relief for the somatic pain of second stage labor. Though far from ideal (see Table 19.3), neuraxial analgesia (lumbar epidural) is often the best pharmacotherapeutic solution to the discomfort of childbirth. Most consider lumbar epidural analgesia to be the gold standard for labor analgesia. It is effective for both first and second stages of labor.

Epidural Analgesia

Continuous lumbar epidural analgesia (see Chap. 13, Regional Anesthesia) is often employed for labor analgesia, with or without patient-controlled bolus dosing. Patient-controlled epidural analgesia has been shown to improve analgesia and decrease the number of provider interventions. The contemporary use of dilute local anesthetics solutions with small doses of epidural opioids provides effective analgesia with minimal motor block and low risk of opioid-related respiratory depression. Main side effects of the labor epidural include
hypotension, motor blockade, and a risk of intravascular or intrathecal local anesthetic injection.

Needle placement in the laboring parturient can be challenging due to ongoing discomfort and increased lordosis. When identifying the epidural space, one must be aware that increases in intraabdominal pressure can transmit to the epidural space. Unintentional dural puncture may result if the needle is advanced indiscriminately during a period of high intraabdominal pressure, as often occurs with uterine contractions. Identification of the epidural space is technically easier in the sitting position. This position may be difficult to achieve in some patients and can render external fetal monitoring difficult. The lateral decubitus position is more conducive to fetal heart rate monitoring and may be more comfortable for the patient.

Spinal Analgesia

When the anticipated duration of labor is short (e.g., grand-multiparous patient with advanced cervical dilation), single injection spinal analgesia may provide sufficient analgesia. Analgesia of a fixed duration can be achieved with intrathecal opioids (e.g., fentanyl, sufentanil, or morphine), with or without small doses of intrathecal local anesthetics (e.g., bupivacaine). Another advantage of spinal analgesia is that it has a faster onset in comparison to epidural analgesia.

Continuous spinal analgesia, planned or unplanned, is highly effective for labor analgesia. An intrathecal catheter permits rapid achievement of

<table>
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<tr>
<th>Attribute</th>
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<tbody>
<tr>
<td>Efficacious and reliable</td>
</tr>
<tr>
<td>Duration of action coincides with duration of labor</td>
</tr>
<tr>
<td>No contraindications</td>
</tr>
<tr>
<td>No side effects (pruritus, nausea, hypotension, urinary retention)</td>
</tr>
<tr>
<td>No complications (nerve injury, high block, epidural hematoma/abscess)</td>
</tr>
<tr>
<td>Produces sensory blockade without motor weakness</td>
</tr>
<tr>
<td>Does not interfere with or prolong labor</td>
</tr>
<tr>
<td>No increased risk of operative delivery</td>
</tr>
</tbody>
</table>

Table 19.3 Qualities of an ideal pharmacotherapeutic technique for labor analgesia.
surgical anesthesia, should it become necessary. Patients who cannot tolerate the hemodynamic effects of a sympathectomy (hypotension, bradycardia) caused by neuraxial local anesthetics or a high spinal may be good candidates for continuous spinal analgesia with opioids alone. Unfortunately, standard-sized catheters must be placed through large bore needles, leading to an unacceptable incidence of post-dural puncture headache (PDPH). Spinal microcatheters can be placed through small bore needles and are less likely to cause PDPH. Unfortunately, spinal microcatheters were associated with an unacceptably high rate of neurologic complications. Local anesthetic neurotoxicity, not microcatheters per se, may underlie the complications of spinal microcatheters.

**Combined Spinal-Epidural Analgesia**

Combined spinal-epidural analgesia has become increasingly popular for labor analgesia. When properly utilized, the technique appears to have a safety profile similar to continuous lumbar epidural analgesia. The principal advantage of the technique is rapid onset of analgesia due to intrathecal injection of opioid and/or local anesthetic.

To perform a combined spinal epidural block, the epidural space is identified with loss of resistance in the low lumbar region. Once the epidural needle is properly positioned in the epidural space, it is stabilized. A long, small-gauge, pencil-point spinal needle is inserted through the epidural needle until a “pop” is detected. CSF is identified, and the desired medications are administered intrathecally. Upon removal of the spinal needle, an epidural catheter is threaded into the epidural space.

**Common Myths Regarding Neuraxial Analgesia**

Over the years, many problems have been attributed to epidural analgesia for labor. Most accusations have been determined to be false. Historical differences in epidural management and difficulties in study design have hampered the battle.

Old data, now refuted, appeared to show that epidural analgesia impairs neonatal well-being and increases the risk of cesarean section. Backache and neuropathy are also frequently blamed on neuraxial blocks. It is well known that all obstetric patients are frequently afflicted by either condition, and obstetric trauma often results in trauma to the lumbosacral trunk. The neurologic exam
may help to distinguish obstetric trauma from block needle trauma. A deficit is more likely due to obstetric trauma if it corresponds to the distribution of a peripheral nerve, whereas a dermatomal distribution may be more likely the result of a neuraxial block.

Though often debated, it is not clear whether or not epidural analgesia prolongs labor. Epidural analgesia was historically avoided in early labor for fear of prolonging it. Fortunately for laboring women, this practice has been largely abandoned.

**Anesthesia for Cesarean Section**

Cesarean section is most commonly performed under regional anesthesia. Though rarely necessary, the operation can be performed under local anesthesia. When choosing the anesthetic, one must consider a number of factors, particularly indication for cesarean, case urgency, and maternal-fetal well-being. Common indications for cesarean section include fetal distress, risk of maternal hemorrhage, dystocia (abnormal labor), and impending maternal death. The qualities of an ideal anesthetic for cesarean section are listed in Table 19.4. Neuraxial anesthesia, though not ideal, usually represents the best option.

<table>
<thead>
<tr>
<th>Table 19.4 Qualities of an ideal anesthetic for cesarean section.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Efficacious and reliable</strong></td>
</tr>
<tr>
<td>Can be achieved instantaneously</td>
</tr>
<tr>
<td>Duration of action coincides with duration of surgery</td>
</tr>
<tr>
<td>Avoids aspiration of gastric contents</td>
</tr>
<tr>
<td>Avoids airway manipulation (negates the risk of difficult intubation)</td>
</tr>
<tr>
<td>Allows maternal participation in delivery</td>
</tr>
<tr>
<td>Conducive to family member presence in operating room</td>
</tr>
<tr>
<td>Does not interfere with neonatal well-being</td>
</tr>
<tr>
<td>Allows for stable hemodynamics</td>
</tr>
<tr>
<td>Does not interfere with hemostasis</td>
</tr>
<tr>
<td>No complications or unwanted side effects</td>
</tr>
<tr>
<td>No contraindications/No technical failures</td>
</tr>
<tr>
<td>Alleviates post-operative pain</td>
</tr>
</tbody>
</table>
Spinal Anesthesia

Single-shot spinal anesthesia produces rapid, reliable surgical anesthesia with a fairly predictable duration. Because peritoneal traction occurs, a T4 sensory level is considered ideal for most patients. Vagal afferents may explain the sensation of visceral discomfort even though the block appears to be “adequate.” Prior to surgical incision, the presence of surgical anesthesia must be verified with objective testing (e.g., pin-prick). Prolonged operations are often best managed with a catheter-based technique (e.g., combined-spinal epidural, continuous epidural, continuous spinal).

Intrathecal injection of small doses of lipophilic opioids (e.g., fentanyl) may help to alleviate some of the visceral discomforts of a cesarean section. Intrathecal morphine can provide good post-operative analgesia, though pruritus, nausea, and respiratory depression limit the enthusiasm of some practitioners for this technique.

Preemptive bolus administration of intravenous fluid may help reduce the hemodynamic consequences of spinal anesthesia. If hypotension occurs, it must be treated aggressively with intravenous fluid, ephedrine, or low dose phenylephrine. As always, attention must be given to proper left uterine displacement, because this aggravates hypotension.

Bradycardia will typically manifest when the block reaches a high thoracic level (T4). Bradycardia and hypotension unresponsive to initial resuscitative attempts must be promptly treated with epinephrine. Respiratory compromise may occur with a high spinal (level above T1).

Epidural Anesthesia

In contrast to spinal anesthesia, epidural anesthesia affords a more gradual onset of hemodynamic changes that may be preferable in some scenarios. Unfortunately, epidural anesthesia is less profound, frequently patchy or unilateral, requires high doses of local anesthetic, and takes more time to establish.

For safety and convenience, epidural anesthesia is usually established via intermittent bolus of an indwelling epidural catheter. With a lumbar epidural, 15–25 mL of local anesthetic (0.5% Bupivacaine, 0.5% Ropivacaine, 1.5–2% Lidocaine, 3% Chloroprocaine) is typically required to achieve surgical anesthesia. If patients are appropriately monitored, epidural morphine may be included for post-operative pain. Epinephrine is often added to epidural local anesthetics to decrease systemic absorption.
A test dose of local anesthetic and epinephrine helps exclude subarachnoid and intravenous administration and is appropriate prior to epidural dosing. Chloroprocaine has a quick onset and is rapidly metabolized by plasma esterases. As such, chloroprocaine offers some protection against systemic toxicity. It is an excellent choice when epidural anesthesia must be induced quickly, such as during fetal distress in a patient with an existing epidural catheter.

Existing labor epidurals that are symmetric and have been controlling labor pain well can be used for surgical anesthesia after bolus dosage with concentrated local anesthetic. Epidural anesthesia is less profound when compared to spinal anesthesia. As with all regional anesthetics, objective testing of block quality must precede surgical incision. Should epidural anesthesia become inadequate during the operation, intravenous supplementation may be helpful (e.g., intravenous opioids, ketamine). Protective airway reflexes must remain intact, however. If the patient requires more than light sedation, general anesthesia should be induced and the airway should be secured.

**General Anesthesia**

Parturients are considered at higher risk for failed endotracheal intubation and aspiration of gastric contents. As a result, general anesthesia is reserved for emergency cases or for those with contraindications to regional anesthesia. If a difficult airway is anticipated, regional anesthesia or awake fiberoptic intubation is usually appropriate.

Prior to induction of general anesthesia, one should administer a non-particulate antacid and consider the administration of metoclopramide and/or an H₂ receptor antagonist. In an effort to minimize fetal depression, general anesthesia is not induced until patient is draped and the obstetrician is prepared to operate.

Since parturients desaturate rapidly, the importance of preoxygenation cannot be overstated. As always, left uterine displacement must be maintained. After adequate preoxygenation, a rapid sequence intubation is performed, most commonly with thiopental or propofol and succinylcholine. Cricoid pressure is applied by an assistant until the endotracheal tube position is confirmed. Prior to delivery, anesthesia is typically maintained with a volatile anesthetic, with or without nitrous oxide. Some advocate the use of 100% oxygen prior to delivery, particularly in the setting of fetal distress. Oxytocin is routinely administered after fetal delivery to promote uterine contracture.
If uterine hemorrhage continues, second-line agents (e.g., methylergonovine or carboprost tromethamine) may be warranted. Regardless of the situation, it is wise to minimize volatile anesthetic concentration after delivery because higher volatile anesthetic concentrations promote uterine atony, leading to hemorrhage. At this stage in the operation, supplemental opioids and nitrous oxide help to achieve a reasonable depth of anesthesia. Relaxation is maintained with small doses of a non-depolarizing neuromuscular blocking drug if needed. It is wise to administer prophylactic antiemetics and empty the stomach with an orogastric tube prior to emergence. As with all patients who are considered to be a “full-stomach,” the trachea must remain intubated until the patient is awake and able to protect her airway.

**Obstetric Hemorrhagic Emergencies**

As stated earlier, the gravid uterus receives up to 12% of cardiac output at term. Not surprisingly, hemorrhage is a leading cause of obstetric morbidity and mortality.

**Antepartum/Intrapartum Hemorrhage**

Placenta previa, abruptio placentae, uterine rupture, and placenta accreta represent major causes of antepartum bleeding.

- **Placenta previa** exists when the placenta is located close to or is even covering the internal cervical os. When hemorrhage occurs secondary to placenta previa, it typically presents as painless vaginal bleeding.
- **Abruptio placentae** is an abnormal separation of the placenta from the uterine wall and may present differently depending on the location and degree of separation. Vaginal bleeding usually occurs with abruption placentae, though significant hemorrhage can be concealed within the uterus.
- **Uterine rupture** is the feared complication of vaginal birth after cesarean section (VBAC), but it also occurs in patients without obvious risk factors. Uterine rupture may present with hypotension, fetal distress, and continuous abdominal pain (e.g., continuous pain unrelieved by epidural analgesia). Significant hemorrhage may be concealed within the abdomen.
- **Placenta accreta** occurs when the placenta invades deeply within the uterine wall, and even if diagnosed antenatally, may place the patient at risk for cesarean hysterectomy.

When obstetric hemorrhage necessitates an emergent cesarean section, general anesthesia is usually most appropriate, because general anesthesia can be more
rapidly attained and regional anesthesia is contraindicated in the setting of hemorrhagic shock. Ketamine causes less hemodynamic depression compared with propofol or thiopental, and is a more useful intravenous induction agent in this setting. Large bore intravenous access, blood products, and fluid warming devices are obvious, life-saving necessities.

Post-Partum Hemorrhage
The most common causes of significant post-partum hemorrhage include uterine atony and retained placenta. Uterine massage and intravenous oxytocin help to prevent uterine atony post-partum. Manual uterine exploration is usually indicated in the setting of a retained placenta. General anesthesia or regional anesthesia may be appropriate, depending on the scenario. If hemorrhagic shock is present, general anesthesia is usually the safest option. Intravenous nitroglycerin and volatile anesthetics facilitate manual uterine exploration via muscular relaxation. Vaginal and cervical lacerations can occur during delivery and may rarely cause overt hemorrhage requiring operative intervention.

Anesthesia for Non-Obstetric Surgery
It is desirable to avoid non-obstetric surgery during pregnancy. Depending on the specific operation, surgical procedures can lead to miscarriage or preterm labor. A medication is generally considered “safe” during pregnancy when adequate, well-controlled studies fail to demonstrate a risk to the fetus. For obvious reasons, this level of evidence is not available for most medications.

Though many agents are believed to be safe, most anesthetics have not been studied to this degree in humans, and safety has only been demonstrated in animal models. As such, it is prudent to avoid unnecessary fetal exposure, especially during the period of organogenesis (1st trimester). Operations should be delayed until the second-trimester whenever feasible. Prior to the administration of any medication, one should weigh the benefit against the potential for fetal harm.

If surgery must be performed, regional anesthesia should be used when possible. It was originally thought that benzodiazepines and nitrous oxide might cause fetal anomalies. However, there is no human data that shows a single exposure to either drug to be unsafe. Yet many providers still choose to avoid benzodiazepines and nitrous oxide during pregnancy.
Whatever technique is chosen, fetal acidosis, hypoxemia, and decreased uteroplacental blood flow must be prevented. Maintenance of normal maternal oxygenation, ventilation, blood pressure, and cardiac output are critical. Depending on the gestational age, it may be useful to monitor the fetus during perioperative period.

Case Study

A 30-year-old otherwise healthy woman presents at 39 weeks gestation with elevated blood pressure for induction of labor. You are consulted when she is 4 cm dilated, contracting regularly, and requesting labor analgesia.

What other information will you seek during your preoperative interview? Besides routine information on comorbidities, NPO status, and obstetric and anesthetic history, you should learn more about the high blood pressure, which may be a sign of preeclampsia. If this diagnosis is suspected, it is prudent to check her laboratory studies, particularly the platelet count, before administering neuraxial analgesia. Her obstetric history is helpful in deciding if she is likely to deliver rapidly (for example, if she is multiparous, with ruptured membranes, and at 8 cm dilation) or more slowly (a nulliparous patient with intact membranes at 4 cm). It is also important to assess the fetal heart rate tracing (FHR) or consult with the obstetrician or obstetric nurse about the status of the baby. This information may guide your selection of analgesic technique.

Your preop shows that she is pregnant with her first child and has intact membranes. Her platelet count is $165 \times 10^9$/L. Other laboratory studies are negative. Her previous medical history is negative and her anesthetic history is unremarkable. Her blood pressure on admission was 150/90 and has remained stable. The FHR shows a reassuring pattern.

What is your anesthetic plan? This appears to be a healthy patient with mild pregnancy-induced hypertension. She is a candidate for epidural or combined spinal-epidural analgesia. Since her hypertension may be a risk factor for cesarean section, some anesthesiologists may prefer conventional epidural analgesia, in order to be certain that the catheter is functioning well (the CSE technique uses intrathecal opioids for the first 90 min or so, potentially masking an inadequate epidural catheter).

You select epidural analgesia.
Describe the technique and your initial choice of drugs.
The patient is positioned after applying standard monitors (pulse oximeter, blood pressure cuff, ECG) either sitting on the edge of the bed or lateral, with knees and hips flexed. The lower back is prepped and steriley draped and the L3-4 (or L4-5 or L2-3) interspace is infiltrated with 1% lidocaine. The epidural needle is inserted into ligament with a slight cephalad angulation and then advanced slowly while checking for resistance to injection of saline or air in a syringe attached to the needle. When a loss of resistance is encountered (typically 4–7 cm from the skin), the catheter is threaded through the needle 3–5 cm into the epidural space, using the marks on the catheter and needle as a guide to depth, and secured with a sterile dressing and tape. A test dose of local anesthetic (with or without 1:200,000 epinephrine) is injected and the patient is asked for signs of intravascular injection (lightheadedness, tinnitus, perioral numbness) or intrathecal injection (immediate onset of profound numbness in the lower extremities). If negative, a loading dose of local anesthetic, often bupivacaine 0.0625–0.125%, often mixed with 2 mcg/ml fentanyl, is injected in divided doses, periodically checking again for signs of intravascular injection.

How will you maintain analgesia once established?
Although there are numerous regimens, patient-controlled epidural analgesia (PCEA) is very popular. A background continuous infusion of 6 ml/h, a demand dose of 6 ml, and a lockout between demands of 15 min, is a typical protocol. The patient is instructed to push the demand button if pain ensues and to have the anesthesiologist paged if relief does not occur after one or two demand doses. Periodic checks of the patient’s comfort and vital signs, the pump, and the FHR should continue even if you are not called!

After 3 h, you are paged because the patient is experiencing discomfort in the perineal area. She has tried pushing the PCEA button. How would you respond?
Sacral pain and the urge to push often herald the beginning of the second stage of labor. Review of the most recent cervical exam with the obstetric nurse can help clarify the situation. A “top-up” dose of local anesthetic (5–10 ml of 0.125–0.25% bupivacaine and/or fentanyl (50–100 mcg), usually given with the back of the bed raised, is often effective.
The patient has reached full cervical dilation and begins pushing. Shortly thereafter, you are paged urgently because of decelerations noted on the FHR tracing.

What are your immediate steps?

First, assess the patient's block and vital signs. Sometimes, hypotension following an additional dose of local anesthetic may precipitate FHR changes. If the BP has declined, give ephedrine, 10 mg, or phenylephrine, 40 mcg, and increase the rate of fluid administration. Put an oxygen mask on the patient and ensure that she is not positioned flat on her back (to avoid aorto-caval compression by the gravid uterus).

Vital signs are normal and the patient is comfortable, but the FHR tracing does not improve. The obstetrician wishes to perform a cesarean section.

How do you extend the epidural block for the operation?

Depending on the urgency of the situation, you administer lidocaine 2% with epinephrine or chloroprocaine, 3%, 10–20 ml in divided doses. Chloroprocaine has a faster onset but shorter duration of action, requiring frequent redosing, and is useful in emergent situations. The speed of onset and depth of the block is augmented by addition of bicarbonate, 1 ml per 10 ml of anesthetic. The goal is to obtain a T4 level (numbness to the level of the nipples) within a few minutes. Vital signs should be monitored during administration, and fluids and ephedrine or phenylephrine are given for hypotension. Fentanyl 50–100 mcg is another useful adjunct to deepen a lidocaine block.

Suggested Further Reading


Physiology and Anesthesia for General and Bariatric Surgery

Rana Badr

For maximum impact, it is recommended that the case study and questions found on page xxvii are reviewed before reading this chapter.

Key Learning Objectives
- Learn the pathophysiology of obesity and endocrine disorders
- Understand the anesthetic considerations for bariatric surgery and common general surgical procedures
- Learn about physiologic considerations that occur during laparoscopy

Obesity

Obesity is a growing problem in the United States and around the world. Over one billion overweight or obese people exist in the world. Sixty-six percent of United States adults are overweight and 32% are obese. Over 300,000 US deaths a year are associated with obesity.

Obesity is the accumulation of extra fatty tissue in the body (more than 25% of body weight for men and more than 35% for women) and is classified based on the body mass index (BMI) as shown in Table 20.1. BMI is calculated as weight (in kg) divided by square of body height (in m²). Normal BMI is 20–25. BMI of 25–30 is considered overweight (class I obesity), BMI of 30–35 is considered obese (class II obesity), BMI of 35–40 is considered severely obese (class III obesity), and BMI over 40 is considered morbidly obese (class IV obesity).
BMI has its limitation and may not be an accurate way of assessing obesity in body builders.

There are two types of obesity: “central-android” type, which is more common in men and “peripheral-gynecoid” type more common in women. The former is also known as apple-shape obesity and the latter is known as pear-shape obesity. It is important to measure abdominal circumference in addition to BMI. Central obesity (waist measurement more than 40 in. for men and more than 35 in. for women) is associated with the respiratory and cardiac co-morbidities. Waist-to-hip ratio (WHR) >0.95 for men and >0.8 for women has been shown to confer higher risk of complications.

**Physiologic Changes Associated with Obesity**

**Cardiovascular System**

Obesity is an independent risk factor for cardiovascular disease. Since adipose tissue needs perfusion, total blood volume and stroke volume will increase to perfuse additional body fat. Cardiac output (C.O.) increases by 0.1 L/min for each 1 kg addition in body weight.

Gradual accumulation of fat between fibers of heart muscle may cause myocyte degeneration and cardiac dysfunction. Lipotoxicity of the myocardium by free fatty acids may also cause apoptosis of lipid-laden cardiomyocytes and contribute to cardiomyopathy. Increased C.O., left ventricular hypertrophy (LVH), and LV diastolic dysfunction all predispose to heart failure. Diabetes mellitus (DM), hypertension (HTN), and coronary artery disease (CAD) are other factors that predispose these people to congestive heart failure.

Increased C.O. with normal peripheral resistance causes hypertension. For every 10 kg increase in body weight, there is 3–4 mmHg increase in systolic pressure and 2 mmHg increase in diastolic pressure. This increase is more prominent with abdominal obesity. Peripheral vascular resistance may also increase due to different substances released from adipocytes and sympathetic

<table>
<thead>
<tr>
<th>Obesity class</th>
<th>BMI</th>
<th>Health risk</th>
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<tbody>
<tr>
<td>Class I (overweight)</td>
<td>25–30</td>
<td>Low</td>
</tr>
<tr>
<td>Class II (obese)</td>
<td>30–35</td>
<td>Moderate</td>
</tr>
<tr>
<td>Class III (severely obese)</td>
<td>35–40</td>
<td>High</td>
</tr>
<tr>
<td>Class IV (morbidly obese)</td>
<td>&gt;40</td>
<td>Very high</td>
</tr>
</tbody>
</table>

**Table 20.1 Classification of obesity.**
nervous system stimulation. Obese people with metabolic syndrome specially have higher risk of CAD. Left atrial (LA) dilation increases risk of atrial fibrillation (AF) in these patients. QT prolongation also occurs in 30% of obese patients and risk of arrhythmia and sudden death also is higher.

Despite high C.O., ventricular filling pressures increase, while the pumping function of leg and calf muscles decreases. Both of these factors contribute to higher risk of deep vein thrombosis (DVT) in obesity. Byproducts of adipose tissue may also cause pro-thrombotic or hypercoagulable state.

**Respiratory System**

Adipose tissue is metabolically active and O₂ consumption and CO₂ production will rise with obesity, as does the work of breathing. Chest wall compliance is decreased in obese people and expiratory reserve volume (ERV) and consequently functional residual capacity (FRC) is significantly reduced. FRC may fall below closing capacity and consequently during normal ventilation small airways may close. Total lung capacity (TLC) is also reduced. Supine position further decreases FRC and TLC. This often results in ventilation–perfusion mismatch. Decrease in FRC means quicker desaturation during periods of apnea and limited available time between induction of anesthesia and intubation. Postoperative atelectasis is more common in this group of patients due to decreased FRC and TLC. Obesity increases the work of breathing due to decrease in both chest wall compliance and decreased respiratory muscle strength. These may lead to dyspnea.

**Obstructive sleep apnea** (OSA) is more common in obese people and is characterized by frequent episodes of apnea and airway obstruction at night, snoring, fragmented sleep, and daytime sleepiness. It may be difficult to ventilate and intubate a patient with OSA. Repetitive sympathetic stimulation at night may be responsible for hypertension in these patients. About 70% of people with OSA are obese and 40% of obese people have OSA.

**Hypoventilation of obesity** (Pickwickian syndrome) is respiratory failure in markedly obese patient characterized by somnolence, daytime hypercapnia (PaCO₂ > 45), hypoxemia, polycythemia, pulmonary hypertension, and cardiac enlargement (cor pulmonale). Most of these patients also have OSA.

**Gastrointestinal System**

Fatty liver (fat accumulation in liver cells >10% of liver weight) is very common in obese patients. Fat accumulation in liver cells may cause inflammation
and wide spectrum of liver disease from simple fatty liver to cirrhosis. Abdomi-
nal obesity is also associated with higher risk of gastroesophageal acid reflux (GERD) and aspiration.

**Endocrine and Metabolic System**

Obesity is associated with hyperlipidemia, hypertension, insulin resistance, and pro-inflammatory and pro-thrombotic states. Extra adipose tissue releases several products including nonsteroidal fatty acids (NSFA), cytokines, plasminogen activator inhibitor (PAI)-1, interleukin-6, and adiponectin. These products are responsible for metabolic complications and are associated with higher risk of coronary artery disease. Treatment should be targeted toward weight reduc-
tion. Figure 20.1 depicts common systemic manifestations of obesity.

**Neurological and Psychological Problems**

Body image may be severely distorted in people with obesity, and obese people may be discriminated against in school and workplace. Depression is common and it is important to be sensitive to these issues. Carpal tunnel and other superficial nerve compression are also more common in obese people, and special attention is necessary during positioning these patients in the

![Diagram](image_url)

**Figure 20.1  Systemic manifestations of obesity. CO = cardiac output; CHF = congestive heart failure; DM = diabetes mellitus; CAD = coronary artery disease; HTN = hypertension.**
operating room to prevent nerve injuries. Also, higher risk of stroke has been recorded in this population.

**Airway Challenges in Obesity**
Excessive soft tissue in the larynx and pharynx, particularly in patients with OSA, should be expected. Increased neck circumference and high Mallampati score may be indicators of a difficult intubation. The incidence of a difficult intubation in obese patients is higher than in general population, although the BMI by itself is not a reliable predictor. These patients may need head and trunk elevation and larger blades for intubation. Even with good positioning, sometimes mask ventilation may be more challenging than intubation. Insertion of an oral airway and two-hand ventilation may improve ventilation.

**Surgery for Obesity**
Gastric banding and gastric bypass are commonly performed for treatment of severe and morbid obesity (see Fig. 20.2). The goal of surgery is gastric restriction and intentional malabsorption. These procedures are being increasingly performed laparoscopically. Procedures performed through a laparoscopy, compared to laparotomy, may result in earlier recovery, and help minimize postoperative problems associated with pain, reduce postoperative pulmonary complications, decrease postoperative infection, and prevent incisional hernias.

![Figure 20.2](image-url) **Figure 20.2** Weight loss procedures. (a) Vertical banded gastroplasty, (b) Laparoscopic adjustable gastric band, (c) Roux-en-Y gastric bypass. (Used with permission. From *Obesity and Diabetes*, Christos S. Mantzoros, Springer, 2006).
Anesthetic Considerations

Preoperative Evaluation
Airway evaluation should be performed before bringing the patient to the operating room, and a fiberoptic intubation (FOI) cart should be available and ready in the OR if difficult intubation is anticipated. Occasionally awake FOI may be necessary. Investigation for co-morbidities, including sleep apnea, hypoventilation of obesity, hypertension, coronary artery disease, and diabetes mellitus, is advised. Intravenous access can be a challenge in obese patients, and central access may therefore be necessary.

If the procedure is being performed through a laparotomy, epidural anesthesia may improve postoperative pain control and respiratory status. Evaluation of the lumbar and thoracic spine area and the feasibility of epidural anesthesia should be addressed in preoperative evaluation. Nerve blocks may also be considered for surgery on extremities.

Intraoperative Considerations
Availability of a bariatric operating table and availability of an appropriately sized bariatric hospital bed for the postoperative period should be addressed before bringing the patient to the operating room. It is also important to obtain an appropriately sized noninvasive blood pressure cuff for accurate measurement of blood pressure. If a cuff is too narrow blood pressure will be overestimated.

Obese patients should be preoxygenated with 100% oxygen for at least 3 min before induction with their head and shoulders optimally positioned prior to intubation. It may be advisable to confirm ability to mask ventilate before administering any muscle relaxant. Discussion about difficult intubation can be found in Chap. 9, Airway Evaluation and Management. A rapid sequence induction (RSI) should be considered in patients with gastrointestinal reflux symptoms.

Positioning difficulties related to patient's body habitus and risk of nerve injury should also be addressed with appropriate padding and positioning. Additional intravenous access, if necessary, should be established soon after induction and before the patient is prepped and draped. As a general rule, medications with weak or moderate lipophilicity can be dosed on the basis of lean body weight (LBW), while highly lipophilic medications with high volume of distribution are usually dosed based on total body weight (TBW). Fluid requirements are usually higher than expected. Sequential compression
devices, stockings, and subcutaneous heparin or low-molecular-weight heparin (if not contraindicated by surgery) should be used to prevent deep venous thromboses (DVTs).

**Postoperative Considerations**

Adequate pain management is important so that the patient can achieve deep breathing (helps prevent lung atelectasis) and early ambulation. Postoperative pain management in abdominal surgery may be achieved with either epidural anesthesia or patient-controlled analgesia (PCA). Long-acting narcotics should be used judiciously due to concern for respiratory depression in patients with OSA. Epidural analgesia utilizing local anesthetic and possibly an opioid may be a good alternative to intravenous analgesia. DVT prophylaxis should be continued until the patient is able to ambulate.

CPAP (continuous positive airway pressure) or BIPAP (Bi-level positive airway pressure) should be considered for postoperative care in patients with OSA. Initiation of CPAP therapy in the recovery room and continuation overnight for prevention of post operative atelectasis has been advocated. Sometimes selected patients are left intubated postoperatively, particularly those who had a difficult intubation.

**Anesthesia Considerations for General Abdominal Surgery**

**Preoperative Evaluation**

Any emergent surgery warrants full stomach precautions, and intraabdominal emergencies are associated with ileus and higher risk of aspiration – even if the patient had nothing to eat or drink for several hours. Signs and symptoms of ileus include nausea, vomiting, and abdominal distention. Some elective abdominal surgeries carry a higher risk of aspiration due to the nature of the disease, as in anti-gastrointestinal reflux surgery or surgery for achalasia. H2 blockers and sodium citrate are often administered before induction in patients at high risk for aspiration. However, metoclopramide is contraindicated in bowel obstruction.

Fluid loss into the gastrointestinal system or into interstitial tissue, in case of bowel obstruction or peritonitis, can be significant and may lead to severe dehydration. Signs and symptoms of dehydration include thirst, dry mucosa, tachycardia, hypotension, and decreased urine output. Fluid resuscitation should be started before induction of anesthesia to decrease chance of hemodynamic
compromise on induction. Blood loss in a patient with gastrointestinal (GI) bleeding may cause significant hypovolemia. Bleeding in the GI tract also increases the risk of aspiration.

Loss of different fluids from the GI system is associated with loss of various electrolytes. For example, loss of stomach secretions either through vomiting or gastric suction is usually associated with decreased H⁺ and Cl⁻ ions leading to hypokalemic, hypochloremic metabolic alkalosis. Elective colon surgery with a bowel prep also can cause electrolyte and fluid imbalance. Consider checking patient electrolytes and hematocrit prior to major abdominal surgeries.

Finally, a patient’s underlying disease should also be considered for each procedure. For example, splenectomy for sickle cell disease has different considerations than splenectomy for Idiopathic Thrombocytopenic Purpura (ITP).

Intraoperative Considerations

Laparoscopic Surgery

Laparoscopic surgery is frequently performed for esophageal fundoplication, Heller’s myotomy, cholecystectomy, hernia surgery, bariatric surgery, and some bowel surgeries. Prior to insufflations, a nasogastric or orogastric tube is placed to decompress stomach, and a Foley catheter to decompress the bladder.

The respiratory system can be affected in laparoscopic surgery by different mechanisms. Effects of pneumoperitoneum (insufflation of the peritoneum by CO₂) include intraabdominal pressure increase, systemic CO₂ absorption, increased end-tidal CO₂, cephalad displacement and impaired movement of the diaphragm, decreased FRC and pulmonary compliance, increased PIPs (peak inspiratory pressures), and ventilatory requirements. Retroperitoneal dissection of CO₂ may cause a pneumothorax. The effects of Trendelenberg or reverse-Trendelenberg positions needed during the procedure should also be considered. Airway pressures including plateau and peak airway pressure may also change.

Effects on cardiovascular system include increases in systemic vascular resistance due to increased sympathetic output from CO₂ absorption, and a neuroendocrine response to pneumoperitoneum. The cardiopulmonary effects of pneumoperitoneum are proportional to the magnitude of intra-abdominal pressure during laparoscopy with significant changes occurring at pressures greater than 12 mmHg. Decreased venous return and bradycardia (due to profound vasovagal reaction) may occur with pneumoperitoneum. Vascular injection of CO₂ can cause air embolism, hypotension,
dysrhythmias, and even cardiovascular collapse. Hemorrhage from vascular injury is another serious complication of laparoscopic surgery.

High intra-abdominal pressure may cause decreased urine output due to decreased blood flow to splanchnic and renal circulation. Hypothermia can occur due to dry gas insufflation, and prevention of hypothermia can be achieved with a fluid warmer, forced air warming devices, and by keeping the OR temperatures high. The use of nitrous oxide is contraindicated in bowel obstruction, because it may cause bowel distention, but otherwise it has been used in other laparoscopic cases.

Laparotomy

Laparotomy (open surgery) is usually performed electively for cancer surgery, solid organ surgeries, and emergency surgeries for trauma and peritonitis. Fluid loss can be significant, even without significant blood loss. Appropriate intravenous access is necessary. Since postoperative pain can interfere with breathing, epidural analgesia may be of benefit in major elective abdominal surgeries. The need for invasive monitoring (arterial, central venous lines) depends on the patient’s coexisting disease and anticipated blood loss. Laparoscopic-assisted mini-laparotomies are usually intended to combine laparoscopic techniques with smaller than usual laparotomy incision for solid organ surgery (e.g., kidney or spleen) to minimize postoperative pain and improve cosmetic appearance.

Postoperative Considerations

A high incidence of postoperative nausea and vomiting warrants the use of prophylactic antiemetics in abdominal surgeries. The use of multiple antiemetics with different mechanisms (“multimodal therapy”) is useful in high-risk patients (see Chap. 7).

In upper abdominal surgery, the possibility of a pneumothorax or hemothorax in the postoperative period should be considered if there is any respiratory compromise. Hemodynamic changes in the postoperative period may also occur due to intra-abdominal bleeding. Shoulder pain in laparoscopic procedures may occur due to phrenic nerve irritation from pneumoperitoneum. Complete evacuation of pneumoperitoneum at the end of procedure will help to decrease this complication. Intraperitoneal and incisional injection of local anesthetic has been used successfully in laparoscopic cases to improve pain control.
At some centers, low-dose ketamine has also been used before incision and during surgery to improve postoperative pain control.

Upper abdominal incisions are painful and are associated with atelectasis. Pain control can be achieved by either epidural anesthesia (for open laparotomy cases) or PCA. A patient’s coagulation status is usually checked before epidural catheter placement.

Advantages of epidural analgesia for abdominal procedures include better postoperative pain control, improved deep breathing and decreased risk of atelectasis, sympathetic blockade, faster resolution of ileus after colonic resection, and improved perfusion of intra-abdominal organs. Disadvantages of epidural analgesia include patient discomfort during catheter placement, incomplete block, catheter migration, small but potentially devastating risk of epidural bleeding and abscess formation, risk of dural puncture and postdural puncture headache. It also has non-procedure-related risks such as hypotension, motor blockade, CNS toxicity, urinary retention particularly after anorectal surgery, and pruritus if an opioid is used within epidural infusion.

The level of epidural catheter placement for pain management after abdominal surgery is either low thoracic or lumbar, depending on the incision site. For upper abdominal surgeries, a low thoracic or upper lumbar (T6–L1) catheter is appropriate, and for pelvic and lower abdominal surgeries mid- to low-lumbar (L2–L5) epidural catheter may provide better coverage (Table 20.2).

PCA (patient-controlled analgesia) as an analgesic option with morphine, hydromorphone, or fentanyl is easier to achieve, can provide pain medication upon patient’s demand, and does not involve a special procedure. However, PCA may prevent the patient from taking deep breaths, may delay ambulation, and can cause respiratory depression and somnolence.

<table>
<thead>
<tr>
<th>Type of surgery</th>
<th>Usual incision</th>
<th>Epidural catheter placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver, pancreas, spleen, stomach</td>
<td>Chevron or upper midline</td>
<td>Low thoracic</td>
</tr>
<tr>
<td>Kidney and ureter</td>
<td>Oblique flank</td>
<td>Upper lumbar</td>
</tr>
<tr>
<td>Colorectal</td>
<td>Low midline</td>
<td>Upper lumbar</td>
</tr>
<tr>
<td>Bladder, uterus surgery</td>
<td>Low transverse or low midline</td>
<td>Low lumbar</td>
</tr>
<tr>
<td>Hernia</td>
<td>Inguinal</td>
<td>Low lumbar</td>
</tr>
<tr>
<td>Hernorrhoid</td>
<td>Anorectal</td>
<td>Caudal</td>
</tr>
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</table>
Anesthetic Considerations for Common Abdominal Surgeries

**Esophageal Surgery**
Surgery for hiatal hernia, achalasia, and GERD is usually performed through a laparoscopic approach. In this group of patients, full stomach precautions and a rapid sequence induction should be utilized. Peripheral IV access and routine ASA monitoring is usually adequate. Patient-specific underlying disease should be considered when deciding on additional hemodynamic monitoring. The possibility of recurrent aspiration pneumonia and diminished pulmonary reserve should be considered in patients with severe gastric reflux.

**Stomach Surgery**
In ulcer surgery, the anesthesiologist needs to pay attention to the patient’s volume status if there is acute bleeding, and consider anemia in chronic bleeding. It is important to have adequate IV access, and blood products for a possible transfusion should be available.

**Small and Large Bowel Surgery**
Common surgeries involving small and large bowel include volvulus, intussusception, perforation, and tumor resection. Important considerations include full stomach precautions, effects of a bowel prep on electrolytes, and increased fluid requirements.

In cases of peritonitis and interstitial swelling, the risk of abdominal compartment syndrome with closing of the incision at the end of surgery should be considered. Increases in peak inspiratory pressure (PIP) with closing of the abdominal incision and hypotension are signs of abdominal compartment syndrome, and should be discussed with the surgical team. It may be necessary to leave incision open and perform a delayed closure of the incision. Patients with inflammatory bowel disease are usually on chronic steroids and often require stress dose steroids before induction. Malignancies may cause anemia from chronic blood loss and also increase the risk of coagulopathy.

**Hemorrhoid Surgery**
Hemorrhoidectomy can be performed in lithotomy, prone, or jackknife positions. General anesthesia and spinal anesthesia are both appropriate. Pressure on the peroneal nerve in the lithotomy position can result in foot drop and attention to appropriate padding is important. Patients in the prone
or jackknife position require chest support to optimize ventilation and venous return. Care must be taken to position extremities and genitals, and avoid pressure on eyes and ears.

**Liver and Biliary Tract Surgery**

Liver surgeries include tumors (primary or metastatic) and bile duct surgeries. Major liver surgeries are usually performed through laparotomy. Liver tumors with vascular involvement may result in major bleeding intraoperatively. Appropriate IV access, monitoring, a rapid volume infusion device, and blood product availability should be considered. If the extent of surgery is not known at the beginning, it may be advisable to establish invasive monitoring (arterial line and CVP) before the start of surgery. Keeping the central venous pressure (CVP) low (between 2 and 5 mmHg) may limit the distention of hepatic veins and sinusoids and reduce blood loss during liver surgery. The liver produces all coagulation factors except factor VIII and coagulopathy may be seen with hepatic insufficiency. Many patients presenting for liver surgery may not be good candidates for epidural catheter placement due to coagulopathy or thrombocytopenia.

Biliary tract surgeries range from simple laparoscopic cholecystectomy to complicated biliary surgeries in extremely ill patients with bile duct tumors. Anesthesia plans should be individualized based on severity of disease and extent of surgery. Gallbladder surgery is usually performed via laparoscopic approach with standard monitors – often on an elective outpatient basis.

**Spleen Surgery**

Elective splenectomy is either performed for hematological disease and thrombocytopenia or for staging of malignancy. Emergent splenectomy is reserved for trauma, ruptured splenic aneurysm, and uncontrollable bleeding. Understanding of the underlying reason for splenectomy is very important for anesthesia care.

In a patient with sickle cell disease, blood transfusion before surgery may be necessary to prevent sickling of red blood cells. For a patient with ITP, platelet transfusion is usually delayed until the spleen is removed. If splenectomy is performed for staging of Hodgkin’s disease, history of medications used for chemotherapy is important, as certain chemotherapeutic drugs can affect kidney, lung, and heart function. Emergency splenectomy usually warrants good intravenous access and availability of blood products.
Pancreatic Surgery
Pancreatic surgery is usually performed for pancreatitis, pancreatic cysts, or tumors. Patients with pancreatitis may have respiratory compromise and sepsis. Severe dehydration and electrolyte imbalance, especially hypocalcaemia, is also common. The need for invasive monitors should be individualized.

Hernia Surgery
Increased intra-abdominal pressure from COPD and chronic cough, bladder outlet obstruction (BPH) or ascites may be some of the predisposing factors for hernias and should be addressed before repair to prevent recurrence. Common hernia types include inguinal, umbilical, and incisional. General, regional or local anesthesia may be used for uncomplicated cases and is usually individualized based on underlying disease, hernia size and location, and patient’s and surgeon’s preferences.

Case Study
A 38-year-old female is scheduled for laparoscopic Roux-en-Y gastric bypass. She is 5 feet, 6 in. tall and weighs 300 pounds. She has tried various diet and exercise plans to lose weight without success. She has hypertension treated with an ACE inhibitor. She wheezes on exertion or in hot weather and uses an albuterol inhaler as needed. She snores loudly while sleeping but has not had a formal sleep study and is not interested in CPAP at home due to a poor experience related by a friend. She does not exercise regularly but she is able to walk on level ground for a few minutes at a time in her work as an office postal worker. She has been told she has “borderline diabetes” but is not currently taking any medication for it. Preoperatively, her examination shows BP 180/95, HR 90, RR 24, scattered end expiratory wheezes, which clear with cough, airway Mallampati class II, thyromental distance 4 fingerbreadths.

How severe is her obesity? Does it matter? Can any other obesity measures help you characterize her health risk further? Her BMI is 48.4, putting her in the morbidly obese category. Although risk is not linearly related to BMI, risk is higher for more obese individuals.
The pattern of obesity, however, may be even more important than the absolute magnitude of her obesity. You could ask her waist size, and if >35 it would correlate with higher risk. Other obesity-related risk factors for perioperative morbidity include her relative inactivity and glucose intolerance. Interestingly, sleep apnea per se probably is not such a risk factor but it has significant anesthetic implications regarding difficulty with intra- and postoperative airway management.

What concerns do you have about her respiratory status? How will these impact your anesthetic plan?
You should be quite concerned. First, she may desaturate with positioning, even before sedative drugs are given, due to lower FRC relative to closing capacity, leading to ventilation–perfusion mismatching. This may worsen with induction of anesthesia, due to limited apneic reserve of oxygen in the lowered FRC. Second, her questionable history of obstructive sleep apnea (snoring) are concerns for possible difficult mask ventilation. Third, you may be concerned that she could have a possibly difficult intubation despite the reassuring airway exam, due to obesity itself. Fourth, she has a history of wheezing, implying she may have reactive airways and thus prone to intraoperative bronchospasm. Finally, though controversial, some consider morbid obesity to be a risk factor for aspiration of gastric contents during induction. In response, you will position her slightly head-up with blankets under her shoulders or a specialized pillow such as the Troop elevation pillow. You will carefully preoxygenate to ensure the longest possible time for intubation. You will have a selection of adjunctive devices available to assist with possibly difficult mask ventilation as well as alternative intubation devices such as a video laryngoscope, which may shorten the time to intubation in obese individuals. Finally, you should have help immediately available should ventilation or intubation prove to be challenging.

How will you monitor her during the anesthetic? Will your plan differ from a normally proportioned patient having laparoscopic surgery?
All ASA standard monitors should be used and will not differ markedly from those used in a normally proportioned patient. The BP cuff must be of appropriate size or you will overestimate blood pressure. An alternative is to place a cuff on the forearm, or consider an arterial line if noninvasive pressure monitoring proves too technically difficult. Depending on your
anesthetic plan, you may choose to use a consciousness monitor such as BIS, particularly if you choose to use TIVA during any part of the case. Temperature monitoring availability is an ASA standard, and morbidly obese patients generally do not lose heat as quickly as thin patients in the OR. However, a large portion of the body will be exposed and the insufflating gas is relatively cool, so she may become hypothermic. Since this is a risk factor for wound infection, you should monitor temperature continuously.

How will you induce and maintain anesthesia?
Although any combination of general anesthetics are possible, you may consider short acting, nonlipophilic drugs to avoid excessive somnolence and respiratory problems at the end of the case. You may choose to avoid nitrous oxide to maximize oxygen delivery, but, conversely, it is rapidly eliminated and thus may facilitate a rapid wakeup. You will have to weigh its use against other adverse effects such as bowel distention in laparoscopic surgery. Some anesthesiologists have advocated TIVA at least at the end of the procedure to allow you to fully wash out inhalation anesthetics. Dexmedetomidine and remifentanil can provide excellent analgesia and sedation with minimal postoperative respiratory depression and is one attractive option. You should avoid large doses of long-acting opioids until her respiratory status can be assessed postoperatively. You will fully reverse neuromuscular blockade prior to emergence to avoid hypoventilation due to even subtle weakness.

How will you manage postoperative pain? Would your plan differ if the procedure were an open Roux-en-Y?
It is important to have good pain control but not oversedate the patient. Pain control is important to avoid splinting and hypoventilation that can cause atelectasis and hypoxemia. Patient-controlled analgesia has been successfully used following bariatric surgery. Some advocate increased vigilance for hypoventilation such as continuous pulse oximetry or frequent respiratory rate monitoring. The surgeon can also infiltrate the laparoscopy incisions with long-acting local anesthetic such as bupivacaine with epinephrine to augment the analgesia. If the procedure were an open laparotomy, placement of a thoracic epidural for postoperative pain control should be strongly considered. This technique allows minimization of systemic opioids and may improve pulmonary outcomes.
Suggested Further Reading


Chapter 21

Anesthesia for Urological Surgery

Payal Kohli

For maximum impact, it is recommended that the case study and questions found on page xxviii are reviewed before reading this chapter.

Key Learning Objectives

- Learn the pertinent urinary system anatomy and physiology
- Understand anesthetic management of common urologic procedures
- Discuss common complications associated with urologic surgery

Anesthesia for urological surgery poses a special challenge for anesthesiologists since patients are often elderly and may have multiple co-morbidities, including renal dysfunction. The scope of the field is broad and ranges from outpatient cystoscopies to major oncological surgeries, so the type of anesthesia needed is variable.

Anatomy

It is critical for the anesthesiologist to be familiar with the anatomy of the genitourinary system in order to understand the technical aspects of the procedure. The kidneys are located retroperitoneally, between T12 and L4, surrounded by perirenal fat and contained within Gerota’s fascia. On gross examination, there is an outer cortex and an inner medulla, which contains calices that drain into the renal pelvis, and eventually taper into the ureter. The ureters run along the psoas muscles and cross the common iliac prior to entering the bladder. Innervation of the upper ureters is carried by sympathetic fibers that enter the cord at T10–L2 and innervation of the lower ureters is by parasympathetics at S2–S4.
This innervation is important when one is administering anesthesia for stone extractions. The bladder holds 400–500 cc of fluid and receives its innervation from the hypogastric plexus (T11–12, S2–4) (Table 21.1).

The blood supply to the kidneys is via a single renal artery, which originates inferior to the SMA. There are, however, many normal anatomical variants in which multiple renal arteries are possible.

### Patient Positioning

There are multiple patient positions utilized in urological surgery and the anesthesiologist must be aware that there are physiological changes that accompany these positions.

The **lithotomy position** (Fig. 21.1) is most commonly used for cystoscopies, transurethral resection of prostate or bladder tumor (TURP or TURBT), or ureteroscopies. Placement in this position for greater than two hours may be a risk factor for development of sensory neuropathies or rhabdomyolysis secondary to compartment syndrome. This position increases upward displacement of intra-abdominal contents, decreasing pulmonary compliance, forced residual capacity and vital capacity, and increasing atelectasis. Elevating the legs also increases venous return, cardiac output, and arterial blood pressure, but these changes may not have clinically significant manifestations.

Placing the patient in the kidney rest position (also called the lateral flexed position) is preferred for better access during renal surgery. Often an axillary roll (usually a rolled towel) is placed between the table and upper chest to ensure that the brachial plexus is free from compression or injury. The lateral decubitus position has profound effects on creating ventilation–perfusion mismatch and causes dependent atelectasis. Hemodynamically,
there is a decrease in systemic arterial pressure, cardiac output, and renal perfusion pressures.

**Preoperative Assessment**

A thorough preoperative assessment is critical in patients undergoing urological surgery and includes all standard preoperative questions including screen for smoking history, medications, cardiac history, and renal function. Lab abnormalities reflective of renal failure include presence of hematuria or proteinuria on urinalysis, elevation in blood urea nitrogen (BUN) and creatinine values, and impaired creatinine clearance. If the patient is found to be in renal failure, the anesthesiologist must discern whether the renal failure is acute or chronic, and determine the etiology: prerenal, intrinsic renal, or postrenal/obstructive.

During surgery, it is critical for the anesthesiologist to avoid nephrotoxic drugs, correct hypovolemia, dose drugs based on renal function, and monitor for causes of urinary outflow tract obstruction. The adult kidney demonstrates
autoregulation, maintaining relatively constant rates of renal blood flow (RBF) and glomerular filtration rate (GFR) over a wide variety of mean arterial blood pressures. Anesthesia can result in decreases in RBF and GFR despite normal blood pressure, and decreases in blood pressure as a result of depression of myocardial activity and sympathetic tone.

### Anesthetic Management

**Cystoscopy/Ureteroscopy/TURBT**

These procedures consist of inserting an endoscope to visualize and intervene upon the lower urinary tract. Indications are varied, and include evaluation of hematuria, need for biopsies, extraction of stones, treatment of strictures, excision of bladder tumors (TURBT), and placement of ureteral stents to relieve obstruction. The patient is usually placed in the lithotomy position and irrigating solution is necessary to optimize visualization and remove surgical debris from the field. Procedures tend to be brief, usually under 1 h, and there is minimal need for postoperative analgesia so short-acting opioids are adequate for pain control.

Anesthesia for these procedures can be highly variable and can range from local anesthesia with monitored anesthesia care/sedation to general anesthesia with an LMA. With the advent of the flexible endoscope, general anesthesia is no longer required for patient comfort for these surgeries except in the case of dilatation of the ureter, which is more stimulating. Occasionally, the surgeon will request muscle relaxation for surgery when working in close proximity to the obturator nerve. In these cases, an endotracheal tube is necessary to secure the airway. If a spinal or an epidural is used, surgery on the lower genitourinary tract mandates a T10 level or higher. These procedures are often outpatient surgical procedures, with discharge home a few hours following surgery. For this reason, general anesthesia is usually preferred to regional. However, a short-acting spinal anesthetic may be appropriate. Disadvantages of regional techniques include awaiting return of urination postoperatively and more dilation of venous sinuses causing a slightly increased risk of TURP syndrome (see Complications of Urologic Surgery below).

**TURP**

Transurethral resection of the prostate (TURP) is commonly done for benign prostatic hypertrophy, which can cause compression of the lower urethra and result in obstructive urinary symptoms. A cystoscope is inserted into the urethra and a resectoscope, which can coagulate and cut tissue, is inserted through the cystoscope
to resect all tissue protruding from the prostatic urethra. This procedure requires continuous irrigation fluid as well, placing the patient at risk for TURP syndrome (see “Complications of Urologic Surgery, pages 348-9).

The patient is positioned in lithotomy and regional or general anesthesia can be used. If general anesthesia is used, muscle relaxation may be indicated or a deep level of anesthesia may be preferred. This will prevent coughing or movement, which may lead to prostatic capsule rupture. Advantages of general anesthesia include positive pressure ventilation, which can decrease the absorption of irrigant solution by increasing venous pressures. Regional anesthesia mandates a T10 level and offers the advantage of an atonic bladder along with the presence of awake patients, in whom TURP syndrome may be detected earlier.

**Laser Surgery in Urology**

Laser surgery in urology allows for treatment of condyloma acuminatum, interstitial cystitis, BPH, ureteral or bladder stricture, contracture or calculi, and superficial carcinoma of the urinary tract or external genitalia. Laser surgery allows for minimal blood loss and postoperative pain. The types of lasers include carbon dioxide, argon, and pulsed-dye lasers. Concern for **ocular injury by lasers** is paramount for the anesthesiologist during these procedures and **eye protection must be worn** by all OR personnel and the patient. Thermal injury by lasers may also be possible and can be avoided by limiting use to one operator and placing the device in standby to allow for cooling between uses. Inhalation of viral particles and smoke can also pose a safety threat; special laser masks that prevent small particles should be worn and the OR should be equipped with a smoke evacuation system.

**Radical Prostatectomy: Open, Laparoscopic, Robotic**

Open radical prostatectomy involves the complete resection of the entire prostate gland, the seminal vesicles, the ejaculatory ducts, and a portion of the bladder neck and is usually performed for prostate cancer. A pelvic lymph node dissection may also be done to aid in cancer staging. The patient is placed in a hyperextended supine position and a midline lower abdominal incision is used. Either general endotracheal anesthesia or regional anesthesia with a T6-8 level may be used for this surgery.

Once the prostatic urethra has been removed and the urethra is reconstructed, diagnostic dyes (methylene blue or indigo carmine) may be requested by the surgeon. A **methylene blue bolus** may lead to hypotension or cause disruption of the pulse oximeter readings; **Indigo carmine** may cause hypertension via $\alpha$-agonist effects. Complications of this surgery can include large
amounts of blood loss, fluid shifts leading to coagulopathy or anemia, and air embolism from Trendelenburg positioning. Large bore IV access is needed and an arterial line or central venous catheter may also be used since urine output will not reliably reflect intravascular fluid status.

**Laparoscopic surgery** or **robotic assisted surgery** is also becoming increasingly popular because of decreased invasiveness. However, retroperitoneal insufflation has been reported in some studies to be associated with increased systemic absorption of carbon dioxide and decreases in urine output, leading to iatrogenic excessive fluid repletion.

**Radical Cystectomy**
Radical cystectomy is indicated in patients with muscle invasive bladder cancer. Other less common indications include neurogenic bladder, chronic urinary obstruction, or pelvic malignancy. In men, the bladder, prostate, seminal vesicles, and urethra is removed. In women, the bladder, urethra, anterior vaginal wall, uterus, and bilateral ovaries and fallopian tubes are removed. A urinary diversion, either to the colon or ileum, is created at the end of the procedure.

Anesthetic considerations and patient positioning are similar to a radical prostatectomy. Bowel surgery introduces additional complications, including longer operative time and increased risk of bacteremia. In addition, in cancer patients, the anesthesiologist must consider effects of previously administered chemotherapeutic agents: doxorubicin has cardiotoxic effects, methotrexate has hepatic toxicity, cisplatin and methotrexate have neurotoxicity and renal toxicity.

**Nephrectomy: Open or Laparoscopic**
Removal of the kidney, fascia, adrenal gland and upper ureter, or a radical nephrectomy, is usually performed for malignancy/neoplasm, transplantation, cystic disease, or severe calculous disease. In about 5% of patients, the tumor extends into the vena cava, which can result in several complications. If the IVC is fully or partially occluded, there may be a decrease in venous return. The IVC may have to be temporarily clamped during resection, potentially requiring vasopressor support. Rarely, cardiopulmonary bypass may be indicated if there is extensive IVC infiltration.

The patient is typically positioned in the kidney rest position for the retroperitoneal approach. This position can cause caval compression and
the patient must be adequately hydrated preoperatively to prevent hypotension. The supine position can also be used if a transabdominal approach is needed. A combined epidural-general anesthetic is often used, and the anesthesiologist must be prepared for large fluid shifts and the potential for large volume blood loss. Laparoscopic nephrectomy is generally done for organ harvest or small tumors (partial nephrectomy) and consists of retroperitoneal insufflation.

**Renal Transplantation**

Recipients of donor organs tend to be patients with end-stage renal disease and a variety of comorbidities including diabetes mellitus, hypertension, coronary artery disease, or autoimmune disease. Such patients have many physiological perturbations such as anemia, coagulopathy, uremia, and electrolyte disturbances. IV access can be difficult and limited, secondary to presence of fistulas or shunts used for hemodialysis. Anesthetic medications must be dosed based upon renal clearance. General anesthesia is usually preferred because of pre-existing coagulopathy, although certain nephrotoxic medications and medications such as succinylcholine may need to be avoided. Maintaining a normal blood pressure is important to preserve renal perfusion, and vasoactive agents, such as dopamine, may be indicated to enhance renal blood flow. The recipient is usually positioned supine for the surgery and the native organs are often left in place. Postoperative pain can be significant, but intravenous opioids used in small doses are preferred to regional techniques.

**Orchiectomy, Orchidopexy, Penile Surgery**

Radical orchiectomy is usually performed for testicular cancer. Most of these patients tend to be young and healthy but may have received preoperative chemotherapy, placing them at risk for chemotherapy-induced systemic toxicity. Bleomycin is a commonly used chemotherapeutic agent for testicular cancer and is associated with pulmonary toxicity. In patients who have received bleomycin, colloid fluid replacement may be associated with less pulmonary complications than crystalloid and lower inspired oxygen concentrations may be beneficial. The patient is positioned supine and either general anesthesia or regional anesthesia is an acceptable option for this procedure. A retroperitoneal lymph node dissection may also be performed and during left-sided dissection, the intercostal arteries may be compromised, leading to loss of blood flow through the artery of Adamkiewicz and resultant spinal cord ischemia.
Other surgery involving the testis and external genitalia can be performed with a variety of techniques, ranging from monitored anesthesia care to general anesthesia with an LMA, depending on the extent of the surgery.

**Extracorporeal Shock Wave Lithotripsy**

ESWL is a minimally invasive technique used for the treatment of renal calculi and ureteral stones. It consists of a lithotripter, which transmits acoustic waves that are reflected and generate internal echoes that create stress to fracture kidney stones. Dysrythmias from incorrect timing of the shock wave (during cardiac repolarization) can be minimized by triggering the lithotripter to send a shock wave 20 ms after the R wave, when the heart is refractory.

The patient is positioned either supine or prone, depending on the location of the stone. For anesthesia, sedation with an ultra-short acting opioid (e.g., remifentanil) is usually adequate for patients since postoperative pain is minimal. IV hydration is recommended and diuretics may be useful in flushing the stone from the collecting system. Postoperatively, nausea, and bradycardia may be seen from excess vagal tone, hematuria may be present, and a subcapsular renal hematoma may occur in patients with hypertension. Patients who are pregnant, at risk for bleeding or have an active infection should not undergo ESWL.

**Complications of Urologic Surgery**

There are many complications unique to urological surgery. **Bladder perforation** during cystoscopy can occur by inadvertent stimulation of the obturator nerve leading to violent thigh muscle contraction or high irrigation pressures. The awake patient would complain of lower abdominal pain and nausea, whereas one might see hemodynamic instability under general anesthesia. The pain can localize to the suprapubic, inguinal, peri-umbilical, or upper abdominal regions, or refer from the diaphragm to the shoulder.

Another rare but serious complication of cystoscopy is **autonomic hyperreflexia**, which usually presents as a hypertensive emergency in spinal cord injury patients with an existing level of injury at T6 or higher. Other signs, such as headache, chest tightness, flushing, and sweating, can also be seen. Treatment is limited to short acting β-blockers or other intravenous agents that can achieve rapid blood pressure control.

The bladder needs to be distended by irrigation fluid to optimize visualization during cystoscopies and TURP procedure. There are a number of choices of irrigating solutions currently used in practice, each with advantages
and disadvantages (Table 21.2). Ideally, one would prefer an isotonic fluid that does not cause hemolysis when intravascularly absorbed, is transparent, non-electrolytic, inexpensive and nontoxic. Since this is not possible, a number of other solutions have been employed and it is critical that the anesthesiologist be aware of the type of solution being used and its associated potential perioperative complications.

**TURP syndrome** is a phenomenon that can be caused by intravascular absorption of irrigation fluid into the venous sinuses of the distended bladder when the pressure of the irrigating fluid exceeds venous pressure. The TURP syndrome is defined as a constellation of signs and symptoms that reflect rapid absorption of irrigating solution, leading to respiratory distress from volume overload, dilution of serum electrolytes and proteins, and resultant cardiopulmonary changes (Table 21.3). Central nervous system manifestations in the awake patient include nausea, agitation, confusion, visual changes, seizures, and even coma. These effects are most likely secondary to hyponatremia leading

### Table 21.2 Commonly used irrigating solutions.

<table>
<thead>
<tr>
<th>Irrigating solution</th>
<th>Relative osmolality</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distilled water</td>
<td>Very hypoosmolar</td>
<td>↑Visibility</td>
<td>Hemolysis, hemoglobinemia, hemoglobinuria, hyponatremia</td>
</tr>
<tr>
<td>Glycine</td>
<td>Hypoosmolar</td>
<td>↓TUPR syndrome incidence</td>
<td>Transient postoperative visual syndrome</td>
</tr>
<tr>
<td>Sorbitol</td>
<td>Hypoosmolar</td>
<td>↓TUPR syndrome incidence</td>
<td>Hyperglycemia, Osmotic diuresis</td>
</tr>
<tr>
<td>Mannitol</td>
<td>Isosmolar</td>
<td>Not metabolized</td>
<td>Osmotic diuresis, may cause intravascular volume expansion</td>
</tr>
</tbody>
</table>

### Table 21.3 Symptoms of TURP Syndrome.

<table>
<thead>
<tr>
<th>Cardiovascular</th>
<th>Neurologic</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>Confusion/disorientation</td>
<td>Hemolysis</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>Seizures</td>
<td>Hyponatremia</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Unresponsive</td>
<td>Hyperglycemia</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>Visual problems or blindness</td>
<td>Hyperammonemia</td>
</tr>
<tr>
<td>Hypoxemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial ischemia</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
to cerebral edema and hyperglycemia causing hyperammonemia (ammonia is a metabolite of glycine). In the anesthetized patient, the anesthesiologist may observe hypertension, bradycardia, dysrhythmias, desaturation secondary to pulmonary edema, and delayed emergence. Coagulopathy can also develop from dilutional thrombocytopenia or disseminated intravascular coagulation.

To treat TURP syndrome, one must begin with the ABCs (Airway, Breathing, Circulation). Once oxygenation and circulatory support have been established, serum electrolytes, arterial blood gases, and electrocardiogram must be checked and fluid restriction with diuresis (usually with furosemide, a potent loop diuretic) must be initiated. If the serum sodium concentration is <120 mmol/L, hypertonic saline can be used but the sodium deficit must be corrected slowly, in order to prevent the development of central nervous system demyelinating conditions. If there is a coagulopathy present, the treatment is supportive and consists of plasma and platelet transfusions to replace factor deficiencies.

**Bacteremia** may also be seen following TURP, given the high-pressure irrigation and because many of these patients have an indwelling foley catheter. Prophylactic antibiotics are usually given prior to the start of the procedure and continued for 2–3 days after the catheter is removed. **Hypothermia** can also be seen in elderly patients who have received large volumes of cool irrigating fluid and have impaired thermoregulatory mechanisms.

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**Case Study**

A 68-year-old man has symptoms of benign prostatic hypertrophy and is to undergo transurethral resection of the prostate (TURP). He has hypertension and hyperlipidemia and takes an ACE inhibitor and atorvastatin (Lipitor). He is physically active and has no symptoms of angina or heart failure.

**What else will you investigate in the preoperative assessment?**

In addition to the usual systems review for any anesthesiology preoperative assessment, you should make certain there are no contraindications to regional anesthesia (anticoagulation, spine abnormalities) and whether there are signs of renal dysfunction. The former may influence the choice of anesthetic technique, and the latter may influence the choice of drugs employed.
Will you recommend regional or general anesthesia? What are the relative merits of each?
Both anesthetics are commonly used and patient preference should be at least one important factor. Spinal anesthesia allows CNS monitoring for signs of the TURP syndrome, may relax the bladder efficiently, and may be associated with less blood loss. Conversely, general anesthesia with positive pressure ventilation increases venous pressure and reduces absorption of irrigation fluid, potentially decreasing the risk of TURP syndrome. In practice, no important differences in outcomes have been demonstrated between the two techniques.

After discussion with the patient, you decide on general anesthesia. How will you induce and maintain anesthesia?
Any reasonable combination of drugs is reasonable for general anesthesia. Most patients stay overnight and thus rapid emergence as required for outpatient surgery is not required. However, shorter acting drugs may allow for easier monitoring in the PACU for signs of fluid absorption and TURP syndrome. Therefore, induction with either thiopental or propofol is reasonable. Maintenance could be with a volatile anesthetic, with or without nitrous oxide, and a modest dose of a short-acting opioid such as fentanyl. Muscle relaxation often used to prevent movement when the resectoscope is in place.

The procedure takes longer than expected due to a very large amount of prostatic tissue requiring resection. At the end of the operation, you extubate the patient and take him to the PACU. He is hypertensive, confused, and agitated. How will you assess him?
Although much attention is paid to it, do not assume it is TURP syndrome! First, check for the common causes of agitation in the PACU, including hypoxia, hypercapnia, pain, and emergence delirium. If you have excluded these causes, you can obtain laboratory studies to help you make the diagnosis. In particular, you can check a serum sodium and possibly an ammonia level (because glycine in the irrigating fluid is metabolized to ammonia).
If you believe he has TURP syndrome, how will you treat him?
The treatment of the syndrome is largely supportive. Begin, as always, with the ABCs: administer supplemental oxygen, ensure a patent airway and adequate ventilation, examine the patient for signs of volume overload and treat hemodynamic derangements with appropriate drugs to lower blood pressure. You will monitor the electrocardiogram for dysrhythmias, and treat them with appropriate drugs if they occur. When you have confirmation that there is hyponatremia, you will then fluid restrict the patient and consider diuresis with a loop diuretic such as furosemide. Rarely you will need to use hypertonic saline to raise the sodium level (generally if severely low, <120, or in the presence of CNS or cardiovascular symptoms). This is done slowly, to avoid myelinolysis. You will also check for the presence of dilutional coagulopathy or anemia and treat if present with factor replacement (fresh frozen plasma) and blood components as needed.

Suggested Further Reading


Chapter 22

Physiology and Anesthesia for Pediatric Surgery

Thomas M. Romanelli

For maximum impact, it is recommended that the case study and questions found on page xxviii are reviewed before reading this chapter.

Key Learning Objectives

- Understand important anatomical and physiologic differences between pediatric and adults patients
- Know how to take an appropriate preanesthetic history for a pediatric patient
- Learn common anesthetic techniques used in pediatric patients

The practice of pediatric anesthesia is often considered challenging because the clinician must address both physical and psychosocial aspects of patient care. A skilled provider must also possess a thorough knowledge of developmental physiology and its alterations in a variety of disease states.

Anatomy

The upper airway in children is markedly different from that of their adult counterparts. Children have a larger tongue relative to the size of their mouth, and the mandible is shorter. The epiglottis is larger, narrower, and slightly stiffer, making elevation with a laryngoscope blade difficult. Figure 22.1 outlines pediatric upper airway anatomy.
The narrowest part of the infant airway is the cricoid cartilage, compared to vocal cords in adults. This circumferential cartilaginous ring is slightly smaller than the glottis – an endotracheal tube may be passed through the vocal cords, but careless advancement may traumatize the subglottic airway (Fig. 22.2).

Uncuffed endotracheal tubes are often used for children less than 10-years-old to help prevent laryngeal edema and postprocedure stridor (a hoarse, “barky” cough indicating the presence of upper airway obstruction). An air leak between 15 and 20 cm H₂O is recommended to ensure an appropriate seal and
limit swelling. Cuffed tubes may still be used safely for young children if the surgery warrants positive pressure ventilation.

The trachea is only 4 cm long in the infant. It is possible that the endotracheal tube may be advanced too far, most often into the right mainstem bronchus. Auscultation of bilateral breath sounds and direct observation of equal chest expansion should always be performed immediately after intubation, and any adjustment of the tube position should be made if necessary.

The trachea is only 4–5 mm in diameter in an infant, and edema caused by rough placement of an endotracheal tube or multiple intubation attempts can significantly increase airway resistance and decrease laminar (nonturbulent) airflow (Fig. 22.3).

**Venous Access**

Small children will often resist any attempts at intravenous catheter placement while they are awake. Therefore, insertion of an intravenous catheter is often aided by an inhalation induction, rendering the young patient quiet and compliant. This process also suppresses withdrawal reflexes and may provide some
helpful vasodilation. Common sites to access include the back of the hand, antecubital fossa, and saphenous veins adjacent to the medial malleoli. Intraosseous routes (a noncollapsible needle is placed within the cavity of the tibia) may be needed in the presence of severe trauma or burn injury.

**Physiology**

**Transition from Fetal to Neonatal Circulation**

Oxygenated blood is delivered to the fetus by the umbilical vein. Intracardiac (i.e., foramen ovale) and extracardiac (i.e., ductus arteriosus and venosus) shunts form a parallel circulatory system that bypasses high resistance of the pulmonary vessels until birth. Figure 22.4 shows a schematic representation of neonatal circulation.

This transition to a normal neonatal circulation occurs after the umbilical cord is clamped and spontaneous breathing begins. As the pulmonary vascular resistance decreases, systemic blood flow is altered. Changes in pressure, plasma oxygen concentration, and diminishing placental prostaglandins help to close the shunts. However, conditions such as sepsis and severe acidosis may cause these shunts to remain open, resulting in persistent fetal circulation.
Respiratory
The architecture of the major conducting airways is established by the 16th week of gestation. Alveoli mature after birth and increase in number until 8 years of age. The chest wall of infants is composed predominantly of cartilage and deforms easily. Accessory muscles are poorly developed and tire quickly. The diaphragm has only a fraction of the typical adult fatigue-resistant type I muscle fibers. These attributes result in paradoxical chest wall movement when increased inspiratory effort is attempted. The increased caloric work is unsustainable, and respiratory fatigue and failure may follow.

Cardiac
There is less contractile tissue than in the adult heart. The chambers are also less compliant, meaning that they cannot significantly increase stroke volume (SV) to compensate for elevated metabolic needs. \textbf{Cardiac output} \((CO = HR \times SV)\) is therefore dependent upon heart rate \((HR)\), and bradycardia in young children is an ominous sign of cardiovascular depression. Factors that contribute to low heart rates (e.g., hypoxia, hypercarbia, surgical manipulation) should be avoided or treated quickly.

Renal
The kidneys are very active in utero and fetal urine output contributes to the volume of amniotic fluid. The glomerular filtration rate (GFR) is lower at birth but quickly matures by the end of one year. A low GFR may result in infants’ and some young children’s inability to remove large amounts of fluids or drug metabolites from their bodies.

Hepatic
Infants, especially those that are preterm or small-for-gestational age, have \textbf{limited glycogen stores} to provide themselves energy. They should be monitored to prevent hypoglycemia, and a maintenance dextrose infusion is often used to prevent this occurrence. Albumin levels are also lower than in adults, and this may alter the binding and activity of certain anesthetic drugs.

Gastrointestinal
Meconium is a mixture of water, pancreatic secretions, and intestinal cells that is usually passed within hours after birth. Premature evacuation, or meconium staining of amniotic fluid, is evidence of a “stressed” fetus and may pose a hazard if this material is aspirated into the immature lungs. The lower esophageal sphincter
may take several weeks to reach the tone normally found in adults. Projectile vomiting after feedings is considered a classic sign of pyloric stenosis.

**Blood**
The estimated blood volume (EBV) is 85–90 ml/kg at term and gradually declines with age (see Table 22.9). The hemoglobin species HbF is most prevalent after birth and has a greater binding capacity for oxygen than HbA (predominant in adults). As HbF is replaced over the first 2–3 months of life, a mild anemia transiently develops (the so-called “physiologic anemia of infancy”).

**Neurologic**
Developmental milestones represent the average rate of neurobehavioral maturation. Deviations from the norm do not necessarily suggest significant disease, and in fact premature infants typically display development delay that is considered “normal” for them. However, some diseases (malnutrition, intracranial trauma) may adversely affect future development.

**Temperature Regulation**
Infants and small children have a large surface area-to-weight ratio, meaning that they lose body heat quickly. They also have limited subcutaneous insulating fat and adipose reserves for generating heat. Infants rely upon a special brown adipose tissue for nonshivering heat generation. This is a catecholamine response which is quickly exhausted and may cause a decrease in peripheral perfusion, increased oxygen consumption, hypoxia and acidemia. The best way to maintain appropriate body temperature is to use ambient warming lamps, adjust the room thermostat, and cover exposed body parts to limit heat loss.

**Pharmacology**
Changes in the volume of fat, muscle, and organ mass are age-dependent and affect pharmacodynamics and kinetics of anesthetic drugs. Since infants and young children have a higher body water content, the volume of distribution is also increased. Enzyme complexes are immature and drugs may have delayed metabolism. Age-related differences in drug responses may be due in part to variations in receptor sensitivities. Most drugs used for pediatric anesthesia have not been formally approved for use in children by the FDA. A weight-based dosing methodology presumes similar clinical responses, but this may be inaccurate. Nevertheless, this paradigm continues to be observed based upon best practice guidelines.
Preoperative Evaluation

Psychological Assessment
Many factors influence how parents and the child will remember their perioperative experience. The preanesthetic interview should be used to gather pertinent information and identify specific causes of anxiety. Potential procedure risks and side-effects should be described using simple, clear language. Setting reasonable expectations for postoperative discomfort and the manner in which it will be alleviated will reassure both parents and patient.

Comfort objects may be brought into the room with the child to ease induction. Parental presence is often helpful to facilitate the acceptance of the induction mask. Some children, especially those with a prior poor surgical experience, may benefit from additional sedation medication, as shown in Table 22.1

Physiological Assessment
An otherwise healthy child who presents for a brief, outpatient procedure rarely requires more than a focused history, pertinent review of systems and a targeted physical exam to assess acute heart or lung dysfunction. Blood tests are often unnecessary and add to the cost of care while providing little benefit. However, the child with a complex past medical history may require a more thorough evaluation. Labs and noninvasive testing (echocardiogram or ultrasound) may be needed. Table 22.2 provides a template for preoperative patient evaluation, and Table 22.3 shows normal vital signs based on patient’s age.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Route</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midazolam</td>
<td>IV</td>
<td>0.05–0.1 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Oral</td>
<td>0.25–0.75 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Nasal</td>
<td>0.2 mg/kg</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>IV</td>
<td>0.5–1 mcg/kg</td>
</tr>
<tr>
<td></td>
<td>Oral (“Actiq”)</td>
<td>10–20 mcg/kg</td>
</tr>
<tr>
<td>Ketamine</td>
<td>IV</td>
<td>1–2 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Oral</td>
<td>5 mg/kg</td>
</tr>
<tr>
<td></td>
<td>IM</td>
<td>2–3 mg/kg</td>
</tr>
<tr>
<td>Methohexital</td>
<td>Rectal</td>
<td>20–30 mg/kg</td>
</tr>
</tbody>
</table>
Table 22.2 Preoperative pediatric history and review of systems.

<table>
<thead>
<tr>
<th>History</th>
<th>Important questions and pertinent findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prenatal care and delivery</td>
<td>Gestational age; Apgar scores at birth; duration of intubation and ventilatory support; associated congenital conditions (BPD, cyanotic heart disease); frequency of hospitalizations; review of growth curves (failure to thrive); persistence of apnea/bradycardia</td>
</tr>
<tr>
<td>Airway</td>
<td>Dysmorphic features (e.g., Pierre-Robin is associated with a difficult airway); micrognathia, loose teeth; advanced caries</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Symptoms c/w acute or recent URI; asthma; sick contacts; second-hand smoke exposure; presence of wheezing, stridor, nasal flaring, cyanosis; sleep apnea</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Murmurs associated with PFO, PDA, or congenital heart disease; frequency/duration of cyanotic spells; tachypnea; poor feeding tolerance</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Repetitive vomiting; delayed meconium passage; abdominal distention</td>
</tr>
<tr>
<td>Hematologic</td>
<td>Bruising; pallor; family history of sickle cell or thalassemia</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Patterns of seizure activity; developmental delay; motor weakness; hypotonia; evidence of elevated ICP</td>
</tr>
</tbody>
</table>

BPD = bronchopulmonary dysplasia; PFO = patent foramen ovale; PDA = patent ductus arteriosus; URI = upper respiratory infection; ICP = intracranial pressure.

Table 22.3 Pediatric vital signs: normal ranges.

<table>
<thead>
<tr>
<th>Age</th>
<th>RR</th>
<th>HR</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm</td>
<td>55–60</td>
<td>120–180</td>
<td>45–60</td>
<td>20–45</td>
</tr>
<tr>
<td>Neonate</td>
<td>40–55</td>
<td>100–160</td>
<td>55–75</td>
<td>20–60</td>
</tr>
<tr>
<td>Infant (&lt;6 months)</td>
<td>30–50</td>
<td>80–140</td>
<td>85–105</td>
<td>55–65</td>
</tr>
<tr>
<td>1 year</td>
<td>30–35</td>
<td>80–120</td>
<td>90–105</td>
<td>55–65</td>
</tr>
<tr>
<td>6 years</td>
<td>20–30</td>
<td>75–110</td>
<td>95–105</td>
<td>50–70</td>
</tr>
<tr>
<td>10 years</td>
<td>20–30</td>
<td>80–100</td>
<td>95–110</td>
<td>55–70</td>
</tr>
<tr>
<td>16 years</td>
<td>15–20</td>
<td>60–80</td>
<td>110–125</td>
<td>65–80</td>
</tr>
</tbody>
</table>

OR Equipment and Setup

Radiant heat loss is the most frequent cause of hypothermia in children. Conservation techniques include radiant warmers, convection blankets (forced heated airflow), increasing the room ambient temperature, and covering exposed body parts. Large-volume IV infusions should be directed through a heating element.

A selection of age-appropriate face masks, laryngoscope blades, oral and nasal airways should be readily available to meet the typical needs of children with a
diverse range of weight and body habitus. Table 22.4 shows the choice of endotracheal tube diameter and length based on patient’s age and weight. Table 22.5 shows the choice of laryngoscopic blade and LMA size based on patient’s age. Table 22.6 shows the most common pediatric emergency drug dosages.

**Intravenous Fluids**

Intravenous fluid management is based upon calculating the sum of the NPO deficit, ongoing maintenance, blood loss (if any), and the potential for surgically induced fluid shifts (also see Chap. 14, Electrolytes, Fluid, Acid–Base and Transfusion Therapy). The formula most often applied is commonly known as the “**4-2-1 rule**” (see below). Crystalloid solutions, normal saline or Lactated Ringer’s fulfill the majority of basic needs. Glucose infusions are used for the newborn or premature infant because of their limited glycogen stores.

Since many young children may still have partially patent shunts, all air bubbles should be evacuated from intravenous tubing prior to administration to prevent paradoxical air embolism and catastrophic cardiovascular collapse.

### Table 22.4 Endotracheal tube sizes and appropriate insertion depths.

<table>
<thead>
<tr>
<th>Age/weight</th>
<th>Internal diameter (mm)</th>
<th>Length (oral) in cm</th>
<th>Length (nasal) in cm</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1.5 kg</td>
<td>2.5</td>
<td>9.0–10.0</td>
<td>12.0–13.0</td>
</tr>
<tr>
<td>1.5–3.5 kg</td>
<td>3.0</td>
<td>9.5–11.0</td>
<td>13.0–14.0</td>
</tr>
<tr>
<td>Term</td>
<td>3.5</td>
<td>10.0–11.5</td>
<td>13.5–14.5</td>
</tr>
<tr>
<td>3–12 months</td>
<td>4.0</td>
<td>11.0–12.0</td>
<td>14.5–15.0</td>
</tr>
<tr>
<td>12–24 months</td>
<td>4.5</td>
<td>12.0–13.5</td>
<td>14.5–16.0</td>
</tr>
</tbody>
</table>

### Table 22.5 Laryngoscopic blade and LMA sizes.

<table>
<thead>
<tr>
<th>Age</th>
<th>Blade</th>
<th>Weight</th>
<th>LMA size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature</td>
<td>Miller 0</td>
<td>≤5 kg</td>
<td>1</td>
</tr>
<tr>
<td>Neonate</td>
<td>Miller 0</td>
<td>5–10 kg</td>
<td>1.5</td>
</tr>
<tr>
<td>1–4 years</td>
<td>Miller 1</td>
<td>10–20 kg</td>
<td>2</td>
</tr>
<tr>
<td>4–10 years</td>
<td>Miller 2, Mac 2</td>
<td>20–30 kg</td>
<td>2.5</td>
</tr>
<tr>
<td>Adolescent</td>
<td>Miller 2, Mac 3</td>
<td>&gt;30 kg</td>
<td>3</td>
</tr>
</tbody>
</table>
Pediatric Drug Preparation

Drugs should be drawn up in an appropriate syringe size that will deliver the desired dose of agent in a minimal volume. All emergency medication syringes should be fitted with a 1.5-inch, 22-gauge needle for IM injection in case vascular access is unavailable.

Techniques

Induction

A smooth anesthetic induction can be achieved in a variety of ways. All methods have their advantages and disadvantages (Table 22.7).

Maintenance

Effective anesthetic depth may be maintained with a number of drug and technique combinations. The selection should be based upon individual needs and guided by the presence of comorbidities, anticipated procedure duration and other case-specific features.

Many clinicians use the “4-2-1 rule” (see Table 22.8) as a guide to fluid replacement (Table 22.8; also see Chap. 14, Fluid, Electrolyte, and Transfusion Therapy). Neonates and infants require additional care to avoid fluid overload and provide appropriate glucose supplementation (D5NS is appropriate). Estimated blood volume (EBV) (see Table 22.9) should always be calculated to guide fluid therapies for surgeries involving significant blood loss. Although young children tend to tolerate a lower hematocrit, it is important to remember that they also have higher metabolic rates and oxygen needs.

Certain procedures (e.g., bilateral herniorrphy) permit the use of supplemental regional anesthesia. Single shot caudals (0.25% Bupivacaine, 1.0 ml/kg up to 20 kg) are popular and relatively easy to perform, and provide hours of analgesia while reducing the potential side-effects of other drugs (e.g., opioids).

### Table 22.6 Common pediatric emergency drugs.

<table>
<thead>
<tr>
<th>Drug</th>
<th>IV</th>
<th>IM/(SQ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>0.01–0.02 mg/kg</td>
<td>0.02 mg/kg</td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>1–2 mg/kg</td>
<td>3–4 mg/kg</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>0.1–0.2 mg/kg</td>
<td>–</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>10 mcg/kg</td>
<td>(10 mcg/kg)</td>
</tr>
</tbody>
</table>
Anesthesia student survival guide

Emergence
A child should be ready for extubation if he is normothermic (body temp > 35.5°C), hemodynamically stable, has no appreciable neuromuscular blockade, and is reasonably comfortable. Although infants and young children will generally not follow verbal commands, they may reach up and try to extubate themselves. Appropriate equipment should be immediately available to secure the airway if extubation fails.

Monitored transport with oximetry is recommended but may not be practical in the emerging, active child. Pulse oximetry is very sensitive to motion artifact, so the recovering child should be closely observed for evidence of airway obstruction. Phonation and crying are actually reassuring signs.
under these circumstances, because they confirm the presence of a patent airway. Always administer supplemental oxygen during transport, though the same practical limitations may apply.

Parents are often invited to stay with their child in the recovery area once the physician and nurses are satisfied with the patient’s clinical status. The parents will provide a reassuring presence and help limit any mild disorientation that may occur. The patient is ready for discharge if he is reasonably comfortable, hemodynamically stable, and has minimal nausea or vomiting. Children may not resume normal oral intake or void prior to discharge, but this does not need to unnecessarily prolong their stay as long as fluid replacement has been adequate. Some common specific operations in children and the associated anesthetic considerations are given in Table 22.10.

| Table 22.9 Estimated HCT and Estimated Blood Volume (EBV). |
|-----------------|-----------------|-----------------|
| Age             | HCT (%)         | EBV (ml/kg)     |
| Premature       | 45–60           | 90–100          |
| Neonate         | 45–60           | 80–90           |
| 3–6 months      | 30–33           | 70–80           |
| 6 months–1 year | 32–35           | 70–80           |
| 1–12 years      | 35–40           | 70–75           |
| Adult           | 38–45           | 60–70           |

**Pediatric Surgical Conditions**

A list of the most common pediatric surgical conditions and their anesthetic implications are shown in Table 22.10.

<table>
<thead>
<tr>
<th>Table 22.10 Common pediatric surgical conditions.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pyloric stenosis</strong></td>
</tr>
<tr>
<td>• Associated findings: Nonbilious, projectile vomiting, hypochloremic metabolic alkalosis, hypokalemia, hyponatremia, dehydration, hypovolemic shock</td>
</tr>
<tr>
<td>• Anesthetic considerations: Usually not a surgical emergency, rehydrate and correct metabolic abnormalities prior to surgery, awake or rapid sequence intubation, decompress stomach with nasogastric tube</td>
</tr>
<tr>
<td><strong>Diaphragmatic hernia</strong></td>
</tr>
<tr>
<td>• Associated findings: Respiratory distress, bowel sounds heard over the chest, decreased breath sounds, scaphoid abdomen</td>
</tr>
<tr>
<td>• Anesthetic considerations: Bowel decompression with a nasogastric tube, avoid mask ventilation because it may cause bowel distension and worsen respiratory status, avoid hypoxia, watch for tension pneumothorax.</td>
</tr>
</tbody>
</table>

(continued)
Table 22.10  (continued)

**Omphalocele and Gastrochisis**

- **Associated findings:** Abdominal contents covered by membrane (omphalocele only), fluid loss, infection, associated anatomic anomalies.
- **Anesthetic considerations:** Volume resuscitation, need for muscle relaxation to allow inserting abdominal contents back into the abdomen, potential for postoperative respiratory failure.

**Tracheoesophageal fistula**

- **Associated findings:** Blind esophageal pouch with a distal fistula near carina (most common type), anatomical anomalies, coughing and choking, dehydration
- **Anesthetic considerations:** Increased risk of aspiration, gastric decompression, maintain spontaneous ventilation to avoid further gastric distention, consider awake intubation

### Case Study

A 5-year-old boy has been vomiting and had little or no appetite for 2 days. He has taken limited amounts of liquids by mouth. He has now developed abdominal pain and is suspected of having acute appendicitis. The surgeons plan a laparoscopic appendectomy. The child is a healthy product of a full-term delivery. Vital signs are HR 120, BP 95/50, RR 24.

**How will you assess his volume status prior to surgery? What metabolic derangement would you suspect him to have?**

The child is likely volume depleted, as assessed by history of little PO intake and vomiting for 2 days. The elevated heart rate and low normal blood pressure for his age imply moderate, but not severe volume depletion. You can estimate the volume loss by the 4-2-1 rule for maintenance fluid requirements and assume that he is depleted up to 2 days’ worth. However, this likely overestimates his volume depletion because people do not consume fluids overnight, and because his vital signs do not reflect a severe deficit. You can also assess other signs such as skin turgor, urine concentration, and urine volume. He likely has hypochloremic, hypokalemic, metabolic acidosis since he has been vomiting and thus losing hydrochloric acid while the kidney wastes potassium in preference to absorbing sodium. However, if the hypovolemia is severe, he may also have volume-associated acidosis.
The child is anxious and teary. How can you help during the preparation for and induction of anesthesia?

If you have an IV in place, you can administer a small dose of a short acting sedative such as midazolam. If not, you can consider sedation via the rectal, intramuscular, nasal, transmucosal, or oral routes. The oral route is less desirable in this child, since absorption may be unreliable. Transbuccal fentanyl, nasal midazolam, rectal methohexital, and intramuscular ketamine have all been used in pediatric sedation. In addition to pharmacological agents, you can consider parental presence to ease the child's anxiety. However, the parents should be carefully counseled regarding what to expect during the induction procedure. Many children in this age group have a favorite toy, stuffed animal, or other “transition object” which comforts them, and you can bring such an object to the OR with you.

Would you perform an inhalation or intravenous induction?

Many children are anesthetized for routine cases with mask inhalation of a volatile agent, prior to starting an IV. In this case, however, there is evidence of abdominal pathology and despite the fact that the patient has not eaten for 2 days, you will likely treat him as at risk for aspiration of gastric contents. Therefore, you should induce with intravenous drugs and secure the airway before beginning positive pressure ventilation.

If you decide on an intravenous induction, how can you facilitate placement of the IV in this frightened child?

Many of the sedative options and other comfort measures noted above are available to you. You can also be careful to infiltrate the IV site with lidocaine or normal saline, using a very fine gauge needle. Another option is to use EMLA cream, a mixture of local anesthetics that can be absorbed directly across intact skin to anesthetize the area around planned IV placement.

How will you induce and maintain anesthesia? What size endotracheal tube will you use?

Once you have placed an IV, you should try to replete the volume deficit at least partly before induction. You will plan a rapid sequence intubation of anesthesia. Thiopental, ketamine, or propofol can be used for induction,
depending on your assessment of volume repletion at the time of induction. In children, use of succinylcholine is somewhat limited due to an increased risk of side effects. However, either succinylcholine or a rapid acting nondepolarizing drug (such as rocuronium) can be used to facilitate intubation. You can use a cuffed or uncuffed endotracheal tube at this age; the usual formula (4 + Age/4 mm internal diameter) is verified empirically at the time of intubation by ensuring that the fit is not too tight. Usually a half size lower is employed when a cuff is used.

How will you know when you are able to extubate the patient at the end of the procedure?
All patients emerging from anesthesia should be breathing spontaneously, fully reversed from neuromuscular blockade, normothermic, hemodynamically stable, and be able to protect their airways. Adults should follow verbal commands such as “squeeze my hand” or “open your eyes” but children of this age are likely to be unable to do so. If you detect signs of purposeful movement (such as reaching for the endotracheal tube) or spontaneous eye opening, you can extubate the patient. You will observe for signs of a patent airway. A strong cry is a good sign in this setting. You should monitor the patient carefully on the way to the PACU, perhaps using a portable pulse oximeter and supplemental oxygen.

Suggested Further Reading


Physiology and Anesthesia for Elderly Patients

Ruma Bose

For maximum impact, it is recommended that the case study and questions found on page xxix are reviewed before reading this chapter.

Key Learning Objectives

- Understand the physiologic changes associated with aging
- Learn the specific considerations for anesthetic management of the elderly
- Understand common postoperative anesthetic complications

The elderly is defined as a person who is over the chronological age of 65 years. This includes a large number of people with varying physical and mental capabilities. However, the basis for defining 65 years as a threshold for old age is still not well-defined. Aging is a natural biological process which is associated with a normal decline in physiological function. In addition, the functional reserve decreases with age, which impacts on the ability of the elderly to recover from major illnesses, surgery, and trauma.

Care of the elderly requires knowledge of the normal age-associated physiologic changes and age-related illnesses. The preoperative management should be focused on identifying and optimizing any comorbid conditions prior to surgery. During the intraoperative phase, one should take into consideration the physiologic changes that occur in the elderly. This often requires the use of shorter acting agents and additional invasive monitoring to maintain hemodynamic stability. Postoperative care should be focused on early identification and treatment of postoperative complications such as postoperative delirium, hypoxia, and hypotension.
For anesthesiologists, it is important to assess preoperatively the baseline physical and mental status of the patient as well as determine the physiological reserves. Functional reserve is the difference between maximal and basal function. Aging inevitably reduces functional reserve even in those individuals who are physiologically “young.” The relationship between maximal and basal physiologic function is shown in Fig. 23.1.

**Physiological Changes with Aging**

**Cardiovascular System**

The cardiovascular system undergoes considerable changes with age and is responsible for most of the perioperative morbidity seen in the elderly. A decrease in arterial compliance leads to an increase in afterload. In response, the left ventricle hypertrophies over time and its compliance decreases. The inability of the left ventricle to relax during diastole is termed “diastolic dysfunction,” which can be quantified by echocardiography. Left ventricular filling then becomes increasingly dependent on preload and atrial contraction. Hence, **maintaining sinus rhythm** is important to ensure adequate left ventricular filling and cardiac output. The venous vasculature also loses some of its compliance and its ability to act as buffer against volume overload. This predisposes the elderly to pulmonary edema with excessive fluid administration.
Conduction system abnormalities are often seen in the elderly because of a decrease in the number and function of atrial pacemaker cells. The most commonly seen abnormalities are right bundle branch block (RBBB) and first degree heart block. The responsiveness of β-adrenergic receptors is also diminished, rendering the elderly unable to initiate compensatory increases in heart rate in response to hypovolemia. Therefore, the elderly are likely to develop orthostatic hypotension. Although cardiac output may remain unchanged, systolic blood pressure increases with age, whereas diastolic blood pressure increases until age 60–65 years and then plateaus or decreases (see Fig. 23.2). Valvular abnormalities are more common due to sclerosis and calcification, and more than 70% percent of the elderly have an audible heart murmur.

Pulmonary Changes
The major changes that occur with aging can be broadly attributed to the following factors:

![Figure 23.2 Changes in blood pressure with age.](image-url)
• blunting of the central nervous system reflexes to hypoxia and hypercarbia
• decrease in the compliance of the thoracic wall
• decrease in alveolar gas exchange surface
• generalized de-conditioning of chest wall musculature

The larger proximal airways tend to dilate with age, causing an increase in dead space. The distal airways tend to collapse, causing an increase in closing volume and air trapping. The compliance of the chest wall decreases (due to stiffening of the costochondral joints). The intercostal spaces are usually decreased because of loss of musculature, which increases the work of breathing and decreases the ability to re-expand atelectatic regions of the lungs. It also hinders the ability to cough and adequately clear secretions.

The central nervous system reflexes in response to hypoxemia and hypercarbia are also diminished. Clinically, all these changes predispose the patients to hypoxemia in the perioperative setting. Finally, the hypoxic pulmonary reflex, which is responsible for shunting blood away from poorly ventilated parts of the lung is diminished, leading to greater ventilation–perfusion mismatching.

Renal Changes

A decline in renal function is seen in the elderly due to a decrease in glomerular filtration rate (GFR) and total renal blood flow (RBF). The serum creatinine may not reflect the extent of renal impairment as muscle mass declines in the elderly. Creatinine clearance can provide a much more accurate reflection of renal function in the elderly. The elderly are also predisposed to dehydration because of diminished compensatory mechanisms, including perception of thirst and the renal response to antidiuretic hormone (ADH) (Table 23.1).

<table>
<thead>
<tr>
<th>Table 23.1  Renal changes in the elderly.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrease in renal blood flow</td>
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<tr>
<td>Decline in glomerular filtration rate</td>
</tr>
<tr>
<td>Decline in ADH response</td>
</tr>
<tr>
<td>Decrease in total body water</td>
</tr>
<tr>
<td>Decreased ability to conserve sodium</td>
</tr>
<tr>
<td>Diminished urine concentrating ability</td>
</tr>
<tr>
<td>Decline in renin-aldosterone levels</td>
</tr>
<tr>
<td>Decreased thirst perception</td>
</tr>
</tbody>
</table>
Nervous System
With age, there is a decline in higher cognitive functions due to **gradual loss of neurons**. This loss is more pronounced in the gray matter than the white matter. Additionally, diminished levels of neurotransmitters (dopamine, serotonin and acetylcholine) predispose elderly patients to cognitive deficits which can be accentuated in the postoperative period. Sensory perception such as vision, hearing and taste also diminishes with age.

Postoperative Cognitive Dysfunction and Delirium
Some of the causes of postoperative mental status changes in the elderly are delirium, cognitive dysfunction, perioperative stroke, and electrolyte imbalances.

**Delirium** is a state of confusion with waxing and waning mental status. It commonly presents acutely in the elderly during hospitalization, and frequently in the postoperative setting. There are **extrinsic** as well as **intrinsic causes** of **postoperative delirium**. Intrinsic factors include preexisting cognitive dysfunction and alcohol abuse. Extrinsic factors include the stress of illness and surgery, an unfamiliar environment, medications (e.g., benzodiazepines, narcotics, anticholinergics), underlying infection, urinary retention, pain, and electrolyte imbalances (e.g. hyponatremia). Some of the most important and treatable causes of postoperative delirium are hypotension, hypoxia, and hypercarbia. Table 23.2 outlines the common causes of postoperative delirium.

**Postoperative cognitive dysfunction** differs from delirium in that the presentation is not acute. In most patients, there is clinically apparent or subclinical

<table>
<thead>
<tr>
<th>Table 23.2 Causes of delirium.</th>
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<tbody>
<tr>
<td>Advanced age</td>
</tr>
<tr>
<td>Preexisting dementia</td>
</tr>
<tr>
<td>Depression</td>
</tr>
<tr>
<td>Hypoxia and hypercarbia</td>
</tr>
<tr>
<td>Hypotension</td>
</tr>
<tr>
<td>Alcohol or sedative withdrawal</td>
</tr>
<tr>
<td>Impaired vision and hearing</td>
</tr>
<tr>
<td>Metabolic disturbances (hyponatremia/hypernatremia)</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
</tr>
<tr>
<td>Infection</td>
</tr>
</tbody>
</table>
cognitive dysfunction at baseline, which can be elicited during the preoperative examination by performing a simple mini-mental status examination. The incidence of postoperative cognitive dysfunction has been stated to be approximately 30% in the immediate postoperative period and 12% after 3 months. Postoperative cognitive dysfunction may be related to increased age, extended duration of anesthesia, low level of education, prior exposure to anesthetics, postoperative infection, respiratory complications, and prior stroke. Patients with postoperative cognitive dysfunction at discharge have been shown to have higher mortality rates during the first year after surgery.

Pharmacokinetic and Pharmacodynamic Changes
With age, there is a progressive change in the constitution of the various body compartments. Total body water diminishes, fat stores increase, and serum albumin decreases. As a result, the volume of distribution of the administered drugs decreases, leading to an increase in drug concentration at the receptor sites. As the lipid stores are increased, lipid-soluble drugs (e.g. morphine) may have a prolonged duration of action. A decline in liver and renal function may also slow down drug metabolism and excretion. Because of these changes, the dosages of most medications should be decreased in the elderly, and the dosing interval should be increased (Table 23.3).

Anesthetic Management
Preoperative Examination
The purpose of the preoperative examination is to (1) determine the baseline physical and mental status of the patient, and (2) identify and optimize any medical comorbidities prior to undergoing a surgical procedure. The elderly patient has on average three or more comorbid conditions at any given time.

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fentanyl</td>
<td>250 min</td>
<td>925 min</td>
</tr>
<tr>
<td>Alfentanil</td>
<td>90 min</td>
<td>130 min</td>
</tr>
<tr>
<td>Diazepam</td>
<td>24 h</td>
<td>72 h</td>
</tr>
<tr>
<td>Midazolam</td>
<td>2.8 h</td>
<td>4.3 h</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>16 min</td>
<td>45 min</td>
</tr>
</tbody>
</table>
The preoperative examination is challenging as geriatric patients may not be able to provide accurate histories due to underlying cognitive dysfunction and memory deficits. “Polypharmacy” is common in the elderly and a detailed list of medications should be obtained. Because the incidence of atherosclerosis and coronary artery disease increases with age, a baseline electrocardiogram is generally recommended in women > 55 years old and men > 45 years old.

Further cardiovascular testing is dictated by a patient’s underlying history and an assessment of the risk of surgery (see Chap. 8, Preoperative Evaluation). For example, a patient with cardiac impairment might be able to proceed for a cataract extraction (a low risk procedure) without extensive preoperative cardiac testing, but the same patient might require further testing (e.g. a stress test) if undergoing a thoracic procedure.

Serum albumin gives an overall indicator of the general state of health of an elderly patient. Low preoperative serum albumin levels have been associated with increased postsurgical morbidity. Finally, a preoperative assessment should allow some determination of the feasibility of ambulatory care versus postoperative hospital admission. This advance planning should be guided by the patient’s baseline level of functioning and the availability of support at home.

Intraoperative Management

Premedications

As has been discussed above, the elderly are more sensitive to benzodiazepines and most of medications have a prolonged duration of action. Premedications should be used judiciously, with decreased doses and titrated to effect. Anticholinergic agents, such as scopolamine and atropine, should be used with caution as they may be contributory to postoperative delirium.

Monitoring

The elderly are predisposed to hemodynamic fluctuations in the intraoperative period. They are more prone to develop cardiovascular complications such as hypotension, arrhythmias, myocardial infarctions, or heart failure. Therefore, close monitoring of vital signs and hemodynamic status with invasive monitoring is critical, especially in cases of intermediate and high-surgical risk.

Intraoperative Management

With age, the minimum alveolar concentration (MAC) decreases (see Chap. 5, Pharmacology of Volatile Anesthetics). The total dose of medications
should be decreased and shorter-acting agents should be used, if possible. **Induction agents** should be titrated to effect. Propofol decreases peripheral vascular resistance and can cause significant hypotension. If hemodynamic stability is a concern, consider using ketamine or etomidate for induction.

Because **thermoregulation** is altered in the elderly, they are at risk for hypothermia and its associated complications (e.g. coagulopathies, myocardial ischemia, poor wound healing). Temperature monitoring is therefore important in the elderly and active rewarming may be required.

There are no data to support the use of one inhalational agent over the other, but shorter-acting agents such as desflurane are preferred to minimize any lingering effects of the more lipid soluble anesthetics.

Shorter-acting opioid medications like fentanyl tend to cause less cumulative effects when compared with longer-acting agents like morphine. Meperidine has been associated with postoperative delirium and should be avoided in elderly patients.

The duration of nondepolarizing muscle relaxants is mildly prolonged in the elderly because of decline in metabolic function, although this is not typically clinically significant. The pharmacokinetics of depolarizing agents (e.g. succinylcholine) are not affected. Muscle relaxants should be adequately reversed and patients should be extubated only after return of muscle strength and airway reflexes. Any residual paralysis can potentiate respiratory depression, hypoxia and hypercarbia.

**General Anesthesia Versus Regional Anesthesia**

Studies comparing general to regional anesthesia in the elderly have not shown a significant difference in outcomes. Because the epidural and spinal spaces decrease in volume with age, a similar dose of epidural local anesthetic in an elderly patient may result in a higher sensory motor loss as compared to a younger patient. While the incidence of postdural puncture headaches (PDPH) is decreased in the elderly, the placement of a neuraxial block may sometimes be difficult due to restrictions in positioning.

**The Postoperative Period**

The elderly are vulnerable to prolonged effects of medications and should be closely monitored for respiratory depression, hypoxia, and hypercarbia. Pain in the elderly may atypically present as **agitation** and **delirium**. Postoperative delirium is commonly seen in the elderly and can be a manifestation
of a variety of conditions – acute hypoxia and hypotension should always be ruled out. Haloperidol is commonly used to control acute delirium and causes minimal respiratory depression. The incidence of postoperative delirium peaks between postoperative days 1–4. With ambulatory procedures, it is very important to assess the physical and cognitive status of the patient prior to discharge. It is also important to know about the support structure at home.

Case Study

An 82-year-old female suffered a fall, fractured her right hip, and is to undergo open reduction and hemiarthroplasty. She has no other injuries and did not lose consciousness. She is a smoker with a 60-pack-year history, but currently smokes just 2–3 cigarettes per day. She has chronic hypertension and an electrocardiogram from last year showed a right bundle branch block and a left anterior hemiblock with a sinus rhythm and rate of 55. She is a retired professor of pathology, a medical school dean, and still serves on your hospital’s faculty council on promotions. She is in mild-moderate pain, which is much worse with movement of the right leg. She has expressed some concern regarding the effects of anesthetics on postoperative cognitive function.

What preoperative assessment will you perform before deciding on an anesthetic plan? How would it differ from the preop you would perform if the patient were having an elective cataract surgery?

In large measure, you will perform the usual preoperative assessment you do for any patient, including review of her airway, pulmonary status, NPO status, physiology, or disease of any other systems. You can ask about her exercise tolerance before the injury to get an idea of her cardiovascular reserve. You will also assess her volume status, because “bones bleed.” A significant fracture, even without external injuries, can lead to a significant volume and red cell loss. This case can be done with a variety of anesthetic techniques, so you will also examine her for suitability for regional anesthesia, including examination of her back and lumbar spine and an assessment of whether she can be positioned without too much discomfort for the placement of a neuraxial block. You may want some laboratory studies, including a complete blood count and a new ECG. In a case such as a cataract done under monitored anesthesia care, there is evidence that routine laboratory studies do not change the anesthetic plan or outcomes, so they can be safely foregone.
How will you address her concern about postoperative cognitive dysfunction? She is medically sophisticated, so you will be clear and discuss the evidence as best as you can from a scientific point of view. In animal models, you can tell her, isoflurane and some other anesthetics that act as gamma aminobutyric acid (GABA) agonists and N-methyl-d-aspartate (NMDA) antagonists can trigger apoptosis or programmed cell death. In elderly animals, isoflurane can increase beta amyloid formation, which is part of the pathophysiology of Alzheimer’s disease. To date, however, no direct human evidence has definitively linked exposure to anesthetics to long term cognitive decline. Nonetheless, there is indeed a theoretical concern. You can offer her an anesthetic excluding isoflurane, though it is certainly possible that other inhalation anesthetics may share this property. You can also offer her TIVA and regional anesthesia. Drugs used for TIVA are also GABA agonists and/or NMDA antagonists, so you cannot absolutely assure her that there are not adverse neurological effects, and indeed they have been shown to have some adverse effects in animal models of developing (neonatal) brain. Regional anesthesia does offer the possibility of avoiding all suspect drugs.

Will you favor regional or general anesthesia?
Given the possibility of pulmonary issues in this chronic smoker and the possibility of avoiding neurotoxic drugs, you should consider regional anesthesia. There are other potential advantages, including less blood loss and risk of venous thromboembolism. Conversely, given the fact that she has suffered some blood loss already, might experience negative hemodynamic effects from spinal or epidural anesthesia, and has disease of her cardiac conduction system, some would consider general anesthesia. Ultimately both are reasonable choices and you should discuss them with the patient.

Will you premedicate the patient prior to anesthesia?
You will ask the patient what she wants, rather than giving drugs reflexively. You will proceed gently, focusing on pain control rather than sedative effects. This may reduce the likelihood of respiratory side effects, as well as reduce postoperative delirium or short term cognitive dysfunction.
If you and the patient agree on regional anesthesia, what type will you perform? There are several approaches to hip fracture. In simple cases, a screw is placed to stabilize the femoral neck; the procedure is short and can be done under isobaric spinal anesthesia. Hemiarthroplasty involves more surgical manipulation and blood loss because the entire femoral head is replaced. This involves reaming of the femur, fitting and cementing a prosthesis. Because of the longer surgical duration, you may consider an epidural block or combined spinal-epidural (CSE). The presence of the epidural allows you to extend the block’s duration should the procedure take longer than the spinal alone lasts. In addition, the epidural may be used postoperatively, which may be helpful in reducing opioid exposure, respiratory depression, and reduce the chance of cognitive dysfunction.

**Suggested Further Reading**


Chapter 24

Ambulatory Surgery and Out-of-OR (OOR) Procedures

Joshua H. Atkins

For maximum impact, it is recommended that the case study and questions found on page xxix are reviewed before reading this chapter.

Key Learning Objectives

- Learn about patient selection, intraoperative management and postoperative care of ambulatory patients
- Understand specific practices of ambulatory anesthesia such as fast-tracking and multimodal management of pain and PONV
- Understand the unique challenges of the OOR environment

Introduction

Surgical procedures performed on an outpatient basis in hospitals, ambulatory surgical centers, or physician offices are increasing in number and complexity. Technically complex interventions such as cerebral aneurysm coiling and cardiac arrhythmia ablation are performed on patients with multiple co-morbidities in sites remote from the operating room. These settings share fundamental differences with anesthesia delivered in the operating room. Moreover, ambulatory surgical centers and office-based surgery practices are subject to strict state regulations. These regulations stipulate types of surgery, patient types, protocols, and necessary emergency resources that are appropriate for each location.
Ambulatory Surgery

Ambulatory surgery generally places a great emphasis on the aesthetics of the patient experience from arrival to discharge and on maximization of efficiency and facility throughput. The goals of ambulatory anesthesia include rapid emergence from anesthesia, expedited discharge by “fast-tracking” patients through the recovery room, prevention and rapid treatment of common postoperative problems such as pain and postoperative nausea and vomiting (PONV), increased operating room efficiency, and patient convenience.

Preoperative Considerations

Ambulatory surgery encompasses all patients, undergoing a surgical procedure, who are planned for discharge on the day of surgery, regardless of the anesthetizing location. However, the range of facilities in which ambulatory procedures occur is diverse and represents an important consideration for patient selection and planning. Unforeseen difficulty can be managed rather routinely when ambulatory surgery is performed in the setting of the full support services of an inpatient hospital. In contrast, even basic problems, such as the need for postoperative bladder catheterization, may not be easily handled in the office-based practice.

Specific preoperative issues to consider for ambulatory surgery patients include:

1. Is the nature of the surgical procedure compatible with same-day discharge?
2. Do patient characteristics or co-morbid conditions (see Table 24.1) predispose the patient to complications that might require hospital admission?

Indeed, even the simplest procedure done on a physiologically complex patient may require hospital admission and overnight observation. Table 24.1 provides representative criteria used to decide whether the patient might be an appropriate candidate for ambulatory surgery. Table 24.2 presents surgery- and procedure-related factors one might consider in deciding whether the proposed

<table>
<thead>
<tr>
<th>Table 24.1 Patient selection factors for ambulatory surgery.</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Caregiver available for transport home and postoperative evaluation</td>
</tr>
<tr>
<td>• Patient willingness to go home the day of surgery</td>
</tr>
<tr>
<td>• Co-morbidities: obesity, obstructive sleep apnea, poorly compensated cardiopulmonary disease, chronic pain, renal failure, urinary retention, significant neurologic disease (myasthenia, Parkinson’s, dementia)</td>
</tr>
<tr>
<td>• Prior anesthetic problems: difficult airway, PONV, postoperative cognitive dysfunction, malignant hyperthermia, poor pain-control</td>
</tr>
</tbody>
</table>
procedure is appropriate for the ambulatory setting. Table 24.3 lists common ambulatory procedures. However, no procedure is always done in an outpatient basis. Even the simplest procedure (e.g., cataract removal) done on a physiologically complex patient may require hospital admission and overnight observation.

Preoperative testing is a controversial topic that requires good judgment. Patients are best evaluated in a preoperative clinic setting well in advance of the planned procedure. Advance assessment allows problem identification and implementation of optimization strategies that may facilitate handling of medically complex patients in the outpatient setting.

Generally, patients planned for same-day discharge should not have active issues that require substantial medical consultation or interdisciplinary planning. If such medical co-morbidities are present, regardless of anesthetic or surgical approach, the risk of peri-operative exacerbation of underlying medical conditions is real. The challenges and dangers intrinsic to the management of sick patients in a stand-alone ambulatory surgery center or office-based practice, in many cases,
outweigh the potential benefits of rapid discharge, patient convenience, and decreased cost. However, a carefully selected patient with medically optimized conditions often does quite well in the ambulatory center.

Preoperative testing focused on specific patient factors is appropriate. Medically informed common sense should guide this decision-making. For example, patients with hypertension or other known cardiovascular disease should have a preoperative ECG; patients on medications that affect electrolyte balance (e.g. furosemide, spironolactone, and potassium) should have a recent preoperative chemistry panel; patients with chronic anemia or recent active bleeding (e.g. menorrhagia, epistaxis, and GI bleed) should have a hemoglobin value measured since the last bleeding episode. A healthy patient generally needs no preoperative testing and “routine” tests such as complete blood count, chemistry panel and chest X-ray should never be ordered without a clear idea of why the test results will be useful in the anesthetic planning and perioperative management of the patient in question.

Certain procedures simply cannot be performed on an outpatient basis; this is primarily due to the need for continuous postoperative monitoring (e.g. measurement of gastric drainage, placement of drains for bleeding, and need for frequent electrolyte studies), ongoing interventions (intravenous medications for pain, fluid resuscitation, and complex dressing changes), or inability to eat, drink, or urinate. Examples are listed in Table 24.4.

<table>
<thead>
<tr>
<th>Table 24.4 Procedure exclusions for outpatient management.</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Requires drain or nasogastric drainage tube to be placed</td>
</tr>
<tr>
<td>o Hysterectomy, bowel resection, neck dissection</td>
</tr>
<tr>
<td>• Oral medications inadequate for postoperative pain control</td>
</tr>
<tr>
<td>o Joint replacement, mastectomy, major abdominal surgery</td>
</tr>
<tr>
<td>• May require postoperative bladder catheterization</td>
</tr>
<tr>
<td>o Ventral hernia repair, bladder tumor resection, ureteral stent</td>
</tr>
<tr>
<td>• Frequently requires intraoperative or postoperative transfusion</td>
</tr>
<tr>
<td>o Hysterectomy, ORIF femur</td>
</tr>
<tr>
<td>• Expectation of postoperative electrolyte shifts</td>
</tr>
<tr>
<td>o Parathyroidectomy, pituitary resection</td>
</tr>
<tr>
<td>• Requires hourly patient assessment</td>
</tr>
<tr>
<td>o Free-flap, craniotomy, patients with severe sleep apnea</td>
</tr>
</tbody>
</table>
Intraoperative Management

Anesthetic management in ambulatory surgery is based on the SAMBA (Society for Ambulatory Anesthesia) S.A.F.E. principles. S.A.F.E. is an acronym that stands for short-acting, fast-emergence anesthetic. General, regional, combined regional/general, and monitored anesthesia care are all compatible with rapid patient discharge. An important consideration is that the anesthetic plan be compatible with patient expectations, surgical needs, and patient-specific factors. Many patients have a preconceived notion that general anesthesia implies delayed emergence and long recovery. These same patients may not appreciate, for example, the delay in discharge that can be associated with time needed for return of motor or bladder function after neuraxial (spinal, epidural) blockade. Patients should participate in the anesthetic planning where appropriate, with their concerns specifically addressed in the preoperative discussion.

Generally speaking, short-acting anesthetic agents are better suited to rapid recovery. Midazolam is preferable to diazepam, propofol to thiopental, and bupivacaine or lidocaine to tetracaine. The inhaled potent agents are all similar in their clinical profiles provided that depth is titrated appropriately, although desflurane, due to its low blood solubility, likely has some clinical advantage in subgroups of patients such as the morbidly obese. In this regard, a processed EEG, such as BIS or SEDLine monitors, may have some utility as a guide to titration of anesthetic depth in order to avoid overdose of agents, which may prolong emergence or recovery.

Adequate postoperative analgesia is of paramount importance. In the absence of effective regional anesthesia, hydromorphone, morphine, and fentanyl are all acceptable opioid options in the intraoperative period. One caveat is that fentanyl is short-acting and may necessitate more aggressive loading with long-acting analgesic agents in the PACU or by mouth at home. Using several analgesics that work by different mechanisms, known as multimodal analgesia may help to reduce narcotic requirements and related side-effects.
Analgesia options in selected patients include low-dose ketamine, intravenous ketorolac, acetaminophen, wound infiltration by local anesthetic, or via single-shot nerve block or continuous catheter.

**Postoperative nausea and vomiting (PONV)** is one of the major reasons for delayed discharge or unplanned admission after elective surgery. In light of the availability of safe, efficacious, and inexpensive agents for PONV prophylaxis (see Chap. 7) there appears to be limited downside to a single dose of a 5HT-3 antagonist (e.g. ondansetron) for most patients. Multimodal PONV prophylaxis should be considered in patients at higher risk. High risk patients include those with prior history of PONV, motion sickness, females nonsmokers, and patients undergoing ear, eye, gynecologic, or abdominal surgery. A scopolamine patch, low-dose dexamethasone, 5HT-3 antagonist, and metoclopramide are likely to have fewer sedating effects than droperidol, prochlorperazine, or promethazine.

**Postoperative Management**

Ambulatory surgery patients and their families desire rapid discharge from the PACU to home. Facilities differ in their discharge criteria, but almost all have well-defined protocols. PACU is often divided into Phase I (immediate recovery with active, ongoing issues such as blood pressure control, pain, and hypoxia) and Phase II (imminently ready for discharge except for voiding, ambulation, or demonstration of oral intake). Some facilities will use established scoring systems like those of Aldrete to objectively manage patient flow and discharge. These scoring systems emphasize pain control and return to baseline neurologic, hemodynamic, and pulmonary function. Most facilities require patients to consume a light snack and beverage and reach reasonable pain control on oral medication prior to discharge. Some still require postoperative voiding while in many centers voiding is not a criterion, provided the patient is not at high risk of urinary retention, has access to support persons at home and can be transported to the ER in the event of a problem.

“**Fast-Tracking**” after ambulatory surgery is a widely accepted practice which involves transferring patients from the operating room to the later stage recovery area (Phase II), by bypassing the early stage (Phase I). The success of fast-tracking depends upon appropriate modification of the anesthetic technique, to allow rapid emergence from anesthesia and the prevention of pain and PONV. Implementation of a fast-track program involves the use of clinical pathways that reduce hospital stay and ensure patient safety.
Inadequate pain control and continued nausea or vomiting with inability to tolerate oral intake are the two most common reasons for discharge delay. These clinical problems should be treated aggressively. PONV in the PACU should be treated with an agent of a different class than used for prophylaxis. Pain should be treated with rapidly acting IV analgesics, and the patient should then be transitioned to oral medications.

**Out-of-OR (OOR) Anesthesia**

**General Considerations**

Provision of anesthesia outside of the operating room is one of the biggest challenges in anesthesia. Demand for anesthesia services in the electrophysiology lab, interventional radiology suite, and endoscopy center is growing rapidly. At the same time, increasingly complex procedures (e.g. percutaneous cardiac valve replacements, cerebral aneurysm embolization, aortic aneurysm stenting, and ICD placement) are performed in these locations on patients with multiple underlying physiologic derangements that range from life-threatening cardiomyopathy to super morbid obesity. The physicians performing these procedures often lack in-depth understanding of the complexities of anesthetic management in patients outside of the OR (OOR), while the demands of the clinical system often emphasize the need for exceptional efficiency. In many instances procedure rooms are not designed to accommodate anesthesia equipment, and the presence of an anesthesia machine with the ability to monitor exhaled carbon dioxide and deliver inhaled potent agents is an uncommon luxury. All of these considerations make the provision of “just another MAC” in the OOR setting a true clinical challenge that requires refined communication skills, deft clinical management, and a high degree of flexibility and accommodation. Table 24.5 shows unique aspects of OOR anesthesia practice.

<table>
<thead>
<tr>
<th>Table 24.5 Unique aspects of Out-of-OR-anesthesia.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Limited anesthesia role in patient selection and preoperative optimization</td>
</tr>
<tr>
<td>2. Unfamiliar procedures and proceduralists</td>
</tr>
<tr>
<td>3. Remote locations with limited equipment availability</td>
</tr>
<tr>
<td>4. Enhanced role of nonanesthesia support staff</td>
</tr>
<tr>
<td>5. Greater potential for miscommunication</td>
</tr>
<tr>
<td>6. High expectations for patient satisfaction, clinical efficiency, and accommodation</td>
</tr>
</tbody>
</table>
Perhaps the most common conundrum in OOR anesthesia pertains to the type of anesthetic. Depth of anesthesia is characterized by a “continuum,” from local (nothing) to general anesthesia with a protected airway. A patient who does not demonstrate purposeful responses to painful stimuli or who requires support of the airway is under general anesthesia, regardless of the medications used or type of airway management.

In many OOR cases the anesthetic may slide along that continuum from moderate sedation to general anesthesia. The key issue is not classification per se, but rather design of an anesthetic that meets the requirements of the procedure while satisfying the patient. Provided that these goals are accomplished and the expectations of the patient are appropriately set, the anesthetic is likely to be satisfactory.

**Gastrointestinal (GI) Endoscopy**

Common GI endoscopy procedures include colonoscopy, upper endoscopy, and endoscopic retrograde cholangiopancreatography (ERCP). Proceduralists typically desire a patient who does not gag or move. Patients often request absolute unconsciousness with amnesia. Procedures are typically brief (5–30 min) with a wide dynamic range of stimulus intensity, requiring near instantaneous adjustment of anesthetic depth. Most procedures are conducted on an outpatient basis and postoperative pain is typically minimal except for gaseous discomfort related to insufflation. Upper endoscopy (EGD) and ERCP require transoral placement of the endoscope. This effectively eliminates the ability to provide positive pressure ventilation via mask without complete interruption of the procedure. Likewise, ERCP is usually performed with the patient in the prone position. Maintenance of spontaneous ventilation is desirable in these circumstances or the use of high FIO₂ with preoxygenation should be considered if anesthetic management may result in intermittent apnea or hypventilation.

A wide variety of anesthetic approaches are suitable for use in GI endoscopy, and individual patient physiology is important. Amnesia and prompt emergence are important. Midazolam, propofol, etomidate, and ketamine are all reasonable agents in selected patient to achieve hypnosis and amnesia. Opioids are useful adjuncts for blunting of reflexes (gag, cough, and pain) during endoscope placement and painful procedures involving stent placement and dilation. Fentanyl (short-acting) and remifentanil (ultra short-acting) are both widely used for this purpose. Remifentanil profoundly suppresses respiration, and when used with propofol can synergistically reduce blood pressure. Ketamine also appears to have modest analgesic properties and a relatively benign
hemodynamic profile. Ketamine does not suppress respiration but may also be associated with unpleasant psychedelic side effects in a subset of patients.

Several possible anesthetic approaches are outlined in Table 24.6, although many others can be conceived.

### Electrophysiology Lab (EP)

The wide variety of procedures performed in the EP lab include pacemaker placement, external cardioversion, internal defibrillator placement and testing, and arrhythmia ablation. By definition, all patients have cardiac disease, and many have severe compromise of cardiac function with low ejection fraction, coronary artery disease, and high propensity toward unstable rhythms. Moreover, the EP lab represents a very foreign environment to the anesthesiologist. There is a large amount of equipment that surrounds the patient and often interferes with immediate access to the airway and intravenous catheterization sites.

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**Table 24.6 Anesthetic options for GI endoscopy procedures.**

<table>
<thead>
<tr>
<th>Option A:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nasal cannula oxygen 2–4 L</td>
</tr>
<tr>
<td>2. Midazolam (1–4 mg) IVP</td>
</tr>
<tr>
<td>3. Glycopyrrolate 0.1–0.3 mg IVP</td>
</tr>
<tr>
<td>4. Propofol/ketamine (1 mg/mL) infusion @ 80–150 mcg/kg/min propofol after 0.8–1.4 mg/kg bolus at induction</td>
</tr>
</tbody>
</table>

*Comment:* Low likelihood of apnea, limited hemodynamic effects, modest analgesia, and possible ketamine side effects

<table>
<thead>
<tr>
<th>Option B:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nasal cannula oxygen 2–4 L</td>
</tr>
<tr>
<td>2. Propofol infusion (120–200 mcg/kg/min) after 1–2 mg/kg induction bolus</td>
</tr>
<tr>
<td>3. Consider addition of fentanyl 25–100 mcg for highly stimulating procedures</td>
</tr>
</tbody>
</table>

*Comment:* High doses of propofol as sole agent may induce apnea when bolused, no analgesic properties, sole agent provides clean anesthetic, and rapid emergence

<table>
<thead>
<tr>
<th>Option C:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Preoxygenate with 100% ( \text{O}_2 ) via facemask</td>
</tr>
<tr>
<td>2. Deliver high ( \text{FiO}_2 ) via Mapleson circuit attached to patient with lubricated nasal trumpet</td>
</tr>
<tr>
<td>3. Propofol/remifentanil (2 mcg/mL) infusion at 60–120 mcg/kg/min propofol after 0.6–1 mg/kg bolus</td>
</tr>
</tbody>
</table>

*Comment:* High likelihood of intermittent apnea, profound analgesia and blunting of reflexes, may be best suited to chronic opioid or benzodiazepine users or patients undergoing painful procedures with high anesthetic requirements, and greater hemodynamic side effects than A or B
The interventional cardiologist commonly controls infusions of vasoactive substances, heparin, and fluids, and has specific target parameters for blood pressure, heart rate, and rhythm.

The majority of EP procedures are performed under sedation or brief (<5 min) general anesthesia. A few more complex procedures, such as ablation of atrial fibrillation and epicardial lead extraction, have high potential for significant complications, and often require an operative field that is nearly devoid of unpredictable movement. Such cases typically last for several hours and are performed under general anesthesia. In some cases, standard sedation provided by the EP team fails to provide adequate comfort or operating conditions and requires unanticipated conversion to general anesthesia. In some centers, all patients undergoing interventional EP procedures will undergo preoperative evaluation and anesthesia consent.

As with GI endoscopy, a wide range of anesthetics is compatible with anesthesia for EP procedures. For patients with preserved ejection fraction, a wide range of induction agents are appropriate, particularly for the brief periods of general anesthesia required for cardioversion or device testing. Etomidate may be used in the presence of impaired cardiac function. For maintenance of general anesthesia, low-dose inhaled potent agents are easily titrated and provide a relatively stable hemodynamic profile with reliable amnesia. The inhaled agent can be combined with low-dose fentanyl to blunt reflexes and provide any necessary analgesia. Intravenous lidocaine or other agents with intrinsic antiarrhythmic properties should be avoided during procedures involving iatrogenic induction of arrhythmias (e.g. VT ablation). Any hemodynamic impact from the anesthetic medications should be treated in consultation with the proceduralist, and vasoactive agents should not be administered without clear communication with the operative team. Dexmedetomidine is a highly specific \( \alpha-2 \) receptor agonist that produces a reduction in sympathetically mediated hemodynamic effects, moderate sedation, and modest analgesia without significant respiratory depression (see Chap. 7). In appropriately selected patients, a dexmedetomidine infusion (1 mcg/kg as a 10 min bolus then 0.3–0.7 mcg/kg/h) may be an optimal agent for sedation in interventional neuroradiology.

**Radiology**

Anesthesiology services are increasingly needed for patients undergoing diagnostic scanning or interventional procedures in the radiology suite. Issues include the use of specialized equipment, the lack of immediate access to the
patient who is in a different room and inside of a scanner, and the need to use total intravenous anesthesia in some cases. Providing anesthesia in the MRI suite is the most cumbersome due to the incompatibility of most standard anesthesia devices (e.g. pumps, blades, stethoscope, and ventilator) and monitoring devices with the magnet. The anesthesia provider must gain working familiarity with the staff, resources, and equipment prior to placement of the patient into the scanner. Similarly, for intubated patients the need for a ventilator and respiratory therapist should be anticipated prior to patient arrival in the scanning area. The anesthetic plan, concerns of the anesthesia team, and patient-specific factors should be explicitly discussed with the radiology team and staff.

Although some anesthetics in radiology involve the provision of sedation and anxiolysis for patient comfort, the greatest concern in these circumstances is respiratory depression inside the scanning device in a patient with an unsecured airway. As discussed above the clinical challenge is that moderate sedation can easily cross into an unplanned general anesthetic. Anesthetic management should include a clear rescue airway plan; sedation should be performed with continuous capnography and if possible, deep sedation should be avoided. A patient who requires aggressive sedation inside a scanner may be best managed with general anesthesia employing an LMA or endotracheal tube.

**Neuroradiology**

Patients present to the neuroradiology unit for diagnostic and interventional procedures that include cerebral angiogram and angioplasty, aneurysm coiling, functional arterio-venous malformation testing, preoperative embolization of vessels feeding intracranial lesions, preoperative assessment of carotid collateralization, and kyphoplasty for pain related to spinal compression fractures. For these delicate procedures, patients must remain still and cooperative. Movement not only risks patient injury, but also significantly impacts the quality of the collected images. Access to the airway is minimal due to the position of radiological equipment over the head. Moreover, the procedure table is frequently repositioned by sliding back and forth, and it is critical that infusion lines, airway devices, and monitors be inspected and has the freedom to accommodate such movement.

Some diagnostic procedures require patient participation, and the level of sedation must be appropriately titratable. These procedures may involve temporary vessel occlusion or intraarterial injection of barbiturate anesthetic in
the vessel(s) of interest. Generally speaking, neuroradiology procedures are minimally painful except for the vascular access of the femoral artery in the groin, which can be performed under local anesthesia. However, the procedures involve highly sensitive areas and the anesthesia team must anticipate the need for rapid conversion to general anesthesia (e.g. in case of aneurysm rupture, embolization of critical feeding vessel).

General endotracheal anesthesia is preferred for nondiagnostic embolization and coiling procedures. These procedures require absolute field quietness and have a higher risk of deleterious side effects or complications. Choice of induction and maintenance strategy should be catered to the specific patient and the pathophysiology under management. Usually, an amnestic dose of inhaled potent agent with maintenance of muscle relaxation is appropriate. Blood pressure should be maintained in an appropriate range as determined by preoperative factors (elevated intracranial pressures, evolving stroke, status of the aneurysm, and baseline hypertension) and after discussion with neurology team. Nicardipine, labetalol, and phenylephrine are all suitable vasoactive agents. Manipulation of the carotid artery (stenting/balloon angioplasty) may activate baroreceptor reflexes that could produce rapid changes in blood pressure or heart rate.

Kyphoplasty is a procedure that may be performed with general anesthesia or sedation with the recognition that patient’s prone position is likely to be uncomfortable, and the procedure itself can be quite painful.

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**Case Study**

A 20-year-old woman is scheduled for breast augmentation surgery. She attends college and works part time as a waitress, and works in the college library. She is strongly motivated to have the procedure performed as an outpatient and to return to work and minimize her time away from school and work. She is generally healthy, though she notes that she has seasonal allergies and occasional wheezing for which she takes an antihistamine and uses a metered dose inhaler (albuterol) as needed. She does not smoke, drinks alcohol on the weekends (3–4 drinks once per week), and does not use recreational drugs. She takes oral contraceptives and also has a history of motion sickness.
Is it appropriate to do this case in an outpatient surgery center? What other information do you need to decide?

Of the various criteria commonly used, she meets most: she is motivated, generally healthy, and has only moderate coexisting disease. The procedure is limited, is not associated with high blood loss, fluid shifts, or the need for drains postoperatively. She will need no special post-op monitoring, and there is no anticipated difficulty with pain control. She needs a caregiver for 24 h, and you will need to make sure her asthma symptoms are not currently active. She is at risk for PONV and needs to be counseled regarding the inability to guarantee that she will not experience nausea and vomiting at home. As with any patient presenting for surgery in any venue, you will need to perform a complete history and certainly must assess her airway. Some centers have cutoff values for maximum BMI.

Is she at high risk of postoperative nausea and vomiting (PONV)?

Yes. According to the criteria proposed by Apfel she meets 3 of 4: she does not smoke, and has a history of motion sickness (or PONV). The fourth factor, use of postoperative opioids, is something we can hope to plan to avoid. With 3 risk factors, her approximate risk of PONV is 60%.

How will you induce and maintain anesthesia?

You will follow the S.A.F.E. principles suggested by the Society for Ambulatory Anesthesia and give short acting, fast emergence drugs. Propofol for induction is a rational and popular choice. You can consider using no muscle relaxants and no intubation, maintaining the airway with an LMA and maintaining spontaneous respiration. Sevoflurane or desflurane are logical choices, given their low solubility and rapid elimination. Nitrous oxide may reduce the need for these agents and is very rapidly eliminated, but it might increase the risk of PONV. You will also avoid large doses of intraoperative opioids and use short-acting drugs such as fentanyl, sufentanil, or remifentanil. Total intravenous anesthesia is a potential alternative which can minimize the risk of PONV, but it will also generally require controlled ventilation, and often endotracheal intubation.

How will you manage postoperative pain?

Your goal is to have a comfortable patient but to minimize opioids. You should discuss local anesthetic infiltration with the surgeon and discuss
the use of NSAIDs, such as single-dose ketorolac, to augment the effect of small doses of short-acting opioids such as fentanyl.

*How will you reduce the risk of PONV?*

Given her relatively high risk for PONV, you will probably administer two- or three-drug prophylaxis. Dexamethasone and ondansetron is a popular combination. You can also consider a scopolamine patch, which has particular efficacy against motion sickness. Often patients do well in the PACU only to experience PONV on the ride home, so this is a good choice for this patient. Importantly, you should also set reasonable expectations with patient, and let her know that is acceptable to experience some nausea and vomiting, even after discharge, as long as she can take oral fluids.

*Anesthesia and emergence are uneventful and you take the patient to the PACU. When can she go home?*

She should meet the ordinary PACU discharge criteria for any patient: alert and oriented, hemodynamically stable, with reasonable control of pain and nausea. This does not imply that she must be 100% pain or nausea free, but she must be comfortable. There are also special considerations for discharge home. She needs a ride home with a responsible adult. She should be able to ambulate and take limited oral intake, which may be defined as fluids only, or fluids and light solids such as crackers. The latter varies by institution and is not an evidence-based standard. Formerly, many outpatients were required to void prior to discharge. However, many surgical patients may have reduced urine production due to the surgical stress response, drug effects, or mild hypovolemia. Many centers have therefore dropped this requirement and discharge patients with a “due to void” instruction and an understanding of what to do if she does not urinate within a few hours after discharge. Finally, she must understand her post-discharge instructions and be comfortable leaving the medical facility. You and her other physicians should have a way to reach her by telephone should any immediate follow-up be required, and she should know how to contact you and your colleagues should problems arise at home.
Suggested Further Reading


Laurito CE. Anesthesia provided at remote sites in clinical anesthesia. In: Barash PG, Cullen BF, Stoelting RK (eds) 4th edn. 3127–1346, Lippincott Williams & Wilkins


Anesthesia for Trauma and Orthopedic Surgery

Roy G. Soto

For maximum impact, it is recommended that the case study and questions found on page xxx are reviewed before reading this chapter.

Key Learning Objectives

- Review key mechanisms of injury that lead to trauma
- Learn the Glasgow coma scale and how to apply it
- Understand the approach to anesthesia for orthopedic surgery

Trauma

Introduction

Patients presenting with traumatic injuries can represent a significant challenge to many aspects of anesthesia practice, including airway, intravenous access, and hemodynamic stability. In this chapter, we will discuss the epidemiology, assessment, and specific concerns for the anesthesiologist taking care of patients with traumatic injury.

Epidemiology

Injuries are the leading cause of death in America for children and young adults, with 150,000 deaths and 450,000 new patients suffering permanent disability each year. One-third of all hospital admissions are related to injuries, and the estimated annual cost of trauma care exceeds $400 billion. Contrary to common
belief, trauma is not a random occurrence, and these patients have an increased likelihood of drug abuse, intoxication, and hepatitis/HIV infection.

Regional trauma care is organized on the premise that most patients die soon after injury, and care received in the “golden hour” after injury is most likely to reduce mortality. Level 1 and 2 trauma centers were developed in an attempt to get “the right patient to the right hospital at the right time” (Table 25.1).

### Patient Assessment

Traumatic injuries seldom occur in isolation, meaning that a dislocated shoulder following a motor vehicle accident is probably not the patient’s only injury. A number of scoring systems have been developed to reduce variability and ensure uniformity in how trauma patients are approached.

Airway/Breathing/Circulation/Disability represents the “A, B, C, and D” approach to initial assessment. The Advanced Trauma Life Support (ATLS) is taught by the American College of Surgeons and is designed to assess a patient in a standardized fashion. The patient’s clothes are removed, IV access is obtained, and the entire body is visually examined for injury.

The Glasgow Coma Scale (GCS) was developed to assess the level of neurologic injury, and includes assessments of movement, speech, and eye opening (see Table 25.2 below). Brain injury is often classified as severe (GCS ≤ 8), moderate (GCS 9–12), or minor (GCS ≥ 13).

Regardless of assessment method, it is vital to remember that these scores do not predict ease of intubation, ventilation, or reflect volume,

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### Table 25.1 Mechanisms of Injury.

<table>
<thead>
<tr>
<th>Trends revealed from the National Trauma Data Bank</th>
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<tbody>
<tr>
<td>1. The majority of reported traumas occur in young males</td>
</tr>
<tr>
<td>2. Case fatality rises with age at time of injury</td>
</tr>
<tr>
<td>3. Motor vehicle accidents are the main cause of injury in young and middle-aged patients, with falls becoming predominant in elderly patients</td>
</tr>
<tr>
<td>4. The vast majority of injuries are blunt</td>
</tr>
<tr>
<td>5. Penetrating injuries have the highest associated mortality</td>
</tr>
<tr>
<td>6. Burns result in the longest hospital stays</td>
</tr>
<tr>
<td>7. America leads the world in firearm related deaths in both adults and children, with an incidence 4× higher than any other industrialized country</td>
</tr>
<tr>
<td>8. Firearm deaths occur predominantly in African-American men</td>
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</tbody>
</table>
pulmonary, or cardiac status. In other words, a patient with a high GCS may still require urgent intubation or may be suffering myocardial ischemia due to injury stress.

**Specific Challenges**

Many hospitals routinely call anesthesia personnel to the emergency department for incoming trauma patients. As a result, anesthesia providers are frequently involved in resuscitation and airway management within minutes of the patient’s arrival. Since initial trauma care occurs on a continuum from the emergency department to operating room, many of the following discussion points are pertinent to both the specialties of emergency medicine and anesthesiology at each locale. As a result, anesthesia providers must be prepared to “work” in a potentially unfamiliar environment with a different (not necessarily better or worse) level of help and equipment than is typically available in a well-stocked trauma operating room.

### Table 25.2 Glasgow coma scale.

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<table>
<thead>
<tr>
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<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To loud voice</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal response</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused, disoriented</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate word</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
<td></td>
</tr>
<tr>
<td>Obey</td>
<td>6</td>
</tr>
<tr>
<td>Localizes</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws (flexion)</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion posturing</td>
<td>3</td>
</tr>
<tr>
<td>Extension posturing</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
</tbody>
</table>
The Trauma Arrest
Patients requiring CPR following trauma have an almost universally poor prognosis. In particular, patients presenting with blunt trauma in cardiac arrest have a mortality rate that approaches 100%. Patients who are young, otherwise healthy, and receive hospital care within a short period of their injury tend to have better outcomes. For any hope of survival, early intubation with appropriate oxygenation and ventilation, as well as aggressive fluid management are required.

The Trauma Airway
Emergency airway management of the trauma can be the single most challenging aspect of anesthesia care, and proper preparation and multiple backup plans are equally important. Airway damage, cervical spine injury, intoxication, and coexisting injury can combine to create a situation requiring experience, expertise, and luck! Fiberoptic intubation may be impossible due to airway blood or patient combativeness. LMA placement may be complicated by a full stomach. The patient may also come with a Combitube (see Chap. 9, Airway Evaluation and Management) or endotracheal tube already in place.

One plan to manage the airway of a combative trauma patient may look something like Fig. 25.1.

![Figure 25.1 Sample plan to manage the airway of a combative trauma patient. RSI = rapid sequence induction.](image-url)
If the airway appears “easy”, then one might proceed with an asleep intubation using in-line neck stabilization. If the airway appears “difficult”, one might either proceed with RSI (rapid sequence induction) with surgery backup (meaning that they are standing nearby ready to assist with a surgical airway) or try to hedge one’s bet and give a dose of intravenous or intramuscular ketamine in hopes that ventilation can be maintained, and an awake intubation technique can be used.

If a patient is stable from a respiratory standpoint and you are truly concerned about being able to manage the airway, consider bringing the patient to the operating room for the intubation. There you can manage your equipment more easily, have the assistance of those who are used to complex airway management, and have a well lit, nonchaotic environment to work in.

Remember: “Good judgment comes from experience, and experience comes from bad judgment.”

Clearing the C-spine

A typical scenario: A patient comes to the operating room from the CT scanner for urgent splenectomy. He has been poked, prodded, and scanned prior to arrival, but is still wearing a C-collar. Is it OK to take it off? Just how does one clear the C-spine definitively? The short answer is that all imaging studies must be negative, and the patient must be able to clearly tell you that nothing hurts (to rule out a distracting injury). That said, patients frequently cannot do that. So in this case, to clear a C-spine, one would need:

1. Cleared films (X-ray, CT, and/or MRI). Note that following an MVA the most likely injuries are to C1 > C5 > C6 > C7, and following a fall C5 > C6 > C7
2. The patient has to be awake, coherent, and cooperative
3. The patient cannot be intoxicated (alcohol, drugs, or otherwise)
4. The patient cannot have a distracting injury that is causing more pain than he may have in his neck
5. The patient cannot have received a significant dose of opioids (just how much is significant is unclear, but if the patient is somnolent, do not trust that opioids have not blunted subjective pain complaints)
6. The patient cannot have tenderness to neck palpation or tenderness to gentle neck flexion/extension

If all of these criteria are not met (they rarely are), then one might still proceed with a rapid sequence induction with in-line stabilization.
(given a normal-looking airway). The job of the person holding stabilization is to not only hold the head/neck in neutral position, but also to inform the person intubating if the neck is moving due to vigorous laryngoscopy. Note that there will be another person holding cricoid pressure during this process, and therefore the anterior portion of the cervical collar should be removed during intubation.

Head Trauma
Patients presenting with head trauma can pose a difficult challenge to anesthesia providers. Laryngoscopy and succinylcholine are associated with increases in intracranial pressure (ICP), and sedation and hyperventilation result in an increase in ICP. The goals include maintaining cerebral perfusion pressure >60 mmHg (MAP minus ICP or CVP), protecting potentially ischemic brain tissue near the area of injury, and keeping the patient’s ICP as low as possible.

Bleeding
Trauma patients frequently need volume resuscitation, blood, and coagulation factors while surgeons attempt to fix whatever was broken, ruptured, or eviscerated. Trauma patients need to have large-bore IV access, ideally via central catheters (see Chap. 15, IV, Arterial and Central Line Gastric Tube Placement Techniques). Consider placing a central line above and below the suspected area of injury.

Progressive hypothermia, coagulopathy, and hypovolemia/acidosis (the so-called “lethal triad”) result in progressive mortality. Resuscitation efforts must be geared towards avoiding all three, and aggressive volume replacement, room and fluid temperature control, and blood product replacement must be addressed simultaneously. Common problems that arise during trauma resuscitation are:

- **Hypothermia**: Trauma ORs should be kept warm, all fluids should be warmed (ideally starting in the ER), and irrigation should be near body temperature. Rapid fluid infusers do a good job of warming fluids quickly, and should be used whenever possible for massive transfusion.

- **Hypovolemia and acidosis**: Commonly administered crystalloid solutions are acidic, with 0.9% normal saline having a pH of 5.0, and Lactated Ringers having a pH of 6.2 (see Chap. 14, Electrolytes, Fluid, Acid–Base and Transfusion Therapy). However, large volumes of crystalloid resuscitation can result in metabolic acidosis. Colloids have not been consistently shown to be better or worse than
crystalloids, and many providers will mix and match in an attempt to avoid giving too much of any one thing. In the face of uncontrolled bleeding, evidence suggests that the goal should be a mean systolic pressure of \( \approx 70–80 \text{ mmHg} \), although existing head trauma and CPP should be kept in mind (Fig. 25.2).

- **Coagulopathy:** Crystalloid/colloid resuscitation can result in dilutional coagulopathy, which can worsen coagulopathy from blood replacement or hypothermia. Rather than giving factors only if laboratory values are abnormal, most hospitals have adopted a massive transfusion protocol aimed at replacing factors at preset intervals. **Recombinant Factor VIIa** has been used by some in trauma resuscitation, and it appears that survival may be improved with its use. The high cost of the drug (\( \approx $5,000/dose \)), however, limits its cost-effectiveness. Finally, calcium replacement must be considered during massive transfusion as calcium is a cofactor in both the intrinsic and extrinsic clotting pathways, and citrate in transfused blood can result in hypocalcemia (see Chap. 14, Electrolytes, Fluid, Acid–Base and Transfusion Therapy). Below is a sample Trauma Blood/Massive Transfusion Protocol (Tables 25.3 and 25.4).

A final word about bleeding. Surgeons are very good at packing wounds to stop them from bleeding, and at times it is important to ask the surgeons to “stop working, pack the wound, and allow us to get caught up on blood loss (please).” Similarly, there are times when an injury should be packed and the patient sent to the ICU, with a plan to bring the patient back another day for a staged repair.
Orthopedic Surgery

Not all trauma is orthopedic, and not all orthopedic procedures result from trauma. Here we shall focus on the special challenges in care of the routine, scheduled, elective orthopedic patient.

Choice of Anesthetic

Orthopedic procedures lend themselves to a wide variety of regional techniques, many of which are detailed in Chap. 13. The question that patients frequently ask is “Are blocks better and safer than general anesthesia?” Unfortunately, the answer is far from clear. A good general anesthetic is always better than a bad regional anesthetic. Does regional anesthesia reduce patient morbidity or improve patient satisfaction?

The answer is an unequivocal “it depends.” Patients with less pain are obviously more satisfied, but again a poorly functioning block (or regional/epidural catheter) will increase pain and aggravation, as well as patient and surgeon dissatisfaction. Ultrasound-guided catheter techniques are revolutionizing ambulatory orthopedic procedures in some hospitals, while others are abandoning

<table>
<thead>
<tr>
<th>Table 25.3 Trauma blood protocol.</th>
</tr>
</thead>
<tbody>
<tr>
<td>• O-neg × 2 units, then</td>
</tr>
<tr>
<td>• O-pos if</td>
</tr>
<tr>
<td>o Male</td>
</tr>
<tr>
<td>o Obviously non-child bearing age</td>
</tr>
<tr>
<td>• Continue with O-neg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 25.4 Massive transfusion protocol.</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Thaw 4 units FFP and 1 unit Cryoprecipitate</td>
</tr>
<tr>
<td>• Crossmatch 6 units PRBC and 1 unit Platelets</td>
</tr>
<tr>
<td>• Deliver to OR</td>
</tr>
<tr>
<td>– Cooler #1: 4 units PRBC</td>
</tr>
<tr>
<td>– Cooler #2: 2 units PRBC + 3 units FFP</td>
</tr>
<tr>
<td>– Bucket: Cryoprecipitate + Platelets</td>
</tr>
<tr>
<td>• Continue to replenish coolers/bucket until told to stop</td>
</tr>
</tbody>
</table>

Table 25.3 Trauma blood protocol.

- O-neg × 2 units, then
- O-pos if
  - Male
  - Obviously non-child bearing age
- Continue with O-neg

Table 25.4 Massive transfusion protocol.

- Thaw 4 units FFP and 1 unit Cryoprecipitate
- Crossmatch 6 units PRBC and 1 unit Platelets
- Deliver to OR
  - Cooler #1: 4 units PRBC
  - Cooler #2: 2 units PRBC + 3 units FFP
  - Bucket: Cryoprecipitate + Platelets
- Continue to replenish coolers/bucket until told to stop
the technique due to failure rates, cost, or administrative complexity. Neuraxial anesthesia (with or without general anesthesia) has been shown to reduce the incidence of DVT (deep venous thrombosis) and potentially resultant PE (pulmonary embolus) in knee and hip surgery, although overall mortality does not seem to be affected in the long run. Bleeding may also be reduced following hip surgery under epidural anesthesia, and recovery room delirium in elderly patients may be less with regional compared to general techniques.

In any event, the skill of the anesthesia provider, wishes and expectations of the patient, presence or absence of peri procedure anticoagulation, requirement for immediate postoperative nerve assessment, duration of the procedure, and degree of anticipated post-operative pain should all be taken into account when discussing anesthetic choice with the patient. Table 25.5 lists the most common orthopedic procedures and their respective anesthetic choices/considerations. Details of regional anesthetics are discussed in Chap. 13: (Anesthetic Techniques: Regional)

**Management of Postoperative Pain**

Pain following orthopedic injury and surgery can be severe, and a well-developed perioperative pain plan is important in the successful management of these patients. As mentioned previously, regional anesthesia can be successfully used, and epidural and regional analgesia catheters can be kept in place for days following surgery. Opioids are still the mainstay of pain therapy, but multimodal

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Anesthetic considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hand surgery</td>
<td>Frequently performed with local anesthesia/sedation only. Also can use Bier or axillary block.</td>
</tr>
<tr>
<td>Wrist surgery</td>
<td>Infraclavicular block.</td>
</tr>
<tr>
<td>Elbow surgery</td>
<td>Difficult to anesthetize. Infraclavicular or axillary block may be adequate.</td>
</tr>
<tr>
<td>Shoulder surgery</td>
<td>Interscalene block (scatheter) provides excellent surgical anesthesia and postoperative analgesia.</td>
</tr>
<tr>
<td>Hip surgery</td>
<td>Spinal/epidural block.</td>
</tr>
<tr>
<td>Knee surgery</td>
<td>Intra-articular local anesthetics for arthroscopy. Spinal ± continuous femoral block or epidural block for knee replacement.</td>
</tr>
<tr>
<td>Ankle surgery</td>
<td>Popliteal fossa block (scatheter) provides excellent surgical anesthesia and postoperative analgesia.</td>
</tr>
<tr>
<td>Foot/toe surgery</td>
<td>Frequently performed with local anesthesia/sedation only. Also can use ankle block.</td>
</tr>
</tbody>
</table>

Table 25.5 Anesthetic options for orthopedic procedures.
management is much more prevalent now. Opioids are often supplemented with anti-inflammatory agents (ketorolac IV or oral COX-2 inhibitors), steroids (dexamethasone), local anesthetics (locally and regionally), acetaminophen, and anticonvulsants (pregabalin or gabapentin). Preemptive analgesia (preoperative prophylaxis for postoperative opioid sparing effect) may have some benefit in orthopedic patients, although further studies are still needed in this area.

Special Considerations
Positioning Injuries
Orthopedic procedures frequently place patients in positions that could potentially lead to nerve or musculoskeletal injury. Regardless of the position, it is important to pad all pressure points and ensure that no undue stretch or compression is placed on joints or neurologic plexes (e.g. axillary rolls can avoid brachial plexus compression). A good rule of thumb is that if you think that your body would be uncomfortable in a particular position, then the patient probably should not be placed that way!

Tourniquet Issues
Tourniquets are used frequently in orthopedic surgery to reduce surgical bleeding and improve operative conditions. Two common problems seen with tourniquet use are pain and reperfusion injury. Tourniquet pain typically begins approximately 45 min after inflation and is frequently described as aching or burning, and is associated with progressive hypertension (which can also be seen under general anesthesia) that resolves quickly with cuff deflation. Prolonged tourniquet inflation (>2–3 h) can also be associated with peripheral nerve injury. Reperfusion injury is due to the release of cold acidotic blood back to the central circulation following tourniquet release, resulting in tachycardia and hypothermia.

Methylmethacrylate Cement
Cement is used to bind joint prostheses to bone. During cement mixing and insertion, the substance expands as it hardens, greatly increasing pressure in the affected bony cavity. As a result, solid cement, marrow, and fat can be forced into the vasculature resulting in microemboli causing hypotension, hypoxemia, and tachycardia – all undesirable, especially in the typical older, sicker orthopedic patients undergoing these procedures. The liquid cement monomer can also
cause direct vasodilation with resultant hypotension and tachycardia. Fluids, vasopressors, and light anesthesia can help lessen these effects.

**Fat Embolism Syndrome**
Patients with long bone fractures are at risk of suffering from this syndrome, characterized by **truncal petechiae, dyspnea/hyoxemia, and mental status changes**. Symptoms typically present within 3 days of injury, and are thought to be due to fatty globules released into the circulation due to bony disruption. Treatment involves bone and joint immobilization to avoid further release and supportive care. The pulmonary and neurologic manifestations of this syndrome can complicate perioperative management, and fat embolism syndrome is associated with a significant increase in patient mortality.

**Blood Loss**
As with trauma patients, elective or urgent orthopedic repairs can be associated with significant amounts of blood loss and coagulopathy. A large amount of blood can be lost into the soft tissues of the thigh or upper arm, making estimates of blood loss difficult. Cell salvage techniques have been used successfully during joint replacement, although care must be taken to avoid its use if the wound is infected or if cement use is imminent.

![Figure 25.3  Hip fracture locations.](image-url)
The location of a hip fracture will significantly impact the amount of blood loss that occurs: subcapital < transcervical < neck base < intertrochanteric < subtrochanteric (see Figure 25.3).

In conclusion, as with all difficult anesthetic cases, vigilance, preparation, and communication with our surgical colleagues is vital to ensure patient safety during trauma and orthopedic cases.

**Case Study**

A 23-year-old male was an unrestrained driver in an automobile crash in an older car without airbags. He and his friends had recently left a party where he had consumed “a couple of beers.” He hit the steering wheel on impact and has multiple contusions on his chest and complains of chest pain with respiration. His left shoulder is dislocated. He also has a broken tibia and is suspected of having a splenic injury. He did not lose consciousness at the scene. His breath smells of alcohol, and he is snoring loudly. He awakens with vigorous shouting and is somewhat combative and confused. He complains of pain in the affected injured area when examined and can move all four extremities on command. He is an otherwise previously healthy college student.

What is his Glasgow Coma Scale score?
The GCS is calculated as 3 for eye opening + 4 for best verbal response + 6 for best motor response = 13.

The patient arrives from the emergency department with two upper extremity peripheral IVs in place infusing room temperature lactated Ringer’s. Do you need additional access? How will you modify the resuscitation strategy in the OR?
You probably have enough IV access for induction of anesthesia. You may want central access for postoperative care, given the possibility of chest trauma and lung contusion and to have “one above and one below” the injuries. You will start warming all intravenous fluids. You will send (or check if already sent) laboratory studies to determine red cell, clotting factor, and platelet needs. You will consider pre-lab administration of blood products per trauma protocols derived from the experience of military trauma resuscitation in Iraq. This protocol, still controversial in civilian trauma, particularly blunt trauma, dictates the use of early RBC, FFP, and platelet transfusions in a 1:1:1 ratio until laboratory studies have returned.
Studies of the aorta have led the surgeons to observe rather than operate for this injury. The cervical spine was found free of fractures or dislocations on head and neck CT scan. The patient is still wearing a cervical collar placed at the scene. He does not complain of neck pain. Can you now remove it prior to facilitate management of the airway?

No. The cervical spine is not cleared yet. First, soft tissue injury is not ruled out because CT does not image soft tissues adequately. Furthermore, the patient’s intoxication and other injuries make the verbal response that his neck does not hurt unreliable. You will need to manage the patient as having a potentially unstable spine. You can remove the anterior portion of the collar after induction to facilitate intubation with manual in-line stabilization.

The patient has not consumed solid food for 8 h and last drank liquids more than 2 h ago. How will you induce anesthesia and secure the airway?

Examine the airway first! Trauma patients are sometimes quite difficult to intubate, and in-line stabilization has been shown to make even easy airways more difficult to manage. Awake intubation may be required if the airway appears challenging, and at any rate you may wish to have additional personnel and equipment available. Assuming you believe the airway exam to be reassuring, you will remove the anterior portion of the collar as anesthesia is induced, and a second experienced operator will provide manual in-line stabilization of the cervical spine to prevent anterior motion with laryngoscopy. In any trauma patient, you should consider having surgical backup to provide an emergency surgical airway if you encounter difficulty. You will treat the patient as a “full stomach” because of the history of trauma, despite the patient’s NPO status. Indeed, the single highest risk group for pulmonary aspiration of gastric contents is trauma patients; some evidence suggests the incidence may be as high as 30%. A rapid sequence intubation with cricoid pressure, generally with thiopental or ketamine and succinylcholine, and placement of a cuffed endotracheal tube without mask ventilation, is customary. Although still considered the standard of care, there are now substantial controversies regarding the efficacy of both in-line stabilization and cricoid pressure. The former has been shown to increase the force required to perform laryngoscopy and to actually increase subluxation of the injured spine in cadaver models. The latter has been shown in imaging studies not to occlude the esophagus in the majority of cases, but instead to displace the esophagus laterally.
What other goals will you have during anesthesia for the case?
In trauma, you hope to avoid the so-called lethal triad of hypothermia, coagulopathy, and hypovolemia. You will keep the patient warm by increasing the room temperature, using convective warmers during the case, even if you have to rearrange them periodically during the procedure, use fluid warmers, and use a passive humidifier in the breathing circuit. You will treat hypovolemia and acidosis with crystalloids, possibly colloids, and blood products. You will also probably place an arterial line and check ABGs periodically. You will check coagulation studies frequently and treat coagulopathy with plasma, cryoprecipitate (for fibrinogen), and platelets as needed. Recombinant factor VIIa (rFVIIa) has been used in cases of refractory coagulopathy with some success (and great expense). Depending on the intraoperative course, you may consider postoperative intubation if fluid shifts are extreme and you suspect airway edema, if ventilation or oxygenation is difficult, if there is ongoing hemodynamic instability, or if there are signs of lung contusion or ARDS at the end of the case.

Suggested Further Reading


Section VI

Postoperative Considerations
For maximum impact, it is recommended that the case study and questions found on page xxx are reviewed before reading this chapter.

Key Learning Objectives
- Understand the basic neurophysiology of pain
- Understand the types of acute and chronic pain (i.e. nociceptive, inflammatory, neuropathic, and dysfunctional)
- Learn the common pain syndromes encountered in the pain clinic, and describe the basic treatment options

Introduction
Pain medicine is a subspecialty composed of anesthesiologists, neurologists, psychiatrists, as well as physical medicine and rehabilitation physicians. The field focuses on the management of patients with both acute and chronic pain arising from physiologic, structural, and psychological pathology.

Basic Pain Sensation in the Normal Individual
Pain, as defined by the International Association for the Study of Pain, is “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” Sensation of pain can be divided into four steps: transduction, transmission, modulation, and
perception. In transduction, the ability of the body to sense noxious stimuli (nociception) depends on the activation of nociceptors (pain receptors). These receptors are divided into thermal, mechanical, and polymodal nociceptors. Thermal receptors are excited by extremes of temperature, mechanical receptors respond to sharp objects that penetrate, squeeze, or pinch, while polymodal receptors respond to the destructive mediators of thermal, mechanical, and chemical stimuli. The chemical stimuli include potassium, serotonin, bradykinin, histamine, prostaglandins, leukotrienes, or substance P, which may lead to activation or sensitization of the polymodal nociceptors.

Following transduction, the nociceptor signal is translated into an electrical signal which allows for transmission of the stimuli via the peripheral nerves. Peripheral nerves are typically classified by their primary function (motor or sensory), diameter and speed of conduction velocity (see Table 26.1). Pain pathways are typically mediated through A delta and C fibers via the dorsal root ganglion and then transmitted through one of three major ascending nociceptive pathways (spinothalmic, spinoreticular, or spinomesencephalic) as shown in Fig. 26.1.

Modulation of pain (suppression or worsening of a painful stimulus) occurs either peripherally at the receptor, at the level of the spinal cord or in

<table>
<thead>
<tr>
<th>Fiber class</th>
<th>Diameter (µm)</th>
<th>Myelin</th>
<th>Conduction velocity (m/s)</th>
<th>Innervation</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>A alpha</td>
<td>12–20</td>
<td>+++</td>
<td>75–120</td>
<td>Afferent to skeletal muscle</td>
<td>Motor and reflexes</td>
</tr>
<tr>
<td>A beta</td>
<td>5–12</td>
<td>+++</td>
<td>30–75</td>
<td>Afferent from cutaneous mechanoreceptors</td>
<td>Vibration, light touch and pressure</td>
</tr>
<tr>
<td>A gamma</td>
<td>3–6</td>
<td>++</td>
<td>12–35</td>
<td>Efferent to muscle spindles</td>
<td>Muscle tone</td>
</tr>
<tr>
<td>A delta</td>
<td>1–5</td>
<td>++</td>
<td>5–30</td>
<td>Afferent pain and thermoreceptors</td>
<td>“Fast”, sharp, intense, lancinating pain, touch and temperature</td>
</tr>
<tr>
<td>B</td>
<td>&lt;3</td>
<td>+</td>
<td>3–15</td>
<td>Preganglionic sympathetic efferent</td>
<td>Autonomic function</td>
</tr>
<tr>
<td>C</td>
<td>0.2–1.5</td>
<td>–</td>
<td>0.4–2.0</td>
<td>Afferent pain and thermoreceptors</td>
<td>“Slow”, dull, burning, achy pain, touch, pressure, temperature, postganglionic autonomic</td>
</tr>
</tbody>
</table>
supraspinal structures (i.e. the brain stem, thalamus, or cortex). Finally, the **perception** of pain takes place at the level of the thalamus, somatosensory cortex, anterior cingulate gyrus, insula, cerebellum, and frontal cortex. The thalamus and somatosensory cortex are thought to allow for the localization of pain, while the anterior cingulate gyrus is involved in the emotional response to the stimulus. The insula, cerebellum, and frontal cortex allow for one to remember and to learn from a painful experience and to develop avoidance behavior.

**General Pain Definitions**
When discussing acute and chronic pain, it is important to have a basic battery of definitions to express the type and description of pain a patient is experiencing.

**General Pain Types**

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nociceptive pain</td>
<td>Normal, acute pain perception evoked by short-lasting noxious stimuli in intact tissue, in the absence of peripheral or central sensitization.</td>
</tr>
<tr>
<td>Inflammatory pain</td>
<td>Pain following tissue injury but with no neural injury.</td>
</tr>
<tr>
<td>Neuropathic pain</td>
<td>Pathophysiologic state of pain after neural injury resulting in peripheral and central reorganization</td>
</tr>
</tbody>
</table>

**Abnormal Pain Descriptor Definitions**

<table>
<thead>
<tr>
<th>Descriptor</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allodynia</td>
<td>The perception of pain by a stimulus that is not normally painful</td>
</tr>
<tr>
<td>Hyperalgesia</td>
<td>The enhanced perception of pain by a normally painful stimulus</td>
</tr>
<tr>
<td>Dysesthesia</td>
<td>Abnormal sensations experienced in the absence of stimulation</td>
</tr>
<tr>
<td>Paresthesia</td>
<td>An abnormal sensation (e.g. burning, pricking, tickling, or tingling)</td>
</tr>
</tbody>
</table>

**Acute Versus Chronic Pain**
The clinical definition of acute versus chronic pain is determined in a temporal fashion with an arbitrary timeframe of 3–6 months defining the cutoff point between acute versus chronic.

Acute pain can be defined as a noxious stimulus caused by injury or abnormal functioning of viscera or musculature. It is usually noted following posttraumatic, postoperative, obstetrical, and acute medical illnesses (i.e. myocardial infarction or nephrolithiasis). It is typically classified as **somatic** or **visceral** in nature. **Somatic pain** is caused by the activation of nociceptors in the skin, subcutaneous tissues,
and mucous membranes. This pain is typically well localized and described as a sharp, throbbing or burning sensation. **Visceral pain** arises from injury of the organs and is typically described as dull, distention, achy and is poorly localized. Acute pain follows the pathways listed above and will resolve within seconds to weeks following resolution of the insult.

Chronic pain can be secondary to lesions of peripheral nerves, the spinal cord, or supraspinal structures. Chronic pain can be complicated by many psychological factors such as attention seeking behavior, and emotional stresses that can precipitate pain (cluster headaches), and pure psychogenic mechanisms.

The types of acute and chronic pain are subdivided into four categories: **nociceptive**, **inflammatory**, **neuropathic**, and **dysfunctional**. **Nociceptive pain** occurs through suprathreshold stimulation of pain receptors and typically serves as a protective mechanism. Typically, no injury or changes to the nervous system are seen in nociceptive pain. This type of pain is typically seen in the acute setting of trauma or following surgery. The pain type works as an adaptive mechanism to allow for protection of the injured body part. Nociceptive pain can be chronic in nature as is seen in certain pathologic states such as osteoarthritis where destruction of the joint can lead to stimulation of the nociceptors with movement.

**Inflammatory pain** is secondary to mediators (e.g. bradykinin, serotonin) released by injured tissues and inflammatory cells. These mediators lead to a decreased threshold for the perception of pain secondary to changes in the peripheral and central nervous system. This pain can be either acute following trauma or surgery or chronic in the setting of cancer or osteoarthritis and as nociceptive pain. Upon the removal of inflammation, the hypersensitivity will typically resolve.

**Neuropathic pain** is secondary to a lesion of the peripheral or central nervous system. These pathologic states can include diabetic neuropathy, thalamic strokes, and postherpetic neuralgia. All neuropathic pain syndromes have positive signs and symptoms (e.g. allodynia, hyperalgesia) and negative symptoms (i.e. weakness, sensory loss, and decreased reflexes). As opposed to inflammatory pain, neuropathic pain will remain long after the resolution of the inciting insult.

**Dysfunctional pain** is a diagnosis of exclusion where no noxious stimuli, inflammation or pathologic lesion can be elucidated. Common diseases included under this heading include fibromyalgia and irritable bowel syndrome.
Treatment of Pain

Acute Pain

Pain is often treated utilizing a *multimodal* approach, meaning multiple treatment methods may be combined to provide analgesia, with the hope of decreasing pain and opioid usage. The treatment of acute pain can often begin prior to the initial surgical insult. In the preoperative period, *preemptive analgesia* is often utilized to decrease or stop nociceptive input. Nonsteroidal anti-inflammatory drugs (NSAIDs) such as celecoxib (PO), ketorolac (IV), and ibuprofen (PO) or acetaminophen can be used preoperatively in combination with other medications such as gabapentin to prevent central sensitization. The main advantage of celecoxib and other cyclooxygenase-2 (COX-2) inhibitors over other NSAIDs include the decreased risk of gastrointestinal bleeding, but other adverse events such as myocardial infarction, stroke, allergic reaction to sulfa, and renal issues may be seen with the use of COX-2 inhibitors.

Preemptive analgesia can also be obtained through neuraxial and regional techniques, such as peripheral nerve blocks of the femoral nerve, and brachial plexus. In those patients with moderate to severe pain, opioid analgesics such as hydromorphone or morphine may be used in combination with acetaminophen or NSAIDs for analgesia. Surgeons may aid in providing pain relief through infiltration of local anesthetics such as lidocaine or bupivacaine at the surgical site.

In those patients not able to take oral medications postoperatively, patient controlled analgesia (PCA) devices allow patients to deliver pain medication through the pressing of a button which allows the medication to be delivered via an intravenous route or an epidural catheter. These devices typically allow patients to deliver a predetermined amount of pain medicine at specific time intervals. There is a lockout period in which the patient can attempt to deliver pain medication, however, none will be given to prevent overdosing on opioid pain medication. A continuous (basal) rate may also be added to provide a baseline level of analgesia without the patient needing to administer the medication.

When assessing postoperative pain, a verbal numeric scale is typically used. The scale typically ranges from 0 to 10 with 0 representing no pain and 10 representing the worst pain imaginable. Important qualitative descriptors of pain to assess are the location, radiation, and the quality (sharp or dull) of the pain.
Chronic Pain

Treatment methods for chronic pain patients are multimodal and include the use of non-narcotic pain medications such as NSAIDs, opioid analgesics, antidepressants, anticonvulsants, and multiple interventional pain procedures. The most common interventional pain procedures are listed in Table 26.2. Additionally, physical therapy, psychiatric evaluation and treatment, and surgical intervention are often coordinated through the pain clinic. Pain physicians are also involved in end-of-life care issues.

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Target</th>
<th>Mechanism</th>
<th>Indicated pain syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidural steroid injection</td>
<td>Nerve root</td>
<td>Injection of steroid to decrease inflammation surrounding the nerve root</td>
<td>Hemiated discs, spinal stenosis, foraminal stenosis</td>
</tr>
<tr>
<td>Medial branch block</td>
<td>Medial branch of dorsal ramus</td>
<td>Local anesthetic injection</td>
<td>Diagnostic test to determine if facet arthropathy is the cause of low back pain</td>
</tr>
<tr>
<td>Radiofrequency ablation</td>
<td>Medial branch of the posterior division of the spinal nerve</td>
<td>Coagulative destruction of the medial branch nerve</td>
<td>Therapeutic intervention if facet arthropathy is determined to be the cause of low back pain after medial branch block</td>
</tr>
<tr>
<td>Trigger point injection</td>
<td>Trigger points</td>
<td>Local anesthetic blocking of sensation from trigger point</td>
<td>Myofascial pain</td>
</tr>
<tr>
<td>Spinal cord simulator</td>
<td>Posterior column of spinal cord</td>
<td>1. Decreased nociceptive input and hyperexcitability through increased neurotransmitter (i.e. GABA and adenosine) in neuropathic pain. 2. Increase coronary blood flow through alteration of sympathetic tone</td>
<td>Neuropathic pain, angina, peripheral ischemic pain</td>
</tr>
<tr>
<td>Intrathecal pumps</td>
<td>Intrathecal space</td>
<td>Decreasing systemic dose of medications such as opioids, thus decreasing side effects</td>
<td>Cancer pain patients</td>
</tr>
<tr>
<td>Neurolytic blocks</td>
<td>Celiac plexus, trigeminal ganglion, lumbar sympathetic chain</td>
<td>Destruction of nerve/plexus via phenol, alcohol or RFA</td>
<td>Palliative care patients</td>
</tr>
<tr>
<td>Stellate ganglion block</td>
<td>Stellate ganglion</td>
<td>Local anesthetic blocking of sympathetic efferent nerves</td>
<td>Complex regional pain syndrome</td>
</tr>
</tbody>
</table>
Common Chronic Pain Medications Classifications

Opioids
The opioids are a diverse classification of medication that typically provide analgesic effect via actions on the $\mu$, $\delta$, and $\kappa$ opioid receptors. The receptors are most abundant in the dorsal horn of the spinal cord and also in the dorsal root ganglion and peripheral nerves. Various natural and synthetic formulations and routes of delivery exist for these medications, including oral, intravenous, buccal, transdermal, and intrathecal. The most common oral agents are listed in Table 26.3. The major side effects of opioids include constipation, nausea, vomiting, pruritus, sedation, and respiratory depression.

Some of the major challenges surrounding opioids include those of tolerance, physical dependence, withdrawal, and addiction. Tolerance is defined as a fixed dose of an opioid providing less analgesia over time that may lead to escalating doses of narcotics to achieve the same pain relief.

Physical dependence is a physiologic state which is manifest by abruptly stopping opioid medications which then results in a withdrawal state. Opioid withdrawal presents with irritability, anxiety, insomnia, diaphoresis, yawning, rhinorrhea, and lacrimation. As time progresses, the symptoms may include fevers, chills, myalgias, abdominal cramping, diarrhea, and tachycardia. Opioid withdrawal is self-limiting and can typically last 3–7 days.

As opposed to physical dependence, addiction is defined by opioid use resulting in physical, psychological, or social dysfunction and continued use of the opioid despite the overlying issues. Behaviors that are most indicative

<table>
<thead>
<tr>
<th>Opioids</th>
<th>Half-life</th>
<th>Duration (h)</th>
<th>Equianalgesic oral doses (mg)</th>
<th>Initial dose (mg)</th>
<th>Dosing interval (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codeine</td>
<td>3</td>
<td>3–4</td>
<td>80</td>
<td>30–60</td>
<td>4</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>2–3</td>
<td>2–3</td>
<td>2</td>
<td>2–4</td>
<td>4</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>1–3</td>
<td>3–6</td>
<td>10</td>
<td>5–7.5</td>
<td>4–6</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>2–3</td>
<td>3–6</td>
<td>7</td>
<td>5–10</td>
<td>6</td>
</tr>
<tr>
<td>Methadone</td>
<td>15–30</td>
<td>4–6</td>
<td>10–20</td>
<td>20</td>
<td>6–8</td>
</tr>
<tr>
<td>Morphine</td>
<td>2–3.5</td>
<td>3–4</td>
<td>10</td>
<td>10–30</td>
<td>3–4</td>
</tr>
<tr>
<td>Propoxyphene</td>
<td>6–12</td>
<td>3–6</td>
<td>43–45</td>
<td>100</td>
<td>6</td>
</tr>
<tr>
<td>Tramadol</td>
<td>6–7</td>
<td>3–6</td>
<td>40</td>
<td>50</td>
<td>4–6</td>
</tr>
</tbody>
</table>

Table 26.3 Common oral opioid pharmacodynamics and dosing.
of addictive behaviors are buying street drugs, stealing money to obtain drugs, attempting to obtain opioids from multiple sources, acts of prostitution to obtain drugs, forging prescriptions, and selling prescription drugs.

**Alpha-2-Agonist: (Tizanidine)**
The alpha-2-agonist tizanidine is commonly used in pain medicine as a muscle relaxant. It causes less significant blood pressure changes compared to clonidine, but can lead to drowsiness.

**Anticonvulsants: (Gabapentin, Carbamazepine, and Oxcarbamazepine, Pregabalin)**
The anticonvulsants work through very diverse mechanisms of actions, including modulation of voltage-gated calcium channels, sodium channels, GABA, and glutamine receptors. FDA-approved pain indications include trigeminal neuralgia (carbamazepine), post herpetic neuralgia (gabapentin, pregabalin), diabetic neuropathy (pregabalin), fibromyalgia (pregabalin), and migraine prophylaxis (divalproex, topiramate).

**Tricyclic Antidepressants: (Nortriptyline, Amitriptyline)**
Tricyclic antidepressants (TCA) contribute to the improvement in pain symptoms through their actions on multiple sites, including serotonergic, noradrenergic, opioidergic, NMDA receptors, adenosine receptors, sodium channels, and calcium channels. The effects of TCAs can include elevation of mood, normalization of sleep patterns, and muscle relaxation. These agents are used for the treatment of neuropathic pain syndromes such as postherpetic neuralgia, diabetic neuropathy, pain secondary to spinal cord injury, cancer-related neuropathic pain, and other pain syndromes such as low back pain, osteoarthritis, and fibromyalgia. The side effects of this class of medications can include dry mouth, drowsiness, dizziness, weight gain, orthostatic hypotension, and lethargy.

**Serotonin-Norepinephrine Reuptake Inhibitors: (Venlafaxine, Duloxetine)**
Serotonin-norepinephrine reuptake inhibitors (SNRI), as their class implies, block the reuptake of norepinephrine and serotonin. Duloxetine is the first antidepressant to have a specific pain indication (diabetic neuropathy) in the United States. These medications have also been demonstrated to be useful in the treatment of fibromyalgia. The side effect profile tends to be lower in the SNRIs than the TCAs.
Cancer Pain
Cancer pain is typically treated in a stepwise fashion via the World Health Organization (WHO) analgesic ladder with the goal of maintaining oral administration of analgesics to allow for the patient to have simplicity, independence, convenience, and lower cost. This is outlined in Fig. 26.2. Mild pain is treated with non-opioids such as NSAIDs and other adjuvants. As the patient’s pain level increases or persists, opioids are added and titrated to the patient’s comfort.

Common Pain Syndromes
Spinal Stenosis
Spinal stenosis is a narrowing of the spinal canal secondary to congenital and acquired pathologies such as disc herniations, facet arthropathy, bone spurs, and ligamentous hypertrophy. It may often lead to low back pain and leg pain, which is worse with standing or walking downhill. Diagnosis can be made via MRI. Treatment ranges from epidural steroid injections, physical therapy, and NSAIDs to surgical decompression via laminectomy and other approaches.
Radicular Pain
The pathology behind radicular pain can be secondary to narrowing of the intervertebral foramina, which leads to compression of the exiting nerve roots. This may be caused by intervertebral disc herniation, osteophyte formation, or spondylolisthesis (a defect in the pars interarticularis). Symptoms typically follow a dermatomal distribution of the exiting nerve root and may manifest with pain, numbness, weakness, and reflex changes. Diagnosis is made through MRI and electromyography. Treatment ranges from epidural steroid injections, physical therapy, NSAIDs, and surgery.

Facet Arthropathy
Facet arthropathy is another cause of chronic low back pain. The facet joints are the articulating bodies of the spine and may develop arthritis over time. Pain may radiate into the scapula, buttocks, or posterior thighs. Diagnosis of the facet joint as the main cause of the patient’s pain may be made through a medial branch block which entails injecting local anesthetic and steroid on the medial branch of the posterior primary division of the spinal nerve. If the patient receives pain relief, radiofrequency ablation may be used at a later time to ablate the nerve.

Discogenic Pain
Discogenic pain is a pathologic process involving the intervertebral disc and often presents in the center of the back, buttocks, or posterior thighs and is worse with mechanical loading, sitting, standing, and bending forward. The diagnosis can be made via a discogram that demonstrates a tear in the annulus fibrosis and concordant reproduction of pain in the back with injection into the disc. Treatment can include conservative treatment with physical therapy and NSAIDs, or more invasive procedures such as intradiscal electrothermal therapy or fusion.

Complex Regional Pain Syndrome
Complex regional pain syndrome type I and II (formally known as reflex sympathetic dystrophy and causalgia, respectively) are chronic pain syndromes that typically affect extremities after trauma. Local trauma to an extremity either without evidence of nerve damage (type I) or with evidence of nerve damage (type II) leads to the maintenance of pain secondary to sympathetic efferent
nerves or circulating catecholamines. A typical extremity affected by CRPS can have edema, loss of range of motion, denudation of hair, a lower temperature and color changes compared to the opposite extremity and allodynia. Diagnosis of CRPS can be made with a sympathetic block of the stellate ganglion or of the lumbar sympathetic plexus. Treatment can consist of medication therapy, physical therapy, psychological therapy, education about the disease process, and regional sympathetic blocks.

**Myofascial Pain**

Myofascial pain is characterized by aching muscles, muscle spasms, stiffness, and weakness which is thought to occur secondary to ischemic microtrauma to a muscle. On exam, patients with myofascial pain will note discrete areas of tenderness (trigger points) that are palpable over the affected muscle. Trigger points may be treated with injections of lidocaine into the trigger point.

**Sacroiliac Joint Dysfunction**

The sacroiliac (SI) joint may cause pain secondary to etiologies such as trauma, spine deformities, facet arthropathy, pregnancy, osteoarthritis, and inflammatory arthropathies. The typical pain distribution is around the SI joint, into the buttock and posterior thigh. Physical exam may demonstrate pain on movement of the joint and limited motion. Local anesthetic injection of the joint may help to elucidate if the SI joint is the true cause of the patient’s pain. Radiofrequency ablation may be used to treat the patient’s symptoms by ablating the nerves providing sensation to the joint.

**Postherpetic Neuralgia**

Acute herpes zoster is caused by reactivation of the latent varicella virus in the dorsal root ganglion. The typical course of the infection is that there is pain for 48–72 h prior to the rash. At this point, a vesicular rash appears in a dermatomal distribution (see Fig 13.3) and lasts for approximately 1–2 weeks. Following resolution of the acute herpes zoster, patients (usually patients greater than 50 years old) may experience sharp, lancinating pain secondary to post herpetic neuralgia. Typical treatment of post herpetic neuralgia involves anticonvulsants, antidepressants, and lidocaine patches.
Physical Therapy
Physical therapy has an important role in the treatment of the chronic pain patient to reduce disability, restore and increase function, and improve strength. Exercise may increase endurance and muscle strength while at the same time decrease the patient’s subjective experience of pain. Passive forms of physical therapy can include electrostimulation, heat and cold therapy, and ultrasound.

Psychological Therapy
Psychological evaluation of the patient may help to diagnose and treat psychiatric issues such as malingering, substance abuse and somatization disorders and other issues such as depression, anxiety, and sleep disorders contributing to the patient’s pain disorder. Early diagnosis and treatment of psychological issues have demonstrated to effect a patient’s pain level, ability to cope, return to work, and medication compliance.

Palliative Care
Palliative care focuses on providing pain relief and care of a terminally ill patient and his/her family over the remainder of the patient’s life. It focuses on pain relief and symptomatic relief of nausea, vomiting, and dyspnea. Care may take place at home (usually through hospice) or in an inpatient palliative care unit, acute care hospital, or nursing home.

Case Study
A 32-year-old woman seeks consultation with you in the pain management clinic. Six months ago she sprained her left elbow and wrist in a fall while roller blading. After recovering uneventfully with splinting of her wrist and wearing a sling for 4 weeks, she has developed severe pain again. She describes it as burning and constant. She describes tingling, “electric shock” sensations over the affected area. It covers the dorsum of her hand, both sides of her forearm, and the posterior aspect of the elbow and lower arm. She notes that she cannot type with her left hand and that she cannot lift her backpack with her left arm. She finds showering painful and keeps the arm out of the water; she avoids long-sleeved shirts because the fabric rubbing against her skin is painful.
On examination, the limb is purplish and mottled, edematous, and cool to the touch. There is less hair than on comparable regions of her right arm. The nails of her left hand are thickened, discolored, and longer than those on her right. Lightly stroking the dorsum of her hand with a fingertip causes pain.

You perform the initial evaluation with your attending. You are asked to dictate the note describing the patient's pain presentation. Which of the four main types of pain will you characterize as hers?

The four main categories of pain are nociceptive, inflammatory, neuropathic, and dysfunctional. This patient's acute injury has long passed, so her pain is probably not nociceptive or inflammatory, and is likely neuropathic. The characteristics of the pain as well (type, pain descriptors) are also consistent with this classification. It is important not to characterize it as dysfunctional until other types have been excluded.

Which pain descriptors will you use to describe her symptoms?

The patient's pain can be described in her own terms (burning), and the location, intensity, and variation in the pain should be noted. For example, behavioral choices she makes (showering, dressing) should be noted. You will also ask her about variation during the day, effect of analgesics, document the duration of her symptoms, and the relationship to her injury. This patient has described *alldynia*, pain elicited by a normally nonpainful stimulus, and *dysesthesia* and *paresthesia*, abnormal sensations occurring spontaneously or in response to stimulation. You have verified alldynia on your exam (stroking her hand) but have not demonstrated *hyperalgesia*, an exaggerated perception of pain in response to a normally painful stimulus, because you wisely did not attempt a painful stimulus.

What is your working diagnosis? How could you verify it?

The patient appears to have complex regional pain syndrome, type I, formerly known as reflex sympathetic dystrophy. We base this diagnosis on the pattern of her pain and its relationship to her injury: it followed local trauma without nerve damage (which might have made it type II, formerly causalgia), she has cutaneous evidence of sympathetic excess and disuse atrophy, and she has
alldynia. She thus meets the International Association for the Study of Pain’s criteria for the diagnosis, which are sensitive but not specific for the disorder. Although not considered definitive, a strongly suggestive diagnostic test is a favorable response to sympathetic blockade of the affected extremity. You could perform a localized chemical sympathectomy of the limb by infusing phentolamine into the arm isolated by a tourniquet. More commonly, you could block the stellate ganglion on the affected side (see below). If evidence of sympathectomy is seen, for example by vasodilation and warming of the extremity, and if some pain relief is observed, the diagnosis is strongly supported.

**What treatment would you offer her?**

A stellate ganglion block is performed by injecting local anesthetic adjacent to the transverse process of C6, palpated medial to the carotid artery at the level of the cricoid cartilage in the neck. Fluoroscopic guidance can improve the efficacy and possibly safety of the block. The spinal and epidural spaces lie close to the correct needle position, as do the carotid and vertebral arteries. If a stellate ganglion block is effective, the block can be repeated several times over the next few weeks. In a fortunate proportion of patients, the pain relief lasts far longer than the effect of the local anesthetic, and may actually lengthen over time. Unfortunately, some patients do not experience progressively longer relief or even relief extending longer than the block, and other treatments will be needed. Multimodal therapy is recommended whether blocks are successful or not. First, the patient needs psychological counseling that her symptoms are not the result of direct tissue damage, and that she can and must begin to use the extremity more as analgesia allows. Physical therapy taking advantage of less painful periods is essential. Anxiety, depression, and sleep disorders should be addressed by counseling and likely medication. Other medications that may be helpful include those directed at neuropathic pain (such as antiepilepsy or antidepressant drugs), opioids, and NSAIDs. The condition can be difficult to treat, so if one therapy fails, a different one should be tried, in order to facilitate rehabilitation efforts.
Suggested Further Reading


Postoperative Anesthesia Care Unit (PACU) and Common Postoperative Problems

R. Dean Nava Jr and Tarun Bhalla

For maximum impact, it is recommended that the case study and questions found on page xxxi are reviewed before reading this chapter.

Key Learning Objectives

- Learn the key elements of a PACU sign-out
- Review the most common postoperative anesthetic complications
- Understand the criteria for discharge from the PACU

Admission

Upon patient admission to the PACU, standard monitors are placed and an initial evaluation, including a set of vital signs is obtained. HR, ECG, BP, RR, oxygen saturation, pain level, temperature, mental status, and level of nausea all need to be evaluated. These should be documented every 5 min for the first 15 min, then at least every 15 min afterwards. Invasive monitors (e.g. CVP, arterial line, PA or Swan-Ganz) are used if indicated by patient condition. Capnography may be used if the patient has an artificial airway or if there is concern for respiratory depression.

A directed yet thorough sign-out to the PACU care team is paramount in the care of patients in the postoperative period. Table 27.1 shows an example of a typical sign-out an anesthesiologist would give to the PACU staff.
Postoperative Respiratory Complications
The most frequent complication in the PACU is airway obstruction. Common causes include:

- the tongue falling against the posterior pharynx (most common)
- laryngospasm (see below)
- glottic edema
- secretions/vomit/blood in the airway
- external pressure on the trachea (e.g. neck hematoma)

A clinical sign of partial obstruction is sonorous respiration. A sign of complete obstruction is absent breath sounds and often paradoxical movement of the chest with respiration.

Treatment modalities include supplemental oxygen, head lift, jaw thrust, oral or nasal airway, or reintubation. If the patient displays signs of extrinsic compression of trachea, such as an expanding hematoma with airway compromise, reopening of the wound and drainage is therapeutic and can be lifesaving.

Laryngospasm (uncontrolled contraction of the laryngeal cords) may also be seen in the PACU. Clinical indicators may include a high-pitched crowing or silence if the glottis is totally closed. This may be more common after airway trauma, repeated airway instrumentation or with copious secretions (including

Table 27.1 Sample PACU sign-out.

<table>
<thead>
<tr>
<th>Preop history</th>
<th>Medications, allergies, past medical history</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Underlying diagnosis</td>
<td></td>
</tr>
<tr>
<td>- Premedications</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intra-operative history</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Anesthesia type</td>
<td></td>
</tr>
<tr>
<td>- Medications &amp; fluids given</td>
<td></td>
</tr>
<tr>
<td>- Estimated blood loss, urine output</td>
<td></td>
</tr>
<tr>
<td>- Intra-operative events/problems</td>
<td></td>
</tr>
<tr>
<td>- Vital sign ranges</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patient status</th>
<th>Airway (preop exam, airway management, ETT position)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Size, location of lines, catheters and invasive monitors</td>
<td></td>
</tr>
<tr>
<td>- Level of consciousness</td>
<td></td>
</tr>
<tr>
<td>- Pain level</td>
<td></td>
</tr>
<tr>
<td>- Intravascular volume status</td>
<td></td>
</tr>
<tr>
<td>- Overall impression</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Postop instructions</th>
<th>Acceptable ranges (blood loss, vitals, urine output)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Potential cardiovascular or respiratory problems</td>
<td></td>
</tr>
<tr>
<td>- Labs or diagnostic studies (CXR, ECG) if necessary</td>
<td></td>
</tr>
<tr>
<td>- Location and physician contact information</td>
<td></td>
</tr>
</tbody>
</table>
blood or vomit in airway). Management includes positive pressure mask ventilation, oral or nasal airway, suctioning, small dose of succinylcholine if refractory, intubation, and finally cricothyroidotomy or jet ventilation if the inability to intubate or ventilate is encountered.

Common causes of hypoventilation in the PACU are residual depressant effects of anesthetics (most common), residual neuromuscular blockade, splinting from pain, diaphragmatic dysfunction after thoracic or upper abdominal surgery, distended abdomen, tight abdominal dressings, and increased CO₂ production (e.g. shivering, sepsis, and hypothermia). The clinical signs may include prolonged somnolence, slow respiratory rate, shallow breathing with tachypnea, and labored breathing. The signs may not become prominent until the PaCO₂ > 60 or pH < 7.25. Treating the underlying cause is the mainstay of therapy, but until that is accomplished, control of ventilation is essential. Intubation may be necessary (hemodynamically unstable, severely obtunded, etc.). Provide an opioid antagonist (naloxone in increments of 0.04 mg IV) if an opioid overdose is a possibility, administer a cholinesterase inhibitor if residual paralysis is suspected. If the patient is splinting, consider increasing pain control measures depending on respiratory rate and mental status.

Common causes of hypoxemia in the postoperative setting are increased intrapulmonary shunting due to decreased FRC (most common), pneumothorax, prolonged ventilation with small tidal volumes, endobronchial intubation, bronchial obstruction by blood or secretions leading to collapse, aspiration, bronchospasm, pulmonary edema, and atelectasis. The early signs usually involve restlessness, tachycardia, and ventricular or atrial dysrythmias. The late signs usually include hypotension, obtundation, bradycardia, and cardiac arrest. The treatment generally includes supplemental O₂, and the patient may need a nonrebreather mask. If symptoms persist, the patient may need intubation until the underlying cause is found and corrected. A chest x-ray should be ordered immediately. Treatment obviously depends on the underlying cause. A chest tube should be placed if a pneumothorax or hemothorax is discovered and bronchodilators (e.g. albuterol) given if bronchospasm is suspected. Consider administering diuretics if there is fluid overload, and performing a bronchoscopy if there is severe atelectasis due to obstructive plugs or aspiration.

**Postoperative Hemodynamic Complications**

The most common causes of hemodynamic compromise in the recovery unit can be differentiated into problems associated with preload, left and right
ventricular function, and afterload. Hypotension can result from one or more of these causes, as outlined in Table 27.2.

The clinical signs of hypotension include a 20–30% baseline decrease in blood pressure, disorientation, nausea, change in consciousness, decreased urine output, and angina. Treatment of hemodynamic compromise should include fluid bolus, vasopressor agents, pleural aspiration if tension pneumothorax is suspected, pericardiocentesis if a cardiac tamponade is suspected, and invasive monitoring (arterial line, CVP, or PA catheter) if necessary. The treatment depends on the patient's clinical picture and underlying cause.

Postoperative hypertension is a frequent occurrence in the PACU. Common causes include noxious stimuli (most common), incisional pain, irritation from the endotracheal tube, distended bladder, previous history of hypertension, fluid overload, metabolic derangements (hypoxemia, hypercapnia, and acidosis), and intracranial hypertension. Clinical signs and symptoms include headache, bleeding, vision changes, angina, and ST changes on ECG. Treatment includes correcting the underlying problem, draining the bladder, providing analgesia, and correcting metabolic derangements. Be aware of the patient's baseline preoperative blood pressure, and use that as a target for titration. Specific medical therapies other than analgesia are listed in Table 27.3 below.

Postoperative tachycardia is often mediated by parasympathetic output or caused by medications such as atropine, glycopyrrolate, and muscle relaxants (e.g. pancuronium). See Table 27.4 for differential diagnosis of tachycardia. Signs and symptoms may include hypertension or hypotension and angina. Treatment

### Table 27.2 Causes of hypotension.

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased preload</td>
<td>• Hypovolemia (most common)</td>
</tr>
<tr>
<td></td>
<td>− “Third spacing” (fluid sequestration)</td>
</tr>
<tr>
<td></td>
<td>− Bleeding</td>
</tr>
<tr>
<td></td>
<td>− Wound drainage</td>
</tr>
<tr>
<td></td>
<td>• Venodilation due to spinal/epidural anesthesia</td>
</tr>
<tr>
<td></td>
<td>• Pericardial tamponade</td>
</tr>
<tr>
<td></td>
<td>• Tension pneumothorax</td>
</tr>
<tr>
<td></td>
<td>• Air embolism</td>
</tr>
<tr>
<td>Left ventricular dysfunction (impaired contractility)</td>
<td>• Severe metabolic derangements (acidosis, sepsis, hypoxemia)</td>
</tr>
<tr>
<td></td>
<td>• Myocardial infarction</td>
</tr>
<tr>
<td></td>
<td>• Volume overload</td>
</tr>
<tr>
<td></td>
<td>• Dysrhythmias</td>
</tr>
<tr>
<td>Arterial vasodilatation (decreased afterload)</td>
<td>• Possible inflammatory response</td>
</tr>
<tr>
<td></td>
<td>• Anesthetic related</td>
</tr>
</tbody>
</table>
includes treating the underlying cause, fluid bolus, draining the bladder, and pain control. Symptomatic treatment may be necessary to allow offending medications to wear off. Cardiac arrhythmias are also common causes of tachycardia. If atrial fibrillation occurs, consider beta blockade, calcium channel blockers, and potentially cardioversion if the patient becomes hemodynamically unstable.

The most common causes of postoperative bradycardia are increased parasympathetic flow or decreased sympathetic output, which may additionally manifest as hypotension concomitant with the bradycardia. In cases of suspected increased parasympathetic output, consider muscarinic blocking agents such as atropine and glycopyrrolate. In cases of decreased sympathetic output, beta-mimetic agents such as ephedrine are useful. Table 27.5 outlines the most common causes of postoperative bradycardia.

Myocardial ischemia should always be part of differential diagnosis in patients with hemodynamic compromise. Risk factors for myocardial
ischemia include CHF, valvular disease, low ejection fraction, smoking history, anemia, hypertension, and emergency surgery. Causes may include tachycardia (decreases time in diastole, leading to coronary hypoperfusion), hypotension, and hypoxemia. Clinical signs are angina, ECG changes, and dysrhythmias. Work-up and treatment includes treating underlying causes (pain control, fluid bolus, and anxiolysis), oxygen, aspirin, nitroglycerine, beta blockade, and morphine. Cardiac enzymes (e.g. troponin levels) should also be checked.

### Postoperative Neurologic and Other Complications

The most common cause of delayed awakening is residual anesthetic, sedative, or analgesic. Less common causes include hypothermia, metabolic derangements, and stroke. Management includes treating underlying causes (e.g. apply a forced air warming blanket, correct metabolic disturbances) or medication reversal. Naloxone reverses opioid effects, although the patient may need repeated doses if the half-life of the opioid is longer than naloxone. Flumazenil reverses benzodiazepine effects.

Another common complication is altered mental status and emergence delirium. Exacerbating factors are listed in Table 27.6.

**Emergence delirium** usually resolves in 10–15 min. Management includes verbal reassurance, adequate analgesia, correcting metabolic derangements,
providing supplemental oxygen, benzodiazepines, arm restraints, and physostigmine if reaction is related to scopolamine or atropine (central anticholinergic syndrome).

Postoperative neuropathy is a less common injury that may present postoperatively. Spinal cord injury can occur with positioning during intubation or with hematoma after neuraxial anesthesia, but this is very rare. More commonly seen are peripheral nerve injuries. These stretch or compression injuries may involve the ulnar nerve (compression of ulnar nerve at the postcondylar groove of humerus), peroneal nerve (compression of nerve against fibular head while in lithotomy), femoral nerve (due to exaggerated lithotomy position with “candy cane” stirrups), brachial plexus (due to over abduction of arms past 90° in the supine position or the neck being too far to one side), and long thoracic nerve (occurs with pneumonectomies, leading to winged scapula and paralyzed serratus anterior muscle). Most symptoms resolve in 6–12 weeks, although permanent injuries may occur.

Corneal abrasions can be caused by ocular drying (eyes open during procedure), contact with eye during facemask ventilation or intubation, or the patient scratching his or her own eye upon awakening (hence the reason we ask the patient not to rub his or her eyes on the way to the recovery room). Signs and symptoms include excessive tear formation, photophobia, and decreased visual acuity. Treatment includes artificial tears, eye closure, and ocular antibiotics. Most corneal abrasions heal within 72 h.

The most common cause of postoperative weakness is residual neuromuscular blockade. Other causes include cerebrovascular accident and preexisting

<table>
<thead>
<tr>
<th>Table 27.6 Causes of postoperative mental status changes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxemia</td>
</tr>
<tr>
<td>Metabolic derangements</td>
</tr>
<tr>
<td>Cerebral hypoperfusion</td>
</tr>
<tr>
<td>Extremes of age</td>
</tr>
<tr>
<td>Emotionally significant operations</td>
</tr>
<tr>
<td>Presence of intraoperative recall</td>
</tr>
<tr>
<td>Scopolamine or atropine</td>
</tr>
<tr>
<td>Substance abuse</td>
</tr>
<tr>
<td>Pain, nausea, pruritus</td>
</tr>
</tbody>
</table>

Table 27.6 Causes of postoperative mental status changes.
neuromuscular disorders (e.g. myasthenia gravis, Eaton–Lambert syndrome, periodic paralysis, and muscular dystrophies). This is clinically evident with poor respiratory effort, shallow breathing, rapid respiratory rate, and subjective skeletal muscle weakness reported by the patient. Treatment includes administration of neuromuscular reversal agents or reintubation until the weakness resolves.

**Postoperative Nausea and Vomiting (PONV)**

20–30% of surgical patients experience some degree of PONV. Risk factors are listed in Table 27.7:

<table>
<thead>
<tr>
<th>Table 27.7 PONV contributing factors.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient related</strong></td>
</tr>
<tr>
<td>• Young women</td>
</tr>
<tr>
<td>• Hiatal hernia</td>
</tr>
<tr>
<td>• Obesity</td>
</tr>
<tr>
<td>• History of postoperative nausea</td>
</tr>
<tr>
<td>• History of motion sickness</td>
</tr>
<tr>
<td>• Non-smokers have higher risk</td>
</tr>
<tr>
<td><strong>Surgical related</strong></td>
</tr>
<tr>
<td>• ENT, abdominal, gynecologic procedures</td>
</tr>
<tr>
<td>• Extraocular muscle traction</td>
</tr>
<tr>
<td>• Middle ear irritation</td>
</tr>
<tr>
<td>• Peritoneal, intestinal irritation</td>
</tr>
<tr>
<td>• Dental procedures</td>
</tr>
<tr>
<td><strong>Anesthesia related</strong></td>
</tr>
<tr>
<td>• Gas in stomach due to facemask ventilation</td>
</tr>
<tr>
<td>• Use of nitrous oxide (controversial)</td>
</tr>
<tr>
<td>• Use of parenteral opioids</td>
</tr>
<tr>
<td>• Use of etomidate</td>
</tr>
<tr>
<td>• Hypotension after spinal/epidural anesthesia</td>
</tr>
<tr>
<td><strong>Postop related</strong></td>
</tr>
<tr>
<td>• Use of parenteral opioids</td>
</tr>
<tr>
<td>• Postoperative oral fluid intake</td>
</tr>
</tbody>
</table>
Treatment includes a number of modalities. An essential part of therapy is treating underlying factors (e.g. hypotension, hypoglycemia, elevated ICP, and GI bleeding). Serotonin receptor blockers, such as ondansetron 4 mg IV, at the end of surgery have few side effects and are commonly used. Dexamethasone, a steroid, is also a useful antiemetic, although its exact mechanism of action is unclear. It is given as a 4–8 mg IV dose just after induction. Droperidol is useful for breakthrough nausea, but may lead to sedation and currently has an FDA mandated black box warning due to Q-T interval prolongation. Compazine, metoclopramide, and phenergan are also available medications (see Chap. 7, Pharmacology of Adjunct Agents). Multimodal therapy (i.e. drugs from several different classes) and prevention is most effective in the treatment of PONV.

### Pain Control

A plan for controlling postoperative pain depends on both patient and surgical factors. Pain medications can be given via intravenous, intramuscular or oral route. The intravenous route is often preferred because medications can be given in smaller doses, have more reliable uptake, and are more easily titrated. Common opioids and their basic properties are listed in Table 27.8. Patients who had a regional block placed may require fewer supplemental medications. Patients who have an epidural placed for postoperative pain may receive both the local anesthetic and the opioid via the epidural catheter.

Non-steroidal anti-inflammatory drugs (NSAIDs), acetaminophen, and COX-2 Inhibitors are other classes of medications that are also very useful in the postoperative setting. Potential benefits include reducing opioid requirements, decreasing the incidence of nausea and vomiting, minimal effect on platelet function (COX-2 inhibitors), and fewer gastrointestinal side effects.

### Table 27.8 Commonly used opioids in the PACU.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Duration</th>
<th>Typical bolus dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fentanyl</td>
<td>Short acting</td>
<td>25–100 mcg IV</td>
</tr>
<tr>
<td>Hydromorphone (Dilaudid)</td>
<td>Intermediate to long duration</td>
<td>0.2–1 mg IV</td>
</tr>
<tr>
<td>Meperedine (Demerol)</td>
<td>Intermediate to long duration</td>
<td>50–100 mg IV/SC/IM</td>
</tr>
<tr>
<td>Morphine</td>
<td>Intermediate to long duration</td>
<td>2–5 mg IV</td>
</tr>
</tbody>
</table>
Ketorolac (Toradol) is a popular analgesic, but potentially deleterious side effects include platelet dysfunction and nephrotoxicity. It should be used with caution in patients with renal dysfunction, the elderly or those with increased risk of bleeding.

**Hypothermia and Shivering**

Hypothermia and shivering can result from a number of causes. Distributive heat loss, evaporation from skin prep, impaired function of normal thermoregulation from anesthetics and the higher rate of heat loss from patients with burns, traumatic injuries, or cachexia can all lead to a significant drop in body temperature and subsequent shivering. The physiologic impairments of both hypothermia and shivering are detailed below in Table 27.9.

The treatment of hypothermia and shivering includes forced air warming devices, patient reassurance, and meperidine in severe cases of shivering.

**Discharge Criteria**

Discharge from the PACU is based on an array of clinical factors outlined below:

**General Condition**

The patient should be oriented to time, place, situation, and follow commands. Patient should be non-cyanotic and non-pallorous, and muscle strength needs to be appropriate. Nausea, pain, and any other major early postop complications should be absent or under control.

<table>
<thead>
<tr>
<th>Table 27.9</th>
<th>Effects of hypothermia and shivering.</th>
</tr>
</thead>
</table>
| **Hypothermia** | • Increased oxygen consumption, carbon dioxide production  
|              | • Elevates peripheral vascular resistance  
|              | • Impairs platelet function, decreased clotting factors  
|              | • Increased infection rates  
|              | • May lead to cardiac dysrhythmias  
| **Shivering** | • Increased oxygen consumption (up to 200%)  
|              | • Increased carbon dioxide production (up to 200%)  
|              | • Impairs monitoring devices  
|              | • May lead to myocardial ischemia  
|              | • May precipitate ventilatory compromise |
Hemodynamics
The patient's blood pressure should be within 20% of baseline preop value. The heart rate and rhythm should be stable for at least 30 min before discharge is considered. It is important to look out for common complications such as cardiac dysrhythmias and myocardial ischemia. Volume status also needs to be stable and hypo- or hypervolemia corrected.

Respiratory Status
A patient's respiratory rate should be around 10–25 breaths/min. Causes of either tachypnea or respiratory depression should be investigated prior to discharge. Secretions need to be coughed up and cleared adequately and the work of breathing acceptable.

Airway
Swallow and gag reflexes should be intact. No obstruction, stridor, or retraction should be present. Artificial airways should no longer be needed prior to discharge to the floor.

Pain Control
The patient should be able to identify and localize pain, and the analgesia for that pain should be adequate. The opioid requirement should be no shorter than every 15 min, and postoperative analgesia orders need to be appropriate for the situation.

Renal Function
It is important to monitor urine output (UO), with UO > 0.5 cc/kg/h in catheterized patients usually considered adequate. In patients without a urinary catheter, voiding prior to discharge is no longer required, unless they had a spinal or have had problems with voiding in the past.

Labs and Diagnostic Tests
If checked, hematocrit needs to be appropriate compared to fluid losses sustained during surgery. Other labs should be checked as indicated, and electrolytes, glucose, coagulation labs, platelets, and hematocrit corrected as needed. Other diagnostic tests, such as ECG and chest x-ray are obtained depending on specific patient indications (chest pain, hypoxia) and need to be evaluated prior to discharge home or to the floor.
Case Study

A 45-year-old woman has just undergone total abdominal hysterectomy. She is generally healthy, does not smoke or drink alcohol, and has not had general anesthesia ever before. She emerged from general anesthesia (thiopental, vecuronium, sevoflurane, and fentanyl) uneventfully. You accompany the patient to the PACU, assist the nurse with settling the patient, and obtain initial vital signs on arrival: BP 148/90, HR 77, SpO₂ 98% on facemask oxygen at 6 L/min.

Describe the elements of the report you will now give to the PACU nurse. You begin with a brief summary of the patient's past medical history, the procedure performed, and a summary of the anesthetic course. You will tell the nurse about preoperative sedation (drugs, total dose, and time), antibiotics, induction agents, mask ventilation and intubation ease or difficulty, maintenance drugs, neuromuscular blockade, and reversal given. You will summarize opioids and other analgesics given and tell the nurse the last dose, amount, and time. You will summarize “ins and outs” by giving estimated blood loss, fluids given, and blood products given. Finally, you will discuss anything special: intraoperative problems, special drugs given (insulin, steroids, antiemetics, etc.), special concerns or requests the patient may have expressed, and plan for postop care if not routine.

After completing your report you leave the bedside to complete your paperwork. Before you return to the operating room, approximately 5 min after your initial arrival in the PACU, the nurse calls you back to the bedside. The patient is agitated, thrashing around in bed and not answering questions or following instructions to lie back and relax. What will be your initial steps in assessing the patient? What is the differential diagnosis?

Although this may be simple emergence delirium you must rule out other more serious problems, including hypoxia or hypercapnia. Check the patient's vital signs, especially looking for hypoxia or extreme hypertension. Make certain that the patient has a patent airway and is breathing, by physical examination. Make certain that the patient is agitated, not seizing.
You exclude emergencies and conclude the patient is experiencing emergence delirium. How will you respond?

Attempt to speak to the patient and calm her. If you are unable to do so, a small dose of short-acting sedative, such as midazolam, 1–2 mg, is reasonable. You and/or the nurse will still need to reassess the patient after getting her more calm.

The patient improves. One hour later you are called back to the PACU. The patient is complaining of pain. How will you assess the patient? What intervention will you recommend? Would your approach be different if the patient had undergone laparoscopic myomectomy and was scheduled to be discharged home later today?

You should speak to the patient and attempt to understand the origin of her pain. Is it incisional? Opioids are usually very effective in this setting. Although individual patient responses may modify your approach (for example, if you noted either unusual sensitivity or resistance to opioids intraoperatively), hydromorphone (Dilaudid), 0.2–0.4 mg boluses, or morphine, 3–5 mg boluses, titrated to effect, are common choices. If the patient is to be discharged home, it may be more prudent to use short-acting opioids such as fentanyl, 50–100 mcg boluses. In both cases you may also consider adjunctive drugs such as the NSAID ketorolac, 30 mg, if not contraindicated by the presence of renal disease or severe bleeding.

The pain is under control 30 min later, but the patient now complains of nausea. How will you respond?

This patient has moderately high risk for PONV, as a nonsmoking female who has received significant opioids (the fourth risk factor in the simplest assessment scale is previous history of PONV or motion sickness). If she has not received any prophylactic antiemetics, ondansetron, 1–4 mg IV, is a reasonable first choice. If she already received this drug for prevention of PONV, then an agent from another class is more prudent. Options include droperidol or haloperidol, prochlorperazine (Compazine), hydroxyzine (Vistaril), promethazine (Phenergan), metoclopramide (Reglan) or scopolamine. The latter is typically given as a transdermal patch, which takes several hours to reach a peak effect.
When can the patient be discharged from the PACU? How would your criteria differ if the patient were being discharged home after laparoscopy instead? The patient should be oriented to person, place, time, and situation. Her pain and nausea should be under reasonable control, but it is not necessary for her to be completely pain free or to be completely without nausea. These symptoms may persist for hours or even days in the case of postoperative pain. The point is to have reached a stable and tolerable equilibrium. She should be fluid replete, as indicated in part by acceptable urine output, and if bleeding has been significant, her hemoglobin should be in a range not requiring transfusion (generally higher than 7 g/dL). Her vital signs should be stable and there should be no respiratory problems other than a possible requirement for supplemental oxygen. If she is to be discharged home, she should have no oxygen requirement, and she should be able to ambulate with minimal assistance. She must have a competent adult to accompany her home.

Suggested Further Reading


Introduction to Critical Care

Beverly J. Newhouse

For maximum impact, it is recommended that the case study and questions found on page xxxi are reviewed before reading this chapter.

Key Learning Objectives
- Review basic concepts of oxygen balance in the body
- Understand the diagnosis and treatment of common conditions encountered in the intensive care setting such as shock, sepsis, and acute respiratory failure
- Learn the basic principles, indications, and complications associated with hemodynamic monitoring techniques such as arterial line, CVP and pulmonary artery catheter.
- Discuss different modes of mechanical ventilation

Initial Assessment of the Critically Ill Patient
In a seriously ill patient, it is often necessary to provide resuscitation before making a definitive diagnosis. Begin with the ABCs (Airway, Breathing, and Circulation) and focus on stabilization as the work-up and diagnosis are ongoing. Ensure a patent airway and stable vital signs, while proceeding further to work-up with history, physical exam, laboratory and radiographic testing, and other diagnostic procedures.

Oxygen Balance
When managing critically ill patients, it is important to have an understanding of oxygen balance, including oxygen delivery to the tissues and oxygen consumption by the tissues.
Oxygen Transport

Oxygen transport involves the loading of blood with oxygen in the lungs, delivery of oxygen from the blood to tissues, and return of unused oxygen to the cardiopulmonary circulation. The amount of oxygen contained in arterial blood can be defined by the arterial oxygen content (CaO$_2$) equation:

$$CaO_2 = [Hb \times 1.34 \times SaO_2] + [PaO_2 \times 0.003]$$

where Hb = hemoglobin concentration, SaO$_2$ = % hemoglobin saturation with oxygen, and PaO$_2$ = partial pressure of dissolved oxygen.

Global oxygen delivery (DO$_2$) to the body depends on this arterial oxygen content (CaO$_2$) as well as cardiac output (CO):

$$DO_2 = CaO_2 \times CO$$

Global oxygen consumption (VO$_2$) is the total oxygen consumption by all of the body’s organs and tissues. Normal oxygen consumption is 3 ml/kg/min O$_2$. The amount of oxygen that is returned to the cardiopulmonary circulation from the venous side is termed the mixed venous oxygen saturation (SvO$_2$). The oxygen extraction ratio (O$_2$ER) is defined as oxygen consumption divided by oxygen delivery:

$$O_2 ER = (VO_2/DO_2) \times 100$$

Under normal conditions, the body extracts approximately 30–35% of the delivered oxygen and the rest is returned to the heart as the mixed venous oxygen. Thus, normal mixed venous oxygen saturation is 65–70%.

The body is capable of increasing oxygen extraction during exercise or periods of stress up to a maximum O$_2$ER of about 70%. Any further increase in oxygen consumption (or decrease in oxygen delivery) will result in cellular hypoxia, anaerobic metabolism, and the production of lactic acid.

Recall from the above arterial oxygen content equation that hemoglobin concentration and hemoglobin saturation (SaO$_2$) influence the oxygen content. The relationship between partial pressure of oxygen in the blood and hemoglobin saturation is defined by the oxyhemoglobin dissociation curve (Fig. 28.1). The position of this curve is affected by pH, temperature, PaCO$_2$, and 2,3-diphosphoglycerate (2,3-DPG). Shifting of the curve to the left or right will alter the ability of hemoglobin to bind oxygen. As the curve shifts to the right, hemoglobin has
less affinity for oxygen, and thus more oxygen will be released to the tissues. As the curve shifts to the left, hemoglobin binds oxygen more tightly and releases less to the tissues. During periods of stress (such as metabolic acidosis), the curve is shifted to the right to allow more oxygen to be delivered to the tissues.

**Markers of Oxygen Balance and Tissue Perfusion**

**Lactate**

When the body’s oxygen balance is such that oxygen demand exceeds oxygen supply, cells become hypoxic and convert to anaerobic metabolism. Lactic acid (lactate) is a by-product of anaerobic metabolism and can be measured in the blood. Elevated lactate levels are associated with tissue hypoperfusion and
poor oxygenation. Although other factors can affect lactate levels, the presence of elevated lactate can therefore be used as an indirect marker of poor tissue perfusion and shock.

**Central Venous and Mixed Venous Blood Oxygen Saturation**

When a central venous catheter is in place, blood can be drawn from the superior vena cava (distal port) and sent to the lab for measurement of central venous blood oxygen saturation. Central venous blood oxygen saturation (ScvO₂) has been shown to correlate well with mixed venous blood oxygen saturation (SvO₂) in most circumstances, and can be used to reflect tissue oxygenation. Normal ScvO₂ is approximately 70% (compared to normal SvO₂ of approximately 65%). Lower than normal ScvO₂ or SvO₂ is an indication of poor tissue oxygenation and the need for improved oxygen delivery and perfusion.

**Hemodynamic Monitoring (also see Chap. 11)**

**Goals**

The goals of hemodynamic monitoring in the critically ill patient are to optimize perfusion and oxygen delivery to tissues, ensure rapid detection of changes in clinical status, and monitor for response to treatment. Although noninvasive monitors (such as a blood pressure cuff) are associated with less risks and complications, it is often necessary to use invasive monitoring techniques to achieve these goals.

**Invasive Arterial Blood Pressure Monitoring**

A common cause of admission to the intensive care unit is hypotension, which may be due to any number of etiologies (see Shock section below). Occasionally, blood pressure monitoring with a noninvasive cuff is adequate, but if the blood pressure is significantly low, it may be undetectable or inaccurate by a cuff. In addition to being the most accurate form of blood pressure monitoring, arterial cannulation allows continuous beat-to-beat monitoring. It also serves as a site for obtaining lab measurements of oxygenation, ventilation, and acid-base status. The most common sites for arterial cannulation are radial or femoral arteries, but other arteries may be used if necessary (see Chap. 15, IV, Arterial & Central Line and Gastric Tube Placement Techniques).

Complications associated with arterial cannulation and precautions to decrease the incidence of complications are listed in Table 28.1.

Volume status can be assessed by evaluating the arterial pressure height during controlled mechanical ventilation. Positive pressure ventilation will lead
to significant systolic variation (>10 mm Hg) of the blood pressure in patients who are hypovolemic (Fig. 28.2).

**Cardiac Output**

Recall that global oxygen delivery (DO₂) to the tissues is dependent on the oxygen content of blood (CaO₂) as well as cardiac output (CO). Cardiac output is equal to the product of heart rate (HR) and stroke volume (SV):

\[
CO = HR \times SV
\]

The variables that affect stroke volume include preload, afterload, and contractility. **Preload** is an estimate of left ventricular volume at the end of diastole. The Frank–Starling curve shows the relationship between preload and stroke volume (Fig. 28.3). In general, increases in preload lead to greater stroke volume. However, a point on the Frank–Starling curve is eventually reached where further increases in preload do not increase stroke volume and may instead lead to decreased stroke volume (as in congestive heart failure). Because it is difficult to measure ventricular volume, ventricular pressure is commonly used to estimate volume and thus preload. Use of a central venous catheter enables

**Table 28.1 Complications associated with arterial cannulation.**

<table>
<thead>
<tr>
<th>Complication</th>
<th>Precautions to Decrease Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematoma</td>
<td>Avoid multiple needle punctures/attempt</td>
</tr>
<tr>
<td></td>
<td>Apply pressure if artery punctured</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Caution in coagulopathic patients</td>
</tr>
<tr>
<td></td>
<td>Apply pressure to bleeding site</td>
</tr>
<tr>
<td>Thrombosis</td>
<td>Avoid multiple needle sticks</td>
</tr>
<tr>
<td></td>
<td>Use continuous flush system</td>
</tr>
<tr>
<td></td>
<td>Avoid prolonged catheterization</td>
</tr>
<tr>
<td>Vasospasm</td>
<td>Avoid multiple or traumatic punctures/attempt at cannulation</td>
</tr>
<tr>
<td>Air embolism</td>
<td>Caution when flushing catheter</td>
</tr>
<tr>
<td>Nerve damage</td>
<td>Avoid sites in close proximity to nerve</td>
</tr>
<tr>
<td>Infection</td>
<td>Use sterile technique</td>
</tr>
<tr>
<td></td>
<td>Avoid prolonged catheterization</td>
</tr>
<tr>
<td>Intra-arterial drug injection</td>
<td>Keep venous and arterial lines well-organized, separated, and clearly labeled</td>
</tr>
<tr>
<td>Ischemia</td>
<td>Avoid traumatized sites</td>
</tr>
<tr>
<td></td>
<td>Avoid prolonged catheterization</td>
</tr>
<tr>
<td></td>
<td>Place pulse oximeter on ipsilateral side to verify perfusion</td>
</tr>
</tbody>
</table>
monitoring of right atrial pressure or central venous pressure (CVP), which is an estimate of right ventricular preload. In a patient without significant pulmonary hypertension or valvular disease, it can be assumed that right ventricular preload correlates with left ventricular preload because the same blood volume that enters the right heart will traverse to enter the left heart. By way of this assumption, CVP is often used as an estimate of left ventricular preload.

**Afterload** refers to the myocardial wall tension that is required to overcome the opposing resistance to blood ejection. Right ventricular afterload is indirectly represented by the pulmonary vascular resistance (PVR) and left ventricular afterload is indirectly represented by the systemic vascular resistance (SVR). SVR may be calculated from the following equation when cardiac output measurements are obtained from a pulmonary artery catheter:
SVR = [(MAP – CVP)/CO] × 80

where MAP = mean arterial pressure.

Contractility refers to the ability of the myocardium to contract and eject blood from the ventricle. Contractility depends on preload and afterload so these variables should be optimized first in order to improve contractility. Contractility can only be directly measured with the use of echocardiography to estimate ejection fraction. However, once preload and afterload are optimized, contractility is often indirectly represented by cardiac output. If cardiac output remains low despite improvements in preload and afterload, the use of inotropic pharmacologic agents may be initiated to improve contractility.

Central Venous Pressure Monitoring
As described above, invasive CVP monitoring allows continuous measurement of right heart pressures, which can be used to reflect preload. Normal CVP during positive pressure ventilation ranges from 6 to 12 mm Hg. A low CVP with hypotension and tachycardia most often corresponds to hypovolemia.
Persistent hypotension following a fluid challenge and higher than normal CVP indicates cardiac congestion (as may occur with cardiac tamponade, tension pneumothorax, or myocardial ischemia).

Cannulation sites for CVP placement include subclavian, internal jugular, and femoral veins (also see Chapter 15, IV, Arterial & Central Line and Gastric Tube Placement Techniques). Complications associated with the placement of a central line are presented in Table 28.2. To reduce the number of cannulation

<table>
<thead>
<tr>
<th>Complication</th>
<th>Precautions to decrease risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematoma</td>
<td>Avoid multiple needle punctures/attempts</td>
</tr>
<tr>
<td></td>
<td>Apply pressure if vein or nearby artery punctured</td>
</tr>
<tr>
<td>Bleeding/Hemorrhage</td>
<td>Caution in coagulopathic patients</td>
</tr>
<tr>
<td></td>
<td>Apply pressure to bleeding site</td>
</tr>
<tr>
<td>Air or thrombotic embolism</td>
<td>Caution with infusions</td>
</tr>
<tr>
<td></td>
<td>Use head-down tilt and avoid open catheter to air</td>
</tr>
<tr>
<td></td>
<td>Avoid prolonged catheterization and use continuous flush</td>
</tr>
<tr>
<td>Carotid artery puncture/cannulation</td>
<td>Use appropriate landmarks ± sonographic visualization</td>
</tr>
<tr>
<td></td>
<td>Use small finder needle; transduce pressure to verify venous</td>
</tr>
<tr>
<td>Pneumothorax/Hemothorax</td>
<td>Use appropriate landmarks</td>
</tr>
<tr>
<td></td>
<td>Avoid multiple needle sticks</td>
</tr>
<tr>
<td></td>
<td>No risk with femoral vein</td>
</tr>
<tr>
<td></td>
<td>Risk with internal jugular &lt; risk with subclavian</td>
</tr>
<tr>
<td>Infection/Bacteremia/Endocarditis</td>
<td>Use strict sterile technique²</td>
</tr>
<tr>
<td></td>
<td>Avoid prolonged catheterization</td>
</tr>
<tr>
<td>Nerve trauma</td>
<td>Use appropriate landmarks and avoid sites in close proximity to nerves</td>
</tr>
<tr>
<td>Thoracic duct damage/Chylothorax</td>
<td>Avoid left subclavian and internal jugular when possible</td>
</tr>
<tr>
<td>Complete heart block</td>
<td>Extreme caution placing PAC in patient with LBBB</td>
</tr>
<tr>
<td>Cardiac dysrhythmias</td>
<td>Use ECG monitoring while placing catheter and avoid prolonged placement of wire/catheter in atria/ventricles</td>
</tr>
<tr>
<td>Pulmonary ischemia/infarction</td>
<td>Do not keep PAC continuously wedged</td>
</tr>
<tr>
<td></td>
<td>Minimize balloon inflation time</td>
</tr>
<tr>
<td>Pulmonary artery rupture</td>
<td>Do not over-wedge PAC; avoid balloon hyperinflation</td>
</tr>
<tr>
<td>Myocardial perforation</td>
<td>Always inflate balloon before advancing catheter, but never inflate balloon against significant resistance</td>
</tr>
</tbody>
</table>

PAC pulmonary artery catheter, LBBB left bundle branch block, ECG electrocardiogram.

²A chest x-ray should always be performed after catheterization to verify correct positioning and absence of pneumothorax/hemothorax.

³Strict sterile technique includes handwashing, sterile gloves, gown, mask, hat, patient drape, and sterile prep with chlorhexidine.
attempts and the risk of inadvertent arterial puncture, a portable ultrasound vessel imaging device is often used.

**Pulmonary Artery Catheter**

As described above, left heart pressures may be estimated from right heart pressures in most circumstances and CVP may be used to approximate pulmonary capillary wedge pressure (PCWP). However, when left ventricular function is impaired, or significant valvular disease or pulmonary hypertension is present, the use of a pulmonary artery catheter (PAC) may be indicated for more accurate estimations of left heart pressures. Use of a PAC allows continuous monitoring of pulmonary artery pressures, intermittent monitoring of PCWP, and thermodilution for estimation of cardiac output and calculation of systemic vascular resistance. PCWP is used as the best estimation of left ventricular end-diastolic volume (preload), analogous to CVP estimation for the right ventricle.

The PAC can also be used to obtain blood samples for mixed venous oxygen saturation in order to evaluate oxygen balance. Risks associated with PAC placement include those associated with central line placement as well as additional anatomic and functional disturbances (Table 28.2). Table 28.4 in the next section shows how the use of a PAC can help in the determination of common hemodynamic disturbances in shock.

**Shock**

Shock is a disorder of impaired tissue perfusion and results when oxygen delivery is inadequate to meet the demands of oxygen consumption or when tissues are unable to adequately utilize delivered oxygen. Hypotension is often present in shock, but shock can also occur without hypotension due to compensatory mechanisms that serve to augment blood pressure. Many other clinical signs of shock may be present, including altered mental status, organ dysfunction such as low urine output, cold extremities, acidosis, tachycardia, tachypnea, and any other sign of impaired perfusion. If not rapidly treated, shock can lead to irreversible tissue injury, organ failure, and death.

**Classification of Shock**

Shock is classified into four main categories. Although this classification can be useful in the diagnosis and management of shock, patients may simultaneously suffer from more than one category of shock. Table 28.3 shows the four main types of shock and lists examples of each.
Table 28.4 presents the most likely hemodynamic disturbances that are associated with each type of shock.

**Management of Shock**

The primary goal in the management of shock is to restore perfusion and oxygen delivery to vital tissues before organ failure develops. This goal is accomplished by improving hemodynamics (including blood pressure and cardiac output) and optimizing oxygen balance. Specific therapy depends on the type of shock. In general, patients with shock will require invasive monitoring to assist in the diagnosis and to monitor response to treatment. Many patients will also require endotracheal intubation and mechanical ventilation, particularly if their work of breathing is increased by metabolic acidosis. Fluid therapy is indicated in almost all forms of shock (with the exception of congestive heart failure and cardiogenic shock) as a means of increasing preload, cardiac output, and blood pressure. **A reasonable blood pressure goal for most patients is a mean arterial pressure (MAP) ≥65 mm Hg.** In patients with a history of hypertension or who already manifest signs of organ failure, a higher blood
pressure may be necessary to optimize tissue perfusion. Beyond fluids, vasoactive agents can be utilized in order to augment blood pressure. Other therapy can be used to improve each of the components of oxygen delivery while at the same time trying to reduce oxygen demand. It is important to search for and treat the underlying cause of shock while continuing resuscitation. Measures of tissue perfusion, including ScvO₂ (or SvO₂ if a pulmonary artery catheter is in place) and lactate can be followed to assess the response to treatment and guide further therapy.

**Vasoactive Agents Commonly Used in Shock**

Vasoactive agents are indicated for management of patients with shock who do not respond adequately to fluid therapy. These medications may include vasopressors, vasodilators, chronotropes, and inotropes. Many of the vasoactive medications used to treat shock have more than one mechanism of action. Table 28.5 lists some of the commonly used vasoactive agents along with their mechanism of action (also see Chap. 7).

**Septic Shock**

Septic shock is a form of distributive shock caused by infection and should be managed in concordance with the formal guidelines that have been devised by the “Surviving Sepsis Campaign.” In addition to the management principles used to treat any form of shock, it is crucial to search for and control the source of infection with the early initiation of broad-spectrum antibiotics and surgical debridement, if necessary. Table 28.6 lists an overview of the management of septic shock.

| Table 28.5 Commonly used vasoactive agents in shock. |
| --- | --- |
| **Agent** | **Mechanism of action** |
| Dopamine | Chronotropy ( β₁), Inotropy ( β₁), Vasocostriction ( α at higher doses) |
| Dobutamine | Chronotropy ( β₁), Inotropy ( β₁), Vasodilation ( β₂) |
| Epinephrine | Chronotropy ( β₁), Inotropy ( β₁), Vasocostriction ( α at higher doses) |
| Norepinephrine | Chronotropy ( β₁), Inotropy ( β₁), Vasocostriction ( α) |
| Phenylephrine | Vasocostriction ( α) |
| Vasopressin | Vasocostriction ( V1) |
Table 28.6  Overview of the management of septic shock.

(1) Resuscitation
   (a) Hemodynamic Goals
      • MAP ≥ 65 mmHg
      • Urine Output ≥ 0.5 ml/kg/h
      • CVP 8–12 mm Hg (12–15 mm Hg if mechanically ventilated)
      • ScvO₂ ≥ 70% (or Svo₂ ≥ 65%)
   (b) Begin with fluid resuscitation if the patient is hypotensive
   (c) Add a vasopressor if the patient is not responding appropriately to fluid resuscitation
      • Use an arterial line if vasopressors are required
      • Norepinephrine or dopamine are first-line vasopressors
      • Vasopressin may be added to norepinephrine
   (d) Consider a blood transfusion (especially if Hb < 7–9 g/dl)
   (e) Consider inotropic support with dobutamine if not meeting hemodynamic goals with above measures

(2) Diagnosis – try to get cultures before giving antibiotics
   (a) Blood cultures
   (b) Sputum culture
   (c) Urine culture
   (d) Culture other sites as indicated by history and physical exam
   (e) Imaging studies (chest radiograph and other studies as indicated by history and exam)

(3) Source Control
   (a) Evaluate for a focus of infection that can be drained or surgically debrided
   (b) Consider foreign bodies as a possible infectious source (such as central lines)

(4) Antibiotic Therapy
   (a) It is vitally important to start antibiotics within the 1st hour of hypotension
   (b) Initiate broad-spectrum antibiotics
   (c) Follow cultures and de-escalate antibiotics as appropriate
   (d) Treat for 7–10 days (unless extenuating circumstances)
   (e) Stop antibiotics if shock determined to be caused by a noninfectious source

(5) Other Supportive Care
   (a) Consider administration of steroids if the patient has a poor response to fluids and vasopressor therapy
   (b) Use low tidal volume ventilation (as defined by the ARDS Network – see Acute Lung Injury and Acute Respiratory Distress Syndrome section)
   (c) Consider recombinant activated protein C
   (d) Glucose management, thromboprophylaxis, stress ulcer prophylaxis (see Supportive Care in the ICU section)
Acute Respiratory Failure
Acute respiratory failure (ARF) is another common disorder leading to intensive care unit admission. Respiratory failure may develop from primary pulmonary disorders or as a result of other systemic disorders. Clinical signs of acute respiratory failure may include altered mental status, tachypnea, increased work of breathing, use of accessory respiratory muscles, decreased oxygen saturation, cyanosis, and other nonspecific systemic signs such as tachycardia and hypertension. ARF may be divided into two types — oxygenation failure (hypoxemic respiratory failure) or ventilation failure (hypercapnic respiratory failure). Patients may also develop combined oxygenation and ventilation failure.

Hypoxemic Respiratory Failure
Hypoxemic respiratory failure is usually a result of mismatched alveolar ventilation (V) and perfusion (Q). Many disease processes can result in areas of alveolar hypoventilation relative to perfusion (termed low V/Q). This is otherwise known as an intrapulmonary shunt (Fig. 28.4). Examples of such disease processes that lead to intrapulmonary shunting include pneumonia, atelectasis, pulmonary edema, aspiration, and pneumothorax. As blood flows to poorly ventilated alveoli, it is unable to pick up adequate amounts of oxygen and thus returns poorly oxygenated blood to the heart. This poorly oxygenated blood dilutes oxygenated blood, causing systemic hypoxemia.

Other causes of hypoxemia include decreased partial pressure of inspired oxygen (such as in areas of high altitude with low inspired oxygen tension), left-to-right cardiac shunting, alveolar hypoventilation, and diffusion abnormalities.

Hypercapnic Respiratory Failure
Hypercapnic respiratory failure results from any disorder that leads to decreased alveolar minute ventilation. Minute ventilation (Va) is defined by the following equation:

\[ Va = f \times (Vt - Vd) \]

where \( f \) = respiratory rate, \( Vt \) = tidal volume, and \( Vd \) = dead space.

Therefore, decreased minute ventilation can result from decreases in respiratory rate or tidal volume (such as occurs with sedation and anesthesia) as well as increases in dead space ventilation. Dead space ventilation includes any area that is ventilated but not perfused. If alveoli are under-perfused, \( CO_2 \) cannot
Diffuse out of the blood via gas exchange and is therefore returned to the circulation, resulting in hypercapnia. Dead space may be anatomic or physiologic. Anatomic dead space results from airways that normally do not participate in gas exchange such as the trachea and bronchi. Physiologic dead space results from alveoli that are ventilated, but not adequately perfused. Physiologic dead space can occur from poor cardiac output resulting in inadequately perfused alveoli. Another example of physiologic dead space occurs with pulmonary embolus, where blood flow to an area of the lungs is obstructed.
Management of Acute Respiratory Failure

While the cause of respiratory failure is being investigated, it is important to ensure a patent airway and support of oxygenation and ventilation. Supplemental oxygen should be provided and if necessary, the patient should be intubated and managed with mechanical ventilation. Appropriate diagnostic tests include history and physical exam, arterial blood gas measurement, chest radiograph, and additional testing based on these findings and the likely etiology of the respiratory failure.

Acute Lung Injury and Acute Respiratory Distress Syndrome

Acute Lung Injury (ALI) is a complex process of injury to the lungs involving cytokines and damage to the alveolar–endothelial barrier, which leads to increased pulmonary microvascular permeability. The most severe form of ALI is the Acute Respiratory Distress Syndrome (ARDS) which leads to severe hypoxemic respiratory failure and is associated with a high mortality rate. Diagnostic criteria for ARDS have been defined by an international consensus committee and include:

- Severe hypoxemia ($\text{PaO}_2/\text{FiO}_2 < 200$)
- Bilateral diffuse pulmonary infiltrates seen on chest radiograph
- Evidence of noncardiogenic pulmonary edema

There are many possible etiologies that lead to ARDS, including both pulmonary and extra-pulmonary causes. In addition, it is known that mechanical ventilation can cause or exacerbate lung injury by volutrauma (overdistention of alveoli), barotrauma (high plateau pressures), and/or atelectrauma (shear stress of opening and closing of alveoli). The ARDS Network found that mortality is significantly reduced when patients with ARDS are ventilated with lower tidal volumes. There has also been much study and debate regarding optimal pressures, levels of PEEP, and modes of ventilation in ARDS. Management of ARDS includes supportive care while treating the underlying cause and avoiding further ventilator-induced lung injury. In addition, salvage therapies may be indicated for patients with such severe ARDS that they cannot maintain adequate oxygenation to support tissue and organ function. Such therapies include the use of high-frequency ventilation, inverse ratio ventilation, prone positioning, inhaled nitric oxide, and extracorporeal membrane oxygenation. While these therapies can improve oxygenation and provide temporary support, none of them has been shown to influence the mortality associated with ARDS.
Mechanical Ventilation
When patients develop respiratory failure such that they cannot maintain adequate oxygenation and/or ventilation, it is often necessary to provide mechanical ventilation.

Indications for mechanical ventilation include:

- hypoxemic respiratory failure
- hypoventilatory (hypercapnic) respiratory failure
- need for sedation or neuromuscular blockade
- need for hyperventilation to control intracranial pressure, and airway protection

Commonly Used Modes of Ventilation

Assist-Control Ventilation (also known as Continuous Mandatory Ventilation)

*Assist-control ventilation* may be delivered with volume-cycled breaths (*volume control*) or time-cycled breaths (*pressure control*). The patient may trigger breaths or breathe over the set rate, but the machine guarantees the minimum number of breaths that are preset. Regardless of whether each breath is patient-triggered or machine-triggered, the patient will receive the full preset tidal volume or preset applied pressure. This serves to decrease the patient’s work of breathing. During volume control ventilation, a preset tidal volume is delivered to the patient at a set rate. The peak pressure may vary per breath depending on the patient’s lung mechanics and compliance. Pressure control ventilation involves a preset inspiratory time and applied pressure instead of a preset tidal volume. Thus, the tidal volume will vary with each breath.

Intermittent Mandatory Ventilation

*Intermittent mandatory ventilation* delivers either volume-cycled or time-cycled breaths at a preset rate. The patient may breathe spontaneously beyond the preset rate, but patient-triggered breaths beyond the set rate are not supported by the machine. Synchronized intermittent mandatory ventilation delivers the preset machine breaths simultaneously with the patient’s inspiratory efforts to avoid patient–ventilator dyssynchrony.

Pressure Support Ventilation

*Pressure support ventilation* allows the patient to breathe spontaneously, but provides a preset level of inspiratory pressure with each triggered breath.
Inspiratory pressure provided by the machine decreases the patient’s work of breathing, but still allows the patient to trigger all breaths and thus controls the respiratory rate. Most modern ventilators will provide a back-up ventilatory rate if the patient becomes apneic, but it is important to ensure that apnea alarms and back-up rates are set appropriately.

**Positive End-Expiratory Pressure**

Positive end-expiratory pressure (PEEP) may be applied during any of the above mechanical ventilatory modes. PEEP functions to keep alveoli open at the end of expiration, thereby reducing atelectasis. PEEP minimizes the cyclic opening and closing of alveoli and reduces shear force which may cause damage to alveoli. By keeping terminal alveoli open, PEEP serves to increase the number of functional lung units that are participating in gas exchange and therefore improves oxygenation.

**Inspiratory Pressures**

During positive pressure mechanical ventilation, pulmonary pressure increases to a maximum at the end of inspiration. This maximum pressure is known as peak inspiratory pressure (Pi) and reflects airway resistance as well as the elastic properties of the alveoli and chest wall. If an inspiratory hold is applied at the end of inspiration, the flow of gas will stop and allow the pressure to drop to a level known as plateau pressure (Pplat). Plateau pressure reflects only the elastic properties of the alveoli and chest wall and is thus the best measure of alveolar pressure. The difference between peak inspiratory pressure and plateau pressure (Pi–Pplat) reflects the resistance of the upper airways.

**Initiating Mechanical Ventilation**

The mode of mechanical ventilation that is chosen is less important than ensuring that the main goals of mechanical ventilation are met. These goals include support of oxygenation and ventilation, synchrony between patient and ventilator, and avoidance of injurious pressures or volumes. Initially, the fraction of inspired oxygen (FiO₂) should be set to 1.0 and can later be titrated down to maintain adequate patient oxygenation. Initial tidal volume should be set at 8–10 ml/kg in patients with normal lung compliance. If the patient has poor lung compliance or is at high risk for ARDS, then tidal volumes should be reduced to 6 ml/kg to avoid volutrauma or barotrauma. If using pressure
control ventilation, the initial peak pressure should be set less than 30 cm H₂O to ensure that plateau pressures remain less than 30 cm H₂O. The set pressure can then be titrated to maintain tidal volumes as above. Initial respiratory rate can be set at 10–15 breaths/min and should be adjusted based on the results of arterial blood gas measurements. PEEP should be used to keep alveoli open at the end of expiration. PEEP of 5 cm H₂O is a reasonable starting level and may be titrated up depending on the patient's underlying pathology or oxygenation requirements.

**AutoPEEP**

AutoPEEP describes the patient's intrinsic positive alveolar pressure that develops at the end of expiration and is caused by incomplete expiration of the tidal volume. AutoPEEP most commonly occurs in patients with obstructive lung disease because they have more difficulty expiring all of the tidal volume before the next breath is initiated. With each breath, more air becomes trapped in the alveoli, leading to a “stacking” of breaths and thus increasing dead space. This increased dead space increases the patient's work of breathing. Strategies to reduce autoPEEP include lowering the respiratory rate or tidal volume to allow more time for expiration or less volume that needs to be expired, decreasing the inspiratory:expiratory ratio to allow more time for expiration, and applying extrinsic PEEP to equalize the autoPEEP and remove the pressure gradient.

**Noninvasive Positive-Pressure Ventilation (NIPPV)**

It is possible to deliver mechanical ventilation without endotracheal intubation in the form of a bilevel positive airway pressure (BiPAP) or continuous positive airway pressure (CPAP) mask. BiPAP uses two levels of positive airway pressure to deliver pressure support during inspiration and PEEP during expiration. These pressures are typically referred to as inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). CPAP delivers a constant pressure during the entire respiratory cycle such that the patient will spontaneously breathe at an elevated baseline pressure, but CPAP does not provide pressure support of ventilation. Note that both forms of NIPPV require the patient to be breathing spontaneously. Therefore, NIPPV is best used in an awake, cooperative patient. NIPPV is contraindicated in patients who are not spontaneously breathing, unable to cooperate, have a high risk of aspiration, or
have facial trauma which precludes the use of a tightly-fitting mask. If a patient does not respond favorably to NIPPV within a few hours, intubation and invasive mechanical ventilation may be required.

**Ventilator-Associated Pneumonia**
Although mechanical ventilation is life-saving when patients develop respiratory failure and cannot support their own oxygenation and ventilation, it is also known that endotracheal intubation with mechanical ventilation is an independent risk factor for the development of pneumonia. Ventilator-associated pneumonia (VAP) is defined as pneumonia that arises after a patient has been intubated >48 h. VAP is a major contributor of morbidity and mortality in the intensive care unit, and the risk of developing VAP is directly proportional to the length of time the patient is intubated. The pathogenesis of VAP is multifactorial but thought to be associated with aspiration of oropharyngeal bacterial pathogens around the endotracheal tube cuff as well as infected biofilm that develops in the endotracheal tube. In addition to being in the intensive care unit and having been intubated for >48 h, patients who develop VAP often have many risk factors for infection with multidrug resistant organisms. Gram-negative organisms (such as *Pseudomonas aeruginosa*) and methicillin-resistant *Staphylococcus aureus* are frequent pathogens in VAP. If possible, lower respiratory tract samples should be obtained for microbiologic culture prior to initiation of broad-spectrum antibiotics. However, if this is not possible in a timely fashion, antibiotic therapy should not be delayed because the failure to initiate prompt appropriate therapy is associated with increased mortality. Significant research efforts continue to focus on reducing risk factors and developing preventive strategies for VAP, but the best possible way to avoid VAP is to treat the underlying cause of respiratory failure and extubate the patient as soon as possible. The most recent guidelines for the management of VAP are included in a document published in 2005 by the American Thoracic Society and the Infectious Diseases Society of America.

**Supportive Care in the ICU**
In addition to the management and treatment of primary underlying disorders, there are supportive and prophylactic measures that have been shown to improve outcomes and help prevent complications associated with critical illness.
Measures to Prevent Nosocomial Infections include
1. Staff education and appropriate hand disinfection
2. Use of sterile technique and precautions during procedures
3. Isolation of patients with multidrug resistant organisms
4. Head of bed elevation to 45° for prevention of aspiration
5. Oral hygiene with chlorhexidine rinse for intubated patients
6. Avoidance of inappropriate use of antibiotics

Sedation Management
Although patients with critical illness often suffer from anxiety and emotional distress, studies have shown that constant deep sedation prolongs ventilator time, increases the incidence of infection, and may lead to worsening delirium. Therefore, the use of sedation protocols as well as daily awakening or lightening of sedation is recommended in the intensive care unit to avoid oversedation. Unless absolutely clinically indicated, neuromuscular paralysis should be avoided as it leads to longer time on the ventilator and is a significant risk factor for the development of prolonged weakness.

Glucose Management
Hyperglycemia is common in critically ill patients, and it is known that severe hyperglycemia is associated with increased morbidity and mortality in certain groups of patients. However, it is also known that intensive insulin therapy to maintain strict normoglycemia increases the risk of hypoglycemia, which is also associated with increased morbidity and mortality. Thus, the optimal target range for blood glucose in critically ill patients is still unclear, but many intensive care units currently use insulin to target a blood glucose <150 mg/dl. The most recent study regarding glucose management found that intensive glucose control (targeting blood glucose of 81–108 mg/dl) led to increased mortality compared to targeting a blood glucose <180 mg/dl.

Thromboprophylaxis
Patients in the intensive care unit often have many risk factors for the development of venous thromboemboli, including:

- prolonged immobility
- venous stasis
- polytrauma
- burns
● spinal cord injury
● malignancy
● obesity
● presence of central venous catheters
● hypercoagulability associated with the perioperative period

Thrombosis of the deep veins can lead to significant morbidity, including embolism of blood clots to the pulmonary vasculature (PE). The majority of clinically significant pulmonary emboli arise from the proximal deep veins in the leg. Because a significant pulmonary embolism is often fatal, prevention of these deep vein thromboses is important. Specific guidelines have been published by the American College of Chest Physicians regarding thromboprophylaxis. In general, all patients in the intensive care unit should receive mechanical prophylaxis (in the form of early ambulation or intermittent pneumatic compression boots) and unless contraindicated, a form of pharmacologic prophylaxis should also be instituted.

**Stress Ulcer Prophylaxis**

Patients with critical illness often develop gastrointestinal mucosal damage that can progress to clinically significant gastrointestinal bleeding, which increases mortality. Strategies for the prevention of stress ulcers decrease the incidence of such bleeding in intensive care unit patients. However, it is important to identify those patients who have risk factors for stress ulcer formation because the indiscriminate use of prophylaxis in all ICU patients may increase the risk of nosocomial pneumonia. Patients with any of the following risk factors should receive stress ulcer prophylaxis:

- Mechanical ventilation >48 h
- Coagulopathy or therapeutic anticoagulation (does not include patients only receiving thromboprophylaxis)
- Use of steroids
- History of active peptic ulcer disease
- Traumatic brain injury
- Major burns
- Severe infection or shock

Recommended prophylaxis may be provided by the administration of either a proton pump inhibitor or an H₂-receptor antagonist. Although proton pump inhibitors may provide more complete suppression of acid secretion, they have
not been shown to be superior to H₂-receptor antagonists in the prevention of gastrointestinal bleeding in critically ill patients.

**Nutrition**

Malnutrition is common in critically ill patients and has detrimental effects on organ function, immune function, wound healing, ventilator weaning, and has been shown to increase mortality. In patients who cannot meet their nutritional needs orally, enteral nutrition is preferable to parenteral nutrition. Enteral nutrition has been shown to have important advantages as well as a lower incidence of complications as compared to parenteral nutrition. Current recommendations support the initiation of early enteral nutrition (within 24–48 h of admission) in critically ill patients who are expected to be unable to tolerate an adequate oral diet, unless there is a contraindication. Contraindications to enteral nutrition include intractable emesis, severe diarrhea or malabsorption, severe gastrointestinal bleeding, peritonitis, mesenteric ischemia, intestinal obstruction, short bowel syndrome, or severe shock. In these situations, it may be necessary to initiate total parenteral nutrition (TPN), especially if the patient is significantly malnourished.

TPN is reserved only for these patients (who have a contraindication to or cannot tolerate enteral feeding) because it is associated with added risks. TPN must be administered into a central vein and as such, confers the risks associated with central venous cannulation and bloodstream infections. In addition, TPN is associated with mucosal atrophy of the gastrointestinal tract which disrupts the normal barrier function of the gut and is associated with bacterial translocation from the bowel lumen into the circulation. Other complications associated with TPN include hepatic dysfunction, cholestasis, and acalculous cholecystitis.

**Ethical Decisions and End-of-Life Care (also see Chap. 31, Ethical Issues in Anesthesia)**

Many patients cared for in the intensive care unit are unable to participate in decisions about their own medical care and are dependent on advance directives or surrogate decision-makers. Healthcare professionals must be able to adequately communicate among themselves and with patients’ families in order to set realistic goals that are consistent with patient and family desires. Sometimes it is determined by the team of healthcare professionals that further medical therapy is unlikely to be beneficial to the patient and this may lead to ethical issues, such as whether aggressive medical care should be continued and/or how end-of-life care should be facilitated.
Case Study
You are called to the PACU emergently to see a 57-year-old patient who has just undergone an aorto-bifemoral bypass graft. On arrival at the bedside, the nurse informs you that the case proceeded uneventfully and the patient arrived in the PACU 1 h ago. The patient underwent general endotracheal anesthesia and was extubated in the OR. Vital signs on arrival had been normal, but the blood pressure had been progressively declining and heart rate had been rising since then. Five minutes ago, the patient’s blood pressure had been 68/40 and heart rate 128. Now the nurse notes that she cannot obtain a blood pressure and cannot feel a pulse. The patient has a peripheral IV infusing lactated Ringer’s and an arterial line in the right radial artery. No blood pressure is seen on the arterial tracing.

What will be your initial response (first 30 s) on arrival?
In any “code” situation, remember ABC’s: Airway, Breathing, and Circulation. These always precede “D” (drugs, discussion, debate…)! From the nurse’s report, this patient appears to be in full cardiac arrest. You will check for a pulse (the lack of arterial pulsations on an otherwise working arterial line trace is confirmatory), and check for breathing by either auscultation or direct inspection.

The patient is found to be apneic and pulseless. What will you do next?
Call for help, either by activating the “code” team or other system in place in the particular hospital where you are working. (Some institutions treat arrests in the OR and PACU differently than on regular nursing units). Open the airway and begin ventilation by bag and mask with 100% oxygen (Airway, Breathing). Ask the nurse, an assistant, or other personnel to begin chest compressions (Circulation). Ensure that someone has secured a defibrillator and emergency medications. Assess the patient’s rhythm on the ECG.

The patient is found to be in ventricular fibrillation. What will you do next?
Current Advanced Cardiac Life Support (ACLS) guidelines recommend immediate DC shock. Any device can be used, including a monophasic defibrillator at 360 J (note that progressive increase in energy is no longer recommended). Alternatively, and possibly more efficacious, one can use a biphasic device at whatever power the machine is designed for (typically 120–200 J).
An automatic defibrillator may also be used at the machine-specific setting. CPR is then immediately resumed for five cycles (or about 2 min) before the next step. The rhythm is checked again during CPR and a second shock (at equal or higher energy if a biphasic device is being used) is given if the rhythm is still VF. Any time after the first or second shock, epinephrine, 1 mg IV (alternative vasopressin 40 U) is also given. CPR is always given for 2 min after a shock or drug dose, to maximize the chances of return of spontaneous circulation. Reintubation is also recommended early in the sequence to facilitate ventilation and allow for continuous chest compressions.

After your initial intervention, sinus rhythm reappears. Inspection of the arterial tracing shows minimal pulsatile activity, and manual blood pressure measurement confirms that the blood pressure remains unobtainable. What are your next steps? The patient has pulseless electrical activity (PEA), likely profound hypotension, as demonstrated by the arterial waveform and lack of noninvasive blood pressure reading. Vasopressors and CPR are continued without interruption while reversible causes are sought. Some recommend vasopressin if it has not already been given. There are several etiologies and a mnemonic, “H and T’s” is sometimes helpful:

- Hypovolemia
- Hypoxia
- Hydrogen ions (acidosis)
- Hypokalemia/hyperkalemia
- Hypoglycemia
- Hypothermia
- Toxins
- Tamponade, cardiac
- Tension Pneumothorax
- Thrombosis (coronary or pulmonary)
- Trauma

In this patient, hypovolemia from internal bleeding should be high on the differential diagnosis. Thromboembolism and pneumothorax are also possible, though less common. Auscultation of the chest will rule out tension pneumothorax, and echocardiography (usually transesophageal) may diagnose massive pulmonary embolism. The electrolyte, acid/base, and other etiologies
are also possible, and history and laboratory studies (recent or sent as part of the evaluation now) may be helpful. You will alert the surgeon immediately if you suspect an anastomotic leak and internal hemorrhage as a etiology, as immediate reoperation will be necessary. Aggressive fluid administration and vasopressors will temporize until definitive intervention can take place.

**Suggested Further Reading**

Society of Critical Care Medicine (2007) Fundamental critical care support, 4th edn. Society of Critical Care Medicine, Mount Prospect, IL


Section VII

Special Topics
Chapter 29

Professionalism, Safety, and Teamwork

Sheila Ryan Barnett and Stephen D. Pratt

For maximum impact, it is recommended that the case study and questions found on page xxxii are reviewed before reading this chapter.

Key Learning Objectives:

- Understand professionalism and its importance for the practice of anesthesiology
- Learn a simple approach to practicing “etiquette based medicine”
- Know how to avoid the common causes of communication failure

During medical school, students are expected to learn a vast amount of complex medical information, requiring hours of concentration and diligent study. However, it is well known that becoming a doctor takes more than book knowledge, and there is much that must be learned from the patients themselves. For anesthesiologists, time spent in the operating room participating in procedures and attaining technical expertise is a vital part of the training, but it is not everything. Anesthesiology is a unique profession, and the training must provide a balance between the development of technical expertise and critical thinking with communication and leadership skills. The anesthesiologist must be skilled at quickly establishing trusting relationships with patients and their families, many of whom are frightened and anxious. At the same time the anesthesiologist must be able to perform complex procedures under pressure – all while remaining ever vigilant in the operating room, always ready to respond to the changing needs of the patient and the surgeon.
A career in anesthesiology is not limited to the operating room, and anesthesiologists have the opportunity to choose diverse careers in areas such as pain management, critical care medicine, or other sub-specialties. Alternately, they may choose to focus on patient safety, medical simulation, or administration. Given the vast array of opportunities awaiting the anesthesiologist, it is not surprising that training must include an emphasis on professionalism, communication, and mutual respect for multiple specialties.

**Professionalism**

Professionalism is hard to define, but ultimately it reflects the “wholeness” of the individual anesthesiologist or the specialty. It embodies the knowledge, technical ability, critical thinking, and interpersonal skills of the provider, and each aspect may alternately enhance or diminish how anesthesiologists are perceived by their patients and the public.

The professional challenges associated with becoming an anesthesiologist mirror many of those encountered in other fields. For instance, the anesthesiologist-in-training must develop a strong understanding of the concepts and theories within anesthesiology, and gain expertise in a vast array of technical procedures. However, there are also unique challenges within anesthesiology that also must be conquered – these are frequently underappreciated by those outside of the field. For example, the importance of communication for anesthesiologists is often downplayed, yet excellence within anesthesiology requires strong leadership and communication skills as well as technical prowess and sound medical judgment.

Defining professionalism is challenging and the concepts and examples shown below in Table 29.1 represent a synthesis of several definitions of Professionalism.

For anesthesiology professionalism, at a minimum, implies competence in the knowledge of anesthesia. The basic curricular elements considered

<table>
<thead>
<tr>
<th>Table 29.1 Professionalism: essential attributes for anesthesiologists.</th>
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<td>(1) Competence in the fundamental elements necessary for the safe delivery of anesthesia, including both technical and non technical aspects.</td>
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<td>(2) Assumes responsibility for the care of individual patients and as such contributes to the well being of society in general.</td>
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<tr>
<td>(3) As a profession anesthesiologists have the right to train, admit, discipline and dismiss its members for failure to sustain competence or observe the duties and responsibilities.</td>
</tr>
<tr>
<td>(4) Exhibits the following humanistic qualities including: altruism, accountability, excellence, duty, honor and integrity, and respect for others.</td>
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</table>
necessary for certification in anesthesia are defined by the American Board of Anesthesiology (ABA) and the Accreditation Council for Graduate Medical Education (ACGME). A board-certified anesthesiologist is a physician who has completed specialized post graduate training in anesthesiology and exhibited an acceptable depth of knowledge in a written and oral examination. The standards for certification examinations are set by the ABA and are constantly reviewed and updated to reflect advances within the field and its specialties.

While the anesthesia profession has a responsibility for the care of individual patients and towards society in general, the care and well being of the patient must take primacy over other considerations. In a busy operating room environment, it can be difficult to put the patient’s needs first, above those of oneself, the surgeon or the schedule, but it is necessary to fulfill the anesthesiologist's professional obligation to the patient. Anesthesiologists have long been recognized as leaders in patient safety, and in 1985 the American Society of Anesthesiologists (ASA) was the first medical society to create a foundation dedicated to patient safety. The Anesthesia Patient Safety Foundation (APSF) was established to raise awareness within the profession and dedicate resources to improve the understanding of safe anesthetic practice. Since that time the ASA and APSF have sponsored numerous research projects and helped to establish guidelines and recommendations that have significantly improved patient safety over the last 20 years. Anesthesiologists have also been instrumental in the advancement of the electronic medical record, team training, and medical simulation. These are examples of how anesthesiologists can fulfill their professional obligations to patients and society in general through active participation with the society and its related organizations.

Anesthesiologists belong to a profession, and as such accept the responsibility to train, admit, discipline, and dismiss its members for failure to sustain competence or observe the expected duties and responsibilities. As indicated above, the public trusts that through education and training an anesthesiologist will have acquired a level of clinical competence and technical expertise matched by intellectual understanding of the needs of the patient preparing for surgery, in the intensive care unit, in labor and delivery or for the patient in pain.

In addition to ensuring competence, as professionals, anesthesiologists are also obligated to ensure the safety of fellow practitioners and their patients. Besides offering national comprehensive educational programs on substance abuse, many state societies have established programs to assist and treat individuals with substance abuse or other professional behavioral issues. State societies work closely with state licensing boards to ensure the development of fair and safe regulations for patients and practicing physicians.
Finally, for individual anesthesiologists professionalism also implies the presence of humanistic qualities that are central to the physician in the role of healer. These key elements of professionalism include: altruism, accountability, excellence, duty, honor and integrity, and respect for others. The basic need for these traits does not differ for anesthesiologists compared to other physicians from other specialties.

“The Etiquette of Medicine”

Another aspect of professionalism that deserves attention is simply put: manners matter! The impression that daily behaviors make on patients and other healthcare providers cannot be underestimated. One author eloquently described the value of “etiquette based medicine”, emphasizing the importance of basic manners and appearance. He points out that it is often these very simple actions that will leave the most lasting impression upon the patient and their family members. The importance of etiquette in medicine is very applicable to anesthesiologists, who often have limited, but intense interactions with patients and other healthcare workers. A modified checklist for behavior is displayed in Table 29.2.

For most patients, surgery is a relatively unique event, and one that is surrounded by significant anxiety and trepidation. The way an anesthesiologist approaches his or her patient before surgery will reflect the professionalism of the individual physician and the profession in general. For example, when a patient arrives at the hospital holding area they frequently encounter a host of healthcare providers in a very short space of time. This is especially true in a

<table>
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<th>Table 29.2 Etiquette-based anesthesia introductions.</th>
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<tr>
<td>(1) Verify with the nurse and the patient that now is an appropriate time to begin the anesthetic interview and preparation.</td>
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<tr>
<td>(2) Introduce yourself – as a physician. First names can come later.</td>
</tr>
<tr>
<td>(3) Look the patient in the eye and shake hands. Introduce yourself to family members – ask their relationship, do not make assumptions.</td>
</tr>
<tr>
<td>(4) Briefly explain your role within the anesthesia team (i.e. a student or resident), name the attending if applicable.</td>
</tr>
<tr>
<td>(5) Verify with the patient the surgery that will be occurring.</td>
</tr>
<tr>
<td>(6) Begin your discussion regarding the administration of anesthesia.</td>
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teaching hospital, where the teams can include medical and nursing students, as well as residents from multiple specialties and various levels of training. In the flurry of activity that ensues, the patient can easily lose track of all the providers. Therefore, it is imperative that anesthesiologists take a few moments to clearly introduce themselves and clarify their role within the anesthesia care team.

Safety and Teamwork

Many students are first attracted to the field of anesthesiology because of the excitement of the operating room and the appeal of the hands-on technical aspects of the field. It is often only later that students begin to appreciate the central – the non-technical – role played by the anesthesiologist during the patient’s operative course. The development of superb communication skills is critical for the anesthesiologist, who will need to be able to speak effectively with patients and multiple healthcare providers, including surgeons, nurses, technicians, and other specialists. In some instances effective communication may prevent significant patient harm.

In 1999, the Institute of Medicine (IOM) published a report indicating that 44,000 to 98,000 patients die in the United States each year due to errors by medical personnel. This placed medical error as the leading cause of accidental death in the country, and sent shock waves through the medical community. Similar data were later published from other countries around the world. More recently, the IOM estimated that there is an average of one medication error per patient per day in hospitalized patients. A growing body of research has now demonstrated that hundreds of thousands of patients are harmed each year due to error, at a cost of hundreds of billions of dollars. The causes of medical error are complex and multi-factorial, but poor communication is the single factor that has consistently been cited as the most common cause of error.

Failure to effectively communicate, to accurately transfer information from one team member to another, is common. In studies performed in the operating room, one author found that about 30% of clinical communication events fail to meet their intended goal. Although not specific to anesthesiology, it is easy to see how important “closed loop communication” could be in a busy operating room.

While it is clear that poor communication is a leading contributor to medical error and adverse events, much less is known about why communication failures continue to occur. One of the most vulnerable times for loss
of information is during the hand-off of patient care from one provider to another, and this has been shown to be the most common time that communication errors lead to patient harm. Contributing factors include differences in communication style, poor or no hand-off structure, and production pressure. Other common causes of communication failure are shown in Table 29.3.

**Team Training**

Formal team training, based on the concepts of Crew Resource Management (CRM), has been recommended to improve communication and to decrease or mitigate the impact of error. CRM was developed during the 1980’s by military aviation after investigations identified poor communication and ineffective coordination of the team as leading causes of aviation accidents. While CRM has evolved through several generations, the tenets have remained largely unchanged. One author has defined a “Big Five” in CRM-based teamwork:

- **Leadership**: A leader who will ensure the proper functioning of the team must be identified (this is not always the same as the clinical leader).
- **Mutual performance monitoring**: Team members must monitor each other’s actions to ensure that plans are followed and to prevent or mitigate errors.
- **Back-up behavior**: Based in part on the performance monitoring, team members must assist each other when needed. This includes advocating for patient safety.
- **Adaptability**: Team members must be able to meet and communicate in order to change plans as the clinical situation dictates.
- **Team orientation**: Team members must understand and trust that the safest way to care for patients is to ensure proper functioning of the team. This is contrary to traditional medical teaching, which emphasizes that a physician has a moral responsibility directly to his/her patients.

These five behaviors are then supported by closed-loop communication, team structure to ensure role clarity, the development of shared mental models (ensuring that all team members have the same understanding of the patient plans), and maintenance of situational awareness (an individual awareness of all factors on the unit that could influence the safe conduct of patient care). The operating room lends itself to the team training model.

Anesthesiologists have led the development of simulation-based courses for teaching CRM-based teamwork concepts to groups of anesthesiologists. In simulation-based team training, the clinician (or clinicians) is placed in a simulated clinical environment (see Chap. 32, Simulation). Classroom-based team training employs didactic education techniques along with low-level simulation, exemplary vignettes, and videos to teach and practice the CRM concepts. The primary advantage of this type of training is that it is relatively inexpensive, and easy to ensure that all staff members are trained in CRM concepts. Classroom-based team training has been shown to improve clinicians’ attitudes toward patient and to improve patient outcomes in obstetrics and emergency medicine. Irrespective of the team training method, it is important to have an implementation plan designed to transfer the teamwork skills from the classroom or simulator to the clinical arena.

**Putting It All Together**

The public trusts that through education and training an anesthesiologist will have acquired a level of clinical competence and expertise that surpasses that of a technician or lesser trained individual. Furthermore, it is expected that the technical expertise will be matched by an intellectual understanding of the needs and requirements of the patient, as well as the ability to communicate effectively and compassionately. The trust exhibited between the anesthesiologists, surgeons, and other healthcare providers and the lay public represents the very essence of professionalism.
Case Study

Peter is your favorite anesthesiology resident. He is amazingly confident, skillful, and aggressive. He loves “big” cases and always volunteers for trauma, cardiac, or messy “whomps.” You have seen him at a couple of social events, and he is the life of the party, joking with everyone, positively lighting up the room. He drives a sports car, regales his friends with stories of his travel adventures, and dates a model. He recently took up skydiving and is working on his private pilot’s license. But he is also amazingly generous. He has covered other residents’ call several times, and he offers to stay late and finish late cases so others can go home. Today, you witnessed an event that seemed totally out of character. One of his assigned cases, one of those big cases he loves, was moved to another room because the first case in his room was running late. He was irritable as he dropped off his patient in the PACU. Then, he sought out the floor leader and lambasted him (an attending with 20 years of seniority) for “taking my case away.” Then, he sought out the resident in the room where the case was transferred and demanded to switch assignments (they had put a breast biopsy in his room). This resident had already begun working with the patient and refused. Peter told the patient that he was more experienced and a better anesthesiologist than the resident now assigned to him, and asked the patient if he would not prefer Peter as his anesthesiologist. The frightened patient was speechless. Peter stormed out of the preoperative area and told the floor leader that he was sick and needed to be sent home.

What lapses in professionalism have you witnessed?

Peter has been impolite, and has personalized a decision made on behalf of patient care and OR efficiency. He has disparaged a colleague in front of a patient. He has been insubordinate to the floor leader. He has placed his own interests above those of the patient, the surgeons, his colleagues, the OR, and hospital. He has feigned illness because he is angry. In each case he has failed to put the broader interests of those he cares for and works with over those of his own interests.

Later, you are discussing the event with another resident and a nurse in the PACU. Both tell you that they are not surprised. “Peter has been pretty volatile lately,” they agree. Another resident says that Peter has recently ended his
relationship with his girlfriend and “is always at the hospital. He sleeps here even when he isn't on call. And he has a great apartment.” How does this knowledge influence your view of the event you witnessed?

Anyone can have a bad day, but Peter is exhibiting a dangerous and worrisome pattern of behavior. Placed in context, his irritability, problems with his personal life, tendency to spend excess time at work, volunteering for big cases and staying late and taking extra call may be indicative of substance abuse, psychiatric illness, or both. It is not unusual for behavior such as his to go unappreciated by any one individual, and it is often not until a crisis occurs that behavior such as this is finally clear enough to lead to intervention.

Despite your suspicions, no action is taken against Peter. Several weeks later, he is on call with you and he is paged for a case. He does not respond to several pages. You are sent to his call room to wake him up and ask him to come to the OR. You knock on his door with no response. You knock more loudly and finally enter the room with your own key. You find Peter in bed, apparently asleep, with the lights and television on. You wake him with great difficulty and when rising he is groggy and somewhat incoherent. He sits up and quickly gathers his belongings into his backpack while muttering something about being exhausted. You believe you have seen several glass ampoules in his bag.

What will you do?

The temptation is to do nothing. After all, you look up to Peter and he has a reputation as a popular and strong anesthesiologist. You are not sure about what you have seen or its implication, and you have not witnessed any of the episodes others have, beyond the one outburst. Yet, you have a responsibility to patients, to the hospital, to the profession, and perhaps most importantly, to Peter, to intervene. You may wish to seek the assistance of senior individuals in the department, such as the program director, clinical director, or chairman. Peter should be confronted directly and firmly. If he has not been abusing substances, he may be offended but will be able to quickly clear himself of any suspicion. If he has been, then denial, anger, and avoidance are likely. Drug testing may be required emergently, as allowing time to pass may obscure the window of opportunity. Peter should ideally not care for patients until the issue is resolved.
Peter is later found to have fentanyl and hydromorphone in his bag and tests positive for opioids in his urine. He admits to having been diverting drugs from the OR for about 3 months, beginning after his relationship began to unravel.

Would random drug testing of all residents have prevented this situation? Possibly, but this has not proven to be a widespread approach. A survey of anesthesia programs found an approximate prevalence of substance abuse of 1% among faculty physicians and 1.6% among residents. Thus, drug testing would unnecessarily test many, many non-using anesthesiologists to discover one who was using. Moreover, the tests (especially for drugs other than opioids) are expensive, prone to misleading results (for example, poppy seed ingestion can lead to a positive opioid test), and defeatable (for example, substituting clean urine). While only a small fraction of departments use this approach, those which have implemented it have reported widespread acceptance. Education, awareness of the risk, and strong support systems within and outside the department are considered the preferred approaches.

Is this problem more common in anesthesiology? This is debatable. Earlier studies showed that among physicians admitted for inpatient substance abuse therapy, anesthesiologists were overrepresented relative to their prevalence among all physicians. This study may have been confounded by better detection of abuse in the specialty. For example, the mean time to discovery when one is abusing fentanyl is only 3 months because tolerance develops so rapidly that the anesthesiologist is not able to divert enough drug (often more than 1,000 mcg or 20 ml per dose) to maintain the addiction. Subsequent work, using different methodology, contradicted this early result and found the incidence to be no higher among anesthesiologists than other physicians. Nonetheless, the daily direct handling of abusable drugs, the ability to mask diversion of drugs (by the use of other agents, such as beta blockers, in patients to mimic the effects of the stolen drugs), and the high stress environment of the OR are all possible reasons for anesthesiologists to become drug abusers. An intriguing but unproven hypothesis holds that exposure to trace quantities of opioids, induction agents, and inhalation agents in the OR can sensitize the anesthesiologist’s brain and predispose it to addiction.
Peter undergoes several weeks of inpatient detoxification and rehabilitation. Should he re-enter the operating room as an anesthesia resident?

This is one of the most controversial topics in the fields of anesthesiology and addiction medicine. Drug use can end and detoxification can occur, but addiction does not end. The direct exposure to drugs in the OR may prove to be a temptation that cannot be overcome by a recovering addict. Conversely, many have recommended that properly motivated recovering abusers be allowed carefully monitored reintroduction into the field. A significant fraction of those who do so are successful. Unfortunately, the presenting symptom of relapse is all too often death. Therefore, many have called for a “one-strike and you’re out” policy, with compassionate counseling towards another field of medicine. Although only a tiny fraction of anesthesiologists succumb to drug abuse, vigilance among all in the field is a professional responsibility.

Suggested Further Reading


Chapter 30

Quality Assurance, Patient and Provider Safety

Arti Ori

For maximum impact, it is recommended that the case study and questions found on page xxxiii are reviewed before reading this chapter.

Key Learning Objectives

- Learn about the need for and history of patient safety
- Discuss anesthesia-related patient safety data
- Understand national initiatives to improve patient safety

Anesthesiologists are responsible for taking their patients safely through the stresses of surgery, while preserving and protecting their vital functions. They become the advocates for the anesthetized patient, who has been rendered unconscious. Patient safety is of utmost concern, and the field of anesthesiology has long been recognized as a leader in patient safety efforts.

The History of Patient Safety

In its early days, anesthesia was perceived to have a high risk of mortality, and medical liability insurance premiums reflected this perception. However, a concerted effort led by the American Society of Anesthesiologists (ASA), in collaboration with a number of other groups, has resulted in paying greater attention to patient safety and the issues of preventable adverse outcomes. The Anesthesia Patient Safety Foundation was formed in 1985 with the vision that “no patient shall be harmed by anesthesia”, and has been a champion for patient
safety ever since. Significant advances in monitoring during anesthesia, such as pulse oximetry, have subsequently been responsible for a decline in adverse events.

**Quality Assurance**
Quality has been described in literature as the product of two factors: the science and technology of health care and the actual application of that science and technology in practice. Quality assurance (QA) refers to the process of determining whether patient services meet or exceed expected standard. QA helps maximize the quality of patient care, so that all patients receive the care they deserve.

In the United States, there is room for improvement in the quality of health care. Although the US spends nearly $2.4 trillion a year on medical care (the most money of all advanced industrialized countries), we still trail some industrialized nations when it comes to many measures of health care quality.

Health care quality and patient safety go hand-in-hand. Issues around safety in healthcare were brought to the forefront of public attention in 1999 with the publication of the Institute of Medicine’s report entitled “To Err is Human.” This widely publicized report estimated that medical errors occur in approximately 7% of all patients, and that between 44,000 and 98,000 deaths occur annually in the US as a result of medical error. This is almost three times the fatality rate on US highways.

While a number of external organizations such as the Joint Commission (formerly known as JCAHO) and state licensing boards evaluate health care quality, the primary responsibility for patient safety and quality of health care provision rests upon anesthesia providers.

**The ASA Closed Claims Study**
The ASA Closed Claims Study, which began in 1985, has played an important role in the identification of anesthesia-related adverse events. This project is an ongoing, detailed analysis of closed anesthesia liability claims to identify significant patterns of injury. The current database contains over 7,700 cases, and the majority of cases are from 1980 to 2001. Most cases involve healthy adults undergoing nonemergency surgery under general anesthesia. These data provide an important opportunity to identify how anesthesia care
contributes to adverse outcomes, since outcomes are not confounded by disease processes.

Table 30.1 shows the most common adverse outcomes listed in the ASA Closed Claims Database with corresponding lists ranges of payments for the claim. It is evident that adverse outcomes occur in groups in a small number of specific categories. More than half of all adverse outcomes are found in three categories: death, nerve damage, and brain damage. The significance of identifying these large groups of injuries is that research and interventions can be more effectively directed at a few large areas of clinical practice, potentially resulting in substantial improvements in patient safety. In the past, this technique was used successfully by the American Society of Anesthesiologists to focus attention on monitoring standards and specific guidelines for high-frequency adverse events, leading to the promulgation of the ASA Standards for Basic Anesthetic Monitoring (see next page).
ASA Standards for Basic Anesthetic Monitoring

Standard 1: Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics, and monitored anesthesia care

Standard 2: During all anesthetics, the patient’s oxygenation, ventilation, circulation, and temperature shall be continually evaluated:

**Oxygenation:**
- Oxygen analyzer for inspired gases
- Observation of the patient
- Pulse oximetry

**Ventilation:**
- Auscultation of breath sounds
- Observation of the patient
- Observation of the reservoir bag
- Capnography (Carbon dioxide monitoring)

**Circulation:**
- Continuous ECG display
- Heart rate and BP recorded every 5 min
- Evaluation of circulation
- Auscultation of heart sounds
- Palpation of pulse
- Pulse plethysmography
- Pulse oximetry
- Intraarterial pressure tracing

**Temperature:**
- Monitor temperature when changes are intended, anticipated, or suspected

The publication of guidelines by the ASA for managing issues with high rates of adverse outcomes has led to a significant decline in these adverse outcomes. For example, difficult airway management during induction of anesthesia has long been regarded as one of the most challenging issues in anesthesia patient safety. However, an analysis of claims associated with difficult airway management during induction of anesthesia shows a marked, statistically significant decrease in the incidence of death and brain damage (62% vs. 35%, \( p < 0.05 \)) in the period after the publication of the ASA Difficult Airway Algorithm (1993–1999), when compared with period before the publication of the airway guidelines (pre-1993). The ASA Difficult Airway Algorithm has been reproduced in Appendix A.
Challenges Facing the Anesthesia Provider
The operating room is a unique environment and presents challenges to even the most vigilant anesthesiologist. Environmental factors such as noise, multiple alarms, and continuous movement through the operating room of members of the team can all distract attention. Human factors like fatigue and sleep deprivation can also affect monitoring and cognitive tasks. In addition, with the emphasis on enhanced productivity, “production pressure” may force errors and compromise patient safety.

Automated information systems that provide automated anesthesia record-keeping have become increasingly popular. They have been shown to be of great benefit in support of patient care and safety, and enhancement of clinical quality improvement programs. These systems are increasingly being implemented in various anesthesia departments to support a number of functions, including real-time clinical decision support.

Steps to Ensure High Quality Anesthesia Care and Patient Safety
In order to optimize patient safety and ensure high quality care, the following principles should be taken into consideration by the anesthesia practitioner.

1. Make patient safety a priority. Be an advocate for your patient, always.
2. Thorough planning. Follow the Boy Scout motto of “Be Prepared.” Practice meticulous preoperative planning, and formulate a plan for intraoperative as well as postoperative care. Have a back-up plan in mind. However, at times, it may not be possible to plan far ahead because of the unpredictable nature of the operating room environment. Even when under pressure, slow down, think things through rationally and clearly and formulate a plan of action.
3. Vigilance. Vigilance. Vigilance. Monitoring the patient involves not only electronic monitoring but also astute clinical observation. Chest rise, mucus membrane color, furrowing of the brow are just a few signs that can provide a wealth of information about the patient. Be aware of what is happening in the operating room at all times, and keep an eye on what’s going on across the drapes. Listen out for indicators of potential problems like for the increasingly frequent sound of the suction catheter heralding an increase in blood loss.
4. **Teamwork** is essential for efficiency and excellence. Make a point to **introduce yourself** to the other members of the team, for it is through the collective efforts of the team striving together toward a common goal that high standards of patient care can be met.

5. **Detailed, accurate record keeping** is a medico-legal requirement. During “Adverse Events” there is often no time to fill out the chart, but do so later in spite of any emotional distress you may be feeling. Keep it brief, factual, and accurate. Remember, if something is not documented, it didn’t happen.

6. **Postoperative patient checks** allow anesthesia providers to document the overall impact of the care they provide. This feedback is critical to understand the downstream effects of the clinical decisions made in the operating room.

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**Common Perioperative Complications**

**Dental Trauma**

Dental injuries are a common complication during anesthesia and pose a significant cost. In a study of 598,904 cases at a large institution, it was found that approximately 1:4,500 patients who received anesthesia sustained a dental injury that required repair or extraction. Half of these injuries occurred during laryngoscopy and endotracheal intubation, and the teeth that were most commonly involved were the upper incisors. Obtaining a dental history and oral examination as part of the preoperative anesthesia assessment can alert one to those patients at high risk of dental injury. It is important to inquire about the presence of crowns, fixed partial dentures or bridges, and porcelain veneers, as teeth with dental work tend to be more fragile. Patients with poor dentition with risk factors for difficult intubation have the highest risk, however even sound teeth can be damaged. The use of mouthguards during intubation is controversial, as this may limit available space and make laryngoscopy more difficult. Being cognizant of the risk of dental injury with every laryngoscopy is the best means of prevention.

**Eye Injury**

Perioperative visual loss is an alarming complication of anesthesia, with the incidence ranging from 0.002% of all surgeries (excluding eye surgeries) to 0.2% of cardiac and spine surgeries. Anterior ischemic optic neuropathy (AION) occurs more commonly with cardiac surgery, while posterior ischemic
optic neuropathy (PION) occurs in patients during spine and neck procedures. Patients present with bilateral visual loss upon awakening from anesthesia. The mechanism for perioperative visual loss is presumed to be ischemia, and risk factors include long duration in the prone position, excessive blood loss, hypotension, anemia, hypoxia, excessive fluid replacement, use of vasopressors, elevated venous pressure, head positioning, and a preexisting vascular susceptibility such as occurs in smokers and patients with diabetes mellitus. Awareness of these risk factors and interventions to minimize them can help limit the frequency of this dreadful complication.

Corneal abrasions are another minor but bothersome complication of anesthesia as they are extremely painful. These may be due to direct trauma to the eye, as can occur with carelessness during mask ventilation. More frequently, they occur due to exposure keratitis due to failure of the eyelids to close fully, resulting in drying of the cornea. Corneal abrasions can be prevented by taping the eyelids closed, or the use of paraffin-based ointments.

**Peripheral Nerve Injuries**

Peripheral nerve injuries can occur during regional or general anesthesia, and can have profound consequences for the patient from the resulting disability. Patient positioning is the usual cause of peripheral nerve injury, with ulnar neuropathy being the most common type of injury. Injuries may be due to external pressure or nonanatomical positioning, and may occur more frequently with old patients, thin patients, and patients with vasculopathies such as smokers and diabetics. When positioning, the head and neck should be kept in neutral position, the arms should not be extended more than 90 degrees and should be supinated. Sand with shoulder abduction and lateral rotation should be minimized to prevent brachial plexus injury. Padding should also be used on pressure points. With meticulous attention to detail during positioning, the occurrence of these injuries can be minimized.

**Intraoperative Recall**

The problem of awareness during general anesthesia has received much public attention recently and is a prime concern with patients. Awareness has been shown to have a frequency of less than 1 in 500 general anesthetics, but the consequences in terms of patient distress are profound. The ASA advises specific interventions to help reduce the risk and impact of intraoperative awareness,
beginning with the preoperative identification of risk factors. These include a prior episode of intraoperative awareness, a history of anticipated difficult intubation, receiving high doses of opioids for chronic pain, substance use/abuse, ASA status 4–5, and limited hemodynamic reserve. In addition, there are certain surgical procedures with an increased risk of intraoperative awareness, such as cardiac, trauma, emergency, and cesarean sections. Some anesthetic techniques can also increase the risk of intraoperative recall, such as using a low MAC of anesthetic or total intravenous anesthesia in the presence of paralysis. The use of brain function monitors for the assessment of the depth of anesthesia has enjoyed increasing popularity, but studies about the actual effectiveness in reducing incidence of awareness remain ongoing.

The Future
The growing burden of healthcare costs has resulted in an increased pressure on anesthesiologists to improve the quality and safety of healthcare in a cost-effective manner. It is recognized that adherence to evidence-based practices may improve outcomes. Evidence-based practice also provides an opportunity for decreasing health care costs by minimizing expensive, preventable complications. Various initiatives have also been instituted as a means of improving quality at lower costs. The Leapfrog Group, which is a consortium of large corporations concerned with improving the “value of the health care dollar,” has a website “dashboard” which shows how well hospitals are progressing in implementing various quality “leaps,” such as rapid response teams and intensivist staffing of ICUs.

Pay-for-Performance
The pay-for-performance concept uses a variety of incentives to encourage delivery of evidence-based practices. It is also a vehicle to promote better patient outcomes as efficiently as possible. In 2006, the Institute of Medicine (IOM) put forward a statement on pay-for-performance, defining which practices should be rewarded, and how they should be implemented. The IOM recommended that rewards be given for high quality clinical care and to those providers who communicate well with patients and coordinate care effectively. Pay-for-performance programs ultimately reward health care that is of high clinical quality, patient-centered, and lower cost. For anesthesia providers, some specific metrics might include on-time antibiotic administration and maintenance of intraoperative normothermia.
Medicare

The Centers for Medicare and Medicaid Services (CMS) have recently implemented a program, where hospitals are evaluated on their performance in multiple clinical areas. These hospitals will be given financial incentives where the top 10% performing centers would receive a 2% bonus, the second 10% would receive a 1% bonus, and the bottom 30% would suffer a 2% decrease in payments in year 3 of the program. Current programs include the Medicare’s Physician Quality Reporting Initiative (PQRI) through which hospitals are eligible for a 1.5% bonus on Medicare cases for 80% compliance in the appropriate timing of prophylactic antibiotics.

With these measures in place, quality assurance, and patient safety have become mandated areas of focus for anesthesia providers. It is important to remember, however, that the ultimate responsibility to ensure that our patients receive the best care lies with each of us.

Case Study

An anxious 48-year-old patient is in the preoperative holding area awaiting outpatient surgery under general anesthesia. With her is her husband, an expert on risk assessment in nonmedical industries, and her father, a retired surgeon in his late 70’s. She is anxious because her father has told her stories of surgery in the 1950s and 60s, when he remembers significant numbers of patients dying or suffering significant morbidity. Her husband has worked in aviation, industrial process design, and is a “six sigma black belt.” All three acknowledge your assurance that the practice of anesthesia is remarkably safer now, but ask you to explain some of the safety advances that characterize anesthesiology today and explain the improvements.

You have just finished setting up the operating room for this case. What safety features of the modern anesthesia machine can you point to in reassuring the patient and her family?

There are quite a few features of a modern anesthesia machine, even those that do not have the most recent electronic controls built in. These include:

- Safety indexed gas lines
- Pin indexed cylinder connectors
- Failsafe valve
- Minimum oxygen flow whenever machine is on
- Knurled flowmeter knobs with standardized textures and positions on the machine
- Oxygen always rightmost in sequence of gas flowmeters to guard against upstream leaks
- Built-in inspired oxygen monitors and alarms
- Low pressure (disconnect) alarm
- All vaporizers standardized to clockwise-off
- Safety fillers for vaporizers
- Vaporizer interlock to prevent multiple agent administration
- Standardized machine checkout, either manual or automatic, before each case

What are some of the monitoring developments since the 1950’s that have improved safety?
Numerous monitors have been added to the manual blood pressure cuff and finger on the pulse of the mid-20th century. Electrocardiography, automatic blood pressure monitoring with alarms, pulse oximetry, capnography, agent and inspired gas monitoring, neuromuscular blockade monitoring, and consciousness monitoring are all routinely found in the modern OR. Interestingly, although without a doubt the introduction of these monitors paralleled the decline in anesthesia-related mortality and morbidity, it has been difficult to prove a causal relationship. For example, a large meta-analysis of randomized trials of pulse oximetry showed that it reliably detected episodes of hypoxemia but did not affect postoperative outcomes! One explanation for this paradox is the concept of “learning contamination bias,” which means that anesthesiologists have learned so much from the use of the monitor that even when it is absent, they employ tactics that prevent episodes of hypoxia. Examples include preoxygenation, use of oxygen during transport to the PACU, and use of high-flow oxygen when discontinuing nitrous oxide administration.

What drug-related advances and procedures have you employed that have enhanced safety?
The use of standardized color-coded drug labels and the use of standardized concentrations of drugs are two practices that help reduce drug errors. Anesthesiologists also have learned from human performance studies to
use safe practices such as “3 looks” when drawing up medications (before drawing, during drawing, after complete before setting down the vial) or positioning drugs in a standardized way on the anesthesia cart. Development of shorter acting drugs (fentanyl and derivatives, low solubility and minimally biotransformed inhalation agents) and drugs with a greater margin of safety between therapeutic and toxic doses have also helped. Other practices include checking blood with two people, pharmacy-mixed drug infusions, computerized infusions pumps with safety programs to limit errors in setting, and in some settings bar codes to verify drug identity.

What communication procedures will you employ that enhance safety?
In nearly every US operating room, the Joint Commission “safety pause” or “time-out” is performed prior to incision, in which the anesthesiologist, surgeon, and circulating nurse (and sometimes the patient) verbally state and agree on the planned procedure. An advancement of this idea is the WHO surgical safety checklist, which adds such practices as “once around the room” checks with all personnel regarding potential concerns. We also have standardized record keeping in the OR, whether manual or electronic and automated, and practice provider-to-provider anesthesia handoff procedures and standardized handoffs in PACU or ICU.

What other safety procedures are routine for all anesthetics in modern practice?
Anesthesiologists note and ensure pressure point and eye protection, assessment of the airway and teeth prior to and following induction, and in some settings temperature, radiation, or laser protection. A key development in the last half-century has been the simple presence of qualified anesthesia personnel in the OR at all times.

The patient’s husband asks if anesthesia is “six sigma?”
Six sigma is a term first coined in industrial process improvement by Motorola. It subsequently spread to many other industries and certification as an expert, or “black belt” is possible from several organizations. The term applies to industrial processes achieving a defect or failure rate of less than 3–4 per million (which is not, ironically, the same as six standard deviations or “sigma” from the mean but is commonly accepted as the working
Motorola pioneered a single-minded attention to quality improvement in the late 1980s and claimed to have achieved this level of quality in many of its manufacturing processes, saving tens of billions of dollars in the act. Virtually no process in medicine even approaches this level of quality but anesthesiology has likely come the closest, at least when defined as anesthesia-related mortality. In the 1940–1950s, Beecher and Todd estimated anesthesia mortality to be about 1 in 2500; by the 1980’s, Eichhorn estimated it to be 1 in 200,000, which is fairly close to the six sigma target. However, others have cautioned that other methodologies put the number at 1 in 46,000. So the answer must be a qualified “maybe” or perhaps “probably” and only vigilant efforts to continue to drive the number toward zero by anesthesia professionals can ensure that the field can earn such an honor.

Suggested Further Reading


Anesthesia patient safety foundation – www.apsf.org


Chapter 31

Ethical and Legal Issues in Anesthesia

Jesse M. Ehrenfeld

For maximum impact, it is recommended that the case study and questions found on page xxxiv are reviewed before reading this chapter.

Key Learning Objectives

- Understand the principle and process of obtaining informed consent
- Learn the definition of medical malpractice and how to avoid frivolous claims
- Know the procedure for addressing DNR/DNI status in the operating room

Informed Consent

Informed consent is a process in which a patient makes decisions and gives consent for procedures and treatments after having achieved a clear understanding of the facts and implications of taking a particular course of action. Contrary to popular belief, informed consent is a process – not just a signed legal document.

Informed consent is only possible when a patient is both (1) able to make rational decisions and (2) has received all of the relevant facts. Typical discussion points should include diagnosis, purpose of the therapy, possible risks and benefits, potential alternative therapies, and risks associated with not receiving the therapy. The process is summarized below.

Guide to Obtaining Informed Consent

- Informed consent is a process – not a signed document
- Informed consent should be obtained prior to administering sedatives
- The patient may accept/refuse any treatment (principle of patient autonomy)
The patient should receive a description of procedure, potential risks and benefits.

- Incapacitated patient (altered consciousness, incompetent, disabled)
  - does not have the ability to provide consent
  - next of kin or a healthcare proxy should provide consent instead

- If obtaining consent via telephone, make sure to obtain a witness

- Under emergency, life threatening situations, consent is implied and may be waived

- Use an official hospital interpreter for non-English speakers whenever possible

- Pediatric patients and minors (<18 years of age) cannot give consent for themselves (except pregnant patients in some states)

**Malpractice**

Medical malpractice is a legal definition for a specific type of negligence, where a professional (e.g. a physician) fails to follow professional standards and causes harm to a patient. In order for medical malpractice to have occurred, these items must be established:

- The physician had a duty to care for the patient
- The duty of care was breached
- The physician deviated from the standard of care (“What a prudent physician would do”)
- The breach caused harm to the patient

Keep in mind that even when physicians act appropriately, patients still may have adverse outcomes. It is therefore important to set appropriate expectations and inform patients about the potential risks of therapy before initiation of treatment. This will prevent confusion, ill-will, and unnecessary malpractice lawsuits.

**Advanced Directives**

Advanced directives are specific instructions given by a patient to direct providers on how to proceed if he/she can no longer make decisions because of illness or other incapacitation. There are a number of different types of advance directives including living wills and health care proxies. A living will provides specific instructions regarding particular treatment courses. For example, a living will may specify that the patient is not to receive specific interventions such as intubation or CPR. A Health Care Power of Attorney differs in that it
appoints another individual to make decisions on behalf of the patient should he/she become incapacitated. It does not provide specific guidance as to what those decisions should be.

**Do Not Resuscitate (DNR)/Do Not Intubate (DNI)**

Some patients will choose to forgo life saving treatments, such as intubation or CPR. Typically this decision is made near the end of a patient’s life or by a patient with a terminal illness. Keep in mind that patients have the right to choose whether or not resuscitative measures should be instituted in case of cardiac arrest.

These choices (DNR/DNI) are not automatically placed on hold should a patient come for surgery. It is therefore imperative that a discussion regarding a patient’s specific preferences be initiated prior to coming into the operating room. In this discussion, the patient should be asked to outline which therapies are acceptable and which are not during the operative period. Treatments typically discussed include intubation, CPR, defibrillation, and vasopressors. The outcome of the discussion and the patient’s choices should be (1) clearly documented in the chart, and (2) communicated to the entire operative team.

**Case Study**

An 80-year-old man has terminal colon cancer. He has metastatic disease with liver and brain metastases. As his condition worsened over the preceding year, he had several conversations with his family and physicians about his end of life care. He has a signed and witnessed advanced directive indicating his desire to be treated as “DNR/DNI” (do not resuscitate, do not intubate). He has now developed bowel obstruction and was admitted with severe abdominal pain. His surgeons have recommended a diverting colostomy for palliative care. They obtained consent for the operation from the patient last night, but anesthesia consent has not yet been obtained. The patient was medicated with hydromorphone and is now somnolent and falls asleep immediately upon waking. The surgeons are eager to operate before the bowel ruptures.

Can you obtain informed consent from the patient? Is surgical consent sufficient? What options do you have?

A somnolent, barely arousable patient can probably not give informed consent. Just waking up the patient long enough to obtain his signature on the
AnesthesiA student survivAl Guide

Informed consent is a process of discussing risks and benefits with the patient and allowing him to make an informed decision that the latter outweighs the former. The signature on the form merely documents the successful completion of the informed consent process. In emergency situations, many anesthesiologists will consider surgical consent to represent implied anesthesia consent, but this case is not such an emergency. There are separate risks and benefits associated with anesthesia and surgery, and there are some unique risks in this patient who has a DNR/DNI order. Separate consent is necessary, therefore. Your options hinge on whether or not the patient has designated a health care proxy to consent on his behalf. If he has, you should approach this person and have a complete discussion regarding the anesthetic risks and options. If the patient has left a detailed advanced directive, you may be able to follow this document and consider proceeding. If not, your options include waiting until the patient is more awake, partially reversing the effect of hydromorphone, or proceeding without consent. The latter option is problematic and should not be contemplated without consulting hospital lawyers or risk management first.

How should you interpret the patient’s DNR/DNI order for the operation, assuming you have obtained consent? You are planning general endotracheal anesthesia for the operation.

DNR/DNI orders usually indicate a patient’s wish in the setting of cardiac arrest or other extreme emergency, which may indicate death at the end of a fatal illness. These orders may not indicate the patient’s wishes in situations such as general anesthesia, when there is a reasonable expectation that the condition requiring intubation or resuscitative efforts is brief and reversible. For example, many patients with a DNR order may choose to undergo surgery with intubation and accept use of pressor agents to correct hypotension. However, they may not wish to be shocked or have CPR in the event of an intraoperative arrest. The main point is that like consent, the interpretation of a DNR/DNI order during surgery follows from a conversation with the patient or his proxy, not a set protocol. Part of the process you should employ to obtain consent is having this discussion. Conversely, some anesthesiologists and surgeons believe that consenting to operation necessarily means suspension of any DNR/DNI order. Many surgeons and anesthesiologists will only take patients to surgery if the DNR/DNI order is suspended. If this is done, then a specific timeframe for the suspension, and plan for resumption of the order, should be defined preoperatively.
If you proceed with surgery with general endotracheal anesthesia, and you are unable to extubate the patient at the end of the case, what will you do? Are you liable for a malpractice claim?

Assuming you have done the consent process properly, you will already know the answer to this question for this patient! In a critically ill patient undergoing abdominal surgery, there is a chance that postoperative intubation and ventilation may be required; your consent procedure should acknowledge this fact and a plan for what to do in this event should be made in advance. Some authorities believe that the operating room is a particularly difficult place for death to occur because surgeons and anesthesiologists routinely intervene aggressively. “Resuscitation” is what we do for a living! Therefore, some have argued, a decision to extubate or discontinue ventilation might be better made in the ICU than the OR, if for no other reason that the patient’s family can be present and participate in the decision-making.

Malpractice claims arise when a physician breaches a duty to a patient and causes harm. Although there is no guarantee that any given situation will not lead to a lawsuit, the mere fact that you are unable to extubate should not constitute malpractice unless you have not adequately counseled the patient and obtained informed consent.

Suggested Further Reading


Chapter 32

Clinical Simulation in Anesthesia Education

Emily M. Hayden

For maximum impact, it is recommended that the case study and questions found on page xxxiv are reviewed before reading this chapter.

Key Learning Objectives

- Understand the different types of simulation and their uses
- Learn how crisis resource management can be used to manage a critical scenario
- Know the expectations of a trainee experiencing a medical simulation

Introduction

Health care training is increasingly incorporating simulation into its curricula. Simulation laboratories provide a “safe” environment for trainees to practice clinical reasoning and procedural skills, where mistakes can be made and from which key lessons can be learned. Both teaching and assessment can occur in these laboratories (Fig. 32.1, Fig. 32.2, Fig. 32.3).

What Is Simulation?

The term “simulation” is a generic term for any technique that allows duplication or imitation of a portion of a clinical encounter. You likely have already learned using simulation during your medical training. If you participated in any form of problem-based learning using paper cases, then you have used simulation. If you have practiced suturing on a pig’s foot, or giving injections on an orange, you have used simulation. If you have been assessed using standardized patient encounters, you have used simulation.
In order to understand simulation better, it is helpful to categorize the various types of simulation. One set of classifications focuses on the objective of the simulation, such as cognitive, procedural, or teamwork practice. Another category focuses on the fidelity, or level of realism, of each simulation. Table 32.1 is a matrix with examples of different types of simulation.

All forms of simulation are used for either instruction (teaching) or assessment (testing). Many medical educators are excited about the possibilities of testing using some of the higher fidelity simulations. In addition, recent pressure from both society and various accrediting bodies has led to a search for examination methods that reliably test skills such as communication and teamwork. It is important to note that some forms of simulation lend themselves to assessment better than others.

**What Is the History Behind Medical Simulation?**

The first medical simulator mannequin was created in the 1960’s for anesthesiologists. It was not until the 1980’s with the advent of smaller and more affordable personal computers that mannequins were developed for mainstream medical training. Around this time, simulation was being used in other sectors,
such as aviation, nuclear power, and the military. Until the start of the twenty-first century, medical simulation was being used mainly in anesthesia and some surgical fields. Since the early 2000’s, medical simulation has spread to all levels of medical training (undergraduate medical school to continuing medical education) and into a number of different specialties.

What Is the Evidence Behind Medical Simulation?

There are several studies showing improved outcomes after the use of simulation. Some of the studies have examined the effect of medical simulation training on patient safety and clinical outcomes (Wayne et. al.), whereas other studies have demonstrated that simulation is an effective training tool for both procedural and cognitive skills (Hall et. al.).

How Is High-Fidelity Simulation Used in Medical Training?

High-fidelity simulation is used for several purposes in medical training. One of the more common uses is for “code” practice training emphasizing Advanced Cardiac Life Support (ACLS) and Advanced Trauma Life Support (ATLS) skills. Many hospitals use the full body mannequins as a platform for teaching, practicing, and assessing these specific skills.

High-fidelity simulation also is used frequently for team training, or crisis resource management. These scenarios bring people together in a simulation and have them practice management of a crisis or a chaotic situation. This is similar to the crew resource management from military aviation and nuclear power plants. In the 1970’s, studies by the aviation industry determined the causes of several airline accidents. From these findings, a program of “crew resource management” was developed. The same ideas from crew resource management were translated into the operating room environment and dubbed

<table>
<thead>
<tr>
<th>Fidelity</th>
<th>Cognitive</th>
<th>Procedural</th>
<th>Teamwork</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>Paper cases</td>
<td>Orange/Pig foot</td>
<td>Table-top exercise</td>
</tr>
<tr>
<td>Medium</td>
<td>Computer case</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>Full-body mannequin/patient actors</td>
<td>Task trainers</td>
<td>Full-body mannequin</td>
</tr>
<tr>
<td>Highest</td>
<td>Virtual reality</td>
<td>Task trainers with haptics (tactile sensation)</td>
<td>Virtual reality</td>
</tr>
</tbody>
</table>

Table 32.1 Matrix of categories of simulation.
“crisis resource management.” As a student, you may participate in some crisis resource management scenarios in the simulation laboratory.

Medical training institutions are beginning to use high-fidelity simulation in remediation or in root cause analysis of medical mistakes. Some institutions are bringing actual cases from morbidity and mortality reports to the simulation room for analysis and reflection.

The specialty of anesthesia was the first to embrace simulation as a part of the training process. Simulation has been used in anesthesia to teach cognitive aspects of the field, including knowledge content and clinical decision making, as well as crisis resource management.

**What Is Crisis Resource Management?**

Crisis resource management training is intended to improve patient safety by emphasizing teamwork and communication. The specific areas within crisis resource management focus on addressing communication, resource management, situational awareness, and role clarity. Key behaviors in crisis resource management include:

- Planning and anticipation of possible problems
- Clear communication
- Defined roles and assertive leadership
- Utilization of the resources available
- Task distribution
- Summoning of additional resources/personnel
- Situation reassessment

**What Is in a Simulation Laboratory?**

Each simulation laboratory is unique. Some have only full-body mannequins, while others have only task trainers for endoscopies or laparoscopies. Some have very realistic accoutrements with newly built or renovated centers, while others may be in very stark environments that do not resemble more than a classroom or a closet.

Most of the simulation centers you will encounter during your anesthesia rotations will involve full-body mannequin simulation. Typically, the physical layout of one of these simulation centers will include several rooms, one with the mannequin, one for the control room, and a debriefing room. The room with the mannequin usually looks similar to a clinical room, such as an operating room, an ICU room, or a floor room in the hospital. Sometimes the room is not
“decorated” as a clinical room, but is just a space with the mannequin and monitor. In this case, one room may hold all three areas: the mannequin, the control area, and the debriefing area. Before the simulation encounter, you should receive enough information to understand the clinical environment in which you are practicing, and thus what resources are available to you (Fig. 32.2).

The simulation room with the mannequin may have other equipment available. Some of this equipment may be for intubation, defibrillation (mannequins are able to receive real electric shocks), and medication administration. If a piece of equipment is not available in the room, you should be able to ask for it. Sometimes it will be given to you, and other times it will be “simulated”.

For all simulations, there is a control area where someone is controlling the voice/responses of the mannequin. This control area may be in direct view of you, in the same room behind a curtain, or in a separate room separated by one-way mirror or through cameras. Usually, you will not spend time in the control room if you are a learner in the sessions (Fig. 32.3).

A debriefing session will occur after the simulation experience. Some simulation centers have a separate room in which this will occur with audio-visual
capabilities to playback the scenario during the debriefing. The simulation leader should disclose if the session is being recorded.

There are several people you may have contact with while in the simulation laboratory. Around the mannequin, there may be other people working on the same case as you. These could be other students learning through the simulation, or they could be actors, or both. Depending on the purpose of the simulation, the actors may be there to try to cause you to make a mistake. One of the benefits of a simulation encounter is to be able to make mistakes and learn from them without harming a patient.

At least one faculty member will be present to lead the debriefing session after a simulation encounter. This faculty member may be a physician, but they also may be a nurse, a paramedic, or an educator. This person may be in the simulation room with you, or may be in the control room.

After a simulation scenario is complete, the faculty member debriefing the scenarios will discuss the case with the participants. The discussion will be driven by the objectives of the scenario. If the purpose of the simulation is teamwork or crisis resource management, then discussion points will revolve around the principles of teamwork. If the purpose of the scenario is
to understand a specific pathophysiologic concept, then the topics of conversation will focus on that concept. All debriefings should focus on discussion of the case. One of the powerful benefits of simulation is that it allows the participant to actively obtain knowledge by participating, rather than passively hearing the information in a lecture-style environment.

What Can a Mannequin Do?
There are a handful of full-body mannequin companies, and each has its own characteristics. Each company has different models of the mannequin, with the more expensive mannequins able to portray responses or exam findings that are more complex. The mannequins range in price from $20,000 to $250,000 each.

In general, mannequins have chest rise, breath sounds, heart sounds, and pulses. Mannequins can often talk, and may blink or have reactive pupils. An important note about the physical exam findings on mannequins is the variability of the fidelity of the findings. If you have a question about a specific exam finding, then you can say that you hear a specific lung finding. A facilitator will be able to help you if this is not the planned physical exam finding. Do be aware that the physical exams are not perfect, and some of the findings may not seem realistic.

The mannequins can usually be intubated, defibrillated, cardioverted, and paced. Be aware that the equipment used to deliver electricity may be live – as the mannequins can receive actual electricity. In some scenarios, you will be required to perform the actual procedure on the mannequin or on a task trainer, such as an intubation head model. In other scenarios, you may be asked to verbalize what you would do for a procedure. You should be notified before the scenario on how you will “perform” a particular procedure in the simulation lab.

The mannequin will have a monitor similar to the monitors available in the ICU or operating room. The mannequin’s monitor may need to be “activated” by either asking for the monitor, or placing a blood pressure cuff or oxygen saturation probe to receive the respective vital signs.

What Should You Expect in the Simulation Laboratory?
The first question you should ask the person leading the simulation is if there will be a formal assessment of your performance during the encounter. As you are keenly aware of your clinical rotations, you may alter your behavior
if you know you are being graded (even though medical educators wish this were not true). You will receive verbal feedback on how you performed in the debriefing scenario, but you may not have formal assessment written as part of your record. If you are being assessed, it will be important to ascertain the rater’s expectations of your performance.

If you are being assessed and a formal evaluation is being given, your performance typically will be videotaped. If your performance is videotaped, a question you should ask is how long the video will be available, how it will be secured, and when it will be destroyed. There has not yet been a medical malpractice suit that has asked for the videotapes of prior years of training for the defendant, but this does not mean that it cannot happen in the future. It also is important for you to have a chance to experience the simulation before the examination, so that your unfamiliarity with the set-up does not compromise your grade.

If your simulation experience will be for practice without formal assessment, then you can relax more and be open to learning. One great advantage of the simulation encounter is for you to practice, reflect upon your actions and thoughts, and have the chance to practice again. In studies of experts in many fields, Ericsson has found that to become an expert, one needs to have opportunities for deliberate practice with coaching and feedback. These scenarios allow you to stretch yourself and have a safe area for you to make mistakes.

Some centers will have you work as an individual in the case, a situation where you are in the “hot seat.” Sometimes you will have more students allowed into the scenario throughout the case, or you may be able to ask for a consultant to help. Other scenarios will have a group of trainees working together to manage the case. If you have multiple cases, you may experience a combination of the above approaches during multiple encounters.

**What Are the Expectations of You, the Learner, in Simulation Scenarios?**

In general, you will be expected to manage the patient in the scenario to the best of your abilities. Do not expect to have seen the case previously. Sometimes scenarios are of rare events that you may have not managed in the past, but should know how to if the situation arises. Again, keep an open mind and do what you think you should do in the encounter. The faculty member observing the case will be able to directly observe your actions and will likely ask for your unspoken thoughts in the simulation scenario. Once you have been a trainee in
the simulation laboratory, you may wish that all of your clinical encounters in the “real” world were observed and debriefed.

Some encounters will have pre-scenario reading or other preparation; however, most encounters will give you no advance idea regarding the case content. Either way, there are reasons behind the approach.

The final expectation of you is feedback on the process and content of the simulation and debriefing. Just as you will receive feedback on your performance, the staff at the simulation laboratory desire feedback on the scenario.

How Do You Make the Best of the Simulation Experience?
The best way to take advantage of the simulation experience is to be open-minded and behave as you would in real life. Again, this is an opportunity to have feedback on your performance and reasoning by someone more expert than you are. There are not many other situations in training where you will be directly observed as you will be in the simulation laboratory.

Case Study
It is the last day of your rotation. You are doing a case completely by yourself in the simulator. You are surprised by how nervous you felt in the beginning, as if the patient you are caring for is not the mannequin in front of you but a real patient. But there is no attending guiding you, and you have heard that sometimes things go very wrong in the simulator. You are not being graded, but you are being videotaped, and you know that your fellow students and the instructors will be reviewing your performance. But so far it has been a quiet case. Your “patient” is undergoing an abdominal operation under general anesthesia. You handled the application of monitors, induction of anesthesia, mask ventilation and endotracheal intubation like a pro. The patient is being mechanically ventilated. You are using desflurane, nitrous oxide, fentanyl, and vecuronium for anesthesia. You are using standard monitors and have a peripheral 18 G IV in place. Blood loss has been about 100 mL but the surgeons anticipate more later in the case, and you have blood available in the blood bank. You are feeling pretty good about yourself, thinking that you might enjoy anesthesiology as a career. After all, you have learned a ton of the basics in your period, and here you are doing a case pretty much by yourself!

Suddenly, all the lights in the room go off and the room falls into an inky blackness and eerie quiet.
It does not stay quiet for long. The surgeon shouts that he has just incised a structure and is concerned that the patient may be bleeding. He is screaming for light and help and accusing you and the circulating nurse of causing a power failure. The circulator is screaming back at the surgeon that she did not do anything (and that neither had you). What are your first steps in assessing the situation?

Find some light. Most anesthesia stations include a flashlight, often kept in one of the drawers in the anesthesia machine. If you cannot find it, reach for your laryngoscope. If your operating room is above ground, there may be natural light in the hallway, so you can open the OR door. Try to take control of the chaos with a firm but calm voice, explaining what you are doing to everyone, preferably by name. Next, quickly survey the patient's condition and that of your anesthesia equipment. If both regular and emergency power are off, nothing electronic without a backup battery will be working. This may include your monitors and some anesthesia machine ventilators, and the desflurane vaporizer. Also communicate with the other OR personnel briefly and directly to make sure no one is injured (especially electrocuted!) and to assess the criticality of the current stage of the surgical procedure.

The surgeon says that the operation is at a critical juncture but that if he can work for 5–10 min, he will be at a stable stage and could end the operation with a quick closure. He is still concerned that the patient may be bleeding. How can you get him enough light to continue?

Since you are ventilating the patient, you can offer him your laryngoscope or flashlight. Every anesthesia set-up includes at least two laryngoscopes, so you can use one and the surgeon can use the other.

You recognize that both the ordinary hospital power supply and the emergency power have failed. Your ventilator is still functioning on battery backup. All of your monitors are not functional except for the BIS brain monitor, which is running on battery power. How will you alter your anesthetic?

You cannot monitor the patient very well so it would be prudent to discontinue nitrous oxide and ventilate the patient with 100% oxygen. Unless the hospital gas supplies fail, you should be able to alter the inspired gases accordingly. Your desflurane vaporizer will be inoperable, because it requires power to heat and store desflurane gas; recall that it is a gas blender, not a true vaporizer. Other vaporizers are purely mechanical devices, so you
can switch to another agent, perhaps sevoflurane because of its rapid onset as desflurane is eliminated from the patient. Although low fresh gas flows are tempting, to preserve the desflurane in the body, this must be tempered by the need to ensure high flow oxygen and eliminate nitrous oxide until you can monitor oxygenation. Your BIS monitor can help you maintain a reasonable plane of anesthesia during this transition. Total intravenous anesthesia would eliminate the need for the anesthesia machine altogether, but it is difficult to manage without infusion pumps. Longer acting intravenous agents such as ketamine are possible backups. Your patient is paralyzed with vecuronium, and you will have to make a judgment regarding the relative merit of continuing its use vs. allowing the patient to regain the ability to breathe spontaneously should the emergency continue.

How will you monitor the patient?
You will have to rely on your senses and manual monitors! Your twitch monitor (neuromuscular blockade monitor) is battery powered and can still be used, whether you continue vecuronium, allow it to wear off, or actively reverse neuromuscular blockade, all of which are reasonable options depending on the surgical requirements and your need for spontaneous ventilation. The BIS monitor will work as long as its battery is charged. Some pulse oximeters have battery backup (but yours does not appear to!). You can attempt to monitor oxygenation grossly by the patient’s color, but this will be difficult without a steady and bright light source. You can monitor blood pressure with a manual cuff, which is present in every properly set-up operating room. You can also use breath and heart sounds as qualitative monitors of respiration and cardiac output, heart rate, and rhythm. Palpating peripheral pulses is always prudent as a qualitative measure of cardiovascular condition.

The battery backup on your ventilator has now run out of power and the ventilator stops. The oxygen flowmeter drops to zero and you realize that the pipeline oxygen supply has failed. How will you proceed?
You will activate the backup oxygen tank supplies in the back of the anesthesia machine by opening the valve on the neck of the green tank. Now, it may be prudent to reduce fresh gas flows to preserve your limited supply. You will manually ventilate the patient through the anesthesia machine; the carbon
dioxide absorbent, vaporizer, and oxygen flowmeter are all still functional. You should locate the manual respirator ("Ambu" bag) in case you need to ventilate the patient for transport or if you run out of oxygen.

*The lights come back on; your instructor walks into the room and announces, "That's a wrap!" Your colleagues break into applause. You have learned a lot indeed!*

### Suggested Further Reading


Appendix A

ASA Difficult Airway Algorithm

Excerpted from “Standards for Basic Anesthetic Monitoring” (Approved by House of Delegates on October 21, 1986, and last amended on October 25, 2005), of the American Society of Anesthesiologists. A copy of the full text can be obtained from ASA, 520 N. Northwest Highway, Park Ridge, IL 60068-2573, USA.
Malignant Hyperthermia

Richard D. Urman

Definition
Malignant Hyperthermia (MH) is an inherited disorder of skeletal muscle, which is characterized by a hypermetabolic state and can be triggered by potent volatile anesthetics (but not nitrous oxide) and depolarizing muscle relaxants such as succinylcholine. Patients with some congenital myopathies may also be at increased risk when exposed to triggering anesthetic agents. However, all intravenous hypnotic agents are considered safe. MH is a potentially fatal disorder if it is not promptly recognized and treated, and the overall incidence during general anesthesia is about 1:50,000–1:100,000. For any patient presenting for anesthesia, a preoperative history should include questions about prior MH episodes or family history suggestive of MH.

Mechanism
In a vast majority of cases, MH-susceptible patients have a defective calcium channel (known as ryanodine receptor) that is located on the sarcoplasmic reticulum membrane. In normal cells, calcium is released into the cell during muscle contraction. In MH, there is a problem with calcium reuptake, and therefore there is a massive increase in intracellular calcium leading to sustained muscle contractions. Consequently, there is an increased demand for oxygen and ATP in the muscle cells, leading to glycolysis and lactic acidosis. If left untreated, this uncontrolled hypermetabolism results in cell hypoxia, rhabdomyolysis, organ failure, and death.
Presenting Signs and Diagnosis

The most common presenting features of MH include significant, unexplained elevation in expired CO₂, tachycardia, steady temperature rise, muscle rigidity, rhabdomyolysis, acidosis, and hyperkalemia. MH may occur at any time during anesthesia and in the postoperative period. The earliest signs are usually tachycardia and an increase in expired CO₂; a rise in temperature may follow. Diagnosis of MH can be made on the basis of these signs, although the variability in the order and time of the onset of signs often makes clinical diagnosis difficult. These signs may present during or after the administration of the anesthetic. Table B.1 outlines possible presenting signs of MH.

Diagnosis is made based on the presenting signs, and other potential conditions that might cause the same symptoms should be ruled out. Genetic testing is also available, which can be done on an outpatient basis at an MH Testing Center. If MH is suspected, treatment should be initiated as soon as possible.

Treatment

_all triggering agents should be discontinued immediately, the surgical procedure should either be aborted or finished quickly, and patient cooling begun_. Dantrolene, a muscle relaxant which abolishes excitation-contraction coupling in muscle cells, is the main drug of choice. Important treatment modalities for MH are outlined in Table B.2.

Over the last several decades, thanks to provider education and increased knowledge about MH, perioperative patient mortality from MH has dropped from 80% to less than 5%. An MH-susceptible patient is still a candidate for any type of anesthetic, including general, regional, or local. If general anesthetic is...
required, a total intravenous anesthetic (TIVA), with or without nitrous oxide would be a safe option.

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