Jesse M. Ehrenfeld
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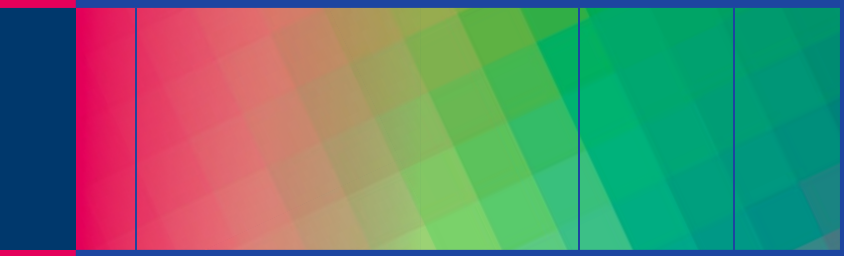

Anesthesia Student Survival Guide

A Case-Based Approach

Second Edition

Springer

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Preface

We are excited to introduce the new and updated second edition of *Anesthesia Student Survival Guide: A Case-Based Approach*. As with the prior edition, our goal was to provide a concise, easy-to-use, and up-to-date introduction to the practice of anesthesiology.

This book is unique in many ways and is primarily intended for students during their anesthesia rotation, although junior residents may also find it useful. The book 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, when reading a particular section, we suggest you first read the case associated with the section, followed by the chapter, and then try to answer the questions in the case on your own *before* reading our sample answer. 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.

As educators, we are indebted to generations of students at our respective institutions – Harvard Medical School, Vanderbilt University School of Medicine, and Tufts University School of Medicine – who inspired us to write this practical "survival" guide, and we are thankful for the support and expertise of our contributors.

We would also like to thank Dr. Joseph Garfield for his outstanding editorial contributions and Drs. Katharine Nicodemus and Zina Matlyuk-Urman for their tireless support, encouragement, and guidance. Finally, a special thanks to our families.

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

Nashville, TN, USA — Jesse M. Ehrenfeld, MD, MPH
Boston, MA, USA — Richard D. Urman, MD, MBA
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Foreword to the First Edition

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. *Anesthesiology 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 anesthesiology specialty. 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.

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 a case-based study tool 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 outside the operating room. It 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, USA

Michelle Szabo, MD
Roger Russell, MD
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Case Studies

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 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 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 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?*
- *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 be used 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 h. 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 in. tall and weighs 260 lb. 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 min, 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 toward 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 Fig. 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 setup?*

- *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 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?*
- *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 l, depending on the findings at operation and extent of major vascular involvement. The estimated surgical time is 6 h. She has a peripheral 14G 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 h ago but had little appetite at the time and so states that it was "just a little." She has a 20G 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. Sixty 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 1930s 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 postoperative 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 ft, 185 lb. 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 165×10^9/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 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?*
- *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 ft, 6 in. tall and weighs 300 lb. 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 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 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 of 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 8 h and last drank liquids more than 2 h 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 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 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 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?*
- *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 min 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 aortobifemoral 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 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?*
- *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 3 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 reenter 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 nonmedical industries, and her father, a retired surgeon in his late 1970s. She is anxious because her father has told her stories of surgery in the 1950s and 1960s, when he remembers a significant number 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 1950s 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 18G 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 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?*
- *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?*

Part 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 future of the medical specialty of 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

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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 (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:

1. Understand basic physiology/pharmacology:
   (a) Review respiratory and cardiac physiology (Chap. 17)
   (b) Review autonomic nervous system pharmacology (Chap. 3)
   (c) Review cholinergic, anticholinergic, cholinesterase actions/Interactions (Chap. 7)
   (d) Review opiate pharmacology (Chap. 5)
   (e) Review local anesthetic pharmacology (Chap. 6)
2. Formulate specific goals:
   (a) Become proficient at mask ventilation
   (b) Intubate 7 patients successfully
   (c) Place LMAs in 3 patients
   (d) Observe at least 1 epidural
   (e) Observe at least 1 spinal
   (f) Observe at least 1 peripheral nerve block
   (g) Spend at least one day in a pain clinic or work with a pain team

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 more frequently becoming direct hospital-paid employees or employees of a larger multispecialty or national group.

Within the specialty, individuals may opt to complete advanced training or fellowships after residency in one of five American Board of Medical Specialties (ABMS) certified areas: critical care medicine, pain medicine, adult cardiothoracic anesthesia, obstetric anesthesiology and pediatric anesthesiology. There are also nonaccredited opportunities for additional training in 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. Finally, there is a relatively new option to obtain board subspecialty certification in Clinical Informatics. This requires completion of a clinical informatics fellowship.

The location, size, and type of practice you choose will ultimately affect your practice model. 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 simply 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 (although this will transition to a PhD requirement within the next decade) 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 six 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 Future of the Medical Specialty of Anesthesiology

Along with the rest of healthcare, the medical specialty of anesthesiology is undergoing some dramatic changes and common questions students ask are: Will the medical specialty of anesthesiology continue to exist, particularly given the growing use of independent nurse anesthetists and anesthesia assistants? What is the role of the physician lead, care team model? And how will technology, including closed loop anesthesia control systems, impact the practice of anesthesia?

It is clear that field of anesthesiology continues to evolve – and while the way in which anesthesia is practiced today, may not be the way it is practiced in a decade from now, there will always be a need for well trained, qualified, anesthesiologists. The growth of the perioperative surgical home model, along with the increasing demand for physicians who can design and create systems that can efficiently deliver high quality, cost-effective care, will drive this demand.

As you contemplate entering the field of anesthesiology, keep an open mind. You very well may be the person that helps define what the field looks like for the next generation of anesthesiologists!

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, whether applying for PGY1 or PGY2 positions. 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.

Program directors take personal statements, Dean's letters, class ranking, grades, and letters of recommendation into account as well as considerations such as geography and medical school reputation, but most will use the step-1 score as an initial screening tool. Most have hundreds of applicants to consider, and generally will interview ~10 candidates per position available, making the selection process complicated.

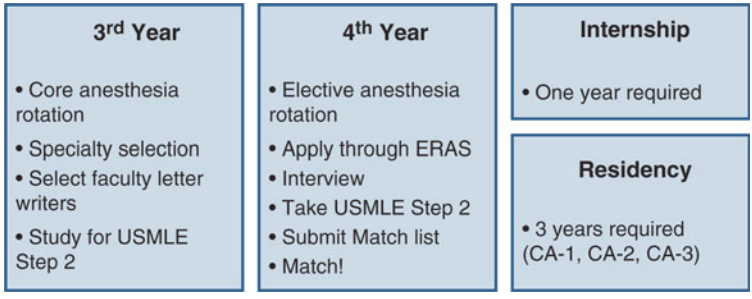

Figure 1.1 Key milestones on the road to the match (Figure Courtesy J. Ehrenfeld)

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 (national average for interview threshold, for those programs having such a threshold, was 208 in 2012, and average step-1 score of successfully matched students was 222). In general, once you've made it to the interview stage, step-1 scores become less important.

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. Having personal experience in a department can be a great way to gain an advantage over competing candidates and can make up for less-than-impressive test scores.

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.

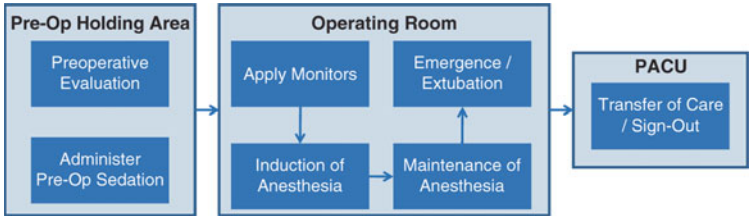

Figure 1.2 Phases of a typical general anesthesia case (Figure Courtesy J. Ehrenfeld)

Prior problems with anesthesia are noted, and physical exam should focus on the heart, lung, and airway (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 s, amnesia, and depression of airway reflexes. Other agents include thiopental (a barbiturate), 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 s), 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 & McGrath video laryngoscopes, 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 (exhaled) CO_2, 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, inhaled 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), and rocuronium (5–10 mg every 30 min as determined by periopheral 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. Most importantly, the patient must be able to protect his/her own airway…you've probably seen anesthesiologists asking patients to "open your eyes!" at the end of the case…no, there's no oculo-airway reflex, but we assume that once a patient is awake enough to follow simple commands, that patient is also awake enough to protect his/her airway.

Volatile agents are rapidly exhaled once inspired vapor is turned off, and most intravenous agents have a short enough half-life to ensure rapid awakening. Paralytics usually are actively reversed with cholinesterase inhibitors (increasing acetylcholine available to compete with the paralytics), 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 many aspects of intraoperative and ICU care.

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. You don't want to be an anesthesiologist? That's OK… just direct your efforts to 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, clinical monitoring, and, above all, safety. They have to establish patient rapport rapidly, allay fear, and educate their diverse patients. Remember to read ahead, ask plenty of questions, and have fun!

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 hemoglobin and whether she has a sample in the blood bank. You will verify that surgical and anesthetic consent forms have been signed before giving her any preoperative medications. You will check the admission vital signs.

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 lungs (and more specifically 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 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 min 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 antimuscarinic (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, 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, IVs, urinary catheter bag, and any other attached items to a stretcher, you are ready for transport to the postanesthesia care unit (PACU; "recovery room").

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

1. Medical Student Anesthesia Primer. http://www.anesthesia-education.com/primer.doc

2. Society for Education in Anesthesia (SEA). www.seahq.org

Chapter 2

History of Anesthesia and Introduction to the Specialty

David C. Lai and Jesse M. Ehrenfeld

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 world 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

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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

Figure 2.1 "Ether day, 1846" pictured are Gilbert Abbott (the patient), John Collins Warren, M.D. (the surgeon), William T. G. Morton (the anesthetist) and Henry J. Bigelow, M.D. (the junior surgeon) (Painting by Warren and Lucia Prosperi. Photograph by Andrew Ryan. Reproduced with permission from Massachusetts General Hospital, Archives and Special Collections)

impact on surgical anesthesia practice solidified after the first successful public demonstration of ether by a Boston dentist, William T.G. Morton (Fig. 2.1). 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

Figure 2.2 The Ether Monument, Boston, Massachusetts (Photograph J. Ehrenfeld, M.D.)

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,

Table 2.1 Historical dates of note

1500 BC	The use of opium-like preparations in anesthesia recorded in the Ebers Papyrus
1275	Ether discovered by Spanish chemist Raymundus Lullius
1540	The synthesis of ether was described by German scientist Valerius Cordus
1665	First intravenous injection of an opiate through a quill
1773	Joseph Priestly introduces nitrous oxide
1842	Crawford W. Long successfully uses ether during neck tumor excision in Jefferson, Georgia
1845	Horace Wells publicly demonstrates the use of nitrous oxide in Boston – however, it is labeled a "failure"
1846	William T.G. Morton shows first successful public demonstration of ether anesthesia at Massachusetts General Hospital, Boston
1847	James Young Simpson uses Chloroform for labor pain
1853	John Snow administers chloroform to Queen Victoria during childbirth
1878	William MacEwan introduces oro-tracheal intubation with a flexible brass tube
1884	Carl Koller discovers local anesthetic properties of cocaine
1889	August Bier describes spinal anesthesia for surgery
1894	Anesthetic charts introduced
1905	Long Island Society of Anesthetists founded
1921	Fidel Pagés describes a lumbar approach to epidural anesthesia
1932	First barbiturate, hexobarbital, used clinically
1941	Robert Miller and Sir Robert Macintosh introduce "Miller" and "Macintosh" blade concepts, respectively
1942	Harold Griffith uses curare for the first time during an appendectomy
1956	Michael Johnstone introduces halothane, a halogenated inhalational agent
1960s	Fentanyl, ketamine and etomidate synthesized
1977	Propofol is synthesized
1970s	Pulse oximeter is developed and becomes widely available for use in 1980s
1980s	Halothane gradually replaced by enflurane and isoflurane
1983	Archie I.J. Brain introduces Laryngeal Mask Airway (LMA)
1986	ASA House of Delegates passes "Standards for Basic Anesthetic Monitoring" resolution
1990s	Sevoflurane and Desflurane introduced into clinical practice
2000s	Anesthesia Information Management Systems (AIMs) come into widespread use
2013	FDA approval of the computer assisted closed loop sedation devices

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.

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

Figure 2.3 **Ether mask and bottles (Photo J. Ehrenfeld, M.D.)**

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 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 anesthesiology 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 anesthesiology 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 anesthesiology. 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 40 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."

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.

Which anesthetics are you most likely to use?
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.

Which intravenous agents will you administer?
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).

How will you administer the anesthetic? How will you manage the airway?
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!

How will you monitor the patient?
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

1. Ellis RH, Sykes WS (eds) (1982) Essays on the first hundred years of anaesthesia, vol 3. Wood Library-Museum of Anesthesiology, Park Ridge

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Part II

Pharmacology

Chapter 3

Pharmacology Principles

Jerome M. Adams, John W. Wolfe, and Jesse M. Ehrenfeld

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.

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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 V_d 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.

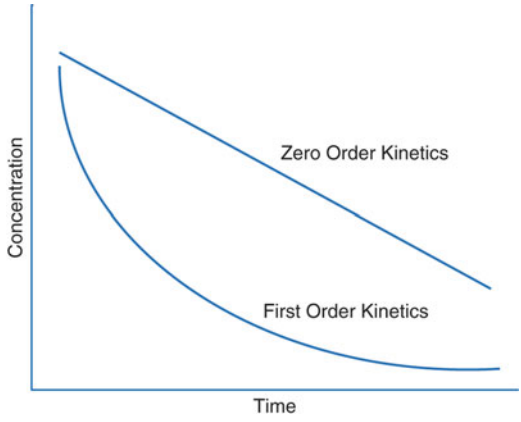

Figure 3.1 Zero vs. first order kinetics (Image Courtesy J. Ehrenfeld)

- ***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).

Table 3.1 Drug remaining at multiples of the half-time

Number of half-times	Percent of drug remaining	Percent of drug removed
0	100	0
1	50	50
2	25	75
3	12.5	87.5
4	6.2	93.8
5	3.1	96.9

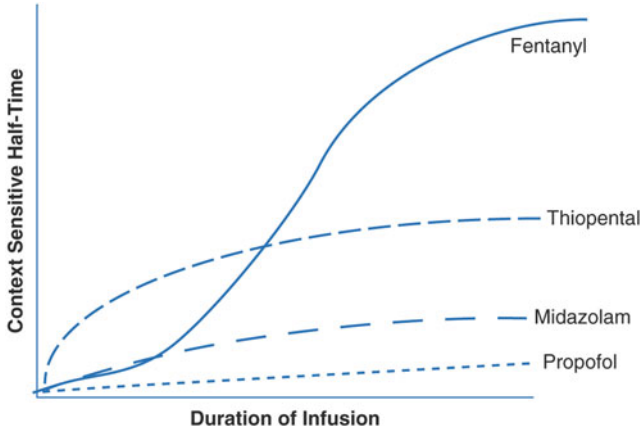

Figure 3.2 Context sensitive half times as a function of duration of infusion (Image Courtesy J. Ehrenfeld)

- ***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.

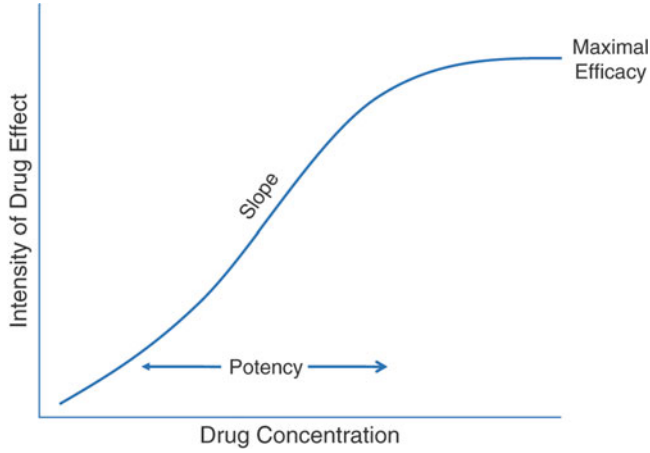

Figure 3.3 Drug dose response relationship (Image Courtesy J. Ehrenfeld)

- ***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.

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 CO_2 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

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Chapter 4

Pharmacology of Intravenous Anesthetic Agents

Jerome M. Adams, John W. Wolfe, and Jesse M. Ehrenfeld

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

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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 4.1 Opioid receptor subtypes & effects

	μ/δ	κ
Analgesia	Supraspinal/spinal	Spinal
Respiratory rate	↓↓	↓
GI motility	↓	
Sedation	↑↑	↑
Dependence	↑↑	↑
Other effects	Euphoria	Dysphoria

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

Opioid	Dose[a] (mg)	Peak (min)	Duration (h)
Morphine	10	20–30	3–4
Meperidine	80	5–7	2–3
Hydromorphone	1.5	15–30	2–3
Fentanyl	0.1	3–5	0.5–1
Sufentanil	0.01	3–5	0.5–1
Alfentanil	0.75	1.5–2	0.2–0.3
Remifentanil	0.1	1.5–2	0.1–0.2

[a]Approximately equianalgesic dosages

IV opioids are the primary means by which pain is controlled for surgical patients. While short-acting opioids such as fentanyl and its derivatives are used 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 (1000 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.

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 CO_2 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 2 min) which antagonizes mu receptors. Other adverse side effects of opioids

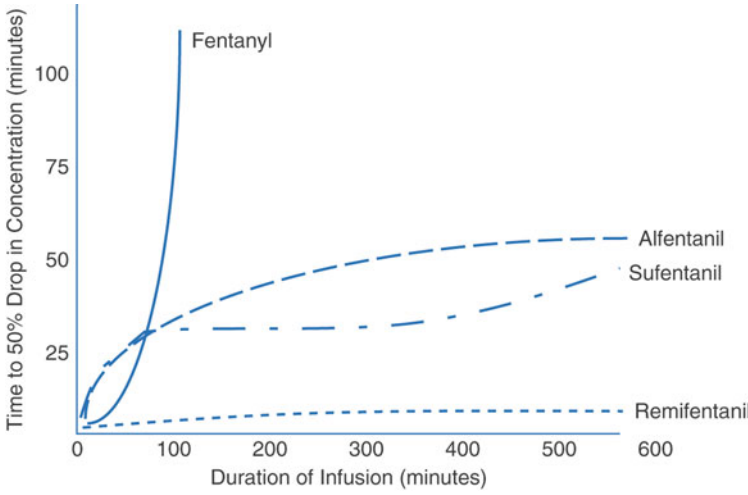

Figure 4.1 Context-sensitive half time for opioid infusions (Image Courtesy J. Ehrenfeld)

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.

Table 4.3 Recommended drug dosages for common IV agents[a]

Benzodiazepines[b]	Induction agents
Midazolam 1–4 mg	Propofol 2–2.5 mg/kg (induction), 25–200 mcg/kg/min (infusion)
Diazepam 2.5–10 mg	Thiopental 3–5 mg/kg
Lorazepam 1–4 mg	Etomidate 0.2–0.5 mg/kg
Opioids (Bolus)[b]	Ketamine 1–2 mg/kg IV, 3–4 mg/kg IM (induction or bolus),
Morphine 1–5 mg	1–2 mg/kg/h infusion
Hydromorphone 0.2–0.5 mg	*Neuromusclular blockers*[c]
Fentanyl 25–100 mcg	Succinylcholine 1–2 mg/kg, 20 mg bolus for laryngospasm
Meperidine 25–50 mg	Rocuronium 0.6 mg/kg
	Vecuronium 0.1 mg/kg
	Cisatracurium 0.15 mg/kg
	Pancuronium 0.1 mg/kg

[a]Adult dosages: always start at low end of range and titrate up
[b]Titration ranges for premedication or intraop/post op bolus dosing
[c]Intubating dosages: divide by 3 for ED95, divide by 5 for maintenance bolus dosing

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 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.

Table 4.4 Cardiovascular effects of IV induction agents

Drug	Mean arterial pressure	Systemic vascular resistance	Cardiac output	Contractility	Heart rate	Intracranial pressure
Propofol	↓↓	↓↓	↓↓	↓↓	↓↓	↓
Thiopental	↓		↓	↓	↑	↓
Etomidate	–	–	–	–	–	↓
Ketamine	↑	↑	–	–	↑	↑

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. 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

Table 4.5 Contraindications to succinylcholine use

Elevated serum potassium levels (>5.5 meq/L)
History of burn injury
History of denervation injury
Known or suspected myopathy
Known or suspected risk for malignant hyperthermia
Known pseudocholinesterase deficiency

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,

Table 4.6 Neuromuscular blocking drugs

Drug	Onset	Duration (min)	Mode of elimination	Important notes/ side effects
Succinylcholine	30–45 s	5	Plasma cholinesterase	Increased duration in pts with pseudocholinesterase
Cisatracurium	2–4 min	30–40	Hoffman degradation	Deficiency
Vecuronium	2–4 min	30–40	Liver/renal	Few side effects
Pancuronium	4–6 min	120–180	Liver/renal	Can cause tachycardia & hypertension
Rocuronium	1–2 min	30–40	Liver/renal	May be used for rapid sequence induction if succinylcholine contraindicated

vecuronium, and pancuronium). NMBs exert their effects by competitively 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.

Table 4.7 Reversal of neuromuscular blockade

Neostigmine 50 mcg/kg paired with glycopyrrolate 10 mcg/kg
Edrophonium 500 mcg/kg *paired with* atropine 20 mcg/kg

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.

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.

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Chapter 5

Pharmacology of Inhalational Anesthetics

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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

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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

Table 5.1 Physical characteristics of inhalational anesthetics

Agent	Vapor pressure (20 °C)	Blood: gas partition coefficient[a]	Fat: blood partition coefficient	Metabolism	Pungency[b]
N_2O	38,770 mmHg	0.46	2.3	0	None
Desflurane	669 mmHg	0.42	27	0.02 %	High
Sevoflurane	157 mmHg	0.65	48	5 %	Low
Isoflurane	238 mmHg	1.46	45	0.2 %	High

[a]The low blood: gas partition coefficients (i.e. low solubility in blood) of nitrous oxide, desflurane, and sevoflurane speed induction and emergence
[b]Due to their low pungencies, nitrous oxide and sevoflurane are excellent agents for inhalational induction of anesthesia by mask

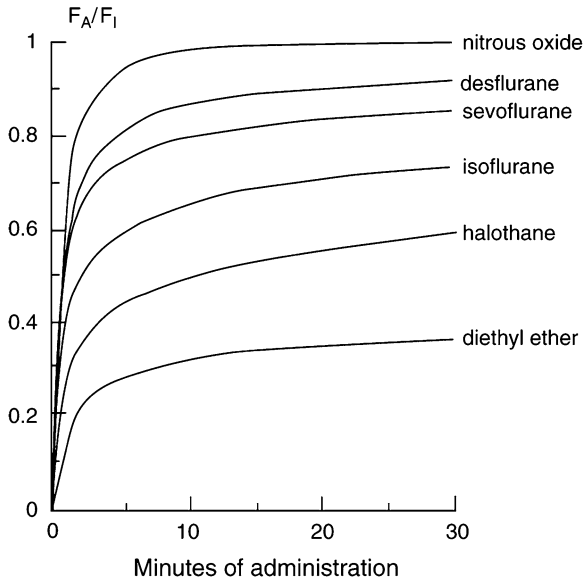

Figure 5.1 Ratio of concentration of anesthetic in alveolar gas to inspired gas. Graph shows how the ratio between the inspired (FI) and alveolar (FA) concentrations of inhalational anesthetics changes with time of administration. The least soluble drugs approach equilibrium (FA/FI) the fastest (From Modern Anesthetics: Handbook of Experimental Pharmacology, by Helmut Schwilden, Springer 2008. Used with Permission)

increases the arterial concentration of the agent, thereby speeding induction of the anesthetic effect. The reverse of this phenomenon is seen on emergence, 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

Table 5.2 Minimum alveolar concentration (MAC) values

Agent	MAC (%)
Desflurane	6.0
Sevoflurane	2.05
Isoflurane	1.15
Halothane	0.75
Nitrous oxide	105

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 MAC), allowing estimation of anesthetic depth in patients receiving more than one agent concurrently (usually a volatile anesthetic and nitrous oxide).

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

Table 5.3 Factors affecting MAC

Increased MAC	Decreased MAC
Children (from infancy to adolescence)	Premature infants and the elderly
Hypernatremia	Hypothermia
Cocaine or amphetamine intoxication	Pregnancy
Chronic alcohol use	Acute alcohol intoxication
MAO inhibitors	Opiates, benzodiazepines, barbiturates, clonidine, dexmedetomidine
Tricyclic antidepressants	

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 %). 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 solubile 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 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 atm 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 N_2O 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.

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Chapter 6

Pharmacology of Local Anesthetics

John W. Wolfe, Jerome M. Adams, and Jesse M. Ehrenfeld

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.

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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 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 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.

Table 6.1 Pain fiber types

Fiber type	Local anesthetic sensitivity	Size	Myelination
A	+	Large	Yes
B	++	Medium	Yes
C	+++	Small	No

Additionally, commercial preparations of local anesthetics typically have 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

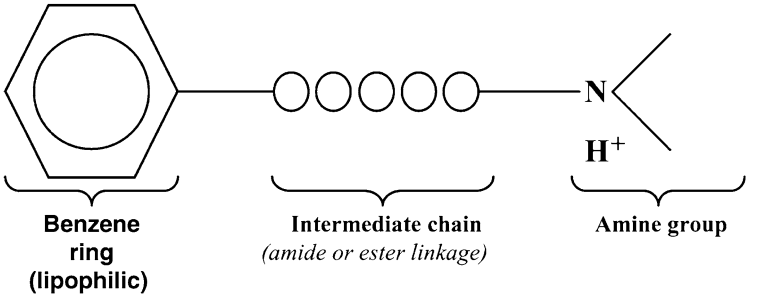

Figure 6.1 Local anesthetic structure

- **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:

intravenous › tracheal › intercostal › caudal › epidural › brachial plexus › sciatic › 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.

Table 6.2 Properties of common local anesthetic agents

	Agent	Onset of action	pK_a (36 °C)	Max dose (mg/kg)[a]	Duration of action (h)
Amides	Lidocaine	Rapid	7.8	4.5 (7 with epi)	1–2
	Mepivacaine	Moderate	7.7	5 (7 with epi)	1.5–3
	Prilocaine	Slow	8.0	6 (9 with epi)	1–2
	Ropivacaine	Slow	8.1	2.5 (3 with epi)	4–8
	Bupivacaine	Slow	8.1	2.5 (3 with epi)	4–8
Esters	2-Chloroprocaine	Very Rapid	9.1	9 (15 with epi)	0.5–1
	Procaine	Rapid	8.9	7 (10 with epi)	0.75–1
	Tetracaine	Slow	8.4	1.5(2.5 with epi)	3
	Cocaine	Rapid	8.7	1.5	0.5

[a]Maximum dose for a single subcutaneous injection

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.

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Chapter 7

Pharmacology of Adjunct Agents

Jerome M. Adams, John W. Wolfe, and Jesse M. Ehrenfeld

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

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Table 7.1 Actions of vasoactive receptor sites

Receptor	Receptor site action
α-1	Glycogenolysis, gluconeogenesis, constricts vascular smooth muscle, relaxes GI tract
α-2	Constricts vascular smooth muscle, decreases insulin secretion and norepinephrine release
β-1	Increases heart rate and contractility, secretion of renin
β-2	Glycogenolysis, gluconeogenesis, relaxes vascular smooth muscle and bronchioles
Δ-1	Increases renin release, dilates vascular smooth muscle
Δ-2	Constricts smooth muscle, inhibits norepinephrine release

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.

Table 7.2 Receptor actions of commonly used vasopressors

Drug	Direct	Indirect	Site of action
Ephedrine	+	++	α, β
Phenylephrine	+		α
Norepinephrine	+		α, β
Dopamine	++	+	α, β, D (dopamine receptor)

Table. 7.3 Vasopressor dosing

Ephedrine	2.5–10 mg IV bolus
Phenylephrine	40–100 mcg IV bolus or 20–150 mcg/min infusion
Norepinephrine	0.01–0.1 mcg/kg/min infusion
Dopamine	2–20 mcg/kg/min infusion

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).

Table 7.4 Commonly used antiemetics[a]

Ondansetron (Zofran)	4 mg IV, may repeat × 1 (0.1 mg/kg up to 4 mg in children)
Dolasetron (Anzemet)	12.5 mg IV, may repeat × 1
Granisetron (Kytril)	0.5–1 mg IV
Promethazine (Phenergan)	12.5–25 mg IV, may repeat × 1
Dexamethasone (Decadron)	4–8 mg IV, best given early during intraoperative period
Droperidol (Inapsine)[b]	0.625 mg IV, may repeat q 10 min × 3

[a]All dosages for adults unless noted
[b]Must monitor ECG for 2 h post administration

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.

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 (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 α2 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 antisialogogue. 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, 1000–1500 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 longacting 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

1. Morgan GE, Mikhail MS, Murray MJ (2005) Chapter 12: Andrenergic agonists and antagonists (p 239–254). In: Clinical anesthesiology. McGraw Hill, United States

2. Blum RH, Heinrichs WL (eds) (2000) Nausea and vomiting: overview, challenges, practical treatments and new perspectives. Whurr, Philadelphia

3. Katzung BG, Furst DE, Ulrich RW, Prakash S (2001) Chapter 36: Nonsteroidal anti-inflammatory drugs. In: Basic and clinical pharmacology. McGraw Hill, New York, pp 635–658

Part III

Preoperative Considerations

Chapter 8

The Preoperative Patient Evaluation

Amit Gupta and Timothy J. Shiveley

For maximum benefit, it is recommended that the case study and questions found on pages xx are reviewed prior to reading this chapter.

Key Learning Objectives

- Understand the key components of a preoperative anesthetic evaluation
- Recognize how cardiovascular risk factors relate to intraoperative morbidity
- Learn the ASA physical status classification system

Introduction

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

- establishing an excellent physician–patient relationship
- obtaining a thorough medical history
- performing a physical exam, including a detailed airway assessment
- ordering and reviewing pertinent tests and consultations
- reviewing the patient's medical records, including previous anesthetic records, if available
- ordering appropriate preoperative medications
- obtaining informed consent

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

J.M. Ehrenfeld et al. (eds.), *Anesthesia Student Survival Guide*,
DOI 10.1007/978-3-319-11083-7_8

The Interview

Among the most vital components 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 patients prior to surgery. A high anxiety state may have a negative impact 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 positive 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 should be implemented to ensure 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 difficult mask ventilation is estimated to be high, careful anesthetic planning (e.g., ensuring that an advanced airway equipment is available) is needed.

Factors affecting mask ventilation
Presence of a beard
BMI > 26 kg/m²
Missing teeth
Age > 55
History of snoring
Adapted from Langeron [10]

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 may increase the patient's risk of dental injury or aspiration of a dislodged tooth or tooth fragment.

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 or cardiac surgery) prior to elective surgery.

Patients should be asked about any history of shortness of breath, dyspnea, chest pain, chest tightness, edema, hypertension, myocardial infarction, cardiac surgery, use of anticoagulants, diuretics, antihypertensive medication, use of antibiotics before dental work, 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 2007 Guidelines on Perioperative Cardiovascular Evaluation & Care for Noncardiac Surgery recommend the following stepwise

Table 8.1 Energy requirements for various activities

1 MET	Eating, getting dressed, working at a desk
2 METS	Showering, walking down eight steps
3 METS	Walking on a flat surface for one or two blocks
4 METS	Raking leaves, weeding or pushing a power mower
5 METS	Walking 4 miles per hour, social dancing, washing car
6 METS	Nine holes of golf carrying clubs, heavy carpentry, using push mower
7 METS	Digging, spading soil, singles tennis, carrying 60 lb
8 METS	Moving heavy furniture, jogging slowly, rapidly climbing stairs, carrying 20 lb upstairs
9 METS	Bicycling at a moderate pace, sawing wood, slow jumping rope
10 METS	Brisk swimming, bicycling uphill, walking briskly uphill, jogging at 6 MPH
11 METS	Cross-country skiing, full court basketball
12 METS	Running continuously at 8 MPH

Adapted with permission from Brigham and Women's Hospital Preoperative Assessment Form

approach to evaluating a patient's cardiac status for patients undergoing noncardiac surgery:

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 surgery is not emergent, then 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 (≤1 month before surgery)
- 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 mmHg, aortic valve area <1.0 cm^2, or symptomatic)

If the patient has one or more of the above conditions, then the cardiac issue should be evaluated, clarified, and treated appropriately. This often involves postponing 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 are unlikely to alter 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.

Table 8.2 Clinical risk factors for increased perioperative cardiac risk ACC/AHA 2007 Guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary. JACC Vol. 50, No. 17, 2007

- History of heart disease (MI, Q waves on ECG, known CAD)
- History of compensated or prior heart failure
- History of cerebrovascular disease
- Diabetes mellitus
- Renal insufficiency

# Clinical risk factors	Surgical Risk	Action
0	Any	Proceed with surgery
1–2	Intermediate risk surgery	Proceed with surgery with heart rate control
	Vascular surgery	or
3^{+}	Intermediate risk surgery	Consider cardiac testing if it will change management
	Vascular surgery	Consider cardiac testing if it will change management

Source: ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery: Executive Summary. JACC Vol. 50, No. 17, 2007.

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.

Pulmonary

Postoperative pulmonary complications may prolong the patient's 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 (Table 8.2).

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)

Table 8.3 Risk reduction strategies for pulmonary complications

Preoperative
Smoking cessation (for at least 8 weeks)
Treat airflow obstruction (patients with COPD or asthma)
Prescribe antibiotics / postpone surgery in presence of respiratory infection
Educate patients about lung-expansion maneuvers
Intraoperative
Limit surgical duration < 3 h
Avoid pancuronium
Consider laparoscopic surgical approach
Postoperative
Encourage incentive spirometry and deep breathing exercises
Initiate CPAP (continuous positive airway pressure) when indicated
Consider epidural analgesia/intercostal nerve blocks

Adapted from Smetana et al. Preoperative pulmonary evaluation. NEJM. Vol 340: 2008

Potential *procedure-related* risk factors include:

- Surgery > 3 h
- General anesthesia
- The type of surgery
- Use of pancuronium[1]

Clinical evaluation should encompass a thorough history (i.e., inquiring about shortness of breath, wheezing, chest pain, recent fever/chills, bronchitis, asthma, emphysema, 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 8.3) can be implemented to optimize patient care.

[1]A prospective study of 691 patients revealed three times as many pulmonary complications among the patients receiving pancuronium from residual paralysis (Smetana, GW. Preoperative Pulmonary Evaluation. NEJM. Vol 340 (12);1999).

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.

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 nausea, vomiting, heartburn, food regurgitation, diarrhea, bloody stools, hiatal hernia, gastric ulcers, viral hepatitis, and alcoholism.

Bleeding Disorders

Bleeding disorders may increase the 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), thrombocytopenia, leukemia, platelet disorders (e.g., Bernard–Soulier syndrome, uremia), certain medications (e.g., warfarin, heparin, clopidrogel), cancer, and liver disease.

Endocrine

Endocrinopathies may 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, and/or insulin regimen) should be assessed, along with a fasting glucose 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 the degree of nephropathy, if present. 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 be scheduled for elective surgery earlier in the day to minimize the impact of prolonged fasting on their glucose management.

Perioperative mortality associated with *pheochromocytoma* and *carcinoid* syndrome can reach 50 % if undiagnosed. Thus, screening patients for any history of thyroid, parathyroid, adrenal, or pituitary disease, and carcinoid syndrome, may help reduce potential perioperative risks.

Renal

History of any kidney disorder holds importance during the preoperative evaluation since abnormal renal function may result in secondary physiologic imbalances, abnormal platelet function (impaired aggregation), anemia, electrolyte imbalances, peripheral neuropathies, and altered drug metabolism and excretion. Investigation of a patient's history of renal insufficiency, renal failure, and dialysis dependence (including timing and frequency) should therefore be undertaken.

Neurologic

When screening a patient for neurologic disease, the anesthesiologist should elicit a history of seizures, convulsions, tremors, headaches, numbness or tingling of an extremity, nerve injuries, and multiple sclerosis. Performance of a neuraxial technique or a regional nerve block requires knowledge of any previous nerve injuries or deficits (and documentation if present).

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 begins with noting the 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, prominent incisors, a beard or moustache, a large tongue, neck masses, tracheal deviation, or if the 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 limited 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, dosage, and timing (including time of last dose) should be noted. In some cases, it is helpful to include the length of time the patient has been taking a given medication – particularly opioids as chronic use may lead to higher perioperative opioid requirements. Additionally, long-term use of steroids may result in adrenal insufficiency and steroid supplementation during surgery may be indicated.

A medication history should also encompass any over-the-counter or alternative medicines (i.e., herbal medications). This is important because many supplements have clinically 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 dysrhythmias).

Patients often state allergies to medications, foods, and environmental agents. It is important to note what allergic reactions are caused by which medications, and the severity of those reactions. For example, does penicillin cause a mild rash or an anaphylactic reaction? Also, it is important to differentiate allergic reactions from 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 amount of a patient's medical history – **of** which the patient may or may not be aware. They may include information that could alter **the anesthetic** plan. For example, a history of a difficult airway may lead to a decision to perform an awake fiberoptic intubation. 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 a family history of anesthetic complications 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 4 billion dollars are spent annually in the United States on preoperative laboratory and diagnostic studies. Therefore, these investigations should be ordered based on the patient's medical history and surgical procedure. Unnecessary testing may result in OR delays or even cancellations due to false positive results. Table 8.4 illustrates the diagnostic and laboratory tests that correspond to specific medical conditions and procedures.

Table 8.4 Suggested indications for preoperative studies

	CBC	T/S and albumin	β-hCG	PT/PTT	Electrolytes	BUN/Cr	Glucose	AST/alkphos	ECG	CXR
Disease-based indications										
Alcohol abuse	x			x				X	x	
Adrenal cortical disease	x				x		x			
Anemia	x									
Diabetes					x	x	x		x	
Hematologic abnormalities	x	x		x						
Hepatic disease				x		x		x		
Malignancy with chemotherapy	x			x		x		x	±	x
Malnutrition	x	x		±	x					
Morbid obesity						x	x		x	
Peripheral vascular disease or stroke	x				x	x	x			
Personal or family history of bleeding	x			x				±		
Possibly pregnant			x							
Pulmonary disease	x								±	x
Renal disease	x				x	x			x	
Rheumatoid arthritis	x								x	x
Sleep apnea	x								x	

Smoking>40 pk-yr	x										x	x
Therapy-based indications												
Radiation therapy	x										x	x
Use of anticoagulants	x			x								
Use of digoxin and diuretics					x		x				x	
Use of statins									x		x	
Use of steroids					x		x	x				
Procedure-based indications												
Procedure with significant blood loss	x	x										
Procedure with radiographic dye							x					

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 8.5 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 a regional technique depends on the patient's co-morbidities and the nature of the operation. Planning to perform an awake intubation instead of intubating after inducing general anesthesia may be indicated for patients with history of 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 versus. ICU). Anesthesia is a *proactive* specialty rather than *reactive* because the pre anesthetic evaluation can prompt change in patient management (see Table 8.6), 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 his/her representative the types of anesthetic options available for the planned procedure and to explain the possible risks and benefits that encompass the anesthetic plan.

Table 8.5 ASA physical status classifications

ASA Class I	A normal healthy patient
ASA Class II	A patient with mild systemic disease
ASA Class III	A patient with severe systemic disease that limits activity, but is ***not*** a constant threat to life
ASA Class IV	A patient with incapacitating system disease that is a constant threat to life
ASA Class V	A moribund patient not expected to survive 24 h with or without surgery
E	Designates an emergency surgical procedure (i.e., Class IE)

American Society of Anesthesiologists Newsletter, 1963

Table 8.6 Formulation an anesthetic plan based on patient history

Patient history	Area to evaluate	Anesthetic considerations
Airway perceived as difficult to intubate or ventilate	Head, eyes, ears, nose, throat: airway; prior anesthesia outcomes	Obtain fiberoptic equipment; obtain skilled help
Asthma	Pulmonary disease	Optimize therapy; use bronchodilators; consider extubating during deep anesthesia
Diabetes, insulin-dependent	Endocrine, metabolic, diabetes	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
Drug abuse	Social history	Consider HIV and Hepatitis testing; prescribe medications to avoid withdrawal symptoms in perioperative period
Gastroesophageal reflux or hiatus hernia	Gastrointestinal disease: hiatus hernia	Administer H_2 antagonists or oral antacids and consider rapid-sequence induction of anesthesia; or use awake intubation techniques and obtain appropriate equipment
Heart disease: valve disease, risk of subacute bacterial endocarditis	Cardiac history and exam, imaging studies	Consider antibiotic prophylaxis. Arrange for antibiotic administration 1 h prior to surgery
Personal malignant hyperthermia history, family history, or suspected potential history	Prior anesthetic/surgical history	Obtain clean anesthesia machine (new CO_2 absorbent, remove vaporizers, flush circuit with 10 L/min for 10 min); use appropriate technique and precautions; have agents to treat malignant hyperthermia available
Monoamine oxidase inhibitors	CNS: psychiatric/medication	Discontinue therapy preoperatively if patient is not suicidal; plan for perioperative pain therapy
Pacemaker or automatic implantable cardiac defibrillator	Cardiovascular disease: electrocardiogram	Evaluate cause of pacemaker implementation; obtain repolarizing equipment or magnet; use electrocautery with Bovie pad placed appropriately (monopolar); use bipolar electrocautery if possible
Peripheral motor neuropathy	CNS disease: neurologic deficit	Avoid depolarizing muscle relaxants; adjust dose of non-depolarizing muscle relaxants appropriately
Pregnancy or uncertain pregnancy status	Genitourinary: pregnancy	Monitor fetal heart rate; use oral antacids; adjust induction of anesthesia; determine status of pregnancy
Pulmonary tuberculosis	Pulmonary disease: tuberculosis	Use disposable breathing circuit or clean equipment; ensure adequate treatment of patient prior to surgery
Renal insufficiency	Genitourinary disease	Monitor fluid status intraoperatively

Adapted from Fischer et al. [11]
***CNS* central nervous system, *HIV* human immunodeficiency virus, *ICU* intensive care unit, *PACU* postanesthetic care unit**

Types of anesthesia typically discussed include general, monitored anesthesia care (MAC), regional, and local. While patients might prefer one type of anesthetic over another, the final decision should involve collaboration of all parties (patient, anesthesiologist, and surgeon).

Some of the more common risks of anesthesia that should be mentioned include infection and bleeding if a regional technique is performed, nerve injury from improper positioning or regional anesthetics, postoperative nausea and vomiting, dental injury, risk of viral hepatitis and HIV from blood transfusions, awareness under anesthesia and a need for postoperative mechanical ventilation (if the patient fails to meet extubation criteria after surgery). The aim of the consent process is to provide the patient with all available anesthetic options and possible risks due to the selected anesthetic technique 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 the patient (or his/her guardian) 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 anesthetic options. It is not merely the signing of a consent form.

Case Study

You are seeing a 64-year-old male 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 multiple 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 his diseases. He is likely not class IV, which implies his

systemic disease 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 underwent stent placement 2 years ago and is no longer taking clopidogrel (Plavix), so the major danger related to stent occlusion and/or anticoagulation has passed. However, we should 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 with elevated cardiovascular risk. He should continue both medications 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, unanticipated 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) is 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 preoperatively, since his surgery is elective. 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 smoking cessation, the immediate preoperative period may not be the optimal time. Studies suggest smokers who abstain for 8 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 postoperative period 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 metformin 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 perioperatively to avoid significant hypo- or hyperglycemia.

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

All patients should undergo a comprehensive airway examination such as determination of the Mallampati class (oropharyngeal structures visualized), thyromental distance, and cervical spine flexibility. 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 essential. 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. One should ensure that the patient and family members have the opportunity to voice any concerns about the surgery or the anesthetic, and an attempt should be made to address them. Finally, anesthesia consent should be obtained, and presence of surgical consent should be verified before proceeding to the OR.

Suggested Further Reading

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Chapter 9

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

- Understand the relevance and importance of airway management in anesthesiology
- 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 intubate and mechanically ventilate, regardless of whether or not such interventions were part of the primary anesthetic plan. A thorough airway

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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.

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 facemask, 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 (Fig. 9.1).

In order to predict difficult mask ventilation or difficult endotracheal intubation, each patient receiving anesthetic care should have a comprehensive

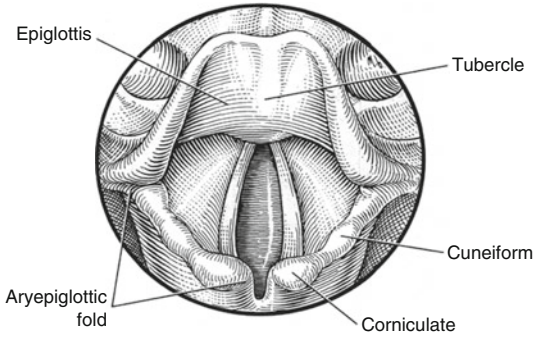

Figure 9.1 The glottis and epiglottis (Reproduced with permission from Finucane and Santora [7])

airway history and physical examination performed (also see Chap. 8). 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, obstructive sleep apnea, 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).

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. 9.2).

Table 9.1 Components of the preoperative airway physical examination

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

Reproduced with permission from Caplan et al. [8]

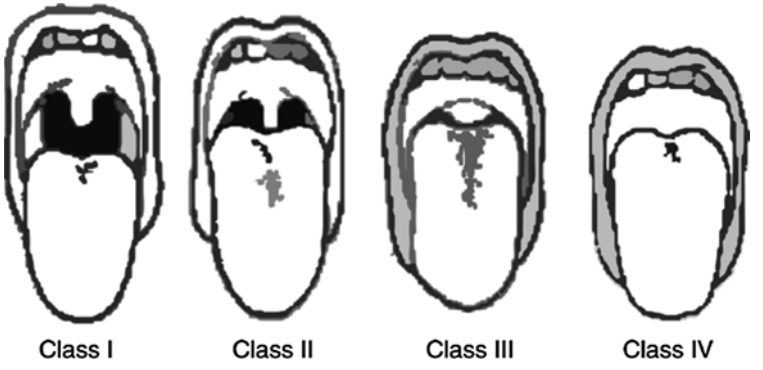

Figure 9.2 Modified Mallampati classification system (Samsoon and Young)

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 facemask 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 facemask and extension of the head as seen in Fig. 9.3. Placement of an oral or nasal airway during facemask ventilation 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. 9.4.
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.

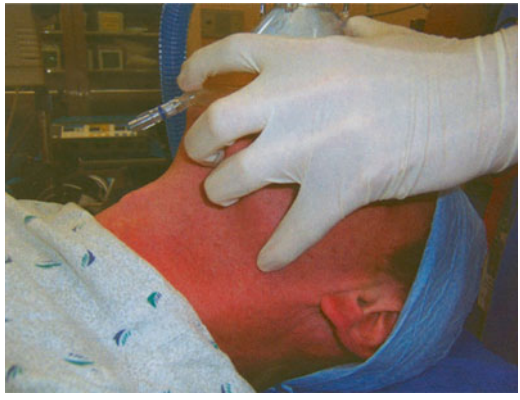

Figure 9.3 Optimal facemask ventilation

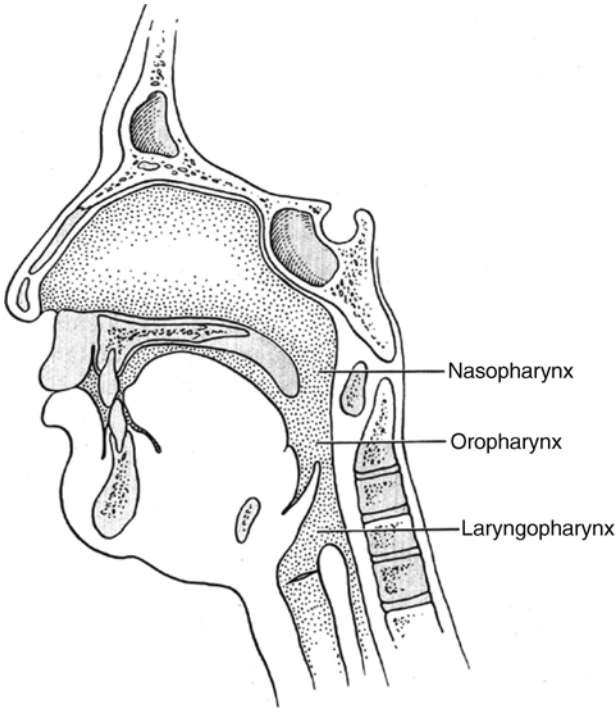

Figure 9.4 Upper airway anatomy (Reproduced with permission from Finucane and Santora [7])

Laryngeal Mask Airway

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 facemask 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.

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.

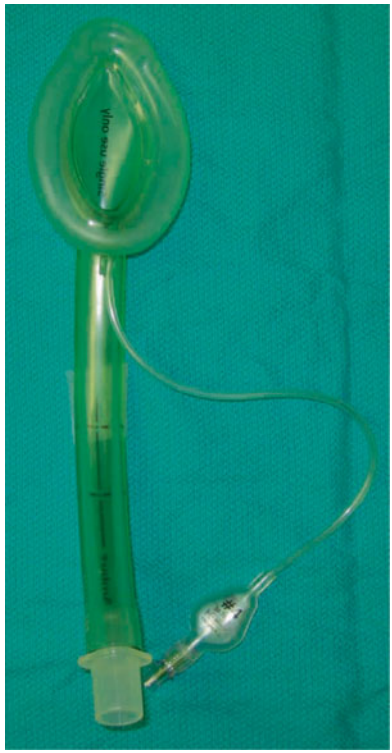

Figure 9.5 Laryngeal mask airway (Photo courtesy J. Ehrenfeld)

The cuff is inflated isolating the gastrointestinal tract from the respiratory tract above the glottis. 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

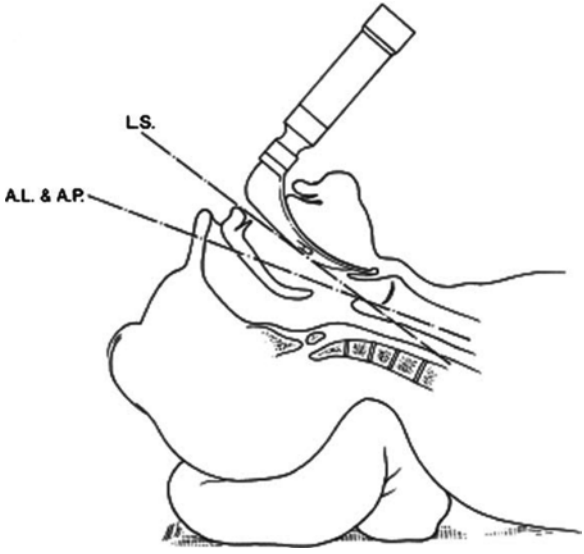

Figure 9.6 Relationship of the oral, pharyngeal, and laryngeal axes for intubation (Reproduced with permission from Ref. [7])

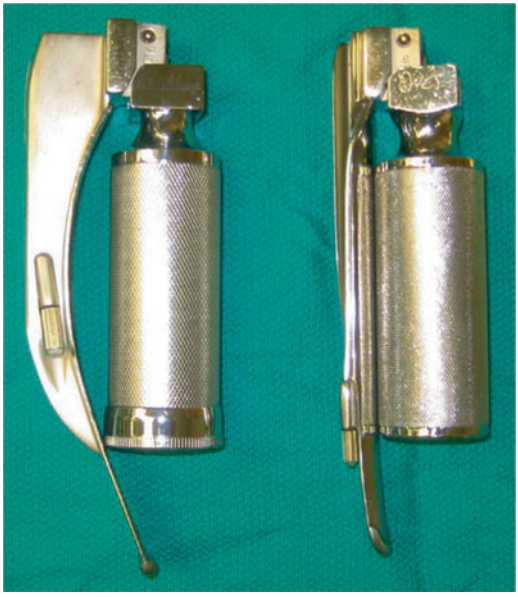

Figure 9.7 Laryngoscopes: Macintosh and Miller blades (Photo courtesy J. Ehrenfeld)

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
- patients with severe 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 with oxygen that can occur with facemask 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, C-MAC)

There have been several airway management tools introduced that combine traditional laryngoscopy with fiber-optic or digital optical technology such as the Glidescope, C-Trach, or C-MAC. One of the benefits of these instruments is that they may allow intubation under conditions such as limited mouth opening that might have been more difficult or 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 and safe approach to utilizing a number of different management techniques for securing the airway. These may 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 lb. 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 will 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. Anesthesiologists often have a preferred approach to situations such as these which is guided by the ASA Difficult Airway Algorithm. It is generally prudent to use a technique that you are experienced 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.

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Chapter 10

The Anesthesia Machine

Alvaro Andres Macias

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 which have been progressively engineered over the last several decades.

The Anesthesia Machine Components

The anesthesia machine can be divided into sub-systems, each with its own function and characteristics. One way to look at the machine is in terms of the pressure of the gases inside of each part of the machine. Using this approach one can divide the machine into a **high** pressure and a **low** pressure system.

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 (Table 10.1).

J.M. Ehrenfeld et al. (eds.), *Anesthesia Student Survival Guide*,
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Table 10.1 Components of the high and low pressure systems

High pressure system
–Pipelines & cylinders for the gases delivered
–Fail-safe valve
–Pressure regulator
–Oxygen flush valve
Low pressure system
–Flowmeters
–Vaporizers
–Flow control valve
–Check valve
–Common gas outlet

Basic Anesthesia Machine Components
Source of gases (O_2, N_2O, Air)
Flowmeters
Vaporizers
Scavenging system
Note: the breathing circuit & CO_2 absorber are separate from the machine.

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 then go into the flowmeters, then into the vaporizers, and finally the anesthesia circuit and patient via the common gas outlet (see Fig. 10.1).

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. 10.2) is a non interchangeable 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.

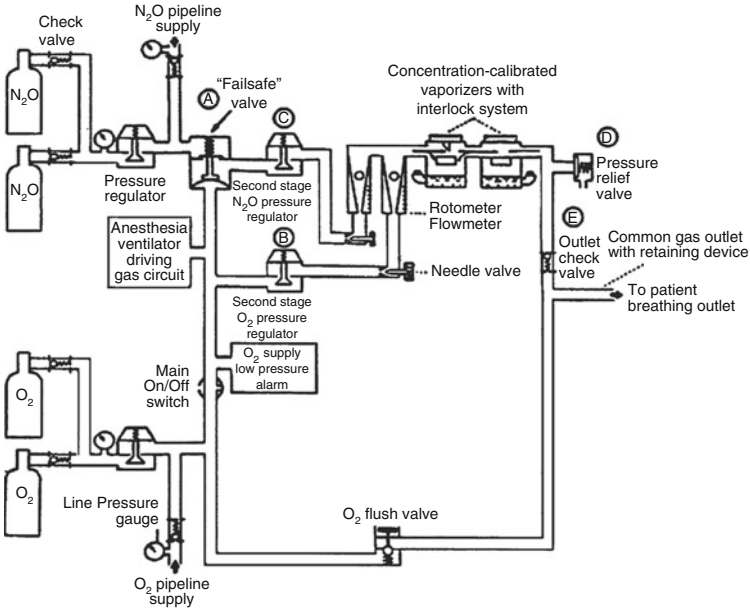

Figure 10.1 Flow arrangement of a basic two-gas anesthesia machine (Reproduced with permission from Check-out: a guide for preoperative inspection of an anesthesia machine, ASA, 1987. Reproduced by permission of the American Society of Anesthesiologists, 520 N. Northwest Highway, Park Ridge, Ill)

Figure 10.2 Diameter index safety system

Cylinder Inlets

Gas cylinders use the pin index safety system (PISS) 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

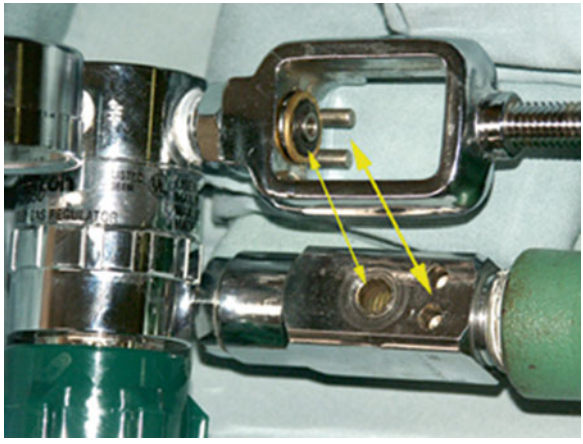

Figure 10.3 Pin index safety system

Table 10.2 Properties of E cylinders

Properties of E cylinders	Oxygen	Air	Nitrous oxide
Color of cylinder	Green	Yellow	Blue
Capacity	625 l	625 l	1590 l
Pressure when full	2200 psi	750 psi	1800 psi
Physical state	Gas	Gas	Liquid & gas

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 (Fig. 10.3).

Cylinders

Cylinders come in a variety of standard sizes. The most commonly used in the operating room are E-cylinders (Table 10.2). By understanding the physical properties of the gases stored in a cylinder, one can calculate the amount of gas (and or time) left in a cylinder.

> *For example: if the pressure gauge on an oxygen cylinder reads 1100 psi and you plan to deliver oxygen at a rate of 6 L/min → one could estimate that there are 312 l remaining in the tank (1100 psi/2200 psi x 625 l). At 6 L/min, one could deliver oxygen for approximately 52 min.*

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 2200 psi and a full nitrous oxide tank delivers gas at 745 psi. In order to ensure 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 still be delivered. This is why inclusion of a working oxygen analyzer (see below) 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 before the flow meters are considered part of the high-pressure circuit whereas those after them are considered part of the low-pressure system. There are three types of flow meters: variable-orifice, electronic and constant-pressure.

Gas flow increases when the flow valve control is turned counterclockwise – delivering the amount gas desired. It is worth mentioning that flow meters 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 U.S.) to reduce the chances of delivering a hypoxic mixture should a leak occur within a flowmeters.

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 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 deliver to the patient.

There are in two types of vaporizers currently in use: variable-bypass 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.

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. For this vaporizer there is no fresh gas flow though it. Instead the vaporizer simply releases the amount desired and then mixes it with fresh gas. Of note is that these vaporizers do not compensate for changes in elevation.

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 time.

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 mis-filling, vaporizers are color coded and have agent-specific keyed filling ports that only accept the correct key or straw (Fig. 10.4).

Gas Outlet

The gas outlet transports fresh gas carrying volatile agent from the machine to the breathing circuit.

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. 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.

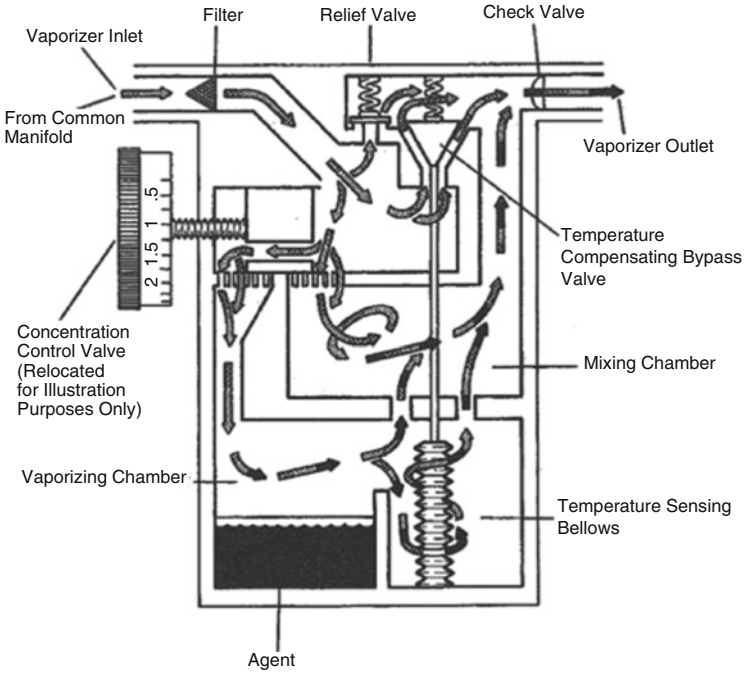

Figure 10.4 Vaporizer schematic (Reproduced with permission from Biomedical Engineering Handbook. Bronzino, J. Springer Press 2000. Figure 84.2, page 86)

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 FiO_2 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 ppm (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 hazards that come with it. If the system becomes occluded, it may deliver excessive positive pressure to the patient increasing the risk of barotraumas. 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, free of volatile agent and at a high flow (400–55 l/min), directly to the breathing circuit at any given time. Looking carefully at Fig. 10.1, 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 the gases from the machine to the patient we need a breathing system. Multiple designs have been developed and they are classified as closed, semiclosed, 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.

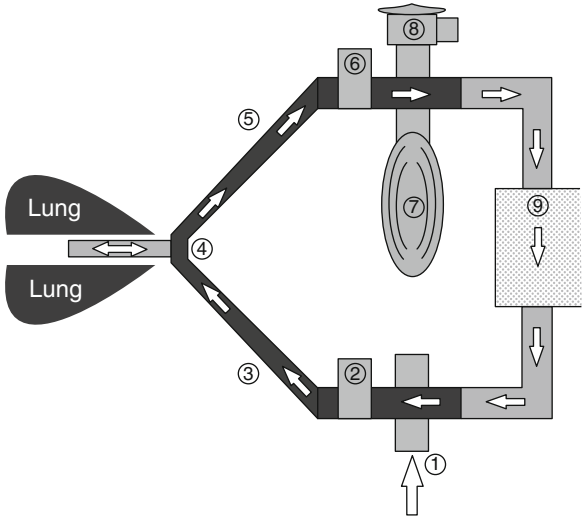

Figure 10.5 Anesthesia machine circle system flow schematic (Image courtesy J. Ehrenfeld)

The most common system in use these days is the **circle system**. The circle system (see Fig. 10.5) is composed of an inhalation check valve, inspiratory limb, Y-piece, expiratory limb, exhalation check valve and APL valve, reservoir bag, bag/vent selector switch and a 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 gas flow delivered to the patient, rebreathing of exhaled gases may occur. In order to prevent rebreathing of CO_2, unidirectional valves and a CO_2 neutralizing system are in place. These devices prevent any CO_2 rebreathing and in many cases reduce the total amount of inhaled anesthetic that must be put into the system.

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. CO_2 absorbent

Humidifiers

Because fresh gas delivered to a patient by an anesthesia machine bypasses all of the natural mechanisms which humidify inspired gases (nasal vessels, 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 any dead space to the circuit, passive humidifiers do.

The Mechanical Ventilator

The mechanical ventilator is a sophisticated and key piece of equipment that has evolved substantially in the last 20 years. There are two basic ventilatory modes, volume control ventilation (VCV) and pressure control ventilation (PCV). Other modes have been developed lately by taking advantage of sophisticated computer algorithms including pressure support ventilation (PSV), synchronized intermittent mandatory ventilation (SIMV), pressure controlled volume guaranteed ventilation (PC-VGV), etc.

In the VCVVolume control ventilation (VCV), the tidal volume to be delivered to the patient is set up by the anesthesia provider and the machine delivers the set tidal volume (TV) each breath, in this mode the pressure delivered by the machine can change with each tidal volume delivered depending on the patient's lung characteristics. Usually there is a high-pressure alarm that will go off if the machine reaches that pressure to deliver the pre set tidal volume. For example let's say one wants to deliver a TV of 600 ml, with each breath the machine will deliver 600 ml of mix gases but the inspiratory pressure may change with each breath.

On the other hand in the PCV mode, the anesthesia provider presets a maximum pressure to be delivered by the machine to reach an approximate

tidal volume. In this case each tidal volume delivered each breath will vary depending on the patient's lung characteristics. Usually there is an alarm that will go off if pre set pressure entered cannot be reached. For example let's say one wants to deliver a peak inspiratory pressure (PIP) of 20 cm H_2O, in this case with each breath the machine will deliver 20 cm H_2O of PIP but the tidal volumes will change with each breath.

Case Study

The Anesthesia Machine

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 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 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 ½ a tank, which should register as 1100 PSI, compared to a full tank at 2200 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 admnistration 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 an 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 figure X.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're off the hook!

Suggested Readings

1. Dorsch JA, Dorsh SE (1999) Understanding anesthesia Equipment, 4th edn. Lippincott, Williams & Wilkins, Philadelphia

2. ASA Machine Checklist. http://www.apsf.org/newsletters/html/2008/spring/05_new_guidelines.htm

3. Morgan GE, Maged SM, Michael JM (2006) Clinical anesthesiology, 4th edn. Mc Graw Hill, New York

4. Stoelting RK, Miller RD (2000) Basics of anesthesia, 4th edn. Churchill Livingstone, New York

5. Barash PG, Cullen BF, Stoelting RK (2006) Clinical anesthesia, 5th edn. Lippincott, Williams & Wilkins, Philadelphia

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 intraoperative monitors
- Understand the underlying principles behind the various monitors
- Know the limitations associated with various types of monitors

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.*

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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)

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) (see Table 11.1).

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.

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

Table 11.2 Monitoring equipment typically employed in general anesthesia cases

Electrocardiogram (***provides information about rate, rhythm, ischemia (ST segments)***)
Blood pressure (***manual, automatic, arterial catheter***)
Pulse oximeter (***usually on fingertip or ear lobe***)
Capnograph (***especially in patients with a supraglottic airway*** (e.g., ***LMA) or ETT)***
Oxygen analyzer (***part of anesthesia machine***)
Anesthetic agent concentration analyzer
Temperature (***usually esophageal, nasopharyngeal or axillary***)
Precordial or esophageal stethoscope (***listen to heart sounds, breath sounds***)
Gas flows/spirometry (***part of anesthesia machine***)
Airway pressure monitor (***part of anesthesia machine***)
Airway disconnect alarm (***part of anesthesia machine***)
Peripheral nerve stimulator (***where muscle relaxants have been used***)
Urometer (***measure urine output – where appropriate***)

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).

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. 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, such may occur when the patient's arms are tucked and the surgeon is leaning against the cuff. This second cuff is usually placed on the arm with the pulse oximeter; when it 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 a pressure transducer via a narrow fluid-filled tube (Fig. 11.2). This arrangement

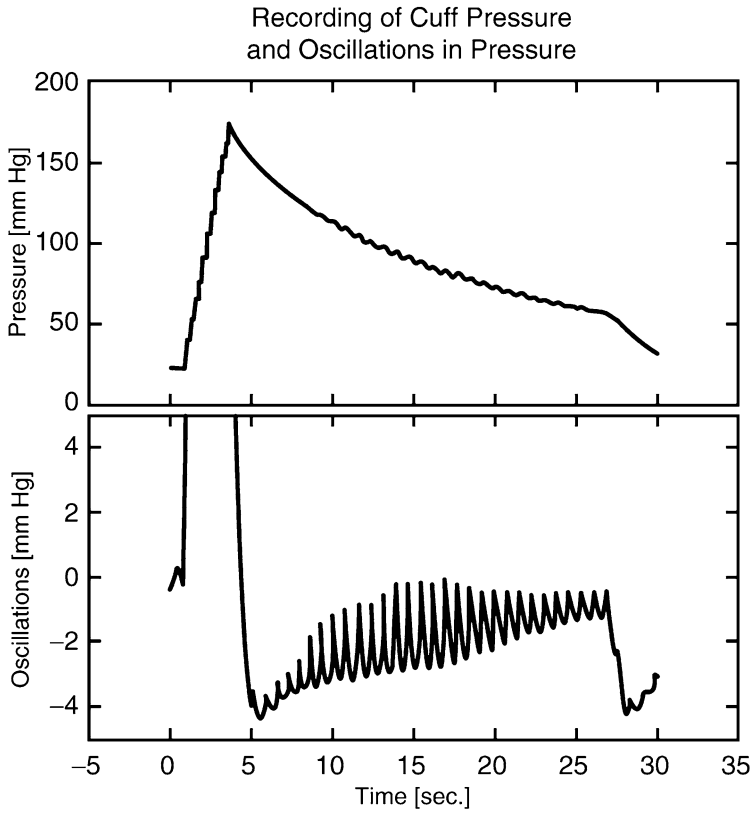

Figure 11.1 Sample blood pressure measurement using oscillometry. The peak oscillation corresponds to the mean arterial pressure (Reproduced with permission from Bronzino [15])

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.

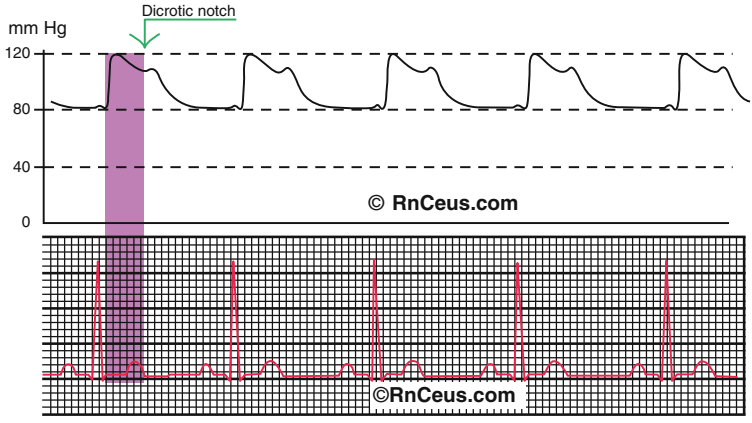

Figure 11.2 Arterial line waveform and its relationship to cardiac cycle (Reproduced with permission from www.rnceus.com/hemo/artline.htm)

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 five-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 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.

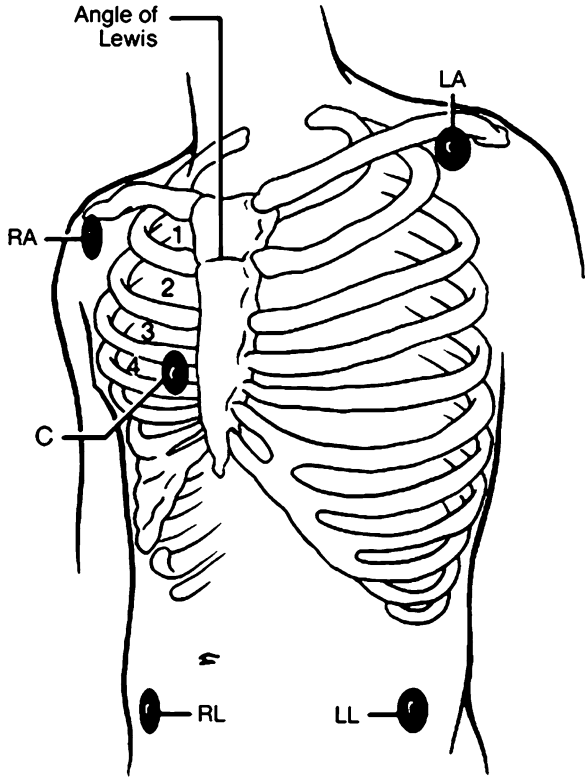

Figure 11.3 Location of the five electrodes used in a typical intraoperative electrocardiographic monitoring setup (Reproduced with permission from American Heart Association [16])

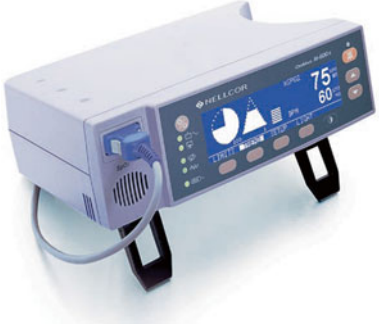

Figure 11.4 A typical pulse oximeter for operating room use, showing a S_pO_2 of 75 % and a heart rate of 60 beats/min. A waveform display allows one to ensure that a quality signal is present (Image courtesy Covidien)

A patient is generally said to be hypoxemic when the SpO_2 falls below 90 %, a point usually corresponding to an arterial 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. Pulse oximetry is mandated in **all** patients undergoing anesthesia. However, it is important to realize that pulse oximeters give no information about the level of arterial CO_2 and are useless in assessing adequacy of patent ventilation. Pulse oximeter units are now available for well under $1000. One design is even capable of providing hemoglobin information, a useful feature in surgical patients expected to undergo large blood losses.

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. Capnography is an ASA mandated monitor for both general anesthesia as well as for moderate sedation. 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.

Capnography is useful in 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)
- Detecting rebreathing of CO_2 (in which case the inspiratory CO_2 level is nonzero)
- Detecting capnograph tracings suggestive of COPD (where no plateau is present in the capnogram)
- Monitoring CO_2 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

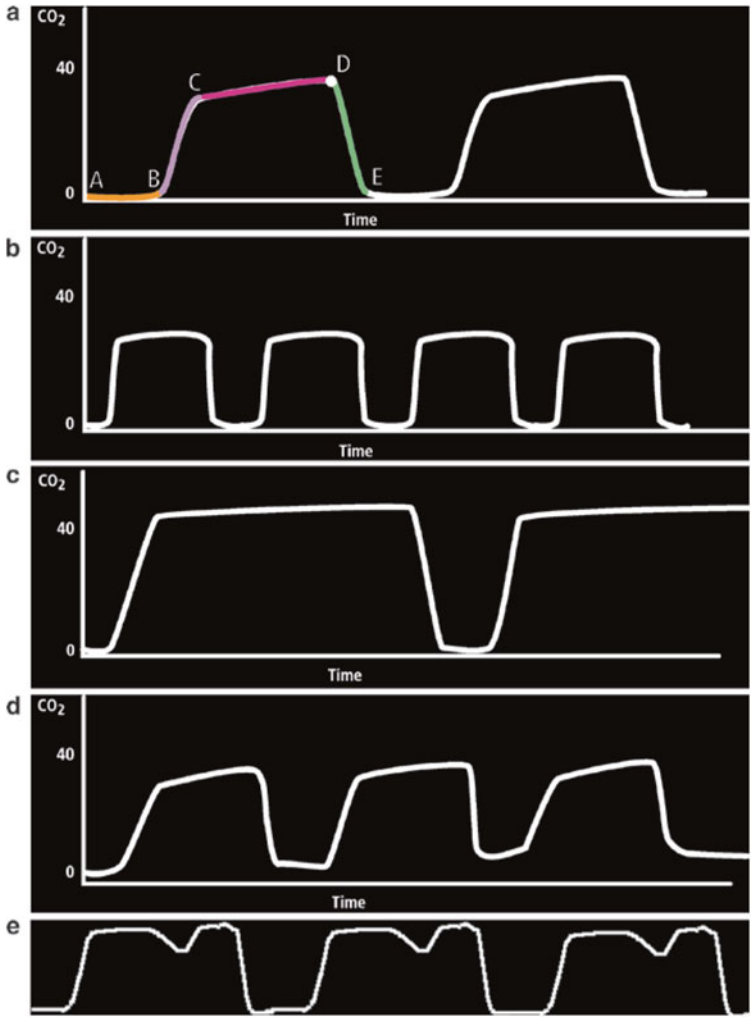

Figure 11.5 (*a*) Normal capnogram. The CO_2 level at point D (end-tidal CO_2) is normally around 40 mmHg. As shown above, the capnogram has four segments that correspond to phases of the respiratory cycle. The first phase (*AB*) is a flat part due to exhalation of dead space. It should always fall to 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 CO_2 from the alveoli. The fourth phase (*DE*) represents the beginning of inspiration (Used with permission from Oridion Medical, Inc. www.oridion.com)

(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 CO_2 is sometimes due to a catastrophic cardiorespiratory event such as circulatory arrest, a large pulmonary embolus, or severe hypotension (such as from extreme blood loss or compression of the inferior vena cava).

In addition to capnography, mechanical ventilation can also be monitored through vigilant monitoring of tidal volumes (V_T) and airway pressures. Tidal volumes may change as the patient's condition changes – for example, when pressure-controlled ventilation mode is used, the V_T 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 V_T 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 by increasing the resistance to gas flow.

(*b*) Hyperventilation. The end-tidal CO_2 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 CO_2 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 CO_2 concentration never falls to zero. A common cause of this is exhausted CO_2 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

Monitoring Muscle Relaxation

Muscle relaxation, or paralysis using neuromuscular blocking agents such as rocuronium 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

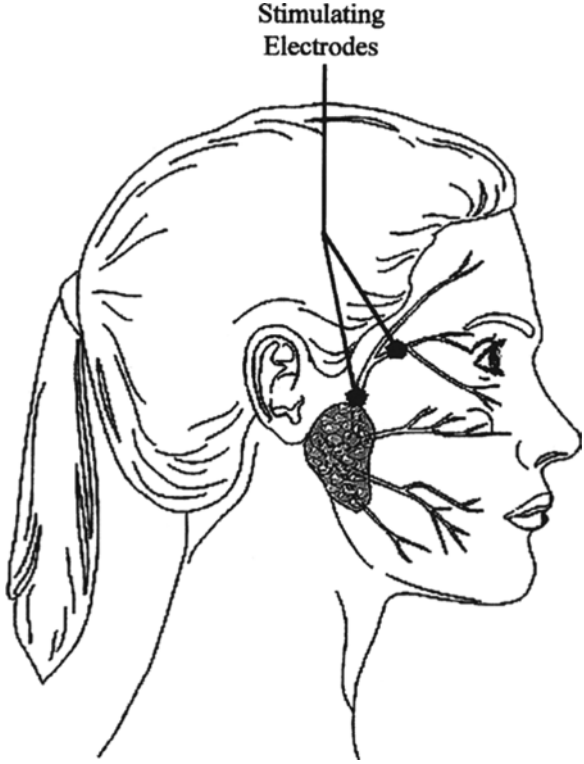

Figure 11.6 Proper electrode placement for stimulating the facial nerve (From O'Donnell and Nácul [17]. Used with permission)

observed "twitch" response to either stimulus sequence allows the anesthesiologist to determine the degree of muscle paralysis. In addition, neuromuscular blockade 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 or sugammadex, as well as a few minutes later to access 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 (electroencephalogram) 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 rate) 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 (CVP) 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.

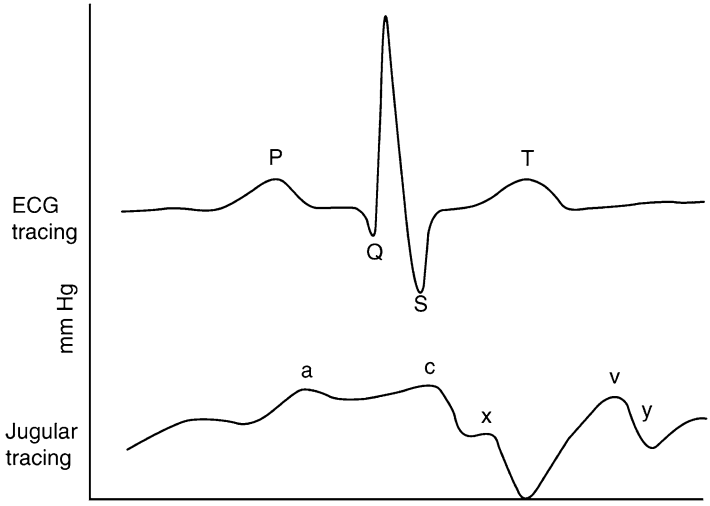

Figure 11.8 **The central venous pressure waveform. +a wave: this wave is due to the increased atrial pressure during right atrial contraction. It correlates with the P wave on an ECG. +c wave: This wave is caused by a slight elevation of the tricuspid valve into the right atrium during early ventricular 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 Norton et al. [18])**

It should be mentioned that absolute CVP measurements have not been as helpful as was originally hoped in identifying which hypotensive patients respond favorably to a fluid bolus. It is now recognized that in patients undergoing positive pressure ventilation, the more variable the systolic pressure across the respiratory cycle, the more likely the patient is to undergo a favorable increase in blood pressure following a fluid bolus.

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

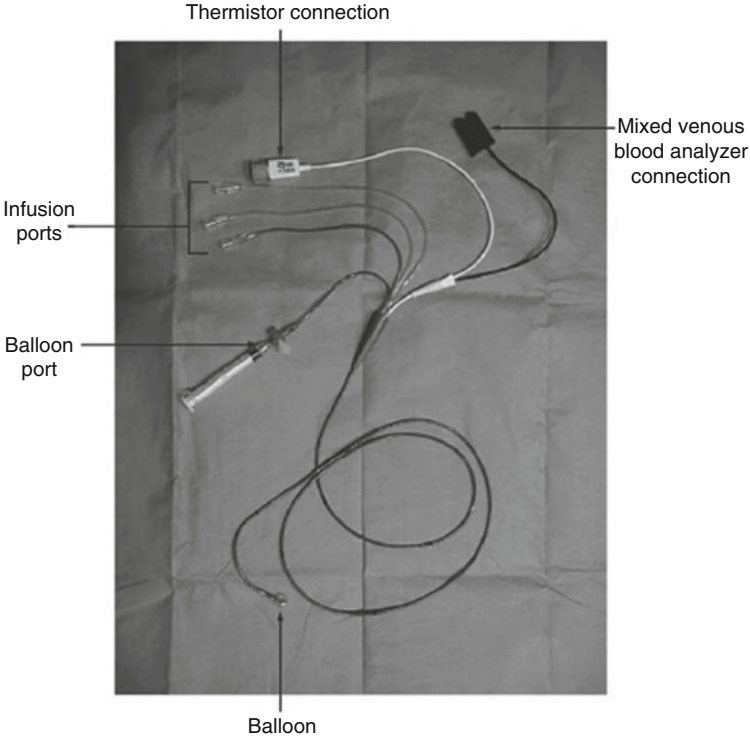

Figure 11.9 A typical pulmonary artery catheter

Table 11.3 Data typically obtained using a PA catheter

	Formula	Typical values
CO	SV × HR	4–8 l/min
CI	CO/BSA	2–5 l/min/m^2
SV	[CO/HR] × 1000	60–90 ml/beat
SI	CI/HR	40–60 ml/beat/m^2
LVSW	[MAP–PCWP] × SV × 0.0136	60–80 g m/beat
RVSW	[MPAP–CVP] × SV × 0.0136	10–15 g m/beat
SVR	[[MAP–CVP]/CO] × 80	800–1500 dyn s/cm^5
PVR	[[MPAP–PCWP]/CO] × 80	100–250 dyn s/cm^5

CO is the cardiac output, the blood ejected per minute from the heart. BSA is the body surface area, typically around 2 m^2 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.

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.

Other Special Patient Monitors

In addition to the patient monitors discussed above, special clinical situations often require specialized monitors. 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 (TEE) to assess heart function, and so on. In recent years TEE has become especially popular as a means to sort out cardiac problems, as it can

Table 11.4 The utility of hemodynamic parameters derived from the systemic blood pressure, and the pulmonary artery catheter in making clinical diagnoses

Diagnosis	Blood pressure	CVP	CO	CI	PCWP	Pulmonary artery diastolic pressure	SVR
Hypovolemia	⇓	⇓	⇓	⇓	⇓	⇓	⇑
Cardiogenic shock	⇓	⇑	⇓	⇓	⇑	⇑	⇑
Septic shock	⇓	⇓	⇓⇒⇑	⇓	⇓	⇓	⇓
Neurogenic shock	⇓	⇓	⇓	⇓	⇓	⇓	⇓
Tamponade	⇓	⇑	⇓	⇓	⇑	⇑	⇑

ß = 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.**

provide real-time information about ventricular filling, cardiac contractility, valvular function and more.

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).

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 20, 2010, is reprinted with permission of the American Society of Anesthesiologists, 520 N. 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 CO_2 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 (with no sedation) and monitored anesthesia care (with no sedation), the adequacy of ventilation shall be evaluated by continual observation of qualitative clinical signs. During moderate or deep sedation the adequacy of ventilation shall be evaluated by continual observation of qualitative clinical signs and monitoring for the presence of exhaled carbon dioxide unless precluded or invalidated by the nature of the patient, procedure, or equipment.

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.*
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."

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 "narcotized" 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 or absent 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.

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Part IV

Intraoperative Considerations

Chapter 12

Anesthetic Techniques: General, Sedation, MAC

Brian C. McLean, Anthony R. Plunkett, and Jesse M. Ehrenfeld

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.

J.M. Ehrenfeld et al. (eds.), *Anesthesia Student Survival Guide*,
DOI 10.1007/978-3-319-11083-7_12

Table 12.1 M.S.M.A.I.D.S. mnemonic

M → Machine
S → Suction
M → Monitors
A → Airway
I → IV
D → Drugs
S → Special

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 7 individual components of the mnemonic are outlined in the discussion below.

The first "**M**" stands for the anesthesia **M**achine. 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 **A**irway 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 2 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 **"I**V" 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 **D**rugs. 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 **S**pecial 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

Table 12.2 Stages of general anesthesia

Stage 1 – Amnesia	Patients should follow commands; respiration pattern typically regular
Stage 2 – Delirium	Period of uninhibited excitation; patients at risk for laryngospasm; pupils often divergent; respirations often irregular
Stage 3 – Surgical anesthesia	Target depth for anesthesia during surgery; respiration pattern typically regular
Stage 4 – Overdosage	Patients at risk for hypotension and cardiovascular collapse

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 4 stages of general anesthesia are outlined in the Table 12.2.

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,

Table 12.3 Action sequence of a general anesthetic

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

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).

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 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).

Table 12.4 ASA continuum of depth of sedation

	Minimal sedation (anxiolysis and analgesia)	Moderate sedation or "Conscious Sedation"	Deep sedation	General anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response following repeated or painful stimulation	Unarousable even with painful stimulation
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

Reproduced with permission from "Continuum of Depth of Sedation: Definition of General Anesthesia and Levels of Sedation/Analgesia." American Society of Anesthesiologists, 2004. http://www.asahq.org/publicationsAndServices/standards/20.pdf.

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

Table 12.5 Important components of a general anesthetic

Hypnosis	Rendering the patient unconscious
Analgesia	Removal of the sensation of pain
Amnesia	Prevention of memory formation
Paralysis	Prevention of movement
Reflex blunting	Prevention of exaggerated autonomic response

Table 12.6 Common drugs used during a basic general anesthetic

Time of administration	Purpose	Example
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, pancuronium

(continued)

Table 12.6 (continued)

Time of administration	Purpose	Example
Maintenance of anesthesia	Volatile anesthetics	Volatile anesthetics Sevoflurane Desflurane Isoflurane Nitrous oxide
	Intravenous anesthetics	IV anesthetics Propofol Ketamine
	Antihypotensives	Sympathomimetics Ephedrine Phenylephrine
	Analgesics	Opioids Morphine, hydromorphone Fentanyl, remifentanil Sufentanil, alfentanil Other Ketamine
Emergence	"Reversal" of neuromuscular blockade	Acetylcholineseterase inhibitors Neostigmine Edrophonium Anticholinergics Atropine Glycopyrolate
Recovery in the PACU	Antiemetics	5HT3 blocker Ondansetron (Zofran) Granesitron (Kytril) Dopamine agonists Metoclopramide (Reglan) Corticosteroids Decadron Histamine blockers Promethazine (Phenergan)

be used to induce general anesthesia if given in large bolus doses. Sedative-hypnotic agents act by binding to and activating $GABA_A$ 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.

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

1. Urman RD, Ehrenfeld JM (2009) Anesthesia techniques. In: Pocket anesthesia, 1st edn. Lippincott, Williams, and Wilkins

2. Barash PG, Cullen BF, Stoelting RK (2001) Monitored anesthesia care. In: Clinical anesthesia, 4th edn. Lippincott, Williams, and Wilkins

3. Longnecker DE, Brown DL, Newman MF, Zapol WM (2008) Total intravenous anesthesia. In: Anesthesiology, 1st edn. McGraw Hill

Chapter 13

Anesthetic Techniques: Regional

Brian C. McLean, Anthony R. Plunkett, and Jesse M. Ehrenfeld

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

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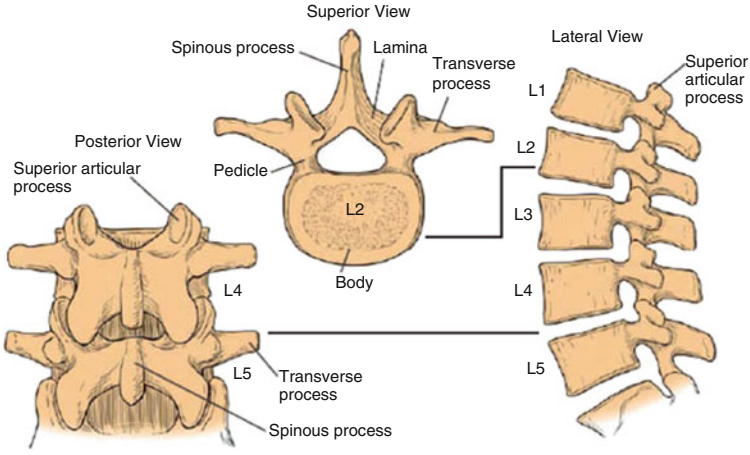

Figure 13.1 Vertebral anatomy (Reproduced with permission from Mathias [5])

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 anesthetic. 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

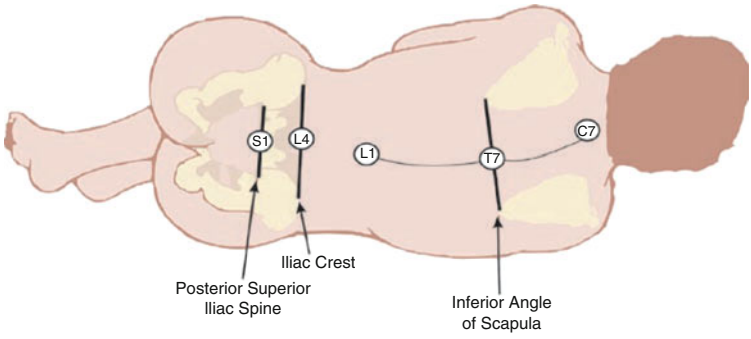

Figure 13.2 Surface anatomy for neuraxial anesthesia

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

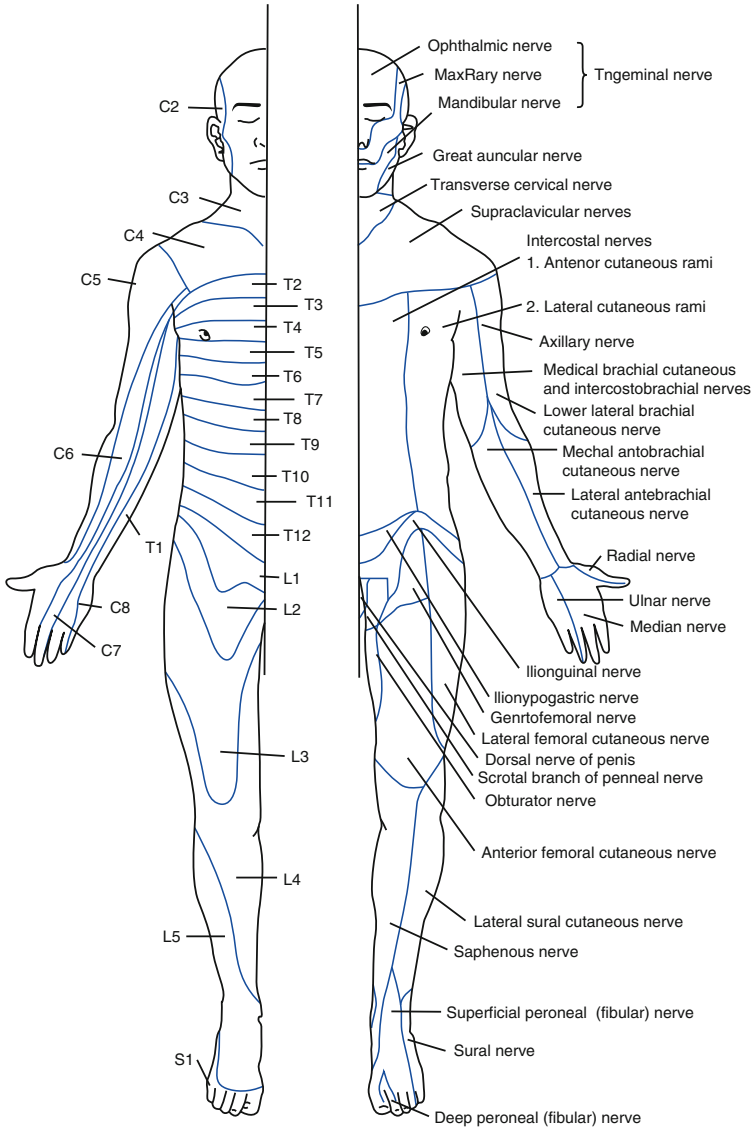

Figure 13.3 Dermatomes (Reproduced with permission from Stewart [6])

Table 13.1 Risks of neuraxial anesthesia

Bleeding
Infection
Nerve injury
Post-dural puncture headache
Failure of block to provide adequate anesthesia

Table 13.2 Contraindications to neuraxial anesthesia

Absolute contraindications	Relative contraindications
Patient refusal	Bacteremia
Infection in the area of needle puncture	Pre-existing neurologic disease (e.g. multiple sclerosis)
Elevated intracranial pressure	Cardiac disease
Uncontrolled bleeding	Abnormal coagulation studies

space (epidural) bathes the nerve root and inhibits synaptic transmission of action potentials. The effect of local anesthetics on nerve fibers varies 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 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

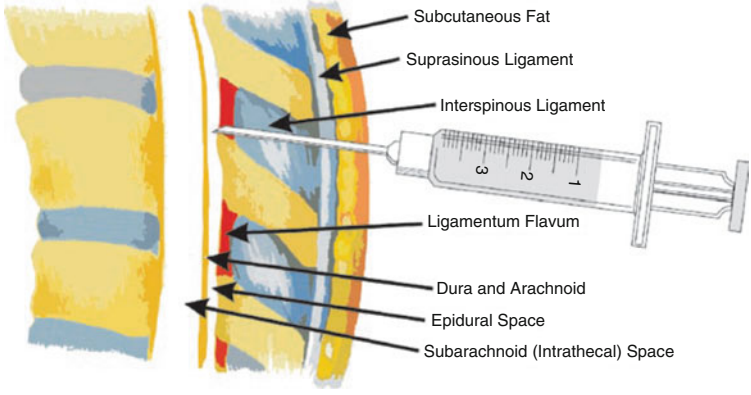

Figure 13.4 Trajectory of epidural anesthesia (Image courtesy J. Ehrenfeld, M. D)

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 commonly cited as approximately 1/150,000 and 1/200,000 after a spinal. However, in an analysis published in 2013 by Ehrenfeld et al. in which 43,200 epidural catheterizations were evaluated, 102 patients (1/430) underwent further imaging studies to exclude or confirm the presence of an epidural hematoma. In that study the overall incidence of epidural hematoma was 1.38 per 10,000 epidural blocks.

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

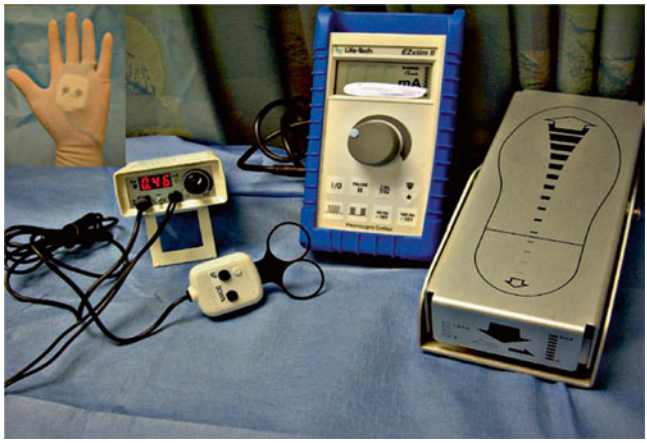

Figure 13.5 Nerve stimulation setup for peripheral nerve block (Reproduced with permission from Tsui [7])

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.

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

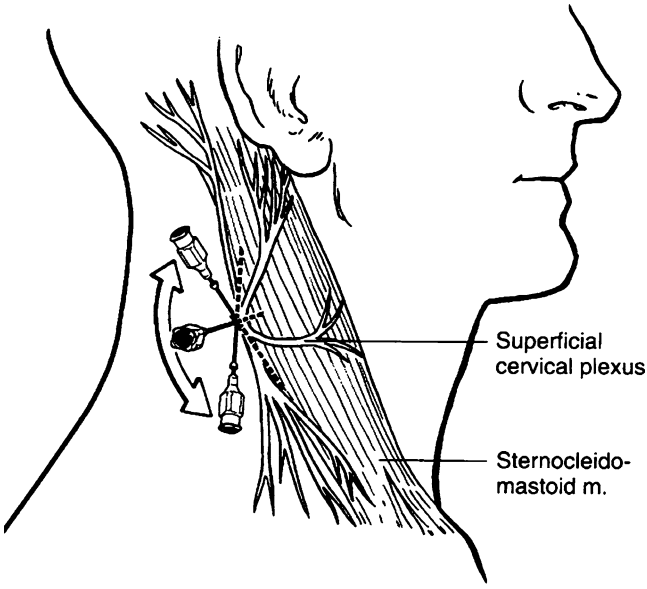

Figure 13.6 Cervical plexus block (Reproduced with permission from Twersky and Philip [8])

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 "**R**andy **T**ravis **D**rinks **C**old **B**eer" with the first letters of each word standing for **r**oots, **t**runks, **d**ivisions, **c**ords, and terminal **b**ranches.

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

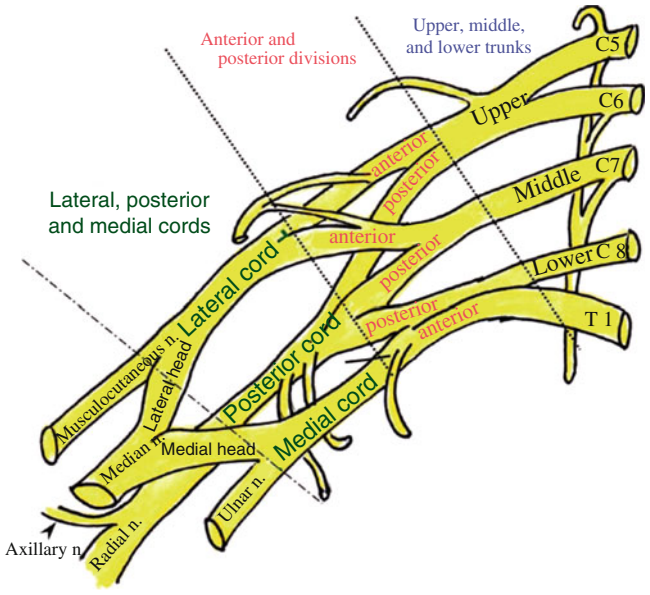

Figure 13.7 Brachial plexus anatomy (Reproduced with permission from Tsui [7])

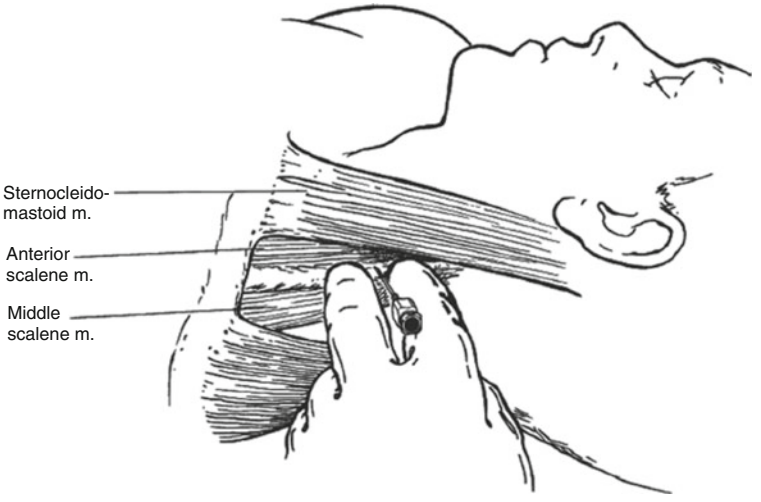

Figure 13.8 Interscalene nerve block (Reproduced with permission from Twersky and Philip [8])

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).

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 pneumothroax, 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

Table 13.3 Summary of upper extremity nerve blocks

Type of nerve block	Indications	Anatomical landmarks	Average needle depth (cm)	Potential complications
Interscalene	Shoulder; upper arm	Between middle and anterior scalene muscles at the level of C6 (cricoid cartilage)	1–2	Hemi-diaphragmatic paralysis; Horner's Syndrome; epidural spread; intravascular injection; ulnar nerve sparing
Supraclavicular	Upper and lower arm; hand	Between the middle and anterior scalene muscles, 1 cm above the clavicle	2–3	Pneumothorax (1 % incidence); intravascular injection
Infraclavicular	Lower arm; hand	2–3 cm caudad from the midpoint of the clavicle	6–8	Pneumothorax (much lower than with supraclavicular); intravascular injection
Axillary	Lower arm; hand		4–6	Intravascular injection; prolonged set-up time; miss the musculocutaneous nerve

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 line

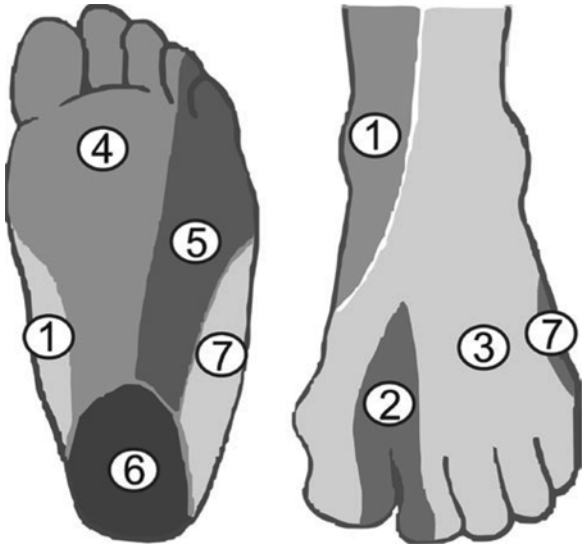

Figure 13.9 Innervation of the foot. *Plantar surface*: 1. Saphenous nerve; 2. Deep peroneal nerve; 3. Superficial peroneal nerve; 4. Medial planar nerve. *Dorsal surface*: 5. Lateral plantar nerve; 6. Tibial nerve (calcaneal branch); 7. Sural nerve (Image courtesy J. Ehrenfeld)

and marked. The block needle is inserted 1–2 cm lateral to the femoral artery pulse. The desired motor response is a quadriceps twitch. 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 (Fig. 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 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 (Fig. 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

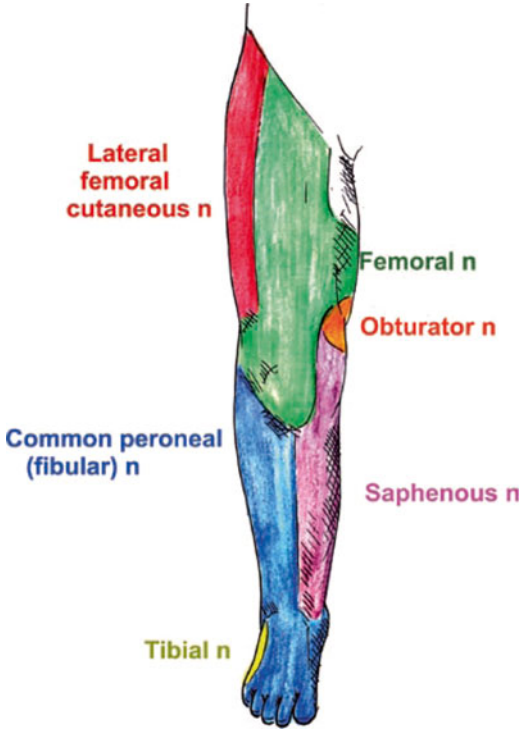

Figure 13.10 Cutaneous distribution of peripheral nerves (Reproduced with permission from Tsui [7])

sensation to the dorsal medial aspect of the foot and the webspace 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.

Table 13.4 Summary of lower extremity nerve blocks

Type of nerve block	Indications	Anatomical landmarks	Average needle depth (cm)	Potential complications
Femoral	Anterior thigh; knee; medial aspect of lower leg (saphenous nerve)	1 cm lateral to palpation of femoral artery along the inguinal ligament	3–5	Intravascular injection; miss obturator and lateral femoral cutaneous nerves
Lumbar plexus (Psoas)	Hip; anterior thigh; knee; medial aspect of lower leg (saphenous nerve)	5 cm lateral from the spinous process (midline) at the level of iliac crest	6–8	Epidural spread; intravascular injection; retroperitoneal hematoma; needle trauma to kidney
Sciatic (posterior approach)	Posterior thigh; below the knee surgery (except saphenous nerve distribution)	Posterior superior iliac spine (PSIS); sacral hiatus; greater trochanter. A line bisecting the midpoint of the PSIS and greater trochanter to intersect with a line drawn from greater trochanter to sacral hiatus	5–7	Intravascular injection; nerve injury
Sciatic (lateral approach)	Below the knee surgery (except saphenous nerve distribution)	Between vastus lateralis and biceps femoris muscles, contact femur, then change needle angle approx. 45°	6–8	Intravascular injection, nerve injury, will miss posterior thigh (tourniquet pain)

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.

Ultrasonography

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 sterilely 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

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Chapter 14

Electrolytes and Acid-Base Analysis

Adam Kingeter and Matthew D. McEvoy

Educational Objectives

1. Understand physiologic regulation of serum electrolytes and describe common abnormalities
2. Understand the importance of acid base regulation in clinical practice
3. Describe the determinants of pH according to both the Stewart and traditional approach
4. Understand physiologic regulation of pH
5. Understand the causes of both metabolic and respiratory acid base disturbances
6. Understand risks and benefits of treatment options available for metabolic acidosis

Introduction to Serum Electrolyte Composition

While blood components contain electrolytes in physiologic concentrations, this is not true for all crystalloids and colloids. As such, this chapter will give the reader more insight into the use of fluids in the perioperative period and how this relates to electrolyte and acid-base physiology. In physiology, electrolytes are defined as compounds that contain an electrical charge when dissolved in a solution such as water and as such are able to conduct electricity. These charged compounds are referred to as ions, and their charge may be either positive (cations) or negative (anions). Tight regulation of electrolytes in both the intracellular and extracellular spaces is essential for normal cellular function in every organ system. The body will seek to maintain serum electrochemical

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neutrality; that is, a balance of all positive and negative charges. In addition to this, the body seeks to maintain acid-base regulation within a very narrow range. These two forces, electrochemical neutrality and acid-base balance, are tightly coupled in order to maintain normal homeostasis. Disruption of this balance leads to cellular dysfunction and can cause fatal pathologic processes such as renal dysfunction, dysrythmias, seizures, and coma.

Physiologic Regulation of Electrolytes

The major serum cations are sodium and potassium, with calcium, and magnesium also being present in lower concentrations. The major serum anions are chloride and bicarbonate, with phosphate and lactate also being present in lower concentrations. Intracellular concentration of these ions is determined by active transmembrane pumps, and to a lesser extent by passive ion-specific channels. Extracellular – including serum – concentration of electrolytes is regulated primarily by the renal system with significant input from the hypothalamic-pituitary-adrenal (HPA) axis.

Sodium

Sodium is the most abundant serum cation, and is a major determinant of serum osmolality. Serum osmolality is tightly regulated between 275 and 290 mOsm/kg.

Serum osmlality is estimated by the following equation:

$$\text{Serum Osmolality} = \left(\text{Serum } Na^{+} \times 2\right) + \left(\text{Serum glucose} / 18\right) + \left(\text{Serum BUN} / 2.8\right)$$

The HPA axis is one of the major organ systems regulating sodium balance and serum osmolality. Serum osmolality is sensed by osmoreceptors in the organum vasculosum of the lamina terminalis (OVLT), located in the portion of the hypothalamus outside of the blood-brain barrier. Changes in osmolality of as little as 1 % are sensed by the osmoreceptors, which send projections to the supra-optic (SON) and para-ventricular (PVN) hypothalamic nuclei. The SON and PVN synthesize anti-diuretic hormone (ADH) and send projections to the posterior pituitary. These projections in the posterior pituitary release ADH into systemic circulation. ADH acts on the medullary collecting duct of the nephron, and causes increased water absorption. This increase in water absorption leads to a subsequent decrease in serum sodium concentration, thus driving down serum osmolality.

The other major system regulating sodium balance is the renin-angiotensin system (RAS). Decreased sodium delivery to the macula densa triggers the synthesis and release of renin from the juxtaglomerular apparatus. Renin converts angiotensinogen to angiotensin I, which is then rapidly converted into angiotensin II by angiotensin converting enzyme (ACE). Angiotensin II acts directly on the vasculature to increase systemic vascular resistance (SVR), increase ADH release, and stimulate the release of aldosterone from the zona golmerulosa of the adrenal cortex. Aldosterone acts on the distal tubules and collecting ducts of the nephron to increase the reabsorption of water and sodium, and increase the secretion of potassium. Abnormalities in either of these systems – HPA or RAS – can lead to disorders of sodium homeostasis.

Hyponatremia

Given that sodium and serum osmolality are tightly linked, and that osmolality is influenced by relative volume status, disorders of sodium homeostasis are categorized by intravascular volume status. Hyponatremia can occur in the context of intravascular hyper-, hypo-, or euvolemia, and patients' intravascular volume status must be evaluated in order to diagnose the underlying cause of the hyponatremia.

Hypovolemic causes are due to the loss of both sodium and total body water as occurs with profuse sweating, prolonged vomiting or diuretic use. A rare cause of hypovolemic hyponatremia is cerebral salt wasting syndrome (CSW). CSW is typically seen in the context of head trauma, or intracranial lesion, and is characterized by decreased renal sodium retention, polyuria, and dysautonomia. Treatment of hypovolemic hyponatremia is mainly supportive and aimed at fluid and electrolyte replacement. Fludrocortisone can be used to increase renal sodium reabsorption. Most cases typically resolve spontaneously within 2–4 weeks, although a few persist longer.

Hypervolemic hyponatremia is due to increased total body water, and typically occurs in the context of decreased effective serum osmolality, and is often associated with peripheral edema. Common etiologies include heart failure, nephrotic syndrome, and liver disease with associated ascites. In these states, plasma osmolality is decreased and oncotic pressure is unable to retain volume in the intravascular space. This leads to decreased effective circulating volume and the body responds by increasing water reabsorption, further decreasing serum osmolality. Treatment for these causes is aimed at the underlying etiology, and usually includes fluid restriction. A new class of medication, the

vaptans, act as vasopressin antagonists and may find clinical use in some of these disease states.

Euvolemic hyponatremia is typically caused by either presence of osmotically active molecules (pseduohyponatremia) or excess secretion of ADH. Pseudohyponatremia occurs when hyperosmolarity from non-sodium osmotically active agents (glucose, triglycerides, immunoglobulins) draws water from the intracellular compartment into the intravascular space with subsequent dilution of sodium content. Syndrome of Inappropriate Anti-Diurectic Hormone (SIADH) is the paradigm for euvolemic hyponatremia, and is seen in a wide array of clinical conditions ranging from intracranial abnormalities to various tumors. SIADH is characterized by inappropriately elevated levels of ADH in the absence of a physiologically appropriate stimulus (i.e. hypotension or hypotonicity). The hallmark of SIADH is inappropriately concentrated urine relative to plasma osmolality. As discussed, one of the physiologic effects of ADH is water retention and these patients develop a pure water excess. Water is not confined to one fluid compartment, it distributes equally throughout all of them, thus these patients appear clinically euvolemic. Classically, water restriction has been the mainstay of SIADH treatment. In cases where water restriction is poorly tolerated, demeclocycline has been used. Demeclocycline is a tetracycline analogue that inhibits vasopressin mediated water reabsorption in the collecting duct. The new class of vasopressin inhibitors, the Vaptans, promises to radically alter the treatment of SIADH.

The deleterious physiologic effects of hyponatremia develop when sodium levels drop enough to significantly lower plasma tonicity, leading to a shift of water into the intracellular space. This leads to cellular dysfunction, particularly in the CNS and accounts for the clinical manifestations of confusion, coma and seizures. In cases of symptomatic hyponatremia, sodium repletion maybe given either orally, or in the form of concentrated 3 % saline. Correction must be gradual, so as to avoid neurologic devastation through central pontine myelinosis, which results from the rapid shift of water out of the intracellular space in the CNS. Plasma sodium concentration should not be raised by more than 0.5 mmol/L/h, and no more than 10 mmol/L in 24 h.

Hypernatremia

Hypernatremia leads to an increase in serum osmolality, with a subsequent shift of water out of the intracellular compartment and cellular dysfunction. As with hyponatremia, the CNS is particularly sensitive to these shifts and patients with symptomatic hypernatremia typically present with weakness, confusion and lethargy. In contrast to hyponatremia, hypernatremia is almost always a result of water loss in excess of sodium loss rather than excess sodium intake relative to water intake. As such, these patients are typically hypovolemic.

Most commonly, hypernatremia occurs via either evaporative losses or by excretion of large volumes of very dilute urine. Evaporative, or insensible losses result from evaporation of pure water from the respiratory tract or other exposed mucosal surfaces. These losses are highly variable and are increased by patients who are febrile, tachypnic, or in warm environments. Surgical procedures that expose large body cavities to room air greatly increase the surface area for evaporative loss, and can cause losses triple to those under normal environmental conditions. The loss of large volumes of dilute urine is characteristic of diabetes insipidus (DI). DI results from either a lack of vasopressin release from the posterior pituitary, or lack of renal response to circulating vasopressin. Patients with DI who are able to regulate their water intake typically do not develop hypernatremia, as their input matches their output. However, patients who are unable to do so can rapidly develop hypernatremia and dehydration.

Treatment of hypernatremia depends on the underlying cause. In patients with central DI, a synthetic vasopressin analogue, desmopressin or DDAVP, is the treatment of choice. DDAVP lacks the hypertensive effects of vasopressin and can be given nasally, intravenously, or orally. In patients with nephrogenic DI, thiazide diuretics have a paradoxic anti-diuretic effect and are the treatment of choice if the underlying cause cannot be reversed. In patients who are hypernatremic secondary to free water loss, the correction of the free water deficit results in correction of the hypernatremia. Free water deficit can be calculated from the following equation:

$$\begin{aligned}\text{Deficit} = \text{TBW}\left(1 - \left[140 / \text{Plasma Na} + \right]\right) &\text{where TBW} \\ &\text{is total body water}\left(\text{L}\right)\text{estimated at} 0.5\times \\ &\text{lean body weight for women and} \\ &0.6\times\text{lean body weight for men.}\end{aligned}$$

Rapid correction of hypernatremia can lead to cerebral edema, therefore no more than half of the deficit should be replaced in the first 24 h of treatment. The remainder can be corrected in the following 24–48 h. Chronic hypernatremia is remarkably well tolerated secondary to compensatory changes which occur at the cellular level. These changes involve intracellular production of osmotically active substances to offset the extracellular hypertonicity.

Potassium

Potassium is the second most common cation in the body following sodium. Ninety-eight percent of the total body potassium content is contained intracellularly. The significant gradient in potassium concentration across the cell membrane leads to a negative electric charge in the intracellular space. This charge, known as a resting membrane potential, is necessary for the proper functioning of excitable tissues such as muscle and nerve. This gradient is maintained by active ion transport pumps located in the cellular membrane, which can account for up to 2/3 of a cell's total energy expenditure. Small changes in extracellular potassium concentration can lead to dysfunction of excitable tissue with potentially catastrophic consequences. Maintenance of potassium homeostasis is accomplished at both the cellular and organ system level; shifts of potassium into and out of cells buffers against acute changes in extracellular potassium, while the renal system maintains a balance between potassium intake and excretion. Multiple disease states affect these systems and can lead to disorders of potassium regulation.

Hyperkalemia

Hyperkalemia maybe classified by duration of the insult as either acute (<48 h) or chronic. Acute hyperkalemia is almost always caused by a shift of potassium out of cells, but rarely can be caused by an excessive intake of potassium. A transcellular shift of only 2 % of intracellular K+ would cause serum K+ levels to double. Dramatic transcellular shifts of potassium are often associated with cell death, such as tumor lysis syndrome or rhabdomyolysis. A metabolic acidosis also results in a transcellular shift of potassium, as H+ displaces K+ from the intracellular compartment. Certain medications are also associated with transcellular shifts of potassium, notably digitalis and succinylcholine. Under normal circumstances, succhinycholine induced fasculations cause a small amount of K+ to leak from muscle. This small leak causes an increase in serum K+ by about 0.5 mmol/L. However, in patients

with prolonged immobilization or paralysis, severe burns, or muscle inflammation this K+ leak can be significantly larger, with potentially fatal consequences. These diseases are associated with proliferation of immature forms of the AchR outside of the motor end plate. These extra receptors are activated by succinylcholine, resulting in more potassium being release from the cells than under normal circumstances. Excessive potassium intake is almost always iatrogenic, as can occur with overly aggressive oral or IV potassium replacement therapy. Banked blood contains a small amount of potassium, however massive transfusions can lead to accumulation of large amounts of potassium and cause symptomatic hyperkalemia. Causes of chronic hyperkalemia include renal failure, Addison's disease, and both aldosterone deficiency and tubular unresponsiveness to aldosterone. Hyperkalemia causes depolarization of cardiac myocytes resulting in dysrhythmias and impaired conduction disorders. Classic EKG changes associated with progressive hyperkalemia include (in order of their appearance): peaked T waves, prolongation of the PR interval, widening of the QRS complex, loss of P wave, "sine wave" appearing ventricular fibrillation, and finally asystole. Hyperkalemia is also associated with paresthesias and skeletal muscle weakness progressing to a flaccid paralysis. Therapy for hyperkalemia is aimed primarily at stabilizing the cell membranes of excitable tissues. Once this has been achieved, therapy is guided towards redistribution of K^+ into cells and enhanced elimination of K^+ from the body. Calcium directly antagonizes the myocardial effects of hyperkalemia and is the treatment of choice for membrane stabilization. Redistribution of potassium into cells can be accomplished by both insulin and albuterol. A dextrose infusion should be started to counteract the hypoglycemic effects of insulin therapy in these patients. Increased elimination of potassium from the body can occur via either the renal or GI route. Renal elimination of potassium is increased by increased flow through the distal nephron, typically accomplished by administration of saline, and is enhanced by loop diuretics. GI losses of potassium are increased by the administration of sodium polystyrene sulfonate (SPS). SPS is a cation exchange resin that exchanges sodium for secreted potassium in the colon. SPS causes constipation and should be given with a cathartic. Cases of colonic necrosis have been reported following administration of SPS, with an estimated incidence of 1.8 % in post-operative patients according to a retrospective analysis. Hemodialysis can also be used to enhance potassium elimination.

Hypokalemia

As with hyperkalemia, hypokalemia may be classified according to duration as either acute (<48 h) or chronic. Acute changes are almost always caused by transcellular shifts of potassium into cells. This inward shift is often seen with treatment of DKA secondary to insulin therapy, and can also be seen in re-feeding syndrome due to increased endogenous insulin production. Other medications, such as β2-agonists, also cause a shift of potassium into cells and can cause acute hypokalemia. Chronic hypokalemia is usually the result of either decreased intake or increased elimination. Elimination can occur from either the GI or renal route. Renal losses of potassium are increased by diuretics, various antibiotics, and mineralcorticoids. Hypomagnesemia is also associated with renal K+ wasting, and often must be repleted along with potassium. Various inborn tubular transport abnormalities, such as Barter, and Gitelman's syndromes are also associated with increased renal losses of K+ and hypokalemia. Hypokalemia leads to cell membrane hyperpolarization, and can cause cardiac arrhythmias and conduction defects. Non specific EKG changes include ST segment depression, T wave flattening and prominent U waves. Neuromuscular signs include weakness, muscle fatigue and myalgias. Treatment of hypokalemia involves replacement of the body deficit and correction of underlying cause when able. Potassium may be given either orally or IV. If given IV, potassium should be administered over 1–2 h to avoid causing a hyperkalemia. Serum potassium levels peak immediately following an infusion, and over the next 2–3 h decrease to the new steady state. Repeat measurements of serum potassium should be taken after the new steady state has been achieved to further guide therapy.

Chloride

Chloride is the predominant anion in the extracellular fluid. As mentioned above, electroneutrality is maintained at all times; that is, the concentration of cations and anions is equal and charges offset. As such, changes in chloride concentration significantly effect the concentration of other anions such as bicarbonate, lactate and other organic acids. Many of these other anions take part in the buffering of serum H+ concentration and help determine plasma pH. Due to the interplay of chloride and other serum anions, chloride physiology and acid base balance are closely related and interdependent.

An example of this sort of interdependence can be seen in patients who have been resuscitated with large volumes of normal saline. 0.9 % normal

saline contains 154 meq of sodium and 154 meq of chloride compared to a normal serum chloride concentration of approximately 100 meq. This increase in serum chloride causes a reciprocal decrease in other serum anions, notably bicarbonate. As serum bicarbonate levels decrease, there is a concomitant increase in unbuffered serum H+ concentration and serum pH decreases resulting in an acidosis. The role of chloride physiology and acid-base balance will be further discussed in the next section.

Introduction to Acid Base Physiology

The tight control of extracellular hydrogen ion concentration is of paramount importance to the function of trans-membrane ion transport pumps and intracellular biochemical reactions. As such the body has developed numerous weak acid buffer systems to maintain a homeostatic pH between 7.35 and 7.45. Deviations in pH beyond this zone are termed acidemia (pH <7.35) and alkalemia (pH >7.45). The presence of acidemia or alkalemia indicates gross metabolic or respiratory abnormalities, which if uncorrected, may lead to end organ dysfunction and death.

Acid-Base balance is a complex physiochemical process. Two different approaches can be used to explain acid-base interaction: anion gap/base excess and strong ion difference (Stewart approach). The Stewart approach introduced by Peter Stewart in 1981 emphasizes two important elements of physical chemistry as the driving forces for acid base balance: electroneutrality and conservation of mass. The primary tenant of Stewart's approach is that serum bicarbonate does not alter blood pH. According to Stewart's theory, pH is the result of the interplay of three variables: Strong Ion Difference (SID), $PaCO_2$, and plasma concentration of weak non-volatile acids such as albumin and phosphate (Atot). The SID equals the difference between completely dissociated cations and anions in plasma. The equation for SID consists of the most abundant ions in plasma and is calculated as such:

$$\begin{aligned} SID = &\left(\left[Na^+\right]+\left[K^+\right]+\left[Ca^{2+}\right]+\left[Mg^{2+}\right]\right) \\ &-\left(\left[Cl^-\right]+\left[Lactate^-\right]\right) = 40-44\ \text{mEq} \end{aligned}$$

Processes that increase the SID cause an alkalemia, whereas processes that reduce it cause acidemia. For example, the loss of gastric fluid, which has a high concentration of Cl^-, increases the SID and thus leads to alkalemia; by comparison, infusion of sodium chloride, a solution with equal parts Na^+ and Cl^- thus an SID of zero, reduces the SID and causes an acidemia.

The anion gap approach is based on the Bronsted–Lowry definition of an acid in which the primary extracellular buffer system is the equilibrium of carbonic acid and bicarbonate represented by the equation below.

$$H_20 + CO_2 <--> H_2CO_3 <--> H^+ + HCO3^-$$

The Henderson–Hasselbach equation describes the relationship between this equilibrium equation and pH as such:

$$pH = pK + \log_{10}\left(HCO_3^- / \alpha_{CO2} \times pCO_2\right)$$

In this equation α_{CO2} represents the solubility coefficient of CO_2 (0.03) and pK represents the equilibrium constant (6.1). A derivative of this equation, known as the Henderson equation, simplifies matters as such:

$$H^+ = 24 \times pCO_2 / HCO_{3^-}$$

From this equation, it is evident that changes in pH may be either the result of a change in pCO_2 (referred to as respiratory) or HCO_3^- (referred to as metabolic). There is much emerging evidence of the likely clinical utility of the Stewart approach and while understanding both approaches is ultimately important, due to its simplicity and widespread use, the anion gap approach will be discussed in this chapter.

Maintenance of homeostatic concentrations of CO_2 and HCO_3^-, including compensatory responses to insult, is a result of the interplay of the pulmonary and renal systems. Consequently, metabolic disturbances are accompanied by a respiratory compensation and visa versa. Compensation results in normalization of pH and can give some indication as to the duration of the insult, with chronic processes being better compensated. Attention must be paid to compensation, as multiple acid-base abnormalities can co-exist in the same patient, which are diagnosed by comparison of anticipated and actual compensatory changes. More than one metabolic abnormality may be present in a patient at one time, however, only one respiratory disturbance is possible at any given moment.

The introduction of blood-gas analysis in the 1950s allowed for the diagnosis and categorization of acid-base derangements and their subsequent treatment. The relatively low cost and ease of collection have led to the use of blood gas analysis in everyday anesthetic practice. Typical blood gas values include pH, pO_2, pCO_2, HCO_3^-, and base excess. Base excess is defined as the amount

Table 14.1 Acid-base disturbances and the expected compensation

Primary disorder	Primary change	Compensatory change	Expected compensation
Metabolic acidosis	↓ HCO_3	↓ $PaCO_2$	$\Delta PaCO_2 = 1.2 * \Delta HCO_3$
Metabolic alkalosis	↑ HCO_3	↑ $PaCO_2$	$\Delta PaCO_2 = 0.9 * \Delta HCO_3$
Respiratory acidosis	↑ $PaCO_2$	↑ HCO_3	*Acute:* $\Delta HCO_3 = 0.1 * \Delta PaCO_2$ *Chronic:* $\Delta HCO_3 = 0.35 * \Delta PaCO_2$
Respiratory alkalosis	↓ $PaCO_2$	↓ HCO_3	*Acute:* $\Delta HCO_3 = 0.2 * \Delta PaCO_2$ *Chronic:* $\Delta HCO_3 = 0.5 * \Delta PaCO_2$

of strong acid (if BE >0) or strong base (if BE <0) which is required to return 1 L of whole blood at a pCO_2 of 40 mmHg to a pH of 7.4. In theory, the base excess represents the metabolic component of an acid base disorder with positive values indicative of a metabolic alkalosis and negative values indicative of a metabolic acidosis. The deviation of BE from 0 can be used as a surrogate measure of severity of the metabolic derangement, with more severe disturbances resulting in values further from zero. Classical blood gas measurements allow for diagnosis of acidemia and alkalemia with levels of compensation as seen in Table 14.1. Further diagnosis and classification necessitates serum electrolytes, hemoglobin and serum lactate concentrations, which are available on most modern blood gas machines.

Metabolic Abnormalities

Metabolic Acidosis

Any process that causes a reduction in the extracellular bicarbonate concentration, via increased loss of bicarbonate or via accumulation of excess acid, is termed a metabolic acidosis. Multiple abnormalities can lead to a metabolic acidosis, and these are grouped according to whether or not they lead to an associated increase in the serum anion gap. The serum anion gap is calculated by the following equation:

$$AG = \left(\text{Serum } Na^+ + \text{Serum } K^+\right) - \left(\text{Serum } HCO_{3^-} + \text{Serum } Cl^-\right)$$

A normal value for anion gap is between 8 and 12 mmol/L and represents the concentration of anions normally unmeasured by a basic metabolic panel

Table 14.2 Causes of non-anion gap metabolic acidosis: HARD-UP

Hyperalimentation
Acetazolamide administration
Renal tubular acidosis
Diarrhea
Ureteroenteric fistula
Pancreaticoduodenal fistula

Table 14.3 Causes of high anion gap metabolic acidosis: MUD PILES

Methanol intoxication
Uremia
Diabetic ketoacidosis
Propylene glycol toxicity
Isoniazid toxicity
Lactic acidosis
Ethylene glycol intoxication
Salicylate toxicity

such as albumin, phosphates, sulfates and organic anions. Due to the large contribution of albumin, the anion gap varies significantly with serum concentration of albumin. Each 1.0 g/dL decrease or increase in serum albumin from 4.4 g/dL results in a corresponding increase or decrease in the anion gap approximately 2.3–2.5 meq/L. Consequently, hypo- or hyper- albuminemia must be considered when calculating anion gap. In a metabolic acidosis with decreased serum bicarbonate, serum electroneutrality is maintained by a compensatory increase in either serum chloride or unmeasured anions present in the anion gap. If electroneutrality is maintained by increasing chloride concentrations, the anion gap remains normal and we refer to the process as a "non anion gap metabolic acidosis" or "hyperchloremic metabolic acidosis". Causes of a non-anion gap metabolic acidosis are outlined in Table 14.2. If, however, electroneutrality is maintained by increasing unmeasured serum anions, the anion gap increases. We refer to this process as a "high anion gap metabolic acidosis" or "hypochloremic metabolic acidosis". Causes of a high anion gap metabolic acidosis are outlined in Table 14.3. It should be noted that

the terms "hypochloremic" and "hyperchloremic" are not in relation to normal laboratory values, but rather in relation to relative ionic composition of the plasma. It is possible to see a non-anion gap metabolic acidosis with a normal serum chloride.

Acidemia leads to cellular and enzymatic dysfunction with multiple deleterious effects including a decrease in cardiac output, hypotension and decreased binding of epinephrine to adrenergic receptors. Several physiologic mechanisms have developed which serve to correct plasma pH. The increase in plasma H^+ is sensed by the carotid bodies, which in turn stimulate the medullary respiratory center to increase minute ventilation and decrease pCO_2. The decreased plasma pH is also sensed by the kidneys, which respond by increasing H^+ secretion in the distal nephron. These compensatory changes take approximately 12–24 h to complete, and can be estimated by Winter's formula:

$$\text{Compensated}\, pCO_2 = 1.5 \times \left[HCO_3^- \right] + 8$$

If the pCO_2 is less than that predicted by Winter's formula, a secondary respiratory alkalosis is present. Similarly, a secondary respiratory acidosis would be indicated by a pCO_2 higher than that predicted by Winter's formula.

Treatment of metabolic acidosis is aimed at correction of the underlying cause. As such, accurate diagnosis of cause is essential. Debate exists as to whether or not correcting the acidemia is necessary. Current guidelines suggest correction of pH less than 7.10 until the underlying process can be corrected. Classically this has been done with sodium bicarbonate to replace the whole body deficit of bicarbonate. However, the volume of distribution for bicarbonate varies significantly with the degree of acidemia from between 50 % and 100 % total body water volume, which makes accurate dosing difficult. Recently the use of sodium bicarbonate as an alkalinizing agent has come under scrutiny. Several studies have demonstrated a lack of benefit, and in some populations increased mortality with the use of sodium bicarbonate to treat acidosis. Numerous adverse physiologic effects can occur from infusing sodium bicarbonate, although no single cause seems to be at fault. Sodium Bicarbonate is usually infused as a hypertonic solution. A 50 mL ampule containing 50 meq of sodium bicarbonate (1000 mmol/L) will raise the serum sodium concentration of a 70 kg person by about 1 meq/L and expand the ECF volume by about 250 mL. In addition, the infused bicarbonate will combine with plasma H+ and dissociate into CO2 and H_2O. In patients with impaired ventilatory

function this will lead to a respiratory acidosis. Alternative alkalinizing agents exist, such as the amino alcohol THAM. The buffering action of THAM does not generate CO_2, and has been used successfully for the treatment of various metabolic acidosis. It is excreted by the kidneys and should be dosed with caution in patients with renal insufficiency. Reported toxicities of THAM include hyperkalemia, hepatic necrosis in neonates, and respiratory depression secondary to an increase in pH and subsequent decrease in CNS CO_2.

Metabolic Alkalosis

Any process that leads to an accumulation of extracellular bicarbonate, via either decreased excretion of bicarbonate or increased loss of non-volatile acid, is termed a metabolic alkalosis. Non-volatile acids are lost either via the upper GI tract (vomiting, naso- or oro-gastric suctioning) or via increased urinary excretion. The kidney is able to excrete large amounts of bicarbonate in response to an alkalosis, thus maintenance of an alkalosis is due to an underlying disease state. These disease states can be grouped into either volume expanded or volume contracted states Table 14.4. Volume contraction reduces cardiac output and subsequently GFR. The reduced GFR leads to decreased chloride delivery to the distal nephron, therefore the $Cl–/HCO_3–$ counter-exchange pumps are unable to excrete $HCO_3–$. Furthermore, in response to the hypovolemia, aldosterone levels are increased which causes increased urinary H+ secretion.

Alkalemia is associated with a variety of negative physiologic effects. The affinity of hemoglobin for oxygen is acutely increased, resulting in a right shift of the oxygen-hemoglobin dissociation curve. This leads to impaired oxygen delivery to peripheral tissues. The increased pH causes a decrease in the

Table 14.4 Causes of metabolic alkalosis

Volume contracted state
Loss of gastric secretions: excessive NGT suctioning or vomiting
Loss of intestinal secretions: villous adenoma, congenital secretory diarrheas
Loop or thiazide diuretics
Sweat losses in cystic fibrosis
Volume expanded states
Primary mineralcorticoid excess
Liddle's syndrome

ionized concentration of calcium. This relative hypocalcemia can cause some of the classic clinical manifestations such as paresthesias and tetany. Other electrolyte abnormalities are seen, such as hypokalemia and hypomagnesemia. The compensation for a metabolic alkalosis is a decrease in ventilation with subsequent rise in pCO_2. Appropriate compensation can be calculated by the following equation:

$$pCO_2 = 0.9 \times \left[HCO_{3^-} \right] + 9$$

Once the underlying mechanism responsible for the alkalosis has been identified, treatment is aimed at correction of the metabolic abnormalities. Treatment of choice for the hypovolemic causes is IV rehydration, preferably with normal saline. Attention must be paid to potassium and calcium hemostasis so as not to worsen existing electrolyte abnormalities. If upper GI losses cannot be controlled, starting an H2 blocker or PPI may be warranted to decrease acid loss. In patients with decreased effective circulating volume, or volume overload, saline resuscitation is not warranted and could be catastrophic. In some cases, acetazolamide can be used to increase renal bicarbonate wasting. Attention must be paid to serum potassium concentrations if acetazolamide is used, as renal potassium wasting also increases. In states of mineralcorticoid excess, spironolactone can be used to antagonize the effects of aldosterone until surgical correction can be obtained. Spironolactone is considered a potassium sparing diuretic, and the potential for hypokalemia is less than with acetazolamide. Infusions of HCl have been used successfully to correct pH in cases of severe, refractory metabolic alkalosis. These infusions must be given slowly via central line with frequent lab measurements to avoid creating an iatrogenic metabolic acidosis.

Respiratory Abnormalities

Respiratory Acidosis

Any process that leads to an increase in pCO_2 due to an imbalance in alveolar minute ventilation and carbon dioxide production is termed respiratory acidosis. A respiratory acidosis may be caused by increased production of CO_2 with insufficient respiratory compensation, or decreased minute ventilation with normal production of CO_2. A third cause unique to the ventilated patient is rebreathing of exhaled CO_2 in the ventilator circuit. Respiratory acidosis is classified as either acute or chronic which can be determined by a large extend

by the degree of compensation. As previously stated, respiratory abnormalities are compensated via metabolic mechanisms. In the case of a respiratory acidosis, the compensation is by increased renal HCO_3^- reabsorption. During the acute phase of a respiratory acidosis, the kidneys are able to raise serum HCO_3– by approximately 1 meq/L for each 10 mmHg rise in pCO_2 above 40. Over time, the kidneys are better able to compensate and can raise plasma HCO_3– by approximately 3.5 meq/L for every 10 mmHg rise in pCO_2. Serum HCO_3– levels not consistent with expected levels of compensation indicate concurrent metabolic abnormalities.

Elevated CO_2 is associated with numerous systemic effects across multiple organ systems. Carbon dioxide is a direct myocardial depressant and acts directly on the vasculature to decrease overall tone. This is offset by increased sympathetic output, which results in elevated heart rate and net increase in cardiac output. Hypercapnia results in a rightward shift of the oxygen-hemoglobin dissociation curve and facilitates oxygen unloading in peripheral tissues. In the central nervous system, hypercapnia results in cerebral vasodilation, which increases cerebral blood flow and ICP. This can be an important consideration in patients with pre-existing elevated ICP or space occupying lesions. In the lungs, hypercapnia results in vasoconstriction and dilation of the small airways. In cases of severe hypercapnia (pCO_2 >90 mmHg), carbon dioxide displaces oxygen in the alveoli resulting in hypoxia, which can be fatal unless FiO_2 is increased.

As with the metabolic abnormalities, treatment of a respiratory acidosis should be aimed at the underlying cause. Occasionally intubation and mechanical ventilation are necessary as temporizing measures until the underlying cause has been reversed. Care must be exercised in patients with long standing respiratory acidosis with metabolic compensation. Increased ventilation and CO_2 elimination in these patients may lead to a relative hypocapnea and resultant metabolic alkalosis.

Respiratory Alkalosis

Any process that leads to a reduction in pCO_2, from increased alveolar minute ventilation relative to production, will result in a respiratory alkalosis. Most often this is secondary to increased alveolar minute ventilation but rarely may be due to decreased production of carbon dioxide with unchanged minute ventilation, such as the hypothermic mechanically ventilated patient. Similarly to a respiratory acidosis, a respiratory alkalosis can be classified as either acute or

chronic given the degree of metabolic compensation. In an acute respiratory alkalosis, every 10 mmHg drop in pCO_2 from 40 is accompanied by a 2 meq/L decrease in serum bicarbonate. A chronic respiratory alkalosis is expected to be compensated by a 5 meq/L decrease in serum bicarbonate for every 10 mmHg drop in pCO_2 from 40. This metabolic compensation is accomplished by decreased renal reabsorption of bicarbonate from the proximal renal tubule and an increase in ammonia excretion. This renal compensation begins within 2 h of a prolonged alkalosis, but is not maximally effective for 2–3 days.

The physiologic effects of decreased pCO_2 inversely correlate to those of an increase in pCO_2. Perhaps the most clinically significant effect of decreased pCO_2 is its effect on cerebral vascular tone. A decrease in pCO_2 results in cerebral vasoconstriction and a subsequent reduction in cerebral blood volume and ICP. In patients with TBI, CBF changes approximately by 3 % for each millimeter of mercury change in $PaCO_2$ over the range of 20–60 mmHg. This can cause a significant reduction in ICP, as a 0.5 mL change in CBF is associated with a 1 mmHg change in ICP. This effect is transient, though, as cerebral vasculature resets to the elevated CO_2. Current consensus does not recommend iatrogenic lowering of $paCO_2$ lower than 30 mmHg due to the increased risk of cerebral ischemia and lack of demonstrable clinical benefit. In addition to changes in CBF, other metabolic derangements occur as well. The decreased serum H+ leads to translocation of H+ from the intracellular to the extracellular space with a concurrent translocation of K+ from extracellular to intracellular space. This relative intracellular alkalosis causes activation of the enzyme phosophofructokinase and increase in glycolysis with generation of H+.

Treatment of the alkalosis is aimed at reversal of the underlying cause. In rare cases intubation and controlled mechanical ventilation may be necessary. However, in cases of chronic metabolic alkalosis correction must be done in a controlled manner allowing for renal compensation, lest a metabolic acidosis result from too rapid of correction.

Evaluating Multiple Disorders

It is not uncommon for multiple acid-base abnormalities to co-exist in the same patient. Diagnosis of these occult disorders requires knowledge of not only the expected direction, but also the expected magnitude of compensatory responses. The interplay of these complex interactions can be explained numerically as follows: in the absence of other metabolic derangements, the fall in the serum HCO_3 should equal the rise in the serum anion gap. We refer

to the difference between changes in serum anion gap and serum HCO_3 as the "Delta Gap." There have been multiple ways to approach this calculation, but one simple way is as follows:

$$\text{Delta Gap} = (\text{Anion Gap}) - (\text{Serum } HCO_3),$$
$$\text{where Anion Gap} = AG_{\text{measured}} - AG_{\text{normal(12)}}$$
$$\text{and Serum } HCO_3 = 24 - HCO_{3\text{measured}}.$$

If the Delta Gap is significantly positive (> +6), a concurrent metabolic alkalosis is usually present because the rise in anion gap is more than the fall in HCO_3. Conversely, if the Delta Gap is significantly negative (< −6), then a second acidosis is present because the rise in anion gap is less than the fall in HCO_3. This is usually a hyperchloremic metabolic acidosis. For example, 3 h into the operation, a blood gas shows an anion gap of 25 and a serum HCO_3 of 18. The Δ Anion Gap is 13 (25−12 = 13) and the Δ Serum HCO_3 is 6 (24−18 = 6), thus the delta gap is 7 (13−6 = 7). Therefore, for this patient we know in addition to the high anion gap metabolic acidosis, a concurrent metabolic alkalosis exists, as the serum HCO_3 would be expected to be lower if the alkalosis were not present.

In conclusion, an in-depth understanding of electrochemical and acid-base homeostasis is very important in the care of the surgical patient. The application of these principles ranges from acute resuscitation of a patient in various forms of shock to perioperative planning for an otherwise healthy patient undergoing major surgery.

Case Study

A 34-year-old man is scheduled to come to your OR for emergent exploratory laparotomy. He presented to the emergency department complaining of abdominal pain for the past 18 h following a 1-day history of diarrhea. His past medical history is significant for insulin-dependent (Type 1) diabetes, ethanol abuse, and poor medical adherence. On physical exam he has dry mucous membranes, a fruity odor to his breath, and has slurred speech. He is guarding his abdomen. Vital signs are: HR: 120, BP: 101/74, RR: 20, SpO2: 99 % on room air, Temp: 101.4 F

What laboratory evaluation would you order preoperatively? Why?
Given the history of diabetes, poor medical adherence, and 2 days of fluid losses and likely little or no oral intake, it is likely that he has some metabolic derangements. You should order a metabolic panel, a complete blood count, and probably a serum or urine ketone test. If you suspect an acid/base disturbance, a blood gas is also needed.

Laboratory values are returned and are shown below. What metabolic disturbance is likely responsible for these values?

Sodium: 134 mmol/L
Chloride: 110 mmol/L
Potassium: 4 mmol/L
Bicarbonate: 13 mmol/L
BUN: 36 mg/dL
Creatinine: 1.10 mg/dL
Glucose: 284 mg/dL

WBC: 21 × 103/mcL
Hemoglobin: 16 g/dL
PCV: 48 %
Platelet: 200 × 103/mcL
MCV: 104 fL
RDW: 16 %

Serum Beta-hydroxybutyrate: 4 mg/dL

ABG: pH 7.21/pCO2 28 mmHg/pO2 99 mmHg/HCO2 13 mmol/L

This patient is in diabetic ketoacidosis (DKA) as confirmed by his hyperglycemia, serum ketones, and acidemia present on ABG. DKA usually presents in patients with IDDM who have a concurrent illness and/or poor insulin regimen adherence.

What acid base abnormalities are present? Is there more than one? Are they appropriately compensated?
The low pH, low bicarbonate and low pCO_2 point to a metabolic acidosis with respiratory compensation. To evaluate the etiology of the metabolic acidosis, we must first calculate the ion gap.

$$\left(\text{Serum Na}^{+} + \text{Serum K}^{+}\right) - \left(\text{Serum HCO}_{3^{-}} + \text{Serum Cl}^{-}\right)$$
$$= (134 + 4) - (13 + 110) = 15$$

As the normal anion gap is 8–12, there is a high anion gap metabolic acidosis present. To determine whether or not we have appropriate compensation, we will use Winter's formula to calculate what his pCO_2 should be.

$$pCO_2 = 1.5 \times \left[\text{HCO}_{3^{-}}\right] + 8 = (1.5 \times 13) + 8 = 27.5$$

This is very close to the measured value of 28, so appropriate respiratory compensation has occurred. To determine whether or not multiple acid base abnormalities are present, we must calculate the delta-gap for this patient.

$$\text{Delta Gap} = (\text{Anion Gap}) - (\text{Serum HCO}_3)$$

$$\text{Anion Gap} = \text{AG}_{\text{measured}} - \text{AG}_{\text{normal(12)}}$$
$$\text{and} \quad \text{Serum HCO}_3 = 24 - \text{HCO}_{3\text{measured}}$$

$$\text{DG} = (15 - 12) - (24 - 13) = -8$$

If a single acid base disturbance is present, the delta gap should be 0+/−6. The delta gap in this case is significantly negative, which is to say we would expect the change in the anion gap to be larger given the change in the serum bicarbonate. Consequently, in addition to a high-anion gap metabolic acidosis, we can also conclude that a non-anion gap metabolic acidosis is present.

What is the likely etiology of the second metabolic acidosis?
The patient demonstrates a combined high-anion gap and non-anion gap metabolic acidosis with appropriate respiratory compensation. His high anion gap acidosis is likely secondary to his DKA, and his non-anion gap acidosis is likely secondary to his diarrhea. In diarrhea, bicarbonate is lost and acidemia occurs; depending on the fluids administered subsequently, chloride may also increase ("hyperchloremic acidosis").

He is started on an insulin infusion and taken to the OR. 15 min into the case he has received 500 mL of normal saline, and the operation is under way. You look up at the ECG and notice ST-segment depression and flattening of the T wave. What is the most likely diagnosis and appropriate treatment?

The patient has most likely developed hypokalemia, which has manifest as ST segment depression and T wave flattening. U waves may also be seen in cases of severe hypokalemia. Patients with DKA may present with normal serum potassium, however they often have a total body potassium deficit. This is due to the transcellular shift of potassium caused by the acidosis and resultant osmotic diuresis from the elevated plasma glucose. This deficit is revealed with the re-introduction of insulin, which stimulates the membrane bound Na^+/K^+-ATPase and causes an intracellular shift of potassium. Treatment is hydration (ideally prior to insulin administration) and repletion of potassium; in severe cases it may be necessary to temporarily pause the insulin infusion.

Halfway through the case, a serum osmolality value is reported by the lab at 340 mosmol/kg. How do you interpret this result? Is this consistent with the calculated serum osmolality?

Calculated serum osmolality is given by the equation:

$$\text{Serum Osmolality} = \left(\text{Serum } Na^+ \times 2\right) + \left(\text{Serum glucose} / 18\right) + \left(\text{Serum BUN} / 2.8\right)$$

In this case: $\left(134 * 2\right) + \left(284 / 18\right) + \left(36 / 2.8\right) = 297$.

Therefore, we can say that an osmolal gap exists in the amount of 43 mosm/kg. An osmolar gap can occur by one of two mechanisms: either an osmotically active solute other than electrolytes, glucose or urea is present, or pseudohyponatremia. In the case of an unmeasured osmotically active solute, the serum osmolality is actually increased and the measured value is the correct value. In the case of pseudohyponatremia, the measured osmolality is spuriously reduced by the presence of increased lipids or proteins (such as triglycerides or immunoglobulins), which reduce the fraction of serum that is water. This represents a measurement artifact and the calculated serum osmolality is the correct osmolality. Osmolar gaps are

important to calculate, particularly in the case of high anion gap metabolic acidosis, as potential causes include toxic alcohols and glycols, which will cause a serum osmolar gap. The most common cause is acute ethanol ingestion, the contribution of which can be estimated by dividing the serum ethanol concentration by 3.7.

You recall that the patient has a history of ethanol abuse and had slurred speech on initial presentation. Using the osmolality you calculated and measured, how would you estimate his blood alcohol concentration?
Assuming the osmolar gap is caused by ethanol, we would multiple the gap by the contribution of ethanol to the osmolality, or 3.7: 43 * 3.7 = 159 mg/dL, or in layman's terms, 0.16 (which is roughly twice the U.S. legal limit for drivers).

A colonic perforation is found, and a subtotal colectomy is performed with end colostomy placement. The abdomen is closed and the patient is transported to the surgical ICU. On arrival to the ICU, your most recent BMP is as follows:

Sodium: 140 mmol/L
Chloride: 108 mmol/L
Potassium: 3.5 mmol/L
Bicarbonate: 21 mmol/L
BUN: 42 mg/dL
Creatinine: 1.3 mg/dL
Glucose: 120 mg/dL

Noting the glucose of 120, the nurse asks if she can discontinue the insulin infusion. What should your response be?
No! When treating DKA insulin administration should be continued until the anion gap is normal, which represents a resolution of ketoacidosis. In this case the anion gap is still 14.5, so insulin therapy should be continued, with IV dextrose administration to prevent hypoglycemia.

Suggested Readings

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Chapter 15

Fluids and Transfusion Therapy

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Educational Objectives

1. Understand physiologic fluid compartments
2. Be able to describe volume status and estimate fluid requirements
3. Describe options available for crystalloid resuscitation
4. Describe options available for colloid resuscitation
5. Understand risks and benefits of tight vs. liberal transfusion strategies

Introduction to Water Physiology

Water represents 60 % of the total body weight of the average adult, and is divided into two major compartments: intracellular and extracellular. The two compartments are separated by the semi-permeable cell membranes that enclose all cell types in the body. Of the two compartments, intracellular is by far the larger and constitutes 66 % of total body water (TBW) with the remaining 33 % residing in the extracellular compartment. The volume of the extracellular compartment is composed of the interstitial space (~75 %) and intravascular space (~25 %). The intravascular space is defined as the volume contained within the lumen of the body's vasculature, including arteries, veins and capillaries. Eighty-five percent of the volume in the intravascular space is contained in the venous system, and the remaining 15 % is contained in the arterial system. The total volume of the arterial and venous system is referred to as "circulating blood volume." The blood volume contains both a cellular and non-cellular component. The cellular component is composed of red blood cells, white blood cells, and platelets, and the non-cellular component

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is referred to as plasma. The plasma component contains approximately 25 % of the extracellular volume. The interstitial component is the volume existing outside of the intracellular compartment that is not contained within the vascular lumen, and constitutes 75 % of the extracellular volume. For example, a 70 kg man contains approximately 40 L of water (1 L water = 1 kg). Of that 40 L, approximately 26 L are contained in the intracellular compartment and approximately 14 L are contained in the extracellular compartment. Of those approximate 14 L contained in the extracellular compartment, roughly 3.5 L reside in the intravascular space with the remaining 10.5 L existing in the interstitial space.

Water travels freely across cellular membranes and between the intravascular and interstitial spaces. The distribution of water is dependent on the interplay of two main forces: hydrostatic and osmotic. Hydrostatic forces are generated by the heart when it beats, and forces blood through the vasculature. Hydrostatic forces can be thought of as pressure that can be measured inside and outside the vasculature, such as blood pressure and compartment pressures. Osmotic forces are generated by the differential distribution of proteins in the various compartments; the higher the protein concentration in a given compartment, the greater the force drawing water into that compartment through diffusion. Protein concentrations are higher in the intracellular compartment and the intravascular space, promoting the movement of water into these areas. Hydrostatic forces are greater than osmotic forces in the arterial side of the intravascular space, which forces water out of the intravascular space and into the interstitial space. However, osmotic forces are greater than hydrostatic forces on the venous side of the intravascular space resulting in water being drawn back into the intravascular space.

In addition to differences in protein content, the electrolyte composition of the extracellular and intracellular compartments varies greatly. The extracellular fluid has a relatively high concentration of sodium and bicarbonate, while the intracellular fluid has a very high concentration of potassium and magnesium. These concentration gradients are maintained by active transport transmembrane pumps, and are essential for the proper functioning of many cellular processes. Electrolyte balance is further discussed in another chapter.

Perioperative Fluid Balance

Significant water losses occur daily via a variety of routes. As water leaves the body, it often takes with it electrolytes and proteins. The electrolyte and protein composition of these losses is highly variable and must be replaced in order

to maintain homeostasis and continue normal physiologic processes. Maintenance fluid requirements can be estimated according to patient body weight using the "4-2-1" rule as follows:

0–10 kg: 4 ml/h per each kilogram of body weight to max 10 kg
11–20 kg: 40 ml + 2 ml/h for each kilogram of body weight in this range
>20 kg: 60 ml + 1 ml/h for each additional kilogram of body weight

For example, maintenance fluid rate for a 70 kg man can be calculated as thus:

60 ml/h for the first 20 kg of body weight leaves 50 kg of body weight unaccounted. Allocating 1 ml/h for each of those kilograms gives us 50 ml/h. Adding these together we get 60 ml/h + 50 ml/h for a total of 110 ml/h maintenance fluid rate. A short cut applying to patients weighing more than 20 kg is to add 40 to the patient's weight in kg to obtain maintenance fluid requirement in ml/h (e.g. 70 kg + 40 = 110 ml/h).

Maintenance requirements do not account for pathologic losses as occur with hemorrhage, diarrhea, or vomiting. In addition, certain disease states and medications can increase fluid loss (e.g. a poorly controlled diabetic with osmotic diuresis from high plasma glucose concentrations). A thorough history and physical exam are vital to determining a patient's volume status and resuscitation requirements. Key elements to be covered in the pre-operative history to assess volume status include but are not limited to: hours NPO, recent diarrhea or vomiting, diuretic use, presence of orthopnea or presence of leg swelling. Physical exam findings suggesting of a fluid deficit include flattened neck veins, tachycardia, dry mucous membranes, and low blood pressure. Physical exam findings suggestive of volume overload include full or distended neck veins, normal to high blood pressure, pitting edema in bilateral lower extremities and auscultation of rales or crackles during inspiration. Once the volume status has been determined, attention can then be turned to selection of resuscitation fluid and administration.

Selection of Resuscitative Fluid

Non-blood product fluid replacement options can be grouped as either crystalloid or colloid. Crystalloid solutions contain water, electrolytes, and occasionally dextrose. Colloid solutions contain water, electrolytes, and large-molecular weight substances that, in theory, remain in the intravascular space for several hours and increase osmotic forces drawing water into the vasculature. Colloids

and crystalloids both are devoid of clotting factors and cellular blood components. Large volume resuscitation with either crystalloid or colloid will cause dilution of blood cellular components and coagulation factors leading eventually to coagulopathy and impaired oxygen delivery. As such, attention must be paid to type of fluid selected and amount of resuscitation needed. More importantly, the electrolyte and acid-base content of the fluid is recently being shown to have profound effects on end-organ dysfunction and possibly mortality in the perioperative period.

Crystalloids

Crystalloid solutions may be classified as hypertonic, hypotonic or isotonic relative to normal plasma osmolarity. Crystalloid solutions distribute freely between intravascular and interstitial compartments, and as such 25–33 % of the intravenously administered crystalloid can be estimated to remain intravascular.

Isotonic solutions include normal saline, so-called "balanced salt solutions," and solutions containing 5 % dextrose. Normal saline (NS, 0.9 % NaCl) contains equal parts sodium and chloride, both of which are higher than physiologically found in plasma. Normal saline has no buffer or other electrolytes, and relative to plasma is slightly hypertonic (308 Osm) and very acidic (pH ~5.0). "Balanced salt solutions" include Plasma-Lyte, Lactated Ringer's (LR) and Normosol. These solutions contain electrolyte compositions similar to those found in plasma, and are buffered to varying degrees. Relative to plasma they tend to be more isotonic and of a more physiologic pH. Dextrose containing solutions typically contain either 5 % or 10 % dextrose, and may or may not contain other electrolytes. In the absence of other electrolytes, dextrose containing solutions are functionally equivalent to free water because the dextrose is metabolized leaving behind only free water. Free water itself cannot be administered intravenously; the hypotonicity would result in lysis of red cells. Dextrose is added to increase the osmolality, however the solution is not buffered and is acidotic relative to plasma pH. Hypertonic solutions contain high concentrations of sodium and chloride and typically range from 3 % to 5 % NaCl. Similar to NS, they do not contain other electrolytes or buffers. They are significantly hypertonic relative to plasma and are typically administered in lower volumes than isotonic or hypotonic solutions. Selection of the appropriate crystalloid depends on the clinical situation and is based on crystalloid electrolyte composition, pH and osmolality. For example, in neurosurgical cases

NS is the preferred crystalloid, as NS is hypertonic relative to plasma and helps limit cerebral edema, whereas LR has 100 mL of free water per liter as compared to normal plasma osmolarity. Transfusion of blood products typically occurs concurrently with crystalloid to help decrease viscosity. LR is contraindicated for this purpose, as the calcium present in LR binds to citrate present in blood products and can occlude IV lines.

Classically, NS has been the crystalloid of choice for virtually all clinical scenarios. However, a growing body of evidence is beginning to demonstrate that it may not be so "normal" after all, and can in fact lead to significant negative clinical outcomes. Hyperchloremic metabolic acidosis can occur with the administration of large volumes of NS and NS-balanced fluids, such as some Albumin 5 % solutions. When compared to NS, balanced salt solutions have been associated with a large variety of beneficial outcomes in the perioperative setting including: greater urinary output, decreased nausea and vomiting in elderly surgical patients, lower incidence of metabolic acidosis, and decreased use of blood products [1–3]. NS has been preferred for patients with renal failure due to the theoretical concern that balanced salt solutions would lead to hyperkalemia. However, a prospective RCT assigning 0.9 % NS or LR for intraoperative use during kidney transplantation found that 31 % of patients receiving NS developed potassium concentrations greater than 6 meq/L vs. 0 % of patients receiving LR [4]. Although a study of this size does not warrant a change in clinical practice, it does challenge the long held belief that saline is superior to balanced salt solutions in patients with renal failure.

Colloid Solutions

Colloid solutions use either a complex sugar or protein suspended in an electrolyte solution and can be grouped accordingly. Multiple preparations of each type of colloid exist and vary according to electrolyte composition and concentration of osmotically active substance. Compared to crystalloids, colloids create a greater amount of intravascular volume expansion if equal volumes are infused. Compared to crystalloids, colloids on average are significantly more expensive.

Albumin is the principal protein based colloid, and is available as either 5 % or 25 % solution suspended in saline solution. The electrolyte composition of albumin is solely sodium and chloride, with 145 meq/L of each. Colloid

albumin is derived from large pools of human plasma that have been processed and sterilized. The intravascular half-life of albumin is highly variable based on capillary basement membrane integrity and ranges from 3 to 16 h [5]. Severe, life-threatening anaphylactic reactions to albumin have been reported, but these cases appear to be extremely rare [6]. Five percentage albumin has a colloid oncotic pressure of approximately 20 mmHg, which close to physiologic plasma oncotic pressure, while 25 % albumin has an oncotic pressure of approximately 100 mmHg. Compared to other colloids, albumin is significantly more expensive.

Complex sugar based colloids include the Dextrans and Hydroxy-ethyl starch (HES). Dextran solutions contained polymerized glucose which is synthesized by certain bacteria. Two preparations of dextran exist differentiated by mean molecular weight: 40 and 70 kDa. Dextran 70 is typically administered in a 6 % solution with saline for use in volume expansion. Dextran 40 is used mainly to improve microcirculatory flow across microvascular anastomosis and seldom is used for volume expansion. Significant allergic responses occur to dextrans in about 1 of every 3300 administrations. Other side effects associated with dextrans include decreased platelet adhesiveness, decreased levels of factor VIII and increased bleeding time [7]. Dextran molecules bind to erythrocyte cell membranes and interfere with blood typing and cross matching. Dextrans are excreted primarily by the kidneys with a plasma half life ranging from 6 to 12 h. HES solutions are classified according to concentration (6 % vs. 10 %), average molecular weight in kDa, and molar substitution. Molar substitution refers to the modification of the original polysaccharide by the addition of hydroxyethyl groups. Higher degrees of molar substitution are associated with greater resistance to degradation by plasma amylase, and therefore longer plasma half-lives. HES concentrations typically result in a volume expansion similar to that seen with 5 % albumin, and lasts 8–12 h. Recently the safety of HES solutions has been brought into question. Most of the early work on HES solutions was found to be fraudulent, and a recent meta-analysis found that HES was associated with a significantly increased risk of mortality and renal failure [8]. In addition to the well-documented deleterious effects of HES on renal function, HES solutions have also been shown to interfere with vWF, factor VIII, and platelet function [7]. Newer formulations of HES are currently in clinical trials and their safety remains to be demonstrated.

Crystalloid Versus Colloid

Much debate has existed as to which resuscitative fluid is superior: crystalloid or colloid. Crystalloid proponents are quick to point out the significantly higher cost of colloids and their associated allergic and hematologic adverse effects. The largest randomized control trial comparing normal saline to 5 % albumin, the Saline versus Albumin Fluid Evaluation (SAFE) trial [10], involved nearly 7000 ICU patients. No difference was noted between saline and albumin in terms of overall mortality and morbidity.

Introduction to Transfusion Therapy and Blood Components

Intraoperative blood losses contain more than water and electrolytes. Oxygen carrying capacity is reduced as red blood cells are lost, and a coagulopathy develops as coagulation factors and platelets are consumed or diluted by aggressive fluid resuscitation. Failure to adequately account for and replace these losses can result in continued hemorrhage with lethal consequences. Replacement of red blood cells and coagulation factors is accomplished by administration of blood components. To obtain blood components, whole blood is collected from donors and separated into a cellular and liquid phase by fractionation. RBC's and platelets are then further isolated from the cellular component, while the liquid component yields FFP and cryoprecipitate. These four components are the most commonly used blood transfusion products.

Blood Groups and Compatibility

There are more than 300 different antigens present on the surface of red blood cells. These have been separated into more than 20 different systems which are used to classify blood groups. Of these 20 different systems, the two most clinically important are the ABO system and the Rh system. ABO typing is determined by the presence or absence of A or B RBC surface antigens. Type A blood has the A surface antigen, type B blood has the B surface antigen, type AB blood has both A and B surface antigens, and type O blood has neither A nor B surface antigen present. Individuals almost universally have IgM antibodies present in their serum against the missing antigen; thus individuals with type A blood have antibodies against B surface antigens and vice versa. The Rh system classifies individuals by the presence or absence of the D Rhesus antigen as Rh-positive or Rh-negative, respectively. In contrast to the ABO antigens, antibodies against the Rh antigen do not typically naturally occur, and develop after Rh-negative individuals are exposed to Rh-positive blood. The probability of

developing anti-D IgG antibodies after a single exposure to the Rh antigen is 50–70 %. Administration of Rh immunoglobulin (Rhogam) to Rh- negative individuals can protect against Rh sensitization following exposure, and is particularly important in Rh-negative mothers with Rh-positive fetuses. Transfusion of incompatible blood leads to reaction of recipient antibodies against donor antigens and leads to compliment activation and intracellular hemolysis. These reactions can be catastrophic, and as such blood products are screened for the type of antigen present prior to transfusion.

Red Blood Cells

Red blood cells are separated from donor whole blood, mixed with an anticoagulant (usually citrate) and can be stored up to 42 days at 1–6 °C. The hematocrit of stored blood is between 70 % and 80 %, and 1 unit of packed Red Blood Cells (pRBCs) is expected to raise the hemoglobin by 1 g/dL and the hematocrit by 3 % in non-bleeding, average sized adults. Stored RBCs have low levels of 2,3-DPG with a leftward shift of their oxygen dissociation curve. This effect is temporary, as 2,3-DPG levels return to normal within 24 h of transfusion. Stored RBCs also leak potassium, and although each unit supplies a relatively small amount of potassium, massive transfusion may result in clinically significant hyperkalemia.

The appropriate hemoglobin at which to transfuse has been the subject of debate. Multiple studies have demonstrated that a restrictive strategy (target hemoglobin 7–9 g/dL) is superior to a liberal strategy (target hemoglobin 10–12) in terms of lower mortality and lower rates of complication. However, subset analyses of these trials show that in older patients and patients with cardiovascular disease a liberal strategy may be more appropriate. The ultimate goal of transfusion therapy is to maintain optimal oxygen delivery, and as such transfusions at higher hemoglobin concentrations may be indicated if signs of end organ ischemia manifest. Transfusion thresholds may be set preoperatively, and the allowable blood loss can be estimated with the following equation:

$$\mathrm{ABL} = \mathrm{EBV} \times \left(\mathrm{Hct_i} - \mathrm{Hct_t} \,/\, \mathrm{Hct_i} \right)$$

where EBV represents estimated blood volume,
$\mathrm{Hct_i}$ is initial hematocrit and $\mathrm{Hct_t}$ is threshold hematocrit for transfusion.
EBV is estimated to be 75 mL/kg for men and 65 mL/kg for women.

Continued large volume blood loss results in the need for massive transfusion. Massive transfusion is defined as transfusion of blood components in excess of 1 blood volume within a 24-h period, which is usually the equivalent of 10 units of pRBC. Hemorrhaging patients lose more than just pRBCs, and replacement of FFP and platelets is necessary in these scenarios. Experience in civilian and military trauma has shown better outcomes if the ratio between products is equal. This 1:1:1 (pRBC:FFP:Platelet) approach mimics whole blood, and is associated with improved outcomes.

Fresh Frozen Plasma

FFP is isolated from whole blood and frozen within 8 h of donation to prevent inactivation of labile coagulation factors. FFP may be stored frozen for up to 1 year. Thawing of FFP is usually accomplished by soaking in a 37 °C water bath for 30–45 min. Once thawed, FFP can be refrigerated for a maximum of 24 h. FFP contains physiologic concentrations of coagulation factors, complement, albumin and globulins. Each unit of FFP raises plasma levels of each clotting factor by approximately 2–3 %. In addition to being used in massive transfusions, FFP is used to correct coagulopathies associated with liver disease, warfarin, or isolated factor deficiencies. ABO-compatibility is ideal, but not mandatory as it is for pRBC transfusion.

Platelets

Platelets are isolated from donor whole blood and suspended in approximately 50 mL of plasma. Individual platelet units can be stored for up to 5 days at room temperature. Platelets are typically pooled from multiple donors prior to administration, and must be used within 4 h of pooling. The usual number of units pooled is 6, and can be expected to raise the platelet count by $30{,}000–60{,}000 \times 10^9$/L in an average sized adult. ABO and Rh compatibility, although desirable, is not necessary for transfusion. However, transfusion of Rh-positive platelets may sensitize an Rh-negative individual due to small concentrations of red cells present in platelet units. The life span of transfused platelets is between 1 and 7 days in the absence of active bleeding or immune mediated destruction. Guidelines for transfusion of platelets are not hard and fast, and continue to change. Patients with platelet counts less than 5×10^9/L are at increased risk for significant hemorrhage and most guidelines propose prophylactic platelet transfusions for counts less than 10×10^9/L. Platelet transfusions are of little benefit, and may be harmful, in cases of thrombocytopenia

due to immunologic processes such as TTP and ITP. If given quickly or through an in line warmer, platelet activation and histamine release can occur leading to hypotension.

Cryoprecipitate

Cryoprecipitate (cryo) is produced by centrifuging frozen FFP that has been thawed to 6 °C and re-suspending the precipitated proteins in 15 mL of supernatant plasma. Similarly to platelets, several units are pooled prior to dosing, and are a concentrated source of factors VIII, XIII, von Willebrand factor, fibronectin, and fibrinogen. The advantage of cryo over FFP is the ability to deliver specific proteins with less total volume. Cryo was historically used for the treatment of inherited coagulopathies such as hemophilia A, Factor XIII deficiency and von Willebrand's disease. Isolated factor concentrates are now used for these diseases and Cryo is now most often administered to replenish fibrinogen. No specific guidelines exist regarding the administration of cryo, but the usual dose for treatment of hypofibrinogenemia is 10 units to start, then 6–10 units every 8 h as necessary to keep fibrinogen above 100 mg/dL. As with platelets, ABO and Rh compatibility is not required for administration.

Complications of Product Transfusion

Complications arising from product transfusion can be classified as either immune mediated or infectious. Immune mediated reactions can be further classified as hemolytic and non-hemolytic. Hemolytic reactions are most commonly due to ABO blood incompatibility with an approximate frequency of 1:38,000 transfusions. These reactions are often severe and can result DIC, shock and death. Fatal hemolytic transfusion reactions occur in about 1 in 100,000 transfusions. Non-hemolytic transfusion reactions include febrile reactions, anaphylactic reactions, and transfusion related acute lung injury. Transfusion related acute lung injury (TRALI) occurs in about 1 in 5000 transfusions and is thought to be caused by damage to alveolar capillaries from transfused anti-leukocyte or anti-HLA antibodies. TRALI manifests as acute hypoxia and non-cardiogenic pulmonary edema within 6 h of blood product transfusion. Treatment is supportive, and TRALI typically resolves spontaneously within 4 days.

Transfusion associated infections may be viral, parasitic or bacterial. The rise of HIV in the 1980s and 1990s led to more stringent donor criteria and increasingly sensitive screening tests. Rates of transmission for HIV

and Hepatitis C are approximately 1:2,000,000 and rates of transmission for hepatitis B are about 1:200,000. Bacterial contamination of blood products is far more common, and is the second leading cause of transfusion-associated mortality. Gram- negative and Gram-positive bacteria can contaminate blood products and cause sepsis in recipients. The prevalence of bacterial contamination varies by blood product, ranging from 1:2000 for platelets to 1:7000 for pRBCs. Transmission of parasites such as malaria, toxoplasmosis and Chagas' disease has been reported, but is extremely rare.

Conclusions

Resuscitative fluids should be thought of as medications; they have specific indications and dosages. Appropriate resuscitation involves understanding the composition of both the resuscitative fluids and the perioperative fluid losses. Most balanced resuscitations consist of multiple types of crystalloid, colloid, and blood products. Much research continues to be done on the ideal composition of resuscitative fluids, and the field will likely continue to evolve quickly in the years to come.

Case Study

Planning for Major Blood Loss

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 l, 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. 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 are you comfortable letting her hemoglobin fall to?
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 patients 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 mmHg 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 hematrocrit 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 Readings

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Chapter 16

Peripheral, Arterial, and Central Lines and Gastric Tube Placement

Jenna L. Walters and Matthew D. McEvoy

Key Learning Objectives

1. Describe the anatomical sites, technical steps, and possible complications of peripheral intravenous catheter placement.
2. Describe the technical steps of radial artery cannulation.
3. Describe the anatomical sites, technical steps, and possible complications of central venous catheter placement.
4. Describe the utility of ultrasound in placement of a peripheral venous, arterial or central venous catheter.
5. Describe the appropriate steps and possible complications of placing a nasogastric or orgastric tube.

Peripheral Intravenous Lines

Peripheral intravenous catheter (PIV) placement is a necessary task for almost every anesthetic. Intravenous access provides the anesthetist the ability to administer fluids, medications, and, if necessary, blood products during the perioperative period.

Site of Cannulation

The site of cannulation should be based on the patient's position during surgery, the site of surgery, and the gauge (size) of the cannula being placed. PIV placement should be avoided in areas where there are signs of infection, burns,

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trauma, previous or present arteriovenous fistulas, radiation exposure, or recent IV infiltration. Along with choosing a site for PIV placement, the size of cannula should also be considered. A decrease in the gauge of the catheter corresponds with an increasing catheter diameter (e.g. a 14G catheter is larger than a 24G). Poiseuille's equation should be considered when the catheter gauge is chosen, as it states that flow through a tube is directly proportional to the pressure difference at the beginning and end of the tube and to the radius to the fourth power, and flow is inversely proportional to the length of the tube and viscosity of the fluid. For all practical purposes when selecting the size of an IV, it is the radius and length that matters, as long as blood products are not being given (i.e. higher viscosity). Additionally, it is difficult to give exact flow rates through any given catheter, but in general larger catheters and higher inflow pressures result in exponentially higher flow rates.

The upper extremities are usually the preferred site of placement as this location allows for easier access to the IV during the surgical procedure and a greater ability to reassess the site during a procedure to check for infiltration. Common sites of cannulation in the upper extremities include the veins of the hand and forearm, which include the cephalic and basilica vein systems. The median antecubital vein can usually accommodate a larger bore PIV, however flow may be limited if the arms are positioned with even a slight bend. Furthermore, a PIV in this position can be frustrating to the patient and care team after surgery as it will consistently cause an infusion pump to alarm if the arm is bent, thus causing the catheter to be kinked within the vessel. The dorsal veins of the foot can also be accessed. However, this is associated with a higher risk of thrombophlebitis, along with patient discomfort. If a lower extremity PIV is needed, the saphenous vein can often be easily palpated just anterior to the medial malleolus and it can typically accommodate a larger gauge catheter if the upper extremities are not accessible.

The external jugular vein is another peripheral intravenous site frequently used by the anesthesiologist because of its reliable anatomical position. It is typically located close to the surface of the skin superficial to the sternocleidomastoid muscle. Placement of this line requires a shallow angle when attempting cannulation. Caution should be taken when placing an external jugular cannula due to the risk of inadvertent puncture of the deeper structures of the neck, including the carotid artery, internal jugular vein, and pleural space. External jugular intravenous catheters also frequently require turning of the head to the contralateral side to run effectively.

Technique

As with any procedure, an explanation of what the patient can expect (mild, temporary discomfort during placement) and the risks, including infection and bleeding, should be discussed with the patient. Gloves should be worn during the placement of any PIV and the area should be thoroughly cleaned with alcohol or chlorhexidine. A tourniquet should be tied tightly proximal to the site of cannulation to promote engorgement of the vein.

The gauge of the intravenous cannula needed for each patient is dependent upon their surgical procedure, likelihood of blood loss or need for vasoactive drugs, their specific comorbidities and the size of the vein being accessed. Once the gauge has been determined, inspect the metal needle and plastic cannula noting the distance between the tip of the needle and the tip of cannula. The amount of exposed needle increases with increasing size of the catheter, thus requiring deeper entry into the vein before the catheter can be advanced off of the needle.

The decision to use local anesthetic at the site for pain depends on the size of the needle being inserted. Although local anesthetic creates a sympathectomy that can prevent venoconstriction, it may also obscure the view of the vein. Lidocaine 1 % is typically used and a volume of 0.5–1 mL at the insertion site is adequate to reduce or eliminate pain.

To stabilize the vein, use your non-dominant hand to pull the skin taught distal to the site of insertion. Use your dominant hand to insert the cannula and needle together at a 5–30° angle to the skin. The idea is simply to put a tibe (catheter) inside another tube (vein). Thus, lining up the axis and angle of the two is the most important step. Once the vein as been entered a "flash" of blood will appear in the reservoir of the catheter. Lower the angle of the catheter so that it is parallel to the axis of the vein and continue to advance the needle and cannula simultaneously an additional 2–3 mm. This ensures that the tip of the cannula has also entered the vein. Next, thread the cannula off of the needle into the vein. Once the cannula has been threaded to its hub, remove the tourniquet and apply pressure proximal to the cannula to occlude the vein in order to prevent back bleeding when the needle is removed. Remove the needle, placing your sharp in a safe location, and attach the intravenous tubing. The intravenous fluid should flow freely into the catheter if the fluids are above the level of the heart. Finally, apply a sterile dressing to secure the intravenous catheter in place.

Troubleshooting

Difficulty locating a vein can be one of the most challenging aspects of PIV insertion. Having the patient open and close their first, letting the arm hang below the level of the heart to encourage venous filling, and tapping the vein can all help increase its size. If no extremity veins are visible or palpable, or if multiple attempts have been unsuccessful, ultrasound can be used to locate the deeper veins in the arm. The basilic vein and deep brachial vein are reliable choices in this setting. A landmark-based approach to these deep veins has been shown to lead to frequent complications, such as arterial puncture and nerve injury. As such, these veins should be located and cannulated with ultrasound guidance. A longer cannula (>2 in.) may be needed to access these veins.

Valves inside a vein can sometimes prevent complete advancement of the cannula. Removing the needle and advancing the cannula further into the vein while flushing it with saline solution creates positive pressure which may help open the valve and allow passage of the cannula past the obstruction.

If a cannula is advanced outside of the vein, there is usually swelling at the site of cannulation when fluid is administered as it infiltrates the extravascular tissue. The cannula should be removed immediately and a new cannula inserted at a different site. Repeated attempts at flushing the catheter or readjusting the position should be avoided.

Arterial Catheters

The indications for arterial blood pressure monitoring include the need for beat-to-beat blood pressure monitoring due to patient co-morbidities or procedure, frequent blood gas analysis, unreliability of non-invasive blood pressure cuff (e.g. the obese patient), or contraindication to use of a cuff (e.g. a patient with extensive burn wounds over the extremities).

Site of Cannulation

The most common site of arterial cannulation is the radial artery. However, the femoral, brachial, axillary, and dorsalis pedis are alternative sites based on the patient's anatomy or surgical site. Contraindications to placement include infection at the site, arterial thrombus, trauma or burn proximal to the vessel, and concern for collateral flow. Arterial cannulas are typically 20 gauge catheters, but smaller sizes may be required for pediatric patients. Their length depends on the site being accessed with longer catheters required for femoral arteries as compared to radial arteries, particularly in the obese patient.

The Allen's Test has been traditionally used to assess the collateral flow to the hand via the ulnar artery. To perform the test, the hand is held in a fist above the level of the heart for 30 s. The radial and ulnar arteries are occluded and the hand is opened and then the ulnar artery is released. The palm is observed to see if the pallor resolves within 10 s. Although some experts still recommend using the Allen's Test, a recent study comparing the Allen's Test to Doppler flow found that it was not reliable in predicting collateral flow.

Technique

Prior to performing the procedure, the patient should be appropriately consented and the procedure explained. Only the technique for cannulation of the radial artery will be discussed in this chapter. Although the incidence of arterial line infection is minimal, it is recommended that a hat, mask, and sterile gloves be worn during the procedure. A small gauze roll is placed under the wrist to slightly extend it and the hand is taped to a table or arm board for immobilization. The radial artery should be located by palpation of the pulse at the wrist between the radius and the flexor carpi radialis tendon. The area should be prepped with chlorhexidine and draped with sterile towels. A small amount of local anesthetic (0.5–1 mL of 1 % lidocaine without epinephrine) can be injected at the entry site to prevent pain during needle insertion if the cannula is placed in an awake patient. This also creates a sympathectomy that reduces vasospasm when accessing the vessel. The needle is held in the dominant hand like a pencil and should enter the skin at a 30–40° angle. Once a flash is seen in the catheter chamber, the needle is advanced 1 mm further and the angle is decreased to 10–15° as the guidewire is advanced into the artery. The cannula can then be passed over the guidewire in a Seldinger technique and the guidewire and needle can be removed. Prior to removing the guidewire, pressure should be applied proximal to the site of cannulation to prevent bleeding through the catheter. The cannula should then be attached to high pressure, low compliance arterial pressure tubing, and sutured or securely taped into place with a sterile, transparent dressing being applied.

Troubleshooting

Frequently there is only a small "flash" of blood into the arterial cannula chamber and the chamber does not fill completely or the chamber fills but the guidewire does not pass easily. In these situations, the artery can be transfixed, meaning the needle and cannula are purposely passed through the artery. The

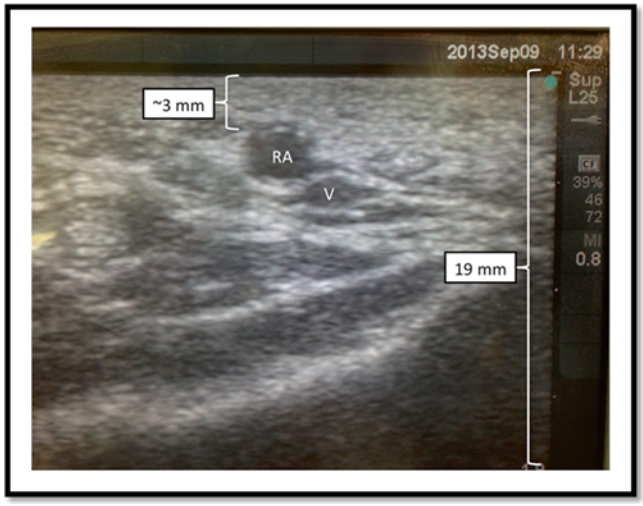

Figure 16.1 This figure shows a short axis view of the radial artery (RA) along with an accompanying radial vein (V), depicting the superficial nature of the artery in relation to the skin [*RA* radial artery, *V* radial vein]

needle is then removed and the cannula is slowly pulled out of the skin until spontaneous arterial flow is identified. The guidewire can then be inserted into the cannula and the cannula advanced further into the artery.

If there is no palpable pulse or multiple attempts at arterial cannulation have been made without success, an ultrasound should be used to provide a visual target. The use of ultrasound can also increase the likelihood of accessing the artery on the first attempt, which is particularly helpful in hypovolemic patients or patients with difficult anatomy, such as the obese, the very thin/frail, or those with advanced peripheral vascular disease (see Fig. 16.1).

Central Venous Catheter Insertion

General Principles

Central venous catheters (CVC) provide reliable intravenous access in preparation for cases where there may be large volumes of blood loss requiring high flow administration of fluids, the need to administer infusions of vasoactive medications, or placement of pulmonary artery catheters. Prior to performing

the procedure, informed consent should be obtained including the risks and benefits of CVC placement. Handwashing should occur immediately before line placement. A sterile gown, hat, mask, gloves should be worn during the procedure. The area should be prepped with chlorhexidine and a full body drape applied to decrease the risk of central line-associated blood stream infection (CLABSI). This series of steps has been shown to significantly reduce CLABSI, and in some instances eliminate it all together. For the placement of an internal jugular or subclavian vein CVC, the patient should be positioned in the Trendelenberg position to promote engorgement of the vein.

Cannula Size and Length

CVCs come in varying sizes, lengths and numbers of ports. The diameter of the catheter is expressed as a French gauge. Adult central lines typically range from 7 to 9 French which equate to a catheter 2.3–3 mm in diameter, respectively. Each catheter can have 1–3 ports, and as the number of ports increases the diameter of each port decreases. If the CVC is placed for volume resuscitation, it is better to have a larger diameter catheter with fewer ports. As noted above, according to Poisuelle's Law the length of the catheter also affects the rate of flow, with shorter catheters allowing for faster flow. The resistance to flow through any catheter is directly proportional to the length of the catheter and the viscosity of the blood and inversely proportional to the radius of the catheter to the fourth power. Therefore a short catheter with a large diameter provides the least resistance to flow.

Ultrasound Guidance

According to the most recent guidelines by the American Society of Anesthesiologists for CVC placement, real-time ultrasound is recommended for all attempts at central line placement. Randomized control trials show that the use of real-time ultrasound increases the first attempt success rate, decreases the time to venous access, results in higher overall successful cannulation and decreases the risk of arterial puncture. Observational studies and expert opinion recommend confirmation of the wire or catheter in the vein before dilation using ultrasound, TEE, continuous EEG, fluoroscopy, pulse-waveform gas or manometry. Figures 16.2a, b and 16.3 show examples of this in short axis and long axis. The long axis is the most reliable method for ensuring that the entire course of the wire remains intravascular (Fig. 16.4).

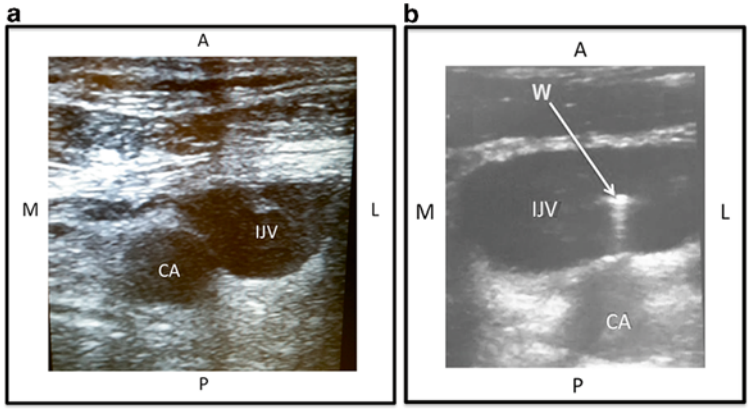

Figure 16.2 (a) and (b) illustrate two different short axis views of the internal jugular vein and the carotid artery. Figure (a) shows the classical description of the anatomic relationships with the artery laying medical to the vein, whereas (b) shows a variant where the artery is posterior to the vein, making arterial puncture more likely if the needle passes too deep [*IJV* internal jugular vein, *CA* carotid artery, *W* wire]

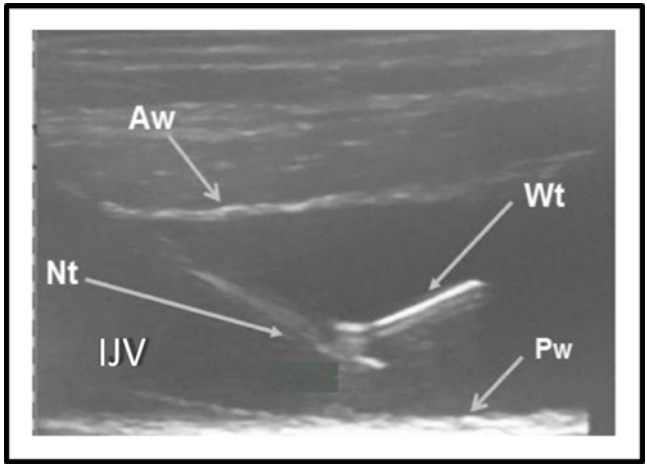

Figure 16.3 This figure illustrates a long axis view of the internal jugular vein (IJV) with the anterior wall (Aw) and posterior wall below (Pw). The needle tip (Nt) can be visualized within the lumen and the needle tip (*Nt*) can be seen exiting the bevel of the needle [*IJV* internal jugular vein, *Aw* anterior wall, *Pw* posterior wall, *Nt* needle tip, *Wt* wire tip]

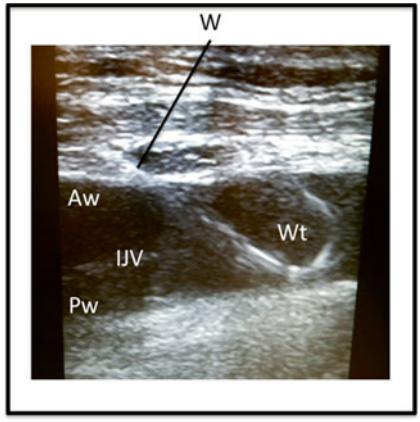

Figure 16.4 This figure also shows a long axis view of the internal jugular vein (IJV) lumen with the anterior wall (Aw) above and the posterior wall (Pw). The wire tip (Wt) is visualized as it is advanced through the needle tip. This ensures that the entire wire is visualized within the internal jugular vein lumen [*IJV* internal jugular vein, *Aw* anterior wall, *Pw* posterior wall, *Nt* needle tip, *Wt* wire tip]

Internal Jugular Vein

The internal jugular vein is tthe most commonly cannulated vein by anesthesiologists due to its ease of access. To find the vessel using anatomic landmarks, locate the cricoid cartilage and move laterally until the carotid artery is palpable. The vein runs just lateral and anterior to the artery.

The head is turned slightly to the contralateral side. The entire side of the neck down to the level of the clavicle should be prepped and draped. The ultrasound probe with a sterile cover should be used to visualize the internal jugular vein and the carotid artery. The vein should be compressible while the artery is pulsatile. If the patient is awake, 3–5 mL of lidocaine 1 % without epinephrine should be infiltrated above the vein. An empty syringe is attached to the needle used to access the vein. Gentle suction is applied to the syringe while the needle enters the skin and subcutaneous tissue at a 30–45° angle. In order to advance the needle and wire under direct ultrasound guidance, an assistant is often required (Fig. 16.5). Once the vein is accessed, the syringe will begin to fill with blood. The syringe should be removed and a guidewire passed through the needle into the vein. Advancing the guidewire too far and into the right atrium can lead to ectopy seen on the ECG and the wire should be pulled

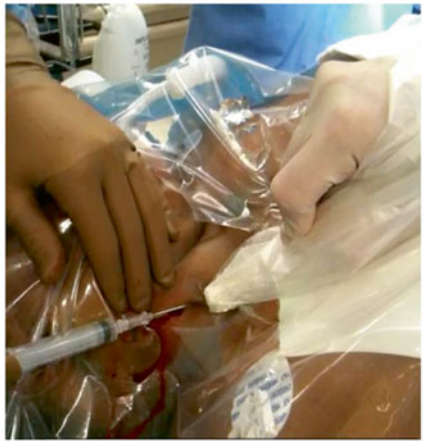

Figure 16.5 This figure shows a two-person technique to access the internal jugular vein using a long axis view of the internal jugular vein. One person advances the introducer needle while another person positions the ultrasound parallel to the course of the internal jugular vein

back. If any resistance is met during insertion of the wire the syringe should be re-attached and confirmation of blood flow confirmed with gentle needle aspiration. If this does not occur, the needle position should be checked with ultrasound. The wire positioning can be checked in the short and long axis view. The wire should be seen traversing through the vein down to the level of the clavicle. These views ensure that the wire has not passed through the vein and into the artery.

Once the wire is positioned in the vein, the needle should be removed. A small cut should be made in the skin at the wire insertion site to allow passage of the dilator. The dilator should be passed over the wire and inserted just through the fascial layer but not farther to avoid sheering the vein. The dilator is removed and the catheter is passed over the wire into the vein. The ports on the line are then aspirated, flushed and capped. The line is sutured in place and a sterile, antibiotic impregnated bio-occlusive dressing is applied. A chest x-ray should be ordered after insertion of all central lines to ensure proper positioning.

Subclavian Vein

The landmarks for accessing the subclavian vein include the bend in the lateral two thirds of the clavicle and the sternal notch. The entire side of the chest above and below the clavicle should be prepped and draped including the sternal notch. The bend in the clavicle is palpated and the needle is inserted 1 cm caudad and 1 cm lateral to this site. Typically, a gap can be palpated between the anterior border of the deltoid and the lateral border of the pectoralis major as an additional anatomical landmark that helps in finding the insertion site. The needle is advanced just underneath the bend in the clavicle aiming at the sternal notch. The vein should be encountered directly below the clavicle. Once the aspiration of blood is confirmed, the syringe should be removed and the additional steps performed as detailed above in the internal jugular vein section followed.

Femoral Vein

The femoral vein is a less common site of central line placement due to concern for infection, however it can be a preferred site of cannulation if there is concern for damage to the superior vena cava or if the neck and subclavian sites would be inaccessible, such as during major head and neck surgery. The vein is located inferior to the inguinal ligament between the superior anterior iliac spine and the pubic tubercle within the inguinal crease just medial to the femnoral artery. The anatomy of this space from lateral to medial is: femoral nerve, femoral artery, femoral vein, and then lymphatics. The contents of the surrounding structures should be understood in order to avoid injury. The entire inguinal region should be widely prepped and draped. Ultrasound should be used to visualize the femoral vein and artery. The needle should be directed cephalad at a 30–45° angle to the skin when entering the femoral vein, and the needle should be directed in a slight medial direction, as the femoral veins follows this course as they turn into the iliac veins and then merge as the inferior vena cava. Once aspiration of blood is confirmed, the syringe should be removed and the additional steps performed as detailed in the internal jugular vein section above followed.

Complications

The complications for all central lines regardless of insertion site include infection and bleeding. Accessing the femoral vein has the highest risk of arterial puncture (6 %), thrombosis (8–34 %) and infection (15 %). However there is

no risk of pneumothorax so this approach may be preferred in patients with severe lung disease in which a pneumothorax could lead to respiratory or hemodynamic instability. Placing a central line in the subclavian vein carries the highest risk of pneumothorax (1.5–3 %) but the lowest risk of infection and arterial puncture (0.5 %). Central lines placed in the internal jugular vein have a relatively low risk of pneumothorax (0.2 %) but a slightly higher risk of arterial puncture (3 %). All central lines carry a risk of damage to surrounding nerves, which can result in a paresthesia and temporary or permanent nerve damage.

Orogastric and Nasogastric Tubes

Orogastric (OGT) and nasogastric (NGT) tubes are frequently placed to decompress the stomach to decrease the risk of aspiration, improve surgical exposure or relieve build-up of gastric and intestinal contents when there is concern for ileus or obstruction. If the patient is awake, a nasogastric tube is typically preferred. This procedure is normally very uncomfortable for the awake patient, so its necessity and procedural steps should be explained to the patient prior to placement.

Nasogastric Tube

To estimate the length of insertion, the distance from the nose to the ear and then to 5 cm below the xiphoid process should be marked. If the patient is awake, they should be encouraged to swallow during the procedure to facilitate successful placement in the esophagus. The tube should be lubricated and directed along the floor of the nostril towards the back of nasopharynx. Do not advance in a cephalad direction, as this can cause epistaxis from injury to the inferior turbinate. Upon entering the nasopharynx, NGT must take a 90° turn to enter the posterior oropharynx and then continue into the esophagus. If the patient is asleep, the non-dominant hand can be used to perform a jaw lift to displace the tongue and guide the tube into the esophagus. If resistance is met during placement the tube should not be forced, as this will help to avoid submucosal placement or epistaxis. The risk of epistaxis can be decreased with the use of a vasoconstrictor nasal spray prior to placement. NGTs should not be placed in patients at high risk for bleeding due to coagulopathy, patients with basilar skull fractures, previous gastric bypass surgery or high-grade esophageal varices. In addition to not being the oral cavity, a major benefit of NGT placement for the awake patient is that it lies behind the tonsillar pillars and is thus associated with less gag reflex.

Orogastric Tubes

The OG approach is preferred if the patient is asleep and will only need the tube while intubated and unconscious. The OGT should be lubricated and directed toward the base of the tongue and through the oropharynx into the esophagus. A jaw lift can help displace the soft tissue if difficulty is encountered when attempting to advance the orogastric tube. The tube should be guided into the esophagus using the index finger of the dominant hand. Contraindications to orogastric tubes include previous gastric bypass surgery and high-grade esophageal varices. As with the NGT, the OGT should not be forced past resistance as this could lead to submucosal placement and significant tissue injury.

Placement of an NGT or OGT should be confirmed. This can be done by attaching the tube to suction and checking for the return of gastric contents. A post-placement x-ray can confirm the location of the tube. Additionally, injecting air through the tube and listening with a stethoscope over the stomach can also be performed, although this method can be associated with false positives. Finally, NGTs and OGTs are often placed on low continuous wall suction or on intermittent suction. A high level of continuous suction is not recommended in order to avoid mucosal damage and bleeding.

Case Study

Do you need better access?

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 this 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 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." Will you proceed?

You can certainly try to 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 blood stream 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 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 (thought the latter causes 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 mmHg, which is above venous but below arterial pressure, may reveal veins by causing mild ischemia-induced vasodilation. Commercial "vein finder" machines use special optics to allow visualization of veins through intact skin that are otherwise not visible. 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, and is considered the standard of care in non-emergency situations.

Suggested Readings

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Chapter 17

Intraoperative Problems

Andrea Westman and Matthew D. McEvoy

Key Learning Objectives

- Discuss a general approach to managing the unstable surgical patient, including formation of a differential diagnosis for the instability.
- Be able to identify the most common intraoperative problems that occur in a systematic manner
- Review management options for the common intraoperative problems encountered
- Discuss an approach to emergence and extubation, including a differential diagnosis for failure to extubate.

General Concepts

Anesthesiologists and nurse anesthetists have the opportunity and challenge of caring for a wide array of patients ranging from the healthy ASA I patient to the moribund ASA 5 patient. Despite the complexity and uniqueness of each patient, there are many common problems that occur in the operating room of which you should be aware. It is important that you have a thorough understanding of both physiology and pharmacology in order to care for patients undergoing anesthesia, and in order to be able to recognize and confidently manage intraoperative events.

Additionally, it is imperative that members of the anesthesia care team act quickly to correct any problems that occur in order to prevent harm to the patient. As opposed to standard Advanced Cardiac Life Support (ACLS) that is primarily built for unwitnessed cardiopulmonary arrest, the problems that

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will be described in this chapter typically occur in a setting in which (a) the problem is witnessed from its onset, (b) the patient is extensively monitored (e.g. ECG, pulse oximeter, $ETCO_2$), and (c) the past medical and surgical history of the patient is known. As such, while many of the underlying principles and goals of ACLS for assessment of patients in unstable, near-arrest, and arrest situations apply, numerous management steps are different. Table 17.1 lists the most common intraoperative problems that may occur during anesthesia. Almost all perioperative urgencies and emergencies will map to one of five areas of concern: cardiac, pulmonary, neurologic, metabolic/endocrine, or toxins. This is certainly true for all common intraoperative patient problems. The only additional category is that of machine or system failure (e.g. oxygen supply failure), which is not discussed in this chapter.

The goals of this chapter include providing you with a framework by which to think about the approach to the unstable patient, how you might construct a differential diagnosis for that instability, and the proper steps for initial management of common intraoperative problems. When there is a concern for an acute change in patient condition under anesthesia, having a structured approach to the patient will be of benefit. First, you should start by assessing the general hemodynamic state of the patient and proceed through a series of decision nodes to define the initial course of action. This should start with the steps of defining whether the patient has a pulse. If not, proceed with advanced cardiac life support (ACLS) management while noting the differences in the operative setting [10, 13]. If the patient has a pulse, then one should define whether there is an acute hemodynamic instability to be addressed. If so, following the steps outlined in the cardiovascular section below is indicated. If the patient appears to have adequate cardiac output, then an assessment can proceed to the other four areas of concern noted above, with pulmonary being most important in order to ensure that adequate oxygenation and ventilation are present. A progression of thought through the other possible major categories should be undertaken in sequence. At each step, one should ask the questions outlined in Fig. 17.1, which will aid in constructing a differential diagnoses, picking a leading diagnosis, and then proceeding with initial management plan. Of note, the initial diagnosis may simply be a patient state (e.g. severe hypoxemia) rather than a particular known cause. Thus, in the acute care setting the first step is to ensure that cardiopulmonary function is at a level that meets basic metabolic requirements in order to avert patient harm and then proceed to defining an exact etiology.

Table 17.1 Common intraoperative problems and differential diagnoses

Problem	Differential diagnosis	Remarks
Cardiovascular		
Hypotension	Hypovolemia	Common causes include blood loss or dehydration from preoperative fasting
	Relative anesthetic overdose	Minimal or a decrease in surgical stimulation can lead to relative anesthetic overdose and hypotension
	Vasodilatation from medication	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
	Low cardiac output	Many anesthetics decrease cardiac output. Other causes include congestive heart failure, myocardial infarction, or tamponade
	Severe bradycardia	This may cause low cardiac output (CO = HR × SV), leading to hypotension
	Severe tachycardia or arrhythmias	If atrial fibrillation or flutter becomes too fast, hypotension may result
	Pneumothorax	Uncommon, but may spontaneously arise during positive pressure ventilation
	Anaphylactic reaction	Most commonly from reaction to muscle relaxants or antibiotics. Give epinephrine to treat
	Sepsis	Sudden decreases in blood pressure may result from sepsis
Hypertension	Pain from surgical stimulus	Always consider “light” or insufficient anesthesia
	Essential hypertension	These patients may be adequately anesthetized, but still markedly hypertensive
	Tourniquet pain	A tourniquet can produce a hard, recalcitrant kind of hypertension called “cuff hypertension
	Light anesthesia	Check for empty vaporizers or medication infusors
	Hypervolemia	Fluid overload from intravenous fluid or blood products; may lead to pulmonary edema and congestive heart failure in patients with heart disease

(continued)

Table 17.1 (continued)

Problem	Differential diagnosis	Remarks
Pulmonary		
Hypoxemia Failure to ventilate	Low inspired oxygen (FiO2)	Always start by increasing the FiO_2, and then continue to look for other causes
	Hypoventilation	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
	Disconnection of breathing circuit	The most common cause of serious hypoxemic accidents
	Atelectasis	Often a result of positive pressure ventilation, intubation and/or hypoventilation. Consider alveolar recruitment maneuvers
	Bronchospasm	Consider administering albuterol
	Mucus Plugging	Perform suction and alveolar recruitment maneuvers
	Right main-stem bronchial intubation	Maximum depth for tracheal tubes measured at teeth: females 21 cm; males 23 cm. May visualize with bronchoscope or auscultate with stethoscope
	Pulmonary thromboembolism (PE)	This may be diagnosed by a sudden drop in end-tidal CO_2, and hypoxemia that does not improve with 100 % FiO_2. Often accompanied by tachycardia and hypotension
	Venous air embolism	Also causes drop in end-tidal CO_2, and an increase in end-tidal N_2 Most likely during ENT or thoracic surgery
	Kinked endotracheal tube	May occur during "light" anesthesia or emergence; Can cause negative pressure pulmonary edema
	Biting on endotracheal tube	The most common cause
	Disconnection of endotracheal tube from circuit or adapter	Can occur rapidly in infants/children whose ETT are narrow (especially when no humidification is used). Suction or replace tube
	Complete endotracheal tube obstruction from mucus or tissue	Most often during laser airway surgery or tracheostomy. Both surgeries also have ↑ risk of airway fire!
	Hole in endotracheal tube or a punctured cuff	

High airway pressures	Bronchospasm	(1) deepen anesthesia, (2) consider neuromuscular blockade, (3) administer beta-agonists, inhaled or intravenous corticosteroids, theophylline or epinephrine
	Kinked endotracheal tube	May require the use of an armored (metal spring reinforced) tube to prevent kinking
	Biting on endotracheal tube	Consider placing a bite block or mouth gag
	Mucus plugging	Common in patients with COPD, asthma and cystic fibrosis. This may require replacement of the ETT
	Stacking or auto-PEEP of mechanical breaths	Occurs when the expiratory phase isn't long enough to allow exhalation. Decrease the respiratory rate
	Dynamic airway obstruction	May be from an airway tumor or mediastinal mass, especially after change in patient position
	Obesity or chest wall rigidity	May be difficult to manage. High opioid dose can cause a "rigid chest syndrome
	Acute Respiratory Distress Syndrome (ARDS)	A common cause of high mean pressures, especially in the ICU
Hypocarbia	Hyperventilation	May see in anxious (awake) or mechanically hyperventilated (anesthetized) patients
	Leak of CO_2 in sampling tubing	This may also cause an abnormality in the capnograph waveform or envelope
	Massive pulmonary embolus	Can impair gas exchange, manifesting as a sudden drop in expired CO_2
	Hypothermia	Most evident in severe hypothermia as during cardiopulmonary bypass
	Cardiac Arrest	Impaired circulation and CO_2 elimination
Hypercarbia	Hypoventilation	Often from opioids, residual neuromuscular blockade, or low respiratory rate/ventilator tidal volumes
	CO_2 insufflation during laparoscopy	May need to increase minute ventilation to overcome hypercarbia
	Malignant hyperthermia	Uncoupling of calcium metabolism in mitochondria from a rare (1:15,000) genetic defect in the ryanodine receptor of the calcium channel

Table 17.1 (continued)

Problem	Differential diagnosis	Remarks
Toxins		
Anaphylaxis	Usually due to neuro-Muscular blockers, latex, or antibiotics	Stop offending agent, support hemodynamics
Transfusion reaction	Many types	Stop agent, support pressure, report to blood bank
Metabolic		
Hypothermia	Convective, conductive, radiative, evaporative losses	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
	Anesthetic effects on hypo-thalamus	Anesthetics cause impaired central thermoregulation due to effects on the hypothalamus
	Administration of unwarmed fluid or blood products	Fluids should be warmed by an FDA-approved device
	Massive blood loss	Difficult to keep patients warm after ≥1 blood volume has been lost
Hyperthermia	Excessive warming	Use the air-warming blanket at a room-temperature setting to cool the patient
	Fever from sepsis or transfusion reaction	Give acetaminophen or ibuprofen in addition to a cooling blanket
	Stroke	Sudden extreme hyperthermia (>105 °F) may be from a stroke to the hypothalamus
	Neuroleptic malignant syndrome	Uncommon side effect of antipsychotic medications (chlorpromazine, haloperidol, olanzepine)
	Malignant hyperthermia	(1) stop anesthetic, (2) give iv dantrolene, (3) call for help (see Appendix B, Malignant Hyperthermia)

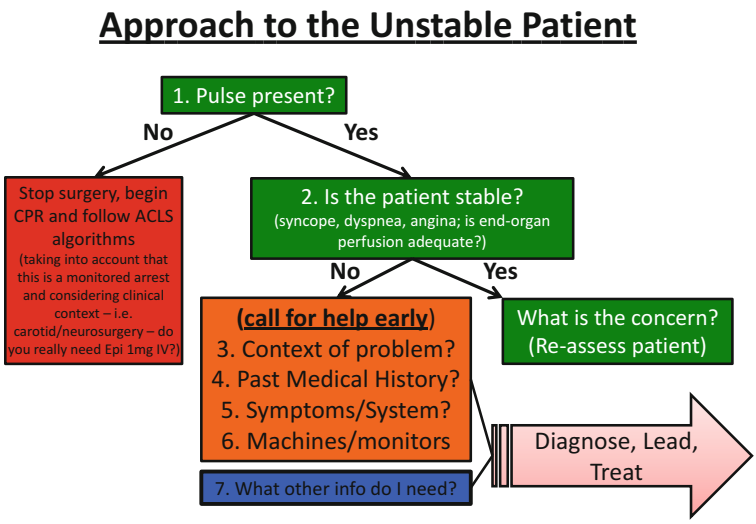

Figure 17.1 Approach to the unstable patient

Cardiovascular

The cardiovascular system is often the source of many intraoperative problems as there are numerous forces at play that can effect normal homeostasis in preload, afterload, chronotropy, and inotropy. Disturbances of the cardiovascular system can be thought of originating from the following etiologies: pipes (systemic vessels), pump (RV/LV inotropy), preload, valves, vessels (coronary arteries), and voltage (conduction system). Systematically reviewing these six categories provides structure within the cardiovascular system to critically evaluate the patients condition, formulate a differential diagnosis, and quickly identify and treat the problem. Below is a brief overview of the six key components of cardiovascular integrity. For a more in-depth discussion of cardiovascular physiology, please see Chap. 18.

Pipes are the peripheral vessels (aorta – >arteries – > arterioles) that carry oxygen and nutrients to the tissues for exchange at the capillary level. They then carry CO_2 and waste back to the liver, for detoxification, and heart by the venous system (venules – > veins – > IVC and SVC). This complex system of vessels in series and in parallel accounts for the majority of **afterload** as systemic vascular resistance. The heart is a **pump** that provides cardiac output

to the body. The effectiveness of the heart as a pump is affected by heart rate, stroke volume, contractility, and afterload, which is the pressure the heart must contract against to generate forward flow. **Preload** can be thought of as the volume of blood in the right and left ventricles at the end of diastole. This volume is important for determining the strength of contraction, which is due to myocardial stretch at the beginning of systole, according to the Frank-Starling law. The extremes of preload (severe hypovolemia and hypervolemia) both result in reduced cardiac output, but by different mechanisms. The heart has four chambers, thus there are four **valves**, which are responsible for both allowing forward flow of blood and halting backward flow, depending on which part of the cardiac cycle is occurring. These include the tricuspid, pulmonic, mitral, and aortic valves. The coronary arteries are the **vessels** that supply blood and oxygen to the myocardium. They consist of the left and right coronary arteries, which become the left anterior descending, left circumflex, and posterior descending arteries, along with a number of smaller branched arteries. **Voltage** refers to electrical conduction through the heart during the cardiac cycle. Normal sinus rhythm is important because it produces maximal atrio-ventricular coupling for effective filling and ejection of blood to produce a stable cardiac output. The most common problems of the cardiovascular system will be discussed below. For an extensive list with many other problems, please refer back to Table 17.1.

Dysrhythmias and Cardiac Arrest

As an extension of what was discussed above, the following steps are helpful during the initial assessment and management of an acute cardiovascular disturbance related to cardiac arrest or acute rhythm disturbance:

1. *Pulse*: present or absent?
2. *Stability*: is patient stable (adequate C.O.) or unstable (signs/symptoms of poor perfusion – angina, SOB, altered mental status, etc)
3. *Rate*: tachycardic (>150, >120 in afib) or bradycardic (<50)
4. *Rhythm*: assess the QRS and rhythm strip
   (a) wide or narrow?
   (b) regular or irregular?
   (c) is a dysrhythmia causing instability?
5. *Diagnosis*
   (a) *Pulseless:* start CPR and commence ACLS, taking into account changes from standard ACLS for some causes, such as local anesthetic systemic toxicity [10, 13].

(b) *Pulsatile*: follow treatment per ACLS or Anesthesia-Centric ACLS based on above questions [10, 13]. For example:
   (i) Unstable bradycardia
   (ii) Unstable, wide complex, regular tachycardia (monomorphic VT)
   (iii) Stable, narrow complex, irregular tachycardia (atrial fibrillation)

6. *Treatment*: as per ACLS protocols for rhythm disturbances

In order for blood to be delivered in an efficient fashion to the peripheral circulation, it is important for the heart to be in normal sinus rhythm. Severe bradycardia and tachycardia can negatively affect the ability of the heart to pump oxygenated blood to tissues. In the community and non-operative health care settings, ACLS algorithms are standardized and followed by all health care professionals [13]. However, the operating room presents a unique setting for the performance of ACLS, which is often modified to a tailored approach for each patient because the anesthesia provider usually witnesses the event, knows the patient's medical comorbidities, and understands the surgical pathophysiology that may have led to the event [10]. See Table 17.2 for common causes of intraoperative dysrhythmias and Fig. 17.2 for the comprehensive anesthesia-centric ACLS algorithm that should be followed.

Bradycardia is defined as a heart rate less than 60. Some patients may have native heart rates that are below this, which is often not a problem as long at they can maintain an appropriate blood pressure. If symptomatic bradycardia develops, you should start by reviewing the Hs and 8Ts (Table 17.3) that are included in the anesthesia-centric ACLS algorithms. In addition to those, some unique surgical causes include a patient who took their beta-blocker preoperatively, administration of vagotonic medications (e.g. fentanyl), reversal of neuromuscular blockade with a cholinesterase inhibitor and insufficient vagolytic agent, and a vagal reaction. Concerning the latter cause, insufflation of the abdomen during laparoscopy may produce severe bradycardia, as may pulling on the eye during ophthalmologic surgery or a high spinal during a caesarian section. If a patient is hemodynamically unstable but still has a pulse, you should give a direct acting chronotrope, such as epinephrine or norepinephrine. Typically 10–25 mcg IV is sufficient to increase the heart rate, but increasing doses and an infusion may be needed depending on the etiology. Anti-cholinergic agents can also be of benefit, but administration of direct-acting chronotropes should not be delayed if the patient is unstable. If chemical chronotropes are not working or if the patient has a 3rd degree AV block, external pacing through defibrillator pads should start immediately.

Table 17.2 Common intraoperative dysrhythmias

Problem	Differential diagnosis	Remarks
Bradycardia	β-blockers	Probably the most common cause
	Hypoxia	Occurs with severe hypoxia
	Myocardial infarction	Likely if the right coronary artery and sinus node are involved in the infarction
	Increased vagal tone	Surgical stimulus on the gut, bladder, or other organs may increase vagal tone. Atropine and, later, deepening anesthesia may be indicated. May also be seen in athletes
	Third degree heart block	The ECG rhythm strip will provide the diagnosis
	Calcium channel blockers	Especially caused by diltiazem (used for this purpose in atrial fibrillation and flutter)
	Reversal of neuromuscular blockade with cholinesterase inhibitors such as edrophonium or neostigmine	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
Tachycardia	Total spinal Increased pain or surgical stimulus	Support hemodynamics and administer vasopressors as needed The most common cause at the start of the surgical procedure. May suggest insufficient anesthesia
	Vasopressors or inotropes	Ephedrine, epinephrine, norepinephrine, isoproterolol can all cause tachycardia
	Myocardial infarction	The most common dysrhythmia associated with MI
	Arrhythmias	Atrial fibrillation, Ventricular Tachycardia
	Malignant hyperthermia	Tachycardia in MH follows an observed increased CO_2 production and precedes hyperthermia
	Atropine, scopolamine, or glycopyrrolate administration	These are commonly given as antisialogogues (dry secretions), vagolytics (increase heart rate) or antiemetics (nausea control)
	β-adrenergic agonists	Bronchodilators, tocolytics, and decongestant medications may cause tachycardia

Premature ventricular contractions	Hypoxia	Always consider hypoxia first
	Myocardial ischemia	Check a 12-lead when feasible
	Metabolic acidosis/alkalosis	Should always be high in the differential. Consider checking a blood gas
	Hypokalemia	Patients on diuretic therapy, without potassium replacement, may have this. Patients on digoxin who are hypokalemic are at particular risk
	Digoxin	Commonly used for atrial dysrhythmia therapy
	Sympathomimetic drugs	Ephedrine or pseudoephedrine (found in cold remedies)
	Hypomagnesemia	Occurs in alcoholism or after prolonged use of diuretics like furosemide
	Hypokalemia	Especially pronounced with digoxin
	Hypothermia	Patients who are post cardiopulmonary bypass or post exposure injury may have this
	Hypercarbia	In postoperative patients with pre-existing PVCs, mild hypercarbia in the PACU may cause increases in rates of PVCs
	Hypocarbia	If severe enough to cause respiratory alkalosis, PVCs may occur
	Myocarditis	PVCs are common with viral myocarditis
	Toxic overdose of drug	Unlikely, but can occur from antidepressants
Ventricular tachycardia	Same causes as PVCs	Ventricular tachycardia may be thought of as three or more PVCs in a row
	Hypoxia or ischemia	Most likely acute causes in the OR
Premature atrial contractions	Hypovolemia	Rapid diuresis and its effect on volume sensors in the atria may cause PACs and atrial fibrillation
	Hypertension	PACs commonly occur with concomitant hypertension
	Previous thoracic surgery	PACs occur in 25–30 % of patients undergoing thoracic (lung, mediastinal, or esophageal) surgery
	Mediastinal infection	Consider anastomotic leak in recent esophagectomy

(continued)

Table 17.2 (continued)

Problem	Differential diagnosis	Remarks
Atrial Fibrillation/ Atrial Flutter	Same causes as PACs	Approach includes: 1. Treat underlying causes 2. Control the heart rate by slowing the ventricular response 3. Convert back to sinus rhythm if possible Useful drugs include digoxin, diltiazem, metoprolol and amiodarone
Asystole	Severe hypoxemia	Treat underlying causes
	Severe hypovolemia	
	Severe electrolyte abnormality	
	Myocardial infarction	May require transcutaneous or transvenous pacing
	Severe metabolic acidosis	Treat underlying causes
	Pneumothorax	
	Pericardial tamponade	
Pulseless electrical activity (PEA)	Severe electrolyte abnormality	Treat underlying causes
	Myocardial infarction	
	Severe metabolic acidosis	
	Pneumothorax	
	Pericardial tamponade	

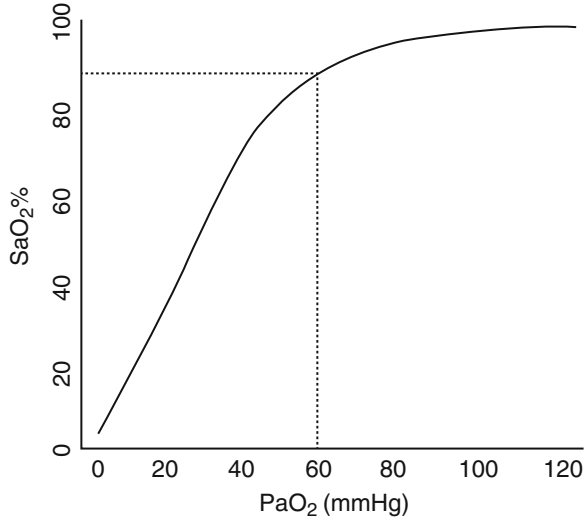

Figure 17.2 Comprehensive anesthesia-centric ACLS algorithm

Table 17.3 Common causes of perioperative pulseless arrest

Hypovolemia	Toxins
Hypoxoia	Tamponade
Hypovolemia	Tension pneumothorax
Malignant Hypothermia	Thrombosis (coronary and pulmonary)
Hypervagal	Trauma
Hydrogen Ion (acidosis)	qT prolongation
Hyper/hypokalemia	Pulmonary hypertension
Hypoglycemia	
Hypothermia	

Tachycardia can occur due to a wide array of causes. Like all dysrhythmias, you should think about the common Hs and Ts (i.e. reversible causes) in the perioperative period, see Table 17.3. Common surgical causes include hypovolemia (due to acute blood loss, preoperative dehydration, etc), pain/surgical stimulus, and medication administration including anticholinergics and chronotropes. If the patient is unstable due to a tachycardic rhythm, they should be cardioverted. If the patient is stable (i.e. adequate perfusion pressure and no signs/symptoms of instability), the QRS complex and rhythm should

be assessed as to whether it is narrow or wide, regular or irregular, as this will direct treatment. If the QRS is narrow complex it should be treated with adenosine, amiodarone, diltiazem, or beta blockade, unless Wolff-Parkinson-White is present. If the rhythm is wide complex, this should be treated with amiodarone and/or magnesium (depending on baseline QT), and the patient should be monitored closely for conversion to normal sinus rhythm or hemodynamic deterioration.

While the ultimate end point of ACLS is the same in the operating room as on the general care ward or out of hospital (administration of medications and energy to regain normal sinus rhythm and normotension), there are some causes of pulseless arrest that are unique to anesthesia. It is important to discuss these because they are known complications that occur perioperatively that should be immediately identified and managed. The first perioperative specific complication is cardiac arrest due to neuraxial anesthesia. Many procedures including orthopedic, urologic, gynecologic, and obstetric procedures are done under either spinal or epidural anesthesia. Approximately 2 out of 10,000 patients who receive neuraxial anesthesia will suffer a cardiac arrest and this often manifests as a high spinal. When this happens, local anesthetic blocks the cardiac accelerator fibers that emerge from spinal levels T1–4 causing bradycardia. The block may affect even higher cervical spinal cord levels, which results in the patient having respiratory and neurologic collapse. Another cause of pulseless arrest in regards to neuraxial anesthesia is unintentional intravascular injection of local anesthetic. This most often occurs with bupivicaine, and symptoms include tinnitus, metallic taste, hypotension and seizures. Should a patient suffer a cardiac arrest that you suspect is due to neuraxial anesthesia, in addition to standard ACLS, you should consider rapid escalation of epinephrine doses as well as treatment with vasopressin. If the patient has suffered an arrest due to local anesthetic systemic toxicity, then a lipid emulsion (10–20 %) should be started immediately with adherence to published guidelines, which require reduction of epinephrine doses and avoidance of vasopressin [12].

Gas embolus is another unique event that occurs in the operative setting that is capable of causing hemodynamic instability. A CO_2 embolus may occur with pneumoperitoneum during a laparoscopic surgery and a venous air embolus may occur when the surgical field is above the right atrium, most commonly during a craniotomy. Treatment should include flooding the surgical field with a crystalloid solution, administering 100 % oxygen, placing the patient in a position where the surgical field is below the heart in order to stop

the entrainment of air. If this occurs during a laparoscopic case, insufflation should be discontinued immediately. Additionally, some reports suggest that placing the patient in the left lateral decubitus position may help trap air in the right ventricle. The patient should be given inotropic and chronotropic support as needed. Two other important perioperative causes of perioperative arrest, anaphylaxis and malignant hyperthermia, will be discussed later in this chapter.

Hypotension

Blood pressure consists of systolic, diastolic, mean arterial pressure, and pulse pressure, with normal being 120/80. Many factors can cause patients in the operating room to develop hypotension, especially during induction of anesthesia. Some of the most common causes of hypotension include vasodilation due to medications that have been given (including intravenous and volatile agents), hypovolemia, and cardiogenic causes.

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. Another notable cause of hypotension is preoperative continuation of angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARB). Many experts disagree about whether or not these should be continued in the perioperative period. Both classes of medications cause pronounced hypotension in the OR, which is often refractory to standard treatment.

Hypovolemia is also a common cause of hypotension during anesthesia. The American Society of Anesthesiology guidelines on preoperative fasting state that patients should be NPO for 8 h prior to surgery. This often results in mild dehydration and hypovolemia. Thus, the resultant hypovolemia may cause hypotension, which may be even more pronounced after induction with medications that cause vasodilation. Additionally, the geriatric population often take diuretic medications in order to control hypertension and heart failure. If these medications are not held prior to surgery they can lead to a hypovolemic state. Finally, hypovolemia may be due to acute blood loss from the surgical procedure. While rapid exsanguination is normally obvious, it is important to routinely assess the suction canisters as well as soaked laparotomy sponges in order to account for ongoing blood loss throughout an operation.

Hypotension after induction is usually treated with IV fluids and small doses of vasopressors, usually phenylephrine (100 mcg increments) and ephedrine (5 mg increments). Other temporary treatments including temporary lightening of the anesthetic depth and placing the patient in Trendelenburg position to increase venous return (i.e. preload) to the heart. If severe hypotension persists, it may be necessary to use stronger alpha and beta agonists including epinephrine, norepinephrine, vasopressin, and dopamine. In patients who took an ACE inhibitors or ARBs on the day of surgery, you can consider small doses of vasopressin, 0.5–1 unit to temporarily improve blood pressure, but a vasopressin drip is often required, as the half-life of ACE inhibitors is approximately 12 h and that of ARBs is roughly 24 h. Finally, other causes of hypotension should be considered, including incorrect placement of the blood pressure cuff, error in medication administration, heart failure, anaphylaxis, and sepsis. If initial measures at resuscitation are not adequate, a transesophageal echocardiogram may be of great benefit in guiding fluid and vasoactive drug therapy.

It is important to remember that one should aim to keep patients intraoperative blood pressure within 15–20 % of their baseline, as this is important for end organ perfusion. Recent studies have shown that intraoperative hypotension, with a greater than 40 % decrease in mean arterial pressure, can be associated with increased perioperative and postoperative morbidity and mortality. As a fixed cutoff point, one recent retrospective trial involving over 30,000 patients demonstrated a strong association between the intraoperative duration below a mean arterial of pressure of 55 mmHg and postoperative acute kidney injury, myocardial injury, and all-cause cardiovascular complications [15].

Hypertension

Hypertension is also a commonly encountered problem during anesthesia; however, brief episodes of hypertension are rarely harmful. The most common causes of intraoperative hypertension include pain, light anesthesia, and baseline hypertension. If pain is suspected, an analgesic can be administered or the level of anesthesia deepened with volatile anesthesia or propofol. Analgesic options include both opioids (fentanyl, hydromorphone, morphine, etc) as well as non-opioids such as ketorolac (NSAID) and ketamine (NMDA antagonist).

If hypertension persists after pain and light anesthesia have been ruled out, treatment with an anti-hypertensive should be considered. Other common etiologies that should be considered include incorrect placement or sizing of

the blood pressure cuff (e.g. a cuff too small for the arm), hypercarbia, hyperthermia, failure to take anti-hypertensives, bladder distension, and tourniquet pain. It is advised to treat hypertension of this sort with an anti-hypertensive rather than using the side effects of intravenous or volatile anesthetics. Common intraoperative medications for treatment of hypertension include beta-blockers (esmolol and labetolol), calcium channel blockers (nicardipine), and vasodilators (nitroglycerin, nitroprusside, and hydralazine).

Myocardial Ischemia/Infarction

Myocardial oxygenation is a balance between oxygen demand and oxygen supply. The key determinants of demand are heart rate, contractility, and wall tension, whereas the key determinants of supply include coronary perfusion, hemoglobin, and dissolved oxygen in the blood. In a normal heart, there is a wide range of hemodynamic parameters at which the heart can still function adequately. However, with an increasingly elderly, obese, and diabetic population, there is a large part of the population with occult coronary artery disease who are susceptible to new ST segment changes consistent with a myocardial ischemia or infarction

If you suspect that the patient is having myocardial ischemia while in the operative room, you should alert the surgeon and discuss your concerns and how to proceed based upon the stage of the operation. You should aim to optimize myocardial supply and demand, with a goal of maintaining a normal blood pressure, especially the diastolic pressure, as this is required for coronary perfusion. A heart rate under 65 bpm is ideal as long as perfusion appears normal. Beta-blockers are a first line therapy for this. You can consider administering nitroglycerin if the systolic blood pressure tolerates this, as it will cause coronary vasodilation. However, care should be taken in the setting of inferior or right-sided ischemia or infarction. Anemia, hypothermia, and shivering should be corrected and the administration of antiplatelet therapy and systemic heparinization should be considered, but these latter measures should be discussed with the surgeon and cardiologist prior to initiation.

Respiratory Disturbances

The pulmonary system consists of the rib cage and muscles of respiration, the tracheobronchial tree, alveoli, and the pulmonary capillaries, which together, are responsible for gas exchange (both oxygen and carbon dioxide). All components must function in harmony to provide for adequate oxygenation and

ventilation. During normal breathing, inspiration requires energy and utilizes diaphragmatic and intercostal muscle activation and expiration is due to passive recoil. Induction of general anesthesia increased pulmonary dead space and decreases functional residual capacity. These changes are usually tolerated relatively well in patients with healthy lungs but even in healthy patients, there are many common respiratory complications that can occur.

Hypoxemia

Normal PaO_2 on room air (21 % O_2) is between 85 and 100, and is a little lower with increasing age. On supplemental oxygen, such as during general anesthesia, the PaO_2 should be greater than 200. If sudden hypoxemia occurs during an anesthetic, one must first determine the severity of the hypoxemia. Severe hypoxemia may be defined as an SpO_2 of less than 90 %, which corresponds to a PaO_2 of under 60 mmHg. An arterial blood gas sample is more sensitive at revealing changes in oxygenation that is an oximeter. This is due to the physiologic characteristics of hemoglobin described by the hemoglobin-oxygen dissociation curve (see Fig. 17.2). Note that at the right side of the curve, the additional PaO_2 from 70 to 100 mmHg and beyond (which represents a significant increase in concentration in the administered O_2) only increases the saturation of hemoglobin slightly. In clinical practice, this means that a patient may have a significant alveolar to arterial oxygen gradient for that is not detected by oximetry alone. Therefore, time is of the essence when responding to hypoxemia as detected by desaturation on an oximeter, as 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.

Common causes of acute hypoxia in the operative room include shunt (e.g. mucus plug, endobronchial intubation, ARDS), diffusion defects (e.g. pulmonary edema), alveolar hypoventilation (e.g. asthma, bronchospasm or neuromuscular disease), significant dead space (e.g. high airway pressures), and decreased oxygen carrying capacity of hemoglobin (e.g. hypothermia or carbon monoxide poisoning). Initial treatment for all of these includes administration of FiO_2 1.0, bag-mask ventilation to assess lung compliance and chest excursion, assessment of ventilator disconnection or kinking in the tube and circuit, assessment of $ETCO_2$ level and the capnograph, auscultation of the chest, suctioning the endotracheal tube, and considering bronchoscopy to assess for an endobronchial pathology (e.g. mucus plug, tube position, etc.).

An additional cause of a low oximetry reading should be noted; namely, administration of intravenous dye used to identify structures during surgery (i.e. indocyanine or methylene blue) may cause an acute drop in oxygen saturation, that is not actual hypoxemia, but the pulse oximeter misreading the dye as deoxygenated hemoglobin. This usually resolves within 30–60 s and is typically not a cause for significant concern.

Hypocarbia

Hypocarbia is a reflection of decreased carbon dioxide (CO_2) levels as measured by either end-tidal monitoring or a blood gas. It can be due to either increased CO_2 elimination or decreased CO_2 production. By far the most common cause is hyperventilation, with decreased metabolic rate (e.g. hypothermia and hypothyroidism), hypovolemia, pulmonary embolism, cardiac arrest, endotracheal tube dislodgement, circuit disconnect, or a disconnected CO_2 sampling line also being possible.

The first step in investigating causes of hypocarbia is to check the breathing circuit in order to rule out any loose connections or other mechanical problems. The next step should be to examine the patient's blood pressure, heart rate, and SpO_2 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, with average settings being a tidal volume of ~6 mL/kg and a rate of 12–16 bpm. Although hyperventilation is the most common cause, other causes of hypocarbia signal significant perturbations, and thus should be addressed first as listed above.

Hypercarbia

Hypercarbia, as measured by end-tidal CO_2 or blood gas analysis, is a common occurrence during general anesthesia. The normal $EtCO_2$ value is 38–42 mmHg. Hypercarbia may result from either increased CO_2 production or decreased CO_2 elimination. Causes of increased CO_2 production include fever, or a hypermetabolic state such as burns, malignant hyperthermia, shivering, and thyrotoxicosis. Causes of decreased CO_2 elimination from the body include hypoventilation, airway obstruction, atelectasis, residual effects of paralytics or opioids, endobronchial intubation, and an exhausted CO_2 absorber. When thinking about the potential causes of hypercarbia, it is often useful to consider when the hypercarbia is occurring. Elevations in CO_2 at the beginning

of a case are more likely to be from improper ETT placement, oversedation, or inadequate ventilator settings, whereas elevated CO_2 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 CO_2 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.

Elevated Peak Airway Pressures

The airway pressures measured during general anesthesia with positive pressure ventilation should ideally be less than 40 cmH_2O. They are lower with spontaneous ventilation than with controlled ventilation. Common causes of elevated peak airway pressures include bronchospasm, endobronchial intubation, a kinked tube or circuit, the patient biting on the tube, light anesthesia, or the surgical manipulation, such as the team on the chest during head and neck surgery or placement of abdominal retractors during an exploratory laparotomy. To address this problem, you should first assess the plane of anesthesia and rule out light anesthesia. You should also listen to the patient's lungs. If you auscultate wheezes, bronchospasm might be the etiology of elevated peak pressures and this can be treated with deepening anesthesia (with propofol or volatile anesthetic), albuterol, or epinephrine. Additionally, diminished breath sounds on one side could signal endobronchial intubation or pneumothorax, which would required repositioning of the endotracheal tube or needle thoracostomy, respectively. Finally, closely examining the circuit and following it from the anesthesia machine to the patient can identify the other causes of elevated peak pressures, including a kinked tube or circuit.

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, pneumonia, acute respiratory distress syndrome, and possibly 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 making sure patients are NPO for 8 h, but if this isn't possible, either using regional anesthesia to avoid putting a patient to sleep, or performing a rapid sequence intubation, without bag-mask ventilation, for general anesthesia are good options. Other measures include administration of a non-particulate acid within 30 min of induction (sodium citrate), an H2 blocker, and metoclopramide to increase gastrointestinal motility and increase lower esophageal sphincter tone. Additionally, you should place a nasogastric or an orogastric tube after induction of anesthesia if you suspect the patient has a full stomach. If you suspect a patient has aspirated, you should place them in head-down position to prevent aspirate from entering the lungs and administer 100 % oxygen. Steroids are contraindicated for gastric acid aspiration and bronchoscopy should only be performed if there are large particles that need to be removed from the lungs, otherwise bronchoscopy with lavage should be avoided.

Metabolic Disturbances

In patients who are not anesthetized, the hypothalamus is responsible for regulating core body temperatures within a tight range. If body temperature becomes elevated, the hypothalamic reflexes induce sweating and vasodilation whereas, at low temperatures, vasoconstriction and shivering occur. Under general anesthesia, these normal mechanisms are ablated because anesthetic agents inhibit central thermoregulation. Thus, both hypothermia and hyperthermia are common problems encountered in the operating room.

Hypothermia

A temperature less than 36 °C defines hypothermia, which is a common occurrence intraoperatively. Cold ambient room temperatures, extremes of age (i.e. poor thermoregulation), large abdominal surgeries, and long procedures are all responsible for hypothermia. Without attempts to actively warm the patient, their temperature will usually decrease 1–2°°C during the first hour of anesthesia and then continue to gradually decline for another hour before stabilizing. Severe hypothermia can cause ventricular fibrillation and cardiac arrest. While hypothermia does reduce cerebral metabolic rate, and is commonly induced during cardiac procedures with cardiopulmonary bypass, it also causes multiple negative physiologic effects. These include impaired renal function, impaired wound healing, increased risk of wound infection, decreased activity of drugs, prolongation of neuromuscular blockade and shivering post-operatively, which

can increase metabolism and oxygen consumption fourfold. Strategies to warm patients include warming the operating room, using convective forced-air warming (Bair hugger), warming fluids, and using low gas flows.

Hyperthermia

While hypothermia is commonly encountered in the operating room, hyperthermia is a more rare occurrence, though it can have deleterious consequences if unrecognized and untreated. A temperature greater than 38 °C defines hyperthermia. Causes include overwarming, malignant hyperthermia, sepsis, neuroleptic malignant syndrome, and a febrile transfusion reaction (discussed later in this chapter). Overwarming can occur during a long case where the patient is covered and forced air is being used. You should be cognizant of this cause and begin to cool the patient if they become febrile. Other treatments should include turning off fluid warmers and setting the convective forced-air warmed to blow ambient air, which effectively cools the patient.

Malignant hyperthermia is a very rare but extremely serious and even fatal complication of general anesthesia. It results from exposure to either a volatile agent or succinylcholine, which causes extreme calcium release from the sarcoplasmic reticulum due to a dysfunctional ryanodine receptor. MH is genetically inherited in an autosomal dominant fashion with variable expressivity and penetrance, and can occur with either the first exposure to a triggering agent, or during subsequent exposures. Findings include muscular rigidity, tachycardia, increased CO_2 production with increase $ETCO_2$ noticed during controlled ventilation, and increased temperature. Treatment includes prompt cessation of all triggering agents with conversion to a total intravenous anesthetic and administration of Dantrolene IV. Neuroleptic malignant syndrome manifests with similar clinical findings to malignant hyperthermia but is caused by dopamine deficiency, which can develops after administration of anti-psychotic drugs.

Toxins

The immune system is a delicate balance of cells whose purpose is to protect the body from foreign pathogens. Classically, there are four types of hypersensitivity reactions, with Type I being an antigen-antibody (IgE) cross-linkage that results in a large release of inflammatory mediators from mast cells. Anaphylaxis is a Type I hypersensitivity. Also included in Type I are atopic reactions, urticarial reactions, and angioedema.

Immune Reactions

Anaphylaxis, a Type I mediated reaction, results when your body is exposed to an antigen to which is has already been sensitized/exposed. Once triggered, mast cells release histamine, leukotrienes, and kallikreins, which cause increased vascular permeability, bronchial smooth muscle contraction, and vasodilation. The result is extreme hypotension, circulatory collapse, and possibly death. In the perioperative setting, the most common cause of anaphylaxis is neuromuscular blockers, both depolarizers (succinylcholine) and non-depolarizers. Other causes include latex and antibiotics.

Anaphylactoid reactions are similar, and often indistinguishable from anaphylactic reactions, but they are not IgE mediated. A drug may cause either cause direct histamine release from mast cells or it can cause activation of the compliment cascade. Regardless of if the reaction is thought to be anaphylactic or anaphylactoid, the treatment is the same. First, discontinue the suspected agent and make sure the patient is receiving 100 % oxygen. Next, assess the cardiopulmonary state of the patient. If wheezes are present, administer albuterol immediately and consider whether low-dose epinephrine is needed. If cardiac instability is present, administer epinephrine 10–25 mcg IV with increasing doses every 30–60 s until the patient is stabilized, noting that an infusion is likely needed due to the bimodal nature of most severe reactions. If the patient is not intubated, you should assess their airway and determine if they need invasive ventilatory support. After initial stabilization is underway, it is also recommended to administer hydrocortisone, an H1-blocker (e.g. diphenhydramine), and an H2-blocker (e.g. ranitidine or famotidine). Finally, continue to provide circulatory support with IV fluids, vasopressors, especially epinephrine, and proceed with ACLS if necessary.

Transfusion Reactions

When patients need blood products in the operating room, they should receive blood that is typed and cross-matched (with the exception of trauma patients who may receive type O, if their type is unknown). The risk of serious reactions from blood transfusion is very rare as our system has become much safer, with many checks and balances, but both major and minor reactions to blood can still occur.

As mentioned above, the most severe reaction is ABO incompatibility, which results in an acute hemolytic reaction. Administration of incorrectly typed blood is usually due to a misidentification of the patient, their blood

type, or the unit transfused. The risk of a fatal hemolytic transfusion reaction is about 1 in every 100,000 transfusions administered. Common signs of an acute hemolytic reaction in an awake patient include chills, fever, nausea, and chest pain. If the patient is anesthetized, signs may include temperature elevation, circulatory instability (hypotension, tachycardia), hemoglobin in the urine, and problems with coagulation (often DIC may develop). Management includes immediately stopping the unit being infused, sending both the patients blood and the unit of blood back to the lab for further testing, diuresing the patient, and providing circulatory support.

Transfusion related acute lung injury (TRALI) presents within 6 h of transfusion and is the most common cause of mortality related to administration of blood products. The patient will become acutely hypoxemic and develop non-cardiogenic pulmonary edema. It most often occurs after administration of FFP, platelets, or after massive transfusion where large volumes of blood products were given. These patients usually require prolonged mechanical ventilation and supportive care. No definitive treatment is available for TRALI, but with supportive care the patient's oxygenation often improves and return to normal in 2–3 days.

Finally, febrile transfusion reactions are non-hemolytic and usually minor. They are allergic reactions and often due to either white cell or platelet sensitization. Patients are often pre-medicated with diphenhydramine and acetaminophen in the non-operative setting in order to avoid concerns. If a patient exhibits signs of significant allergy, the transfusion should be stopped immediately and they

Post Operative Complications

The neurologic system is the one system not discussed above in which there can be perioperative complications. Most of these are not able to be noticed intraoperatively due to the inability to assess the neurologic status of a patient under general anesthesia. Stroke, blindness, corneal abrasions, and neuropathies due to positioning are all possible complications.

The most common of these complications, corneal abrasions occur during intubation, before the eyes are taped, or during emergence, when patients tend to scratch their eyes. These injuries are usually mild, are not recognized until the patient is in the recovery room, and usually heal within several days. Treatment includes the use of eye lubricant or antibiotic eye drops.

Neuropathies are also commonly encountered post-operatively. Positioning during general anesthesia is extremely important and vigilance with positioning can help avoid many neuropathies. The most common neuropathy encountered is ulnar, which usually happens when the arm is pronated with compression of the ulnar nerve within the cubital tunnel. Common peroneal neuropathy is the most common lower extremity neuropathy, and occurs from compression of the nerve against the fibular head during lithotomy position. There is little evidence that nerve injury 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 hyper-extend the elbow and to be cognizant of some patients' flexion deformities, which may make it impossible to fully extend some joints.

Postoperative visual loss (POVL) and stroke are rare but devastating complications. POVL is associated with specific patient risk factors such as lengthy surgery, prone position, anemia, edema of the 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 documented.

Stroke is also a devastating complication in the operative setting. Should a patient not proceed through emergence from anesthesia as expected, this diagnosis should be considered. When considered, other common causes of delayed emergence should be ruled out, including residual neuromuscular blockade, hypoglycemia, hypoxemia, and hypercarbia. If stroke is still being considered, a neurologic consult should be requested immediately, a non-contrasted CT scan should be ordered STAT, and the surgical team should be notified of the concern.

Emergence and Extubation

Emergence is the period of time where the patient begins to awaken from anesthesia. At this point in the case, neuromuscular blockade should be reversed, the volatile or intravenous anesthetic turned off, and the patient should begin to have the return of spontaneous breathing. Most importantly, the anesthesia

provider must decide if it is safe to extubate the patient's trachea, which should be based upon the confirmation of proper **oxygenation**, **ventilation**, **mechanics and cognition/airway control**.

To evaluate the **oxygenation** status of a patient, one should first examine the pulse oxymetry reading, with a reading >95 % is desirable. If there is a concern in regards to oxygenation, several steps can be undertaken. First, although it is commonplace to extubate with a high fraction inspired oxygenation concentration (FiO_2), you can reduce the FiO_2 to 21 % (room air) in order to examine the oxygen saturation at that state. During the reduction in FiO_2, if the pulse oximeter reading drops to 90 %, you should note the FiO_2 at which this happens, because at this hemoglobin saturation level the PaO_2 is 60 mmhHg. As such, an easy calculation of P/F ratio (P_aO_2/FiO_2) or A-a gradient can be performed. If this is not possible, an arterial blood gas can be obtained. A P/F ratio of <300 indicates acute lung injury (ALI) and a P/F ration of <200 is suggestive of acute respiratory distress syndrome (ARDS). If the P/F ratio is <300 after attempting recruitment maneuvers and ensuring that the endotracheal tube is in the proper location (e.g. not a mainstem intubation), then the patient should remain intubated until proper oxygenation status has been restored and the cause of the poor oxygenation status has been confirmed. Extubation of a patient with poor oxygenation places them at risk of respiratory failure and hypoxemia that will require reintubation. However, in most patients, appropriate oxygenation can be confirmed and the next parameter can be assessed.

To evaluate the **ventilation** status of a patient, one should first examine the end tidal CO_2 (ET_{CO2}). Once spontaneous ventilation has returned, you should ensure that a patient is able to maintain a stable ET_{CO2} in a normal range (<55 mmHg) prior to extubation. Moderate to severe hypoventilation, ET_{CO2} >60 mmHg, is an indication that the patient cannot adequately support their own ventilation and that extubation is likely not safe (unless the patient has a confirmed, chronic high $PaCO_2$). Furthermore, if there is concern for hypercarbia, an ABG can be obtained in order to assess whether the problem is acute or chornic. A pH <7.25 indicates significant respiratory acidosis in the setting of elevated $PaCO_2$ and is further evidence that the patient does not meet extubation criteria. If this condition is present, the patient should remain intubated until the cause of poor ventilation status (e.g. residual NM blockade, bronchoconstriction/increased work of breathing, etc) has been identified.

Extubation of a patient with poor ventilation places them at risk of respiratory failure and worsening academia that will require reintubation. However, in most patients, appropriate ventilation can be confirmed and the next parameter can be assessed.

To evaluate the **respiratory mechanics** of a patient, one should examine the respiratory rate, tidal volume, work of breathing, and strength of the patient. This component of the patient assessment is intimately linked to oxygenation and ventilation, but is not fully contained therein. During emergence, patients should be taking adequate tidal volumes (normal tidal volumes are approximately 4–6 mL/kg) and have a regular respiratory rate, which in an adult patient is normally >8 and <25 breaths per minute. Although there is controversy over the best test of strength other than respiratory mechanics, the ability to maintain sustained head lift (>5 s) and sustained tetany without fade on peripheral electrical nerve stimulation are commonly used.

Finally, evaluation of the **status of cognition and airway control** is extremely important, with the major goal in this area being to assess whether a patient can control their secretions and protect their airway if the endotracheal tube is removed. This is easy to confirm by asking them to follow basic commands such as squeezing your hand, wiggling their toes, and giving you a thumbs up. However, not all patients will be able to perform these responsive motor tasks, such as a patient with cerebral palsy. In these cases, assessing bulbar reflexes such as the gag reflex, strong coughing, and observation of the patient attempting to swallow, can be used as evidence that they possess adequate airway control to extubate the trachea.

Thus, extubating a patient is not simply what happens during emergence form anesthesia. Rather, there are a discrete set of parameters that should be assessed concerning respiratory function from gas exchange (oxygenation/ventilation) to mechanics to airway control that should be assessed. If patient fails the above assessment, or if a patient has respiratory compromise after extubation and requires re-intubation, it is helpful to think about the differential diagnosis in a stepwise fashion that follows the criteria for extubation.

Hypoxemia: If the patient is hypoxemic, reviewing the five causes of hypoxemia are critical (V/Q mismatch, shunt, low FiO_2, hypoventilation, and diffusion defects), with shunt due to atelectasis being common after general

anesthesia, as is hypoventilation due to residual neuromuscular blockade, overdose of opioids (slow, large breaths), and pain with respiration (rapid, shallow breaths). Other causes, such as wheezing (bronchoconstriction), stridor (vocal cord dysfunction), mucus plugging, pulmonary edema, and pneumothorax, should also be considered as the patient's overall history and anesthetic course is reviewed.

Ventilation (**Hypercarbia**): Hypoventilation with subsequent hypercarbia is also a common cause of failure of extubation and is usually due to an overdose of narcotics, which may present without hypoxemia if a high FiO_2 is being used. The most common cause of this in the postoperative period is narcotic overdose. Opioids, benzodiazepines, and residual anesthetics reduce the respiratory drive and make patients less responsive to elevations in CO_2, causing a reduction in minute ventilation, with opioids causing deep, slow breathing accompanied by pupillary constriction.

Mechanics: Failure to extubate due most often relates to either insufficient reversal of neuromuscular blockade or no reversal at all. This is a common cause of reintubation in the PACU. Additionally, pain can fall under mechanics as well, for the reasons mentioned above.

Cognitive and Control: Airway control is an important aspect of failure to extubate, most often due to decreased consciousness, which is often seen with narcotics overdose and residual anesthesia. However, with an aging population, consideration of emergence delirium, stroke, and baseline cognitive state should be undertaken (e.g. severe dementia). Additionally, in a population that is increasingly overweight, post-extubation respiratory failure due to airway collapse is not uncommon in the obese patient who has, or is at risk for, obstructive sleep apnea. Finally, the type of surgery should be considered, as loss of airway control after thyroid surgery could be due to neck hematoma, acute hypocalcemia, or hoarseness/stridor due to damage to one or both of the recurrent laryngeal nerves.

A thorough assessment of the patient's medical history, the anesthetic course, the surgery, and the presenting signs and symptoms should be undertaken in order to fully construct and consider the differential diagnosis of failure to extubate or need for re-intubation in the immediate perioperative period.

Case Study

Now that you have a thorough understanding of common problems encountered during anesthesia, it's time to test your knowledge. The algorithm below is a helpful was to think through each problem encountered and can be used as a framework to evaluate the case discussed below.

WOULD LIKE TO INSERT DR. MCEVOYS CRITICAL THINKING ALGORITHM HERE

A 52-year-old male is undergoing proctocolectomy for rectal cancer. He was admitted this morning for the operation after undergoing a bowel prep at home the day before. Vital signs were as follows: BP 130/84, HR 80, RR 14, O2 98 % on room air. He does not take any medications. Anesthesia was induced with propofol 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 (propofol) 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, 100–200 mcg.

The differential diagnosis also includes rarer but serious causes, including anaphylaxis from the antibiotic cefazaolin or the neuromuscular blocking drug, vecuronium, or pneumothorax from central line placement. One should also rule of artifact by comparing the tracing on the arterial line to the 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 opioid would be prudent.

The patient's hemodynamic status has stabilized and the case is proceeding. 15 min 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 FiO_2. You can then check for adequacy of ventilation by observing the CO_2 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 airway 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 FiO_2, 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. You should be cognizant that inhaled beta-agonists will also cause tachycardia, as some patients might not tolerate an increased heart rate.

Wheezing resolved 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 their 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.

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Part V

Systems Physiology and Anesthetic Subspecialties

Chapter 18

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.

J.M. Ehrenfeld et al. (eds.), *Anesthesia Student Survival Guide*,
DOI 10.1007/978-3-319-11083-7_18

Table 18.1 Coronary artery supply

Source	Branch #1	Branch #2	Supply
Left main	Circumflex	Obtuse marginal branches	Left ventricle lateral & posterior walls
	Left anterior descending (LAD)	Septal branches	Majority of interventricular septum
		Diagonal branches	Left ventricular surface, especially anterior wall
Right main	Acute marginal branches		Right ventricle
	AV nodal & SA nodal		AV & SA nodes
	Posterior descending		Inferior & posterior wall of left ventricle, right ventricle

There are 2 **main coronary arteries** that serve as blood supply to the heart (see Table 18.1). 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 while perfusion to the right coronary artery occurs during both systole and diastole.

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 from the left ventricular cavity also provide a route for venous drainage.

There are 4 heart valves that promote unidirectional flow (see Fig. 18.1). 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.

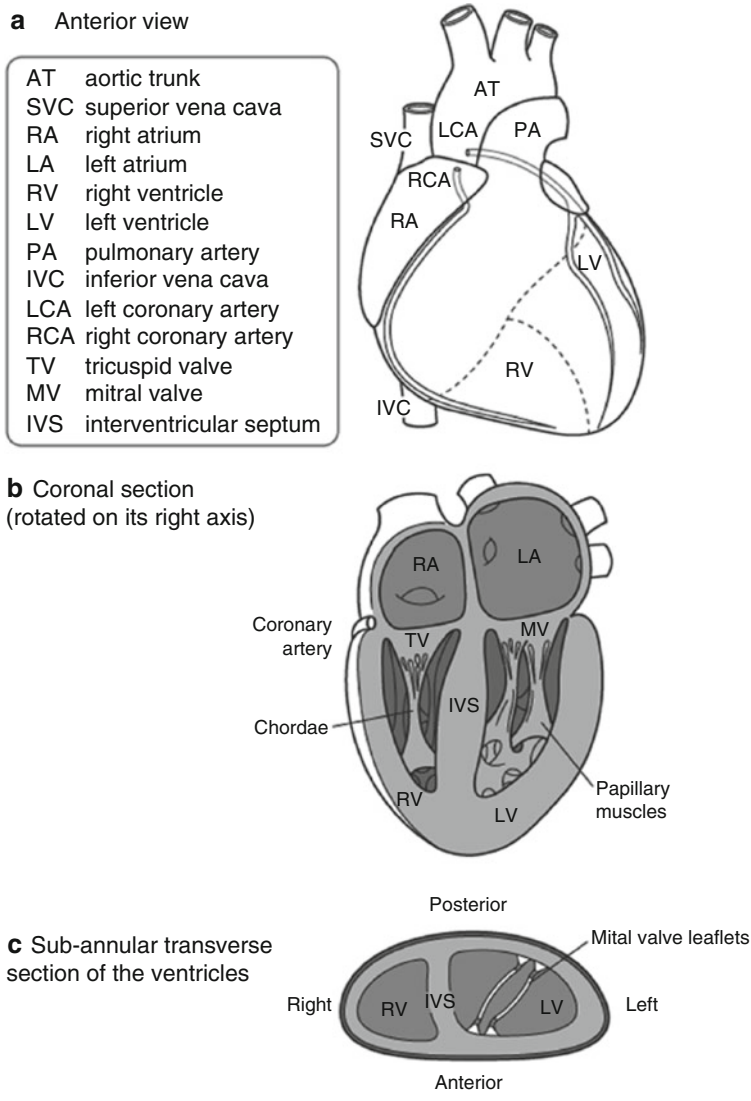

Figure 18.1 Anatomy of the heart (Reproduced with permission from Allen [7])

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 **atrioventicular** (**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**. Sympathetic and parasympathetic nerves innervate the heart. β-adrenergic stimulation increases cyclic AMP levels and enhances Ca^{2+} influx which causes depolarization of conduction cells. Cholinergic signals, via parasympathetics (vagus nerve), oppose β-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. 18.2). **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

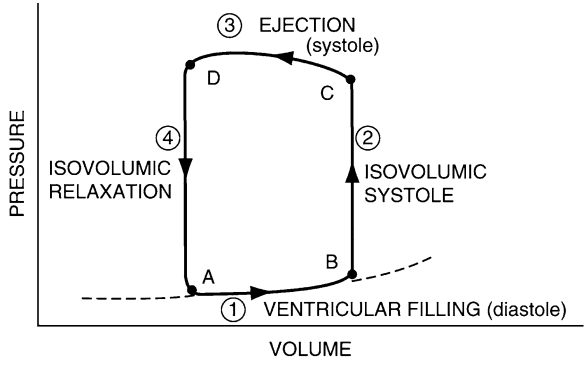

Figure 18.2 Cardiac cycle (From Fung [8]. Used with permission)

Table 18.2 Other cardiac cycle definitions and equations

Cardiac output (CO)	Volume of blood pumped per minute. CO = heart rate × stroke volume
Stroke volume	Amount of blood pumped with each contraction
Preload	Volume of blood in the ventricle before systole. Usually measured as left ventricular end-diastolic pressure (LVEDP), which estimates left ventricular end-diastolic volume (LVEDV)
Afterload	Resistance to ejection of blood by each ventricle
Starling's Law	Contractility depends on muscle fiber length
Coronary perfusion pressure	CPP = Aortic diastolic blood pressure – LVEDP
Left ventricular wall tension	Wall tension = (interventricular pressure × chamber radius)/(thickness × 2)
Fick equation	C.O. = O_2 Consumption/([Arterial $O_{2\ content}$] – [Venous $O_{2\ content}$])

heart sound). **Isovolumetric ventricular contraction** occurs when atrio-ventricular valves are closed but pressure in the ventricles has not yet exceeded 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 18.2.

Common Disease States Affecting the Heart

Ischemic Heart Disease

There are many manifestations of ischemic heart disease (see Table 18.3). Determinants of myocardial perfusion depend on the relationship of supply and demand. Myocardial supply is provided by coronary perfusion pressure, heart rate, PaO_2, and coronary diameter. Myocardial demand parameters are myocardial oxygen consumption, heart rate, left ventricular wall tension, contractility, conduction, and relaxation.

Table 18.3 Ischemic cardiac disease

Disease	Definition
Coronary artery disease	Narrowing of coronary arteries from atherosclerosis
Ischemic heart disease	Myocardial O_2 demand not met by coronary blood flow
Acute coronary syndrome/myocardial ischemia	Term that includes any of the life threatening conditions below which represent acute myocardial ischemia
Angina pectoris	Myocardial ischemia with chest discomfort
Stable angina	Exercise induced chronic angina pectoris
Unstable angina	Myocardial ischemia at rest or with minimal exertion
Variant angina	Discomfort from coronary artery vasospasm
Non-ST-segment elevation myocardial infarction (NSTEMI)	Myocardial ischemia from a partially occlusive coronary thrombus
ST-segment elevation myocardial infarction (STEMI)	Myocardial ischemia from a totally occlusive coronary thrombus

Myocardial revascularization procedures such as percutaneous coronary interventions (i.e. coronary stenting) and coronary artery bypass graft surgery (CABG) are performed to relieve symptoms, or prevent future morbidity and mortality related to myocardial ischemia and infarction. The specific indications of when to use each of these techniques is complex, and the literature is evolving as new information comparing the two modalities becomes available. Coronary revascularization may be indicated for patients symptomatic with persistent anginal episodes, unstable angina, NSTEMI, STEMI, and patients in cardiogenic shock. Indications also include significant stenosis of multiple coronary arteries, significant left anterior descending, left main disease, or left main equivalent disease (left anterior descending and left circumflex artery disease).

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 cm^2 and critical MS is ≤1 cm^2. 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, chordal elongation, perforations in mitral valve leaflets, or flail segments of the mitral valve. Other causes of mitral regurgitation include ischemic heart disease, which can result in left ventricular enlargement with restriction of mitral valve leaflets, mitral annulus abnormalities, or necrosis of papillary muscle structures. Other less common etiologies include endocarditis, rheumatic heart disease, congenital clefts, and hypertrophic cardiomyopathy, which can lead to systolic anterior motion (SAM) of the anterior leaflet of the mitral valve. Acute MR leads to high pulmonary pressures and pulmonary congestion, whereas chronic MR can 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, or a percutaneously placed clip which holds the posterior and anterior leaflets together. *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. AS is a valve area great than 1.5 cm^2, moderate is 1.0–1.5 cm^2, severe is 0.7–0.99 cm^2, and critical is <0.7 cm^2. Treatment is percutaneous balloon valvuloplasty, transcatheter aortic valve replacement (TAVR), or surgical 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 or symptoms of heart failure. 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.*

Arrythmia Management

Cardiovascular Implantable Electronic Devices (CIEDs) include pacemakers that *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 (preferred). 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.

CIEDs that are *Implantable Cardioverter-Defibrillators* (ICDs) are indicated for survival of sudden death episode, sustained ventricular tachycardia (VT), syncope from VT, low ejection fraction, or hypertrophic cardiomyopathy. All current ICDs have a pacemaker function. To avoid unnecessary shocks due to interference by electrocautery, tachytherapies (ICD function) should be turned off with a magnet or programming (preferred). Pacemaker function is maintained even after defibrillation capabilities are turned off with a magnet. 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 usually 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 decreased afterload improving forward cardaic output.* There are many different types of left ventricular assist devices (LVAD) that can be placed surgically or in the cardiac catheterization lab.

Table 18.4 Key features of minimally invasive cardiac procedures

Off-pump coronary artery bypass surgery (OPCAB)	Median sternotomy, but no CPB, no cardioplegic arrest
Minimally invasive direct CABG (keyhole CABG, MICS) or Robotic assisted MIDCAB	Small left anterior transverse thoracotomy, usually for single left internal mammary to LAD. No CPB
Minimally invasive valve replacement/repair or robotic	Smaller incision, may be thoracotomy, uses CPB

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. It is also possible that the surgery will be done without CPB or with a minimally invasive technique which may require lung isolation (Table 18.4)

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 an evaluation of cardiac anatomy such as a CT scan optimized for coronary vessels or a cardiac catheterization performed – all of which will provide an understanding of areas of areas of the myocardium which are at risk for ischemia during the perioperative period.

Monitoring

Monitoring should include a pre-induction arterial line, a central venous line, and sometimes a PA catheter will be placed before or after induction depending on the severity of the patient's disease (See Chap. 11 on Patient Monitoring).

After induction, a transesophageal echocardiography (TEE) probe is frequently 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

After adequate intravenous access is obtained with at least one large bore (16–18 gauge or greater) peripheral IV line, or central venous access, induction is typically performed with a combination of medications such as high dose opiates (fentanyl 5–50 mcg/kg) with either etomidate, or propofol depending on the patient's underlying disease state. Sevoflurane and isoflurane are acceptable, provided that hemodynamics are well controlled. Pancuronium, cisatracurium, rocuronium and vecuronium are good choices for paralytics, although pancuronium may cause tachycardia which is undesirable in coronary artery disease, stenotic valvular disease, and hypertrophic cardiomyopathy. Succinylcholine can be used carefully when indicated often with a precurarizing dose of non-depolarizing neuromuscular junction blocker. Ketamine should generally be avoided, Ketamine can increase the risk of myocardial ischemia, and can cause cardiac dysfunction in patients who are already catecholamine-depleted. Ketamine can be helpful to induce patients in pericardial tamponade. During CPB procedures, nitrous oxide is avoided because of its ability to expand the size of gas emboli that can arise in the bypass pump.

Pre-Bypass Considerations

During sternotomy, deflate the lungs to prevent injury during chest opening using the sternal 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 mmHg 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

Table 18.5 Etiology of ischemia and treatments

Etiology	Treatments
Hypotension	– Vasopressors/inotropes, treatment of underlying problem Phenylephrine
Hypoxia	– Increase the FiO_2, increase PEEP
Tachycardia	– β-blockers, increase depth of anesthesia
Vasospasm	– Nitroglycerin
Anemia	– Transfusion of red blood cells
Thrombosis	– Heparin other anticoagulants as indicated
Hypovolemia	– Fluid administration

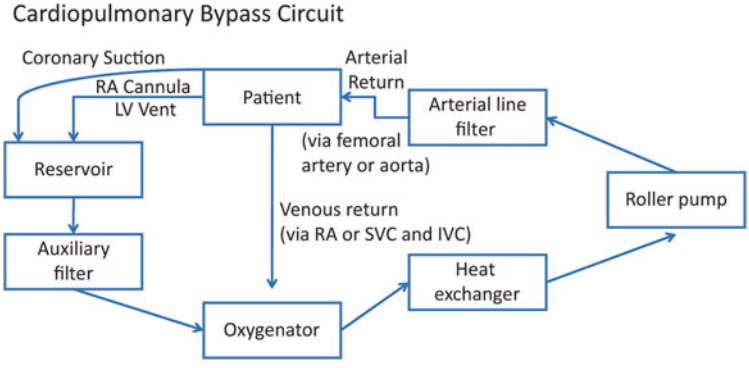

Figure 18.3 Cardiopulmonary bypass circuit (Image courtesy J. Ehrenfeld)

often made based on ECG or echocardiogram findings. Therapy will be guided by the specific mechanism of ischemia as shown in Table 18.5:

Cardiopulmonary Bypass (CPB)

A basic cardiopulmonary bypass circuit (see Fig. 18.3) consists of venous drainage by gravity often 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 physician 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 small IV bolus. Isoflurane 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, myocardial dysfunction, and renal dysfunction.

Weaning From Cardiopulmonary Bypass

The patient's blood is rewarmed 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

Most 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 18.5).

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. 18.4), 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. 18.5). 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

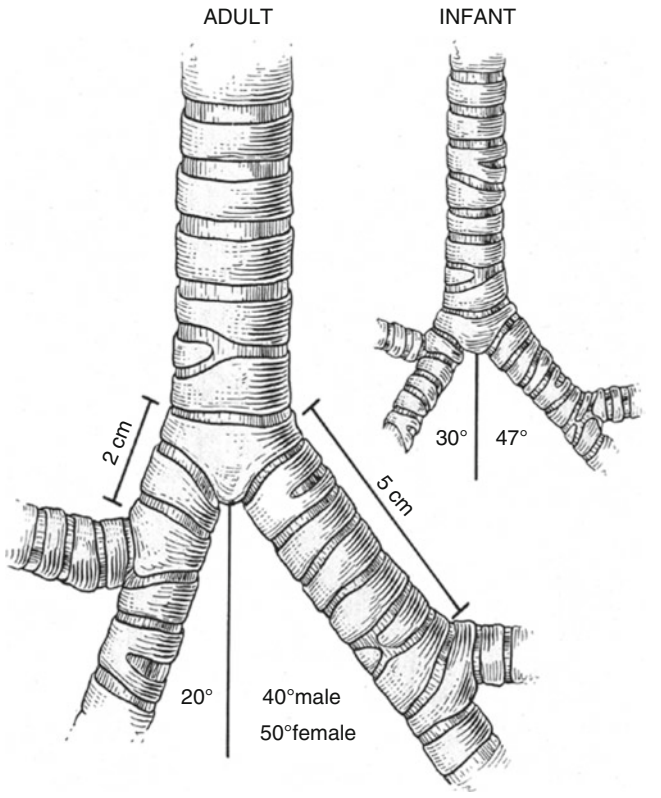

Figure 18.4 Adult and pediatric tracheae and bronchi (With permission from Finucane and Santora [9])

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.

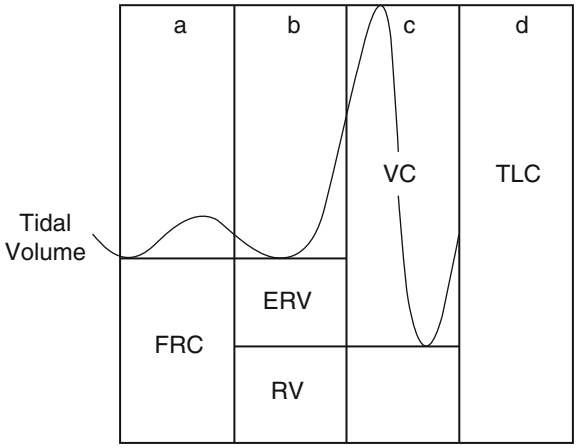

Figure 18.5 Schematic depiction of lung volumes. *VC* vital capacity, *FRC* functional residual capacity, *ERV* expiratory reserve volume, *RV* residual volume, *TLC* total lung capacity (From Bittar [10]. Used with permission)

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 abcess, 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 thoracoscopic 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 vasconstriction 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. 18.6). 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 maintstem 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. 18.4).

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

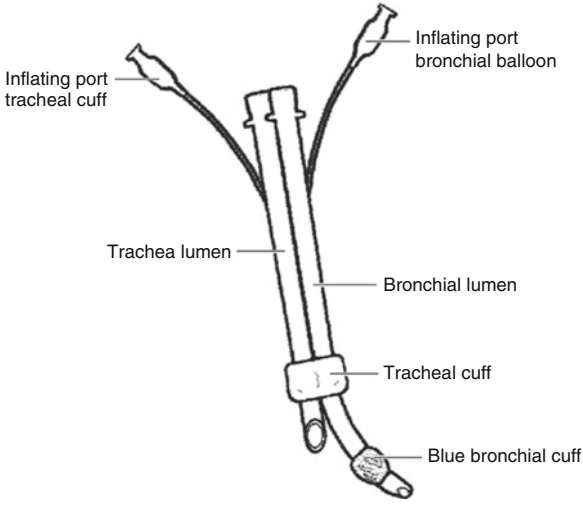

Figure 18.6 Left sided double lumen endotracheal tube (From Criner and D'Alonzo [11]. Used with permission)

cords, the tube is rotated 90°, the stylet removed, and the tube is advanced into the left or right main stem 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 30 s 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 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

1. Ehrenfeld JM, Walsh JL, Sandberg WS (2008) Right and left sided Mallinckrodt double lumen tubes have identical clinical performance. Anesth Analg 106(6):1847–1852

2. Ehrenfeld JM, Mulvoy W, Sandberg WS (2009) Performance comparison of right- and left-sided double-lumen tubes among infrequent users. J Cardiothorac Vasc Anesth 24:598–601

3. Hensley FA, Martin DD, Gravlee GP (2008) A practical approach to cardiac anesthesia. Lippincott Williams & Wilkins, Philadelphia

4. Kaplan Joel A, Reich DL, Lake CL, Konstadt SN (2006) Kaplan's cardiac anesthesia. Saunders Elsevier, Philadelphia

5. Libby P, Bonow RO, Mann DL, Zipes DP (2008) Libby: Braunwald's heart disease: a textbook of cardiovascular medicine, 8th edn. Saunders Elsevier, Philadelphia

6. Searl CP, Ahemd ST (2009) Core topics in thoracic anesthesia. Cambridge University Press, New York

7. Allen DC (2004) Histopathology specimens: clinical, pathological and laboratory aspects. Springer, New York

8. Fung YC (1997) Biomechanics: circulation. Springer, New York

9. Finucane B, Santora A (2003) Principles of airway management, 3rd edn. Springer, New York

10. Bittar EE (2002) Pulmonary biology in health and disease. Springer, New York

11. Criner, D'Alonzo (2002) Critical care study guide: text and review, Springer, New York

Chapter 19

Physiology and Anesthesia for Neurologic, ENT, and Ophthalmologic Surgery

Joshua H. Atkins and Jesse M. Ehrenfeld

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, PaO_2, and $PaCO_2$
- 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 mmHg. Cerebral blood flow (CBF) is a function of mean arterial blood pressure (MAP) and ICP or

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DOI 10.1007/978-3-319-11083-7_19

central venous pressure (CVP), and is defined as **CPP = MAP-ICP** (or CVP, whichever is greater). A cerebral perfusion pressure (CPP) of 55–70 mmHg 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 (↓venous drainage, ↑ 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°
2. Optimization of jugular venous drainage
3. Direct drainage via a lumbar drain or intraventricular catheter
4. Hyperventilation (P_aCO_2 25–30 mmHg) to decrease CBF
5. Osmotic diuresis (mannitol, hypertonic saline)
6. Deep intravenous anesthesia (propofol/barbiturate infusion).

Cerebral Blood Flow

Under normal conditions cerebral blood flow is autoregulated in the range of MAP 50–150 mmHg. 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 19.1 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,

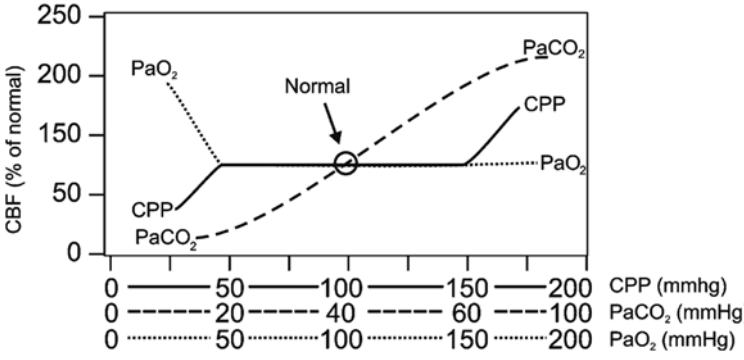

Figure 19.1 Cerebral blood flow/carbon dioxide diagram (Image courtesy J. Ehrenfeld)

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), near infrared monitoring (NIRS), transcranial doppler (TCD), 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 19.1)

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.

Table 19.1 Effects of anesthetic agents on cerebral physiology

Agent	CBF	CMR	EP	Comments
Halogenated potent agents	↑	↓	↓↓	<0.5 MAC generally suitable "luxury perfusion" uncoupling of CBF/CMR relationship
N_2O 60 % (alone)	↑↑	↑	↔	
N_2O + potent agent	↑	↔	↓↓	Effects ↑ with ↑ MAC of the potent agent
N_2O + 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
Dexmedetomidine	↓	↓	↔	Some controvery for MEP, anesthetic sparing + an algesia
Midazolam	↔	↓	↔	May ↓ EPs in >0.2 mg/kg bolus dose

***EP* evoked potential, *CBF* cerebral blood flow, *CMR* cerebral metabolic rate**

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 19.1). 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 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

(1) Neuroprotection
  (a) Optimization of CBF/CMR balance
  (b) Control of ICP
  (c) Temperature regulation (avoid hyperthermia)
(2) Provision of optimal operating conditions, including neuromonitoring and "relaxed" brain
(3) Maintenance of normal glucose and electrolyte balance
(4) Prompt emergence from anesthesia to facilitate neurologic assessment

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. Transient cardiac standstill using IV adenosine is occassionally used after rupture to facilitate surgical exposure and control of bleeding. Blood loss can be substantial and sudden. Hypotension can be problematic during AVM resection due to low flow venous outflow pathway.

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?*

See Haelewyn B, David HN, Rouillon C, Chazalviel L, et al. Neuroprotection by nitrous oxide: facts and evidence. Crit Care Med 2008;36(9):2651–9.

(b) *Are deliberate hypothermia or EEG burst suppression useful methods of neuroprotection during neurovascular surgery or after traumatic brain injury?*

See Baughman VL. Brain protection during neurosurgery. Anesthesiol Clin North America 2002;20(2):315–27.

(c) *Does neuromonitoring in aneurysm surgery reduce complications?*

See Szelényi AM, Langer D, Kothbauer, K, Bueno de Camargo, A, et al. Monitoring of muscle motor evoked potentials during cerebral aneurysm surgery: intraoperative changes and postoperative outcome. J Neurosurg 2006;105(5):675–681 and Neuloh G, Schramm J. Monitoring of motor evoked potentials compared with somatosensory evoked potentials and

microvascular Doppler ultrasonography in cerebral aneurysm surgery. J Neurosurg 2004;100:389–399.

(d) *Is hypertonic saline better than mannitol for ICP management?*

See Diringer MN, Zazulia AR. Osmotic therapy: fact and fiction. Neurocrit Care 2004; 1(2):219–233.

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 19.2.

Table 19.2 Anesthetic considerations in neurologic diseases

Myasthenia gravis (MG)	
Etiology	Autoimmune antibodies against nicotinic cholinergic receptors
Symptoms	Dysphagia, dysarthria, ptosis
Treatment	Anticholinesterases, steroids, plasmapheresis, thymectomy
Preoperative considerations	Assess degree of weakness & duration of symptoms
	Optimize patient prior to surgery; maintain home anticholinesterase therapy
	Consider PFTs, ECG (can see myocardial changes), electrolytes
	Anticholinesterase overdose can lead to ***cholinergic crisis*** –Diagnosis = worsening of symptoms with edrophonium (10 mg) –Treatment = anticholinergic administration (i.e., atropine)
Anesthetic management	Minimize sedatives/respiratory depressants; consider regional
	Consider rapid sequence induction (patients at risk for aspiration)
	Patients at risk for postoperative respiratory failure
	Avoid muscle relaxants if possible
	Use caution when using neostigmine (risk of cholinergic crisis)
Multiple sclerosis	
Etiology	CNS disorder leading to demyelinated nerve plaques
Symptoms	Visual disturbances, limb weakness, paralysis, respiratory failure
Treatment	Steroids, interferon, baclofen, dantrolene
Anesthetic management	Increased risk of aspiration
	Increased risk for postoperative respiratory failure
	Spinal associated with worsening symptoms (epidurals are ***not***)

(continued)

Table 19.2 (continued)

Guillain–Barre syndrome	
Etiology	Acute demyelinating polyneuropathy (often after minor infection)
Symptoms	Limb weakness, decreased reflexes, autonomic instability
Treatment	IVIG, plasmapharesis
Preoperative considerations	Patients may require ventilatory support
	Increased aspiration risk
Anesthetic management	Consider RSI, avoid succinylcholine, minimize muscle relaxants & opioids
Neuroleptic malignant syndrome (NMS)	
Etiology	Derangement of dopaminergic receptors in hypothalamus
	Associated with psychotropic drug use (phenothiazines, butyrophenones)
Symptoms	Hyperthermia, muscle rigidity,
Treatment	Discontinue neuroleptic meds, control temperature, hydrate
	Administer dantrolene, bromocriptine, amantadine
Anesthetic considerations	NMS has a slower onset than malignant hyperthermia (see Appendix 2) and muscle rigidity is a central, not peripheral, effect in NMS
Parkinson's disease	
Etiology	Loss of dopaminergic fibers leads to unopposed acetylcholine activity
Symptoms	Uncontrollable tremors, slow movements, muscle rigidity
Treatment	Levodopa, anticholinergics, antihistamines, MAO inhibitors
Anesthetic management	Patients at high risk of aspiration, consider RSI
	Avoid dopamine and acetylcholine antagonists (droperidol, promethazine, prochlorperazine, metoclopramide, scopolamine, high dose glycopyrrolate)
	Patients may exhibit dysrhythmias and intravascular volume depletion

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**" **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 19.3.

Table 19.3 Perioperative considerations for ENT surgery

General considerations
Higher incidence of a difficult airway
Be prepared for an airway emergency
Airway is often shared with the surgeon: two-way communication is essential
Procedure-specific considerations:
Neck dissection
Lengthy procedure with increased risk of air embolus
Usually performed under general endotracheal anesthesia with controlled ventilation
Brachial plexus nerve monitoring may preclude the use of paralytics
Higher incidence of anesthesia circuit disconnect and significant bleeding
Endoscopic sinus surgery
Short-duration procedure
Pituitary tumors may have associated conditions (acromegaly, diabetes insipidus)
Usually performed under general anesthesia
"Smooth" wakeup to minimize hypertension and bleeding is recommended
Inner ear surgery
Anesthetic techniques include general anesthesia or sedation (stapedectomy)
Generally minimal blood loss and postoperative pain
Facial nerve monitoring precludes the use of paralytics
Higher incidence of postoperative nausea and vomiting
Airway surgery
Higher incidence of a difficult airway (awake intubation sometimes needed)
Increased risk of airway fire during the use of electrocautery
Postoperative intubation and ventilation may be needed

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 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. Post-operative ICU monitoring may be necessary.

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. Use of an air-oxygen blender should be considered if sedation is employed. High-frequency jet ventilation through the surgical laryngoscope can be helpful for surgical exposure. 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

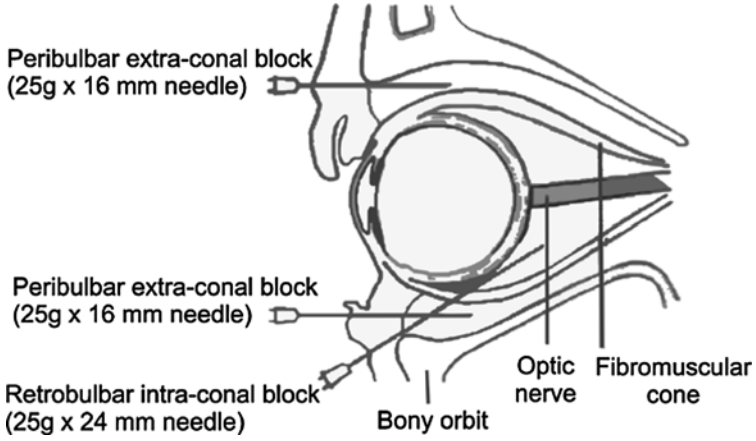

Figure 19.2 Approach to retrobulbar and peribulbar blocks

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. 19.2), 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 ft, 185 lb. BP 185/90, HR 55, SpO296% 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°. Hyperventilation to $PaCO_2 = 30$ mmHg 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, $PaCO_2$ 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 propofol 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. A pre-induction ventriculostomy, placed under local anesthesia, can be considered.

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. The former may increase ICP from hypercapnia or hypoxia. The latter risks aspiration of gastric contents. Again there is no one best answer. If endotracheal intubation can be accomplished quickly with an alternative technique, this is probably reasonable. If not, then ventilation by mask or laryngeal mask airway (second generation LMA provide gastric outlet port) with as low a pressure as possible is prudent.

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

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Chapter 20

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 20.1 summarizes some of the important changes.

J.M. Ehrenfeld et al. (eds.), *Anesthesia Student Survival Guide*,
DOI 10.1007/978-3-319-11083-7_20

Table 20.1 Summary of physiologic changes of pregnancy at term

Total blood volume	Increases
Serum cholinesterase activity	Decreases
Cardiac output and stroke volume	Increases
Minute ventilation	Increases
Functional residual capacity	Decreases
Oxygen consumption	Increases
$PaCO_2$	Decreases
Systemic vascular resistance	Decreases
Blood pressure	Decreases
Hematocrit	Decreases
Serum creatinine	Decreases
Serum albumin	Decreases

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 **20 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 **gestational hypertension** (systolic bp >140 mmHg or diastolic >90 mmHg), **pre-eclampsia** (hypertension + proteinuria), or **eclampsia** (pre-eclampsia + seizures). The definitive 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 (1000 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 deep venous thrombosis, pulmonary emboli, and other thromboembolic events.

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. H_2-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 nonpregnant 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 nonpregnant state.

Uteroplacental 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 α-agonists (phenylephrine) because of supposed increases in uterine vascular resistance. However, more recent human studies have shown that phenylephrine is superior to ephredrine for the treatment of hypotension following neuraxial block for cesarean section, as evidenced by better hemodynamic control and more favorable umbilical cord gases.

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 acidotic 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. Fetal heart rate, though nonspecific, may be a useful surrogate for fetal oxygen delivery. In one meta-analysis, continuous electronic fetal heart rate monitoring reduced the risk neonatal seizures as compared to intermittent monitoring but did not reduce the intrapartum fetal death rate or reduce the risk of fetal neurologic injury. However, the incidence of operative delivery was higher with continuous electronic fetal heart rate monitoring. With either strategy, the baseline fetal heart rate (FHR) should be between 120 and 160 beats per minute (Fig. 20.1). 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. 20.2, 20.3 and 20.4).

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.

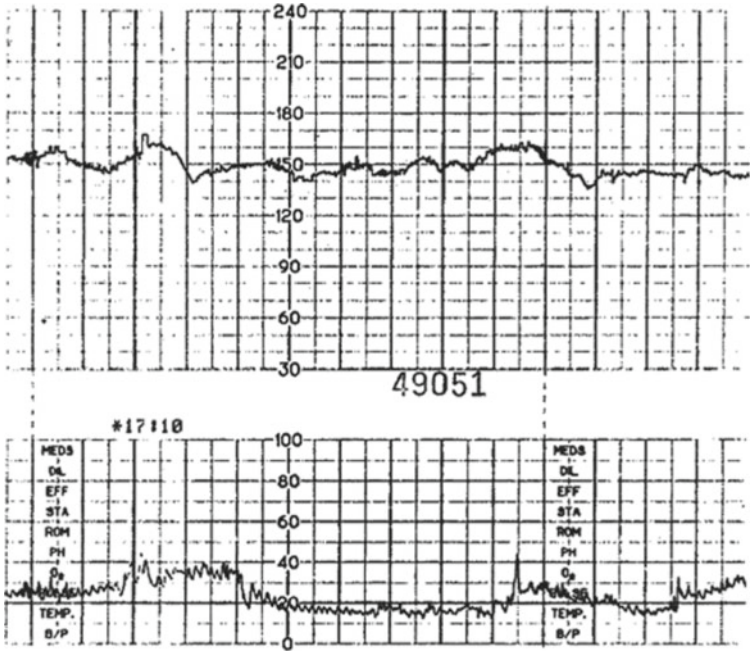

Figure 20.1 Normal fetal heart rate pattern. The heart rate (140 beats/min) variability is normal. There are no periodic changes (From Ref. [13]. Used with permission)

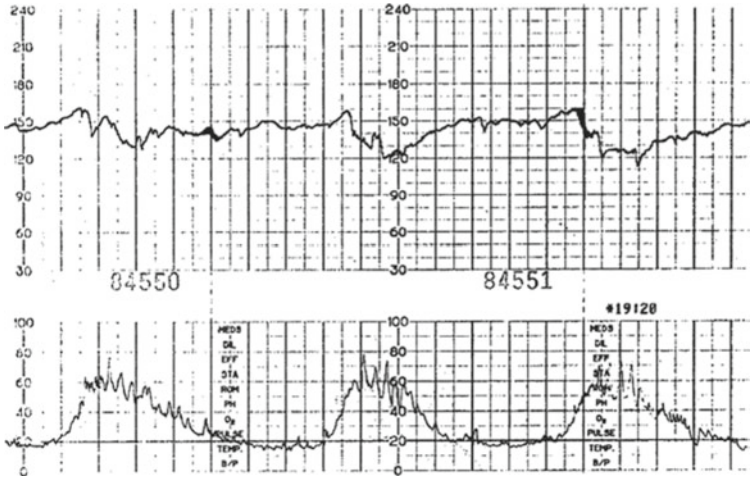

Figure 20.2 Early decelerations (From Datta [13]. Used with permission)

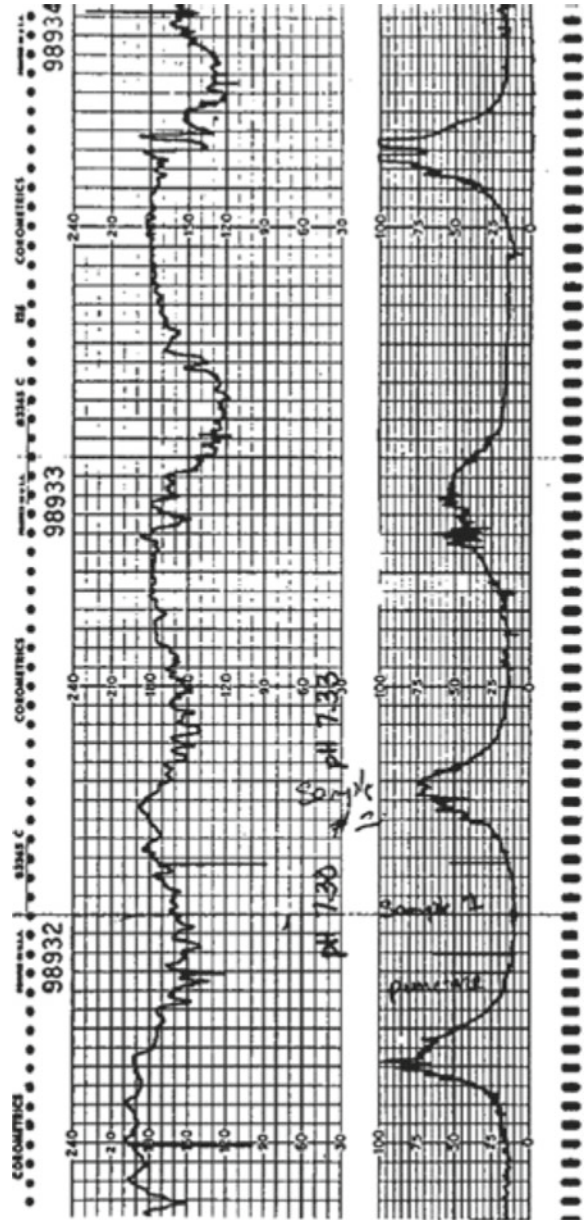

Figure 20.3 Late decelerations, with decreased variability of the fetal heart rate (FHR) between contractions (From Datta [13]. Used with permission).

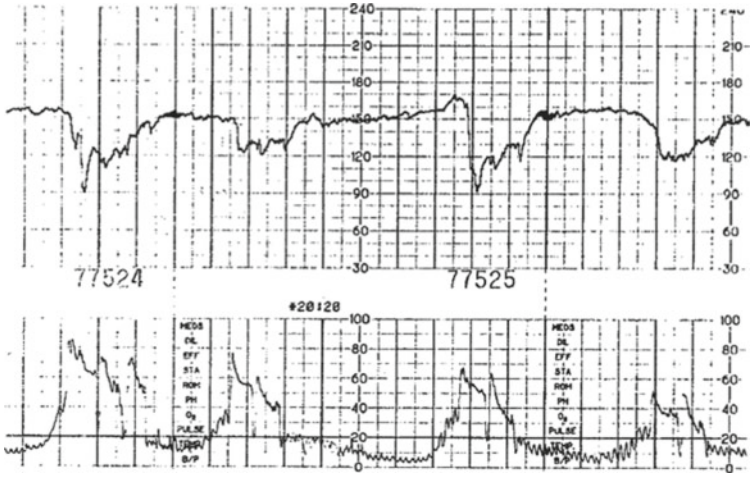

Figure 20.4 Mild to moderate variable decelerations with pushing during the second stage of labor (From Datta [13]. Used with permission)

Table 20.2 Apgar score

	0 points	1 point	2 points
Appearance	Completely blue	Extremities blue	Pink
Pulse	Absent	<100 bpm	>100 bpm
Grimace	No response to stimulation	Grimaces when stimulated	Pulls away when stimulated
Activity	None	Some flexion	Moving actively
Respiration	None	Weak	Good

Neonatal Evaluation: The Apgar Score

Once the fetus has been delivered, the Apgar Score (Table 20.2) can be used to evaluate its well-being. Named after Virginia Apgar (an anesthesiologist who developed the system in the 1950s), 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 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). Figure 20.5 depicts pain pathways in the parturient.

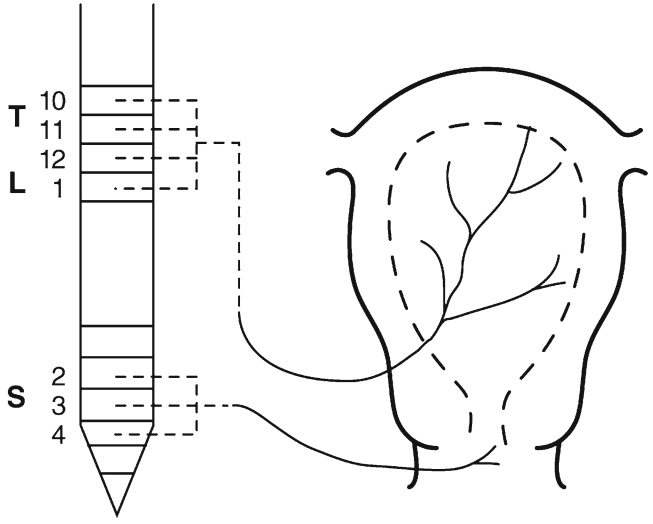

Figure 20.5 Pain pathways for the first and second stages of labor (From Datta [14])

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. 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 20.3), neuraxial analgesia (lumbar epidural) is often

Table 20.3 Qualities of an ideal pharmacotherapeutic technique for labor analgesia

Attribute
Efficacious and reliable
Duration of action coincides with duration of labor
No contraindications
No side effects (pruritus, nausea, hypotension, urinary retention)
No complications (nerve injury, high block, epidural hematoma/abscess)
Produces sensory blockade without motor weakness
Does not interfere with or prolong labor
No increased risk of operative delivery

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 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. Many obstetric patients are afflicted by either condition whether or not they have had a neuraxial block. It is important to remember that obstetric trauma can injure 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 20.4. Neuraxial anesthesia, though not ideal, usually represents the best option.

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."

Table 20.4 Qualities of an ideal anesthetic for cesarean section

Efficacious and reliable
Can be achieved instantaneously
Duration of action coincides with duration of surgery
Avoids aspiration of gastric contents
Avoids airway manipulation (negates the risk of difficult intubation)
Allows maternal participation in delivery
Conducive to family member presence in operating room
Does not interfere with neonatal well-being
Allows for stable hemodynamics
Does not interfere with hemostasis
No complications or unwanted side effects
No contraindications/no technical failures
Alleviates post-operative pain

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 continuous 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 hemodymanic consequences of spinal anesthesia. If hypotension occurs, it must be treated aggressively with intravenous fluid and phenylephrine. Maintain proper left uterine displacement and avoid the supine position because it 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.

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, 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., intraveneous 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 to be 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 may be appropriate.

Prior to induction of general anesthesia, one should administer a nonparticulate antacid and consider the administration of metoclopramide and/or an H_2 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 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. 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 an acceptable 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 and etomidate cause less hemodynamic depression as compared to propofol, making them good intravenous induction agents for 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/or 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. Surgical procedures can lead to miscarriage or preterm labor and many medications have not been well studied during pregnancy. 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 (first 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 165×10^9/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 gestational 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 sterilely 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 6–8 ml/h, a demand dose of 5 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 phenylephrine, 60 mcg, or ephedrine, 5–10 mg, 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 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 phenylephrine are given for hypotension. Fentanyl 50–100 mcg is another useful adjunct to augment a lidocaine block.

Suggested Further Reading

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Chapter 21

Physiology and Anesthesia for General and Bariatric Surgery

Rana Badr and Jesse M. Ehrenfeld

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 21.1. BMI is calculated as weight (in kg) divided by square of body height (in m^2). 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

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Table 21.1 Classification of obesity

Obesity class	BMI	Health risk
Class I (overweight)	25–30	Low
Class II (obese)	30–35	Moderate
Class III (severely obese)	35–40	High
Class IV (morbidly obese)	>40	Very high

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 comorbidities. 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 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 O_2 consumption and CO_2 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 ($PaCO_2$ >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. Abdominal obesity is also associated with **higher risk of gastroesophageal acid reflux** (GERD) and aspiration.

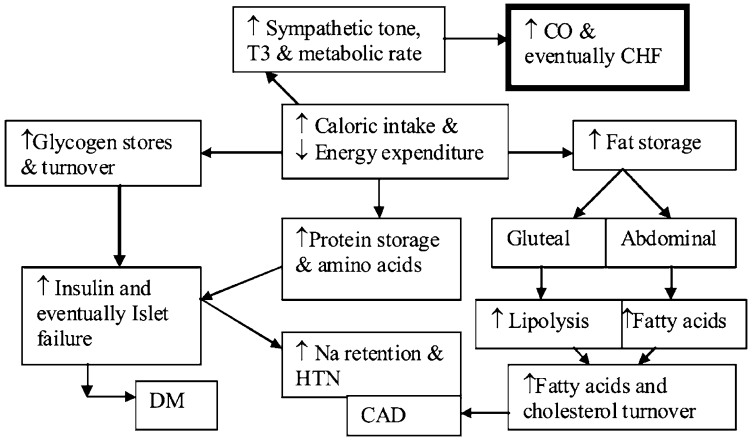

Figure 21.1 Systemic manifestations of obesity. *CO* cardiac output, *CHF* congestive heart failure, *DM* diabetes mellitus, *CAD* coronary artery disease, HTN hypertension

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 reduction. Figure 21.1 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 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. 21.2). 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.

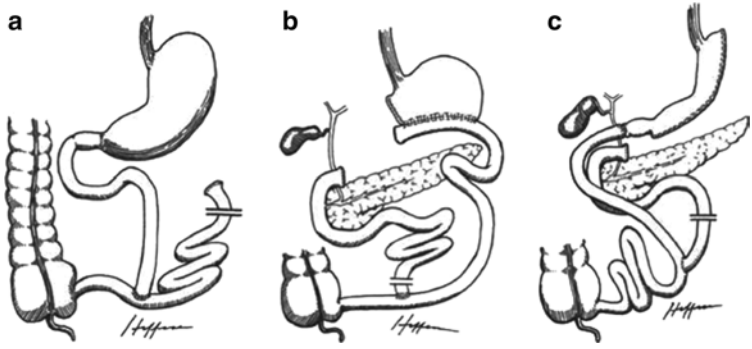

Figure 21.2 Weight loss procedures. (a) Vertical banded gastroplasty, (b) Laparoscopic adjustable gastric band, (c) Roux-en-Y gastric bypass (Used with permission. From Mantzoros [14])

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 CO_2) include intraabdominal pressure increase, systemic CO_2 absorption, increased end-tidal CO_2, cephalad displacement and impaired movement of the diaphragm, decreased FRC and pulmonary compliance, increased PIPs (peak inspiratory pressures), and ventilatory requirements. Retroperitoneal dissection of CO_2 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 CO_2 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 CO_2 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 laparatomy 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.

Table 21.2 Epidural catheter placement level for postoperative pain control

Type of surgery	Usual incision	Epidural catheter placement
Liver, pancreas, spleen, stomach	Chevron or upper midline	Low thoracic
Kidney and ureter	Oblique flank	Upper lumbar
Colorectal	Low midline	Upper lumbar
Bladder, uterus surgery	Low transverse or low midline	Low lumbar
Hernia	Inguinal	Low lumbar
Hemorrhoid	Anorectal	Caudal

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 pruritis 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 21.2).

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.

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 spelenic aneurysm, and uncontrollable bleeding. Understanding of the underlying reason for spelenectomy 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 ft, 6 in. tall and weighs 300 lb. 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.

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Chapter 22

Anesthesia for Urological Surgery

Jesse M. Ehrenfeld

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

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Table 22.1 Spinal pain segments for the genitourinary system

Organ	Sympathetics	Pain pathways
Kidney	T8-L1	T10-L1
Ureter	T10-L2	T10-L2
Bladder	T11-L2	T11-L2 (bladder dome) S2–4 (bladder neck)
Prostate	T11-L2	T11-L2S2–4
Penis	L1 and L2	S2–4
Scrotum		S2–4
Testes	T10-L2	T10-L1

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 22.1).

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. 22.1) is most commonly used for cystoscopies, transuretheral resection of prostate or bladder tumor (TURP or TURBT), or ureteroscopies. Placement in this position for greater than 2 h 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

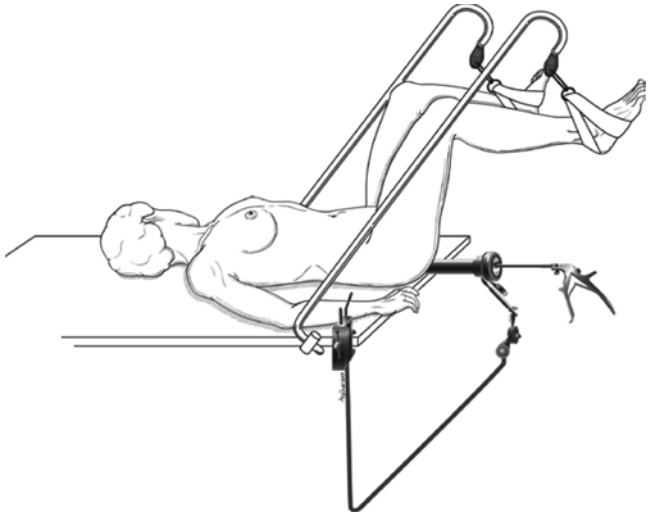

Figure 22.1 Patient positioned supine in the lithotomy position (Used with permission. From Cataldo and Buess [7])

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 section "Complications of Urologic Surgery" below).

TURP

Transuretheral 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").

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 α-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 preexisting 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

Table 22.2 Commonly used irrigating solutions

Irrigating solution	Relative osmolality	Advantages	Disadvantages
Distilled water	Very hypoosmolar	↑ Visibility	Hemolysis, hemoglobinemia, hemoglobinuria, hyponatremia
Glycine	Hypoosmolar	↓ TUPR syndrome incidence	Transient postoperative visual syndrome
Sorbitol	Hypoosmolar	↓ TUPR syndrome incidence	Hyperglycemia, osmotic diuresis
Mannitol	Isosmolar	Not metabolized	Osmotic dieresis, may cause intravascular volume expansion

Table 22.3 Symptoms of TURP syndrome

Cardiovascular	Neurologic	Other
Hypertension	Confusion/disorientation	Hemolysis
Arrhythmias	Seizures	Hyponatremia
Congestive heart failure	Unresponsive	Hyperglycinemia
Pulmonary edema	Visual problems or blindness	Hyperammonemia
Hypoxemia myocardial ischemia		

choices of irrigating solutions currently used in practice, each with advantages and disadvantages (Table 22.2). 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 22.3). 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 to

cerebral edema and hyperglycemia causing hyperammonemia (ammonia is a metabolite of glycine). In the anesthetized patient, the anesthesiologist may observe hypertension, bradycardia, dysrythmias, 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.

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 fenanyl. 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

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4. Hedican SP (2004) Complications of hand-assisted laparoscopic urologic surgery. J Endourol 18(4):387–396

5. Lynch M, Anson K (2006) Time to rebrand transurethral resection of the prostate? Curr Opin Urol 16(1):20–24

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7. Cataldo PA, Buess GF (2009) Transanal endoscopic microsurgery: principles and techniques. Springer, New York

Chapter 23

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 23.1 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. 23.2).

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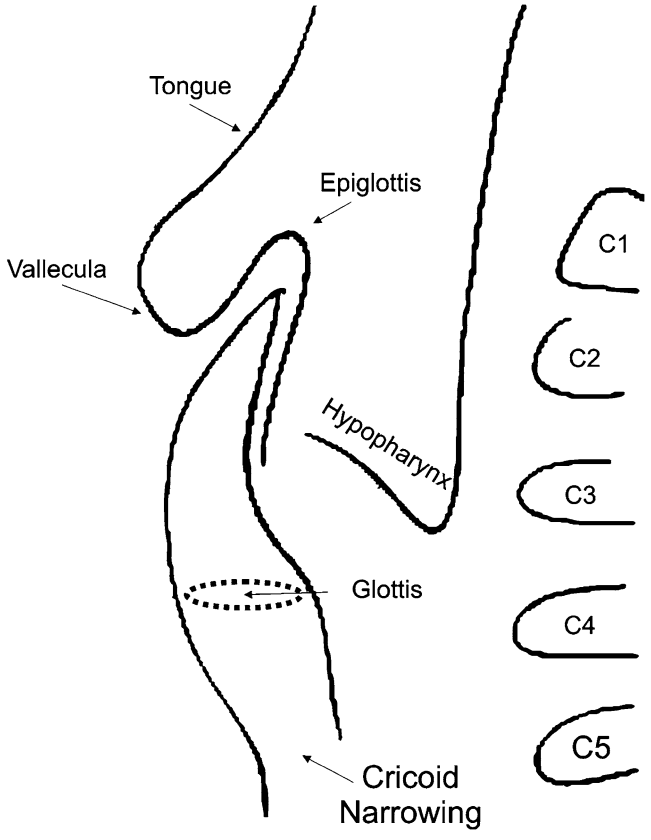

Figure 23.1 Pediatric upper airway anatomy (Image adapted from Cote et al. [3])

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 H_2O 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

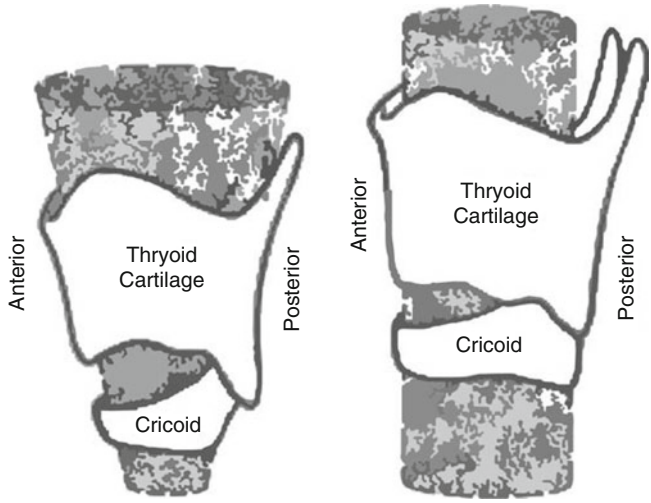

Figure 23.2 Comparison of pediatric (*left*) and adult (*right*) tracheal structures (Adapted from Cote et al. [3])

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. 23.3).

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.

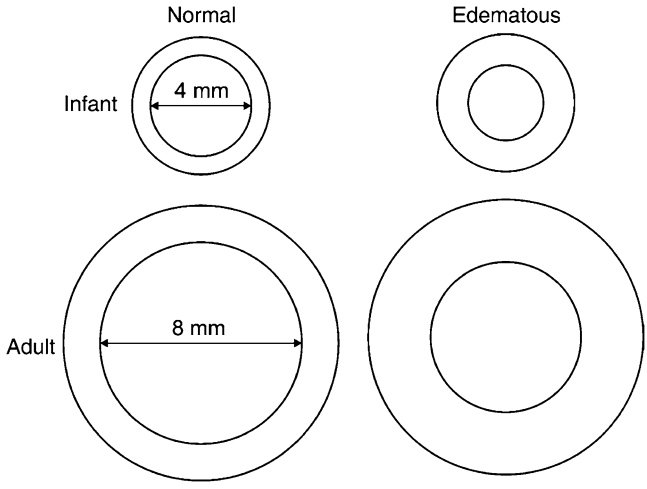

Figure 23.3 Tracheal diameters: Infant (*top*) and Adult (*bottom*) in both normal (*left*) and edematous (*right*) states (Adapted from Cote et al. [3])

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 23.4 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.

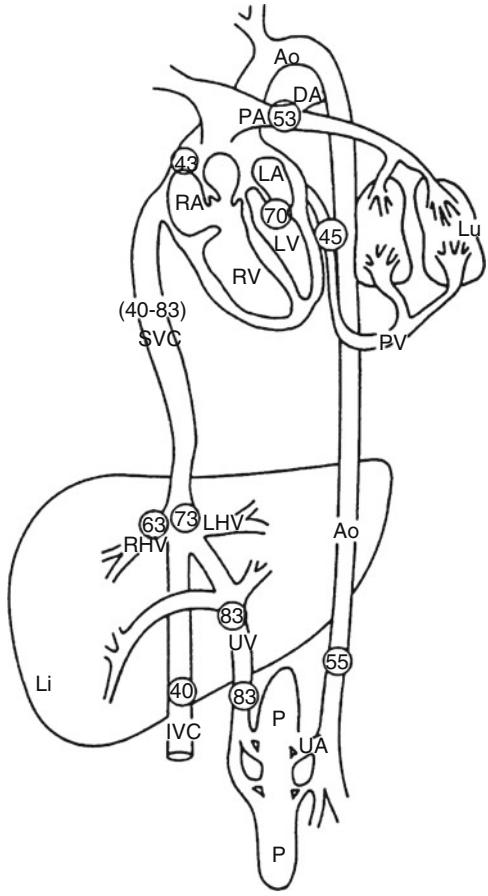

Figure 23.4 Neonatal circulation. Normal fetal circulation with major blood flow patterns and oxygen saturation values (*circled numbers* indicate percent saturation). *IVC* inferior vena cava, *P* placenta, *Li* liver, *RHV* and *LHV* right and left hepatic veins, *SVC* superior vena cava, *RA* and *LA* right and left atria, *RV* and *LV* right and left ventricles, *DA* ductus arteriosus, *PA* pulmonary artery, *Ao* aorta, *Lu* lung, *DV* ductus venosus, *PV* pulmonary vein, *UV* umbilical vein, *UA* umbilical artery (Reproduced with permission from Datta [5])

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. **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 1 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 **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. 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.

Table 23.1 Preoperative sedation drugs and dosing

Drug	Route	Dose
Midazolam	IV	0.05–0.1 mg/kg
	Oral	0.25–0.75 mg/kg
	Nasal	0.2 mg/kg
Fentanyl	IV	0.5–1 mcg/kg
	Oral ("Actiq")	10–20 mcg/kg
Ketamine	IV	1–2 mg/kg
	Oral	5 mg/kg
	IM	2–3 mg/kg
Methohexital	Rectal	20–30 mg/kg

Comfort objects may be brought into the room with the child to ease induction. Parental presence in the OR may help 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 23.1

Physiological Assessment

The 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 23.2 provides a template for preoperative patient evaluation, and Table 23.3 shows normal vital signs based on patient's age.

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.

Table 23.2 Preoperative pediatric history and review of systems

History	Important questions and pertinent findings
Prenatal care and delivery	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
Airway	Dysmorphic features (e.g., Pierre-Robin is associated with a difficult airway); micrognathia, loose teeth; advanced caries
Respiratory	Symptoms c/w acute or recent URI; asthma; sick contacts; second-hand smoke exposure; presence of wheezing, stridor, nasal flaring, cyanosis; sleep apnea
Cardiac	Murmurs associated with PFO, PDA, or congenital heart disease; frequency/duration of cyanotic spells; tachypnea; poor feeding tolerance
Gastrointestinal	Repetitive vomiting; delayed meconium passage; abdominal distention
Hematologic	Bruising; pallor; family history of sickle cell or thalassemia
Neurologic	Patterns of seizure activity; developmental delay; motor weakness; hypotonia; evidence of elevated ICP

BPD **bronchopulmonary dysplasia,** ***PFO*** **patent foramen ovale,** ***PDA*** **patent ductus arteriosus,** ***URI*** **upper respiratory infection,** ***ICP*** **intracranial pressure**

Table 23.3 Pediatric vital signs: normal ranges

Age	RR	HR	SBP	DBP
Preterm	55–60	120–180	45–60	20–45
Neonate	40–55	100–160	55–75	20–60
Infant (<6 months)	30–50	80–140	85–105	55–65
1 year	30–35	80–120	90–105	55–65
6 years	20–30	75–110	95–105	50–70
10 years	20–30	80–100	95–110	55–70
16 years	15–20	60–80	110–125	65–80

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 23.4 shows the choice of endotracheal tube diameter and length based on patient's age and weight. Table 23.5 shows the choice of laryngoscopic blade and LMA size based on patient's age. Table 23.6 shows the most common pediatric emergency drug dosages.

Table 23.4 Endotracheal tube sizes and appropriate insertion depths

Age/weight	Internal diameter (mm)	Length (oral) in cm	Length (nasal) in cm
<1.5 kg	2.5	9.0–10.0	12.0–13.0
1.5–3.5 kg	3.0	9.5–11.0	13.0–14.0
Term	3.5	10.0–11.5	13.5–14.5
3–12 months	4.0	11.0–12.0	14.5–15.0
12–24 months	4.5	12.0–13.5	14.5–16.0

Table 23.5 Laryngoscopic blade and LMA sizes

Age	Blade	Weight	LMA size
Premature	Miller 0	<5 kg	1
Neonate	Miller 0	5–10 kg	1.5
1–4 years	Miller 1	10–20 kg	2
4–10 years	Miller 2, Mac 2	20–30 kg	2.5
Adolescent	Miller 2, Mac 3	>30 kg	3

Table 23.6 Common pediatric emergency drugs

Drug	IV	IM/(SQ)
Atropine	0.01–0.02 mg/kg	0.02 mg/kg
Succinylcholine	1–2 mg/kg	3–4 mg/kg
Ephedrine	0.1–0.2 mg/kg	–
Epinephrine	10 mcg/kg	(10 mcg/kg)

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.

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-in., 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 23.7).

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 23.8) as a guide to fluid replacement (Table 23.8; also see Chap. 14, Fluid, Electrolyte, and Transfusion

Table 23.7 Pediatric induction methods

Technique	Advantages	Disadvantages
Mask induction (sevoflurane)	Brief onset (2–3 min) Avoids awake IV Spontaneous respirations Parental participation Facilitates IV start via Vasodilatation	Breath-holding/laryngospasm Contraindicated for full stomach Can trigger malignant hyperthermia Unprotected airway Gases are cold and dry Requires good seal with the mask
Intravenous (propofol)	Rapid onset (<30 s) Minimize duration of | unprotected airway	Anxiety about "shots" Pain upon injection Malfunction Extravasation
Intramuscular (ketamine)	Brief onset (2–4 min) Can inject at multiple sites Does not require cooperation	Pain upon injection Difficulties with obese children Secretions with ketamine Unprotected airway Nerve injury
Rectal (methohexital)	Rapid onset (1–2 min) Quick clearance	Only useful in young children No pre-packaged delivery device Unprotected airway

Table 23.8 Pediatric maintenance fluid (per hour)

Weight	Rate
‹10 kg	4 ml/kg/h
10–20 kg	40 ml/h + 2 ml/kg/h for each kg ›10 kg
›20 kg	60 ml/h + 1 ml/kg/h for each kg ›20 kg

Table 23.9 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

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 23.9) 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 herniorraphy) 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).

Emergence

Extubation criteria should include normothermia (body temp > 36 °C), hemodynamic stability, resolution of neuromuscular blockade and evidence of appropriate analgesia. 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/she 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 23.10.

Table 23.10 Common pediatric surgical conditions

Pyloric stenosis

—***Associated findings:*** Nonbilious, projectile vomiting, hypochloremic metabolic alkalosis, hypokalemia, hyponatremia, dehydration, hypovolemic shock

—***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

Diaphragmatic hernia

—***Associated findings:*** Respiratory distress, bowel sounds heard over the chest, decreased breath sounds, scaphoid abdomen

—***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.

Omphalocele and gastroschisis

—***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

Pediatric Surgical Conditions

A list of the most common pediatric surgical conditions and their anesthetic implications are shown in Table 23.10.

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 alkalosis 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

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Chapter 24

Physiology and Anesthesia for Elderly Patients

Ruma Bose and Jesse M. Ehrenfeld

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 physiological changes that occur in the elderly. This often requires the use of shorter acting agents and additional invasive monitoring to

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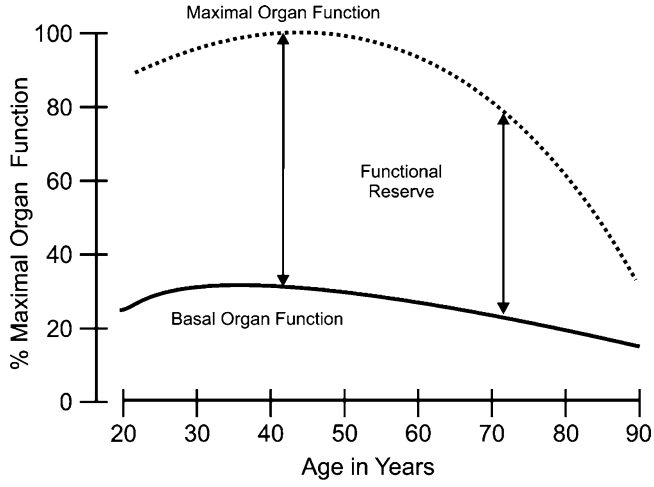

Figure 24.1 Relationship between age and organ function

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. 24.1.

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. 24.2). Valvular abnormalities are more common due to sclerosis and calcification, and more than 70 % percent of the elderly have an audible heart murmur.

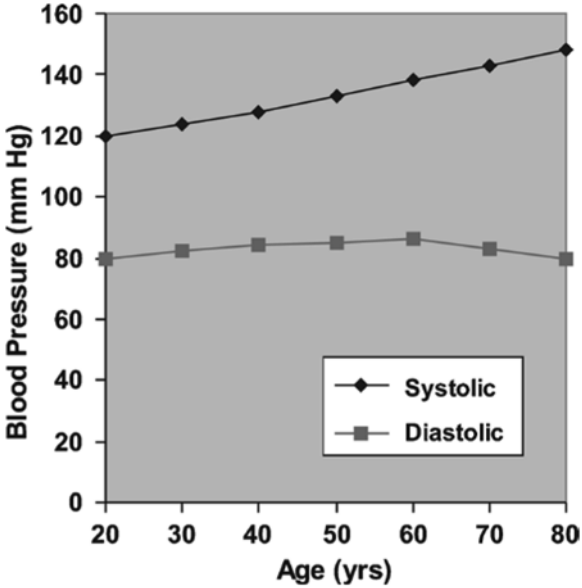

Figure 24.2 Changes in blood pressure with age

Pulmonary Changes

The major changes that occur with aging can be broadly attributed to the following factors:

- 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 24.1).

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.

Table 24.1 Renal changes in the elderly

Decrease in renal blood flow
Decline in glomerular filtration rate
Decline in ADH response
Decrease in total body water
Decreased ability to conserve sodium
Diminished urine concentrating ability
Decline in renin-aldosterone levels
Decreased thirst perception

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 24.2 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 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.

Table 24.2 Causes of delirium

Advanced age
Preexisting dementia
Depression
Hypoxia and hypercarbia
Hypotension
Alcohol or sedative withdrawal
Impaired vision and hearing
Metabolic disturbances (hyponatremia/hypernatremia)
Acute myocardial infarction
Infection

Table 24.3 Comparative elimination half-life of drugs

	Young	Old
Fentanyl	250 min	925 min
Alfentanil	90 min	130 min
Diazepam	24 h	72 h
Midazolam	2.8 h	4.3 h
Vecuronium	16 min	45 min

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 24.3).

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. 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 >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, The Preoperative Patient 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.

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.

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Chapter 25

Ambulatory Surgery and Out-of-OR (OOR) Procedures

Joshua H. Atkins and Jesse M. Ehrenfeld

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

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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 25.1) 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 25.1 provides representative criteria used to decide whether the patient might be an appropriate candidate for ambulatory surgery. Table 25.2 presents surgery- and procedure-related factors one might consider in deciding whether the proposed procedure is appropriate for the ambulatory setting. Table 25.3 lists common ambulatory procedures. However, *no* procedure is *always* done in an

Table 25.1 Patient selection factors for ambulatory surgery

Caregiver available for transport home and postoperative evaluation
Patient willingness to go home the day of surgery
Co-morbidities: obesity, obstructive sleep apnea, poorly compensated cardiopulmonary disease, chronic pain, renal failure, urinary retention, significant neurologic disease (myasthenia, Parkinson's, dementia)
Prior anesthetic problems: difficult airway, PONV, postoperative cognitive dysfunction, malignant hyperthermia, poor pain-control

Table 25.2 Procedure-related considerations for ambulatory surgery

Duration of surgery (no absolute cutoff)
Intraoperative fluid shifts and bleeding
Risk of serious postoperative complications (bleeding, infection, airway compromise)
Extent of postoperative pain and analgesic needs
Need for intravenous medications or inability to tolerate oral intake

Table 25.3 Common ambulatory procedures

Local lesion removal (cyst, melanoma, breast biopsy/part, mastectomy)
Orthopedic procedures not involving major fractures
Basic ENT procedures (sinus/tonsillectomy/tympanoplasty, cochlear surgery, single lobe thyroidectomy)
Limited plastics procedures (blepharoplasty, scar revision)
Limited urologic procedures (cystoscopy, biopsy, vasectomy, circumcision)
Ophthalmologic procedures (excluding vitrectomy and enucleation)
Limited GYN procedures (hysteroscopy, D&C/D&E, cone biopsy, tubal ligation)

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 25.4.

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 *s*hort-*a*cting, *f*ast-*e*mergence 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

Table 25.4 Procedure exclusions for outpatient management

Requires drain or nasogastric drainage tube to be placed	Hysterectomy, bowel resection, neck dissection
Oral medications inadequate for postoperative pain control	Joint replacement, mastectomy, major abdominal surgery
May require postoperative bladder catheterization	Ventral hernia repair, bladder tumor resection, ureteral stent
Frequently requires intraoperative or postoperative transfusion	Hysterectomy, ORIF femur
Expectation of postoperative electrolyte shifts	Parathyroidectomy, pituitary resection
Requires hourly patient assessment	Free-flap, craniotomy, patients with severe sleep apnea

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

General, regional, combined regional/general, monitored anesthesia care, and local anesthesia are all compatible with rapid patient discharge. However, anesthetic plan should be compatible with patient expectations. 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 to discharge that can be associated with time needed for the return of motor or bladder function after neuraxial blockade.

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 25.5 shows unique aspects of OOR anesthesia practice.

Perhaps the most common conundrum in OOR anesthesia pertains to the type of anesthetic. Depth of anesthesia is characterized by a "continuum," from

Table 25.5 Unique aspects of Out-of-OR-anesthesia

1. Limited anesthesia role in patient selection and preoperative optimization
2. Unfamiliar procedures and proceduralists
3. Remote locations with limited equipment availability
4. Enhanced role of nonanesthesia support staff
5. Greater potential for miscommunication
6. High expectations for patient satisfaction, clinical efficiency, and accommodation

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 FIO_2 with preoxygenation should be considered if anesthetic management may result in intermittent apnea or hypoventilation.

Table 25.6 Anesthetic options for GI endoscopy procedures

Option A:

1. Nasal cannula oxygen 2–4 L
2. Midazolam (1–4 mg) IVP
3. Glycopyrrolate 0.1–0.3 mg IVP
4. Propofol/ketamine (1 mg/mL) infusion @ 80–150 mcg/kg/min propofol after 0.8–1.4 mg/kg bolus at induction

Comment: Low likelihood of apnea, limited hemodynamic effects, modest analgesia, and possible ketamine side effects

Option B:

1. Nasal cannula oxygen 2–4 L
2. Propofol infusion (120–200 mcg/kg/min) after 1–2 mg/kg induction bolus
3. Consider addition of fentanyl 25–100 mcg for highly stimulating procedures

Comment: high doses of propofol as sole agent may induce apnea when bolused, no analgesic properties, sole agent provides clean anesthetic, and rapid emergence

Option C:

1. Preoxygenate with 100 % O_2 via facemask
2. Deliver high FiO_2 via Mapleson circuit attached to patient with lubricated nasal trumpet
3. Propofol/remifentanil (2 mcg/mL) infusion at 60–120 mcg/kg/min propofol after 0.6–1 mg/kg bolus

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

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 25.6, 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. 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 α-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 quiescence 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.

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 three of four: 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 three 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

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3. Apfelbaum JL, Walawander CA, Grasela TH et al (2002) Eliminating intensive postoperative care in same-day surgery patients using short-acting anesthetics. Anesthesiology 97:66–74

4. Dean M, The Royal College of Radiologists, et al (2003) Safe sedation. Analgesia and Anaesthesia within the Radiology Department. http://www.rcr.ac.uk/index.asp?PageID=310&PublicationID=186

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Chapter 26

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 its implications for anesthesia care
- Understand the approach to anesthesia for orthopedic surgery

Trauma

Introduction

Patients presenting with traumatic injuries can represent the entire gamut of anesthesia challenges, including airway, 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.

J.M. Ehrenfeld et al. (eds.), *Anesthesia Student Survival Guide*,
DOI 10.1007/978-3-319-11083-7_26

Table 26.1 Mechanisms of Injury

Trends revealed from the National Trauma Data Bank
1. The majority of reported traumas occur in young males
2. Case fatality rises with age at time of injury
3. Motor vehicle accidents are the main cause of injury in young and middle-aged patients, with falls becoming predominant in elderly patients
4. The vast majority of injuries are blunt
5. Penetrating injuries have the highest associated mortality
6. Burns result in the longest hospital stays
7. America leads the world in firearm related deaths in both adults and children, with an incidence 4× higher than any other industrialized country
8. Firearm deaths occur predominantly in African-American men

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 26.1).

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. 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 ("fingers and tubes in every orifice").

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 26.2 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, pulmonary,

Table 26.2 Glasgow coma scale

Eye opening	
Spontaneous	4
To loud voice	3
To pain	2
None	1
Verbal response	
Oriented	5
Confused, disoriented	4
Inappropriate word	3
Incomprehensible sounds	2
None	1
Best motor response	
Obeys	6
Localizes	5
Withdraws (flexion)	4
Abnormal flexion posturing	3
Extension posturing	2
None	1

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. Also, post-traumatic patients frequently have a change of status, and frequent reassessments are mandatory as they might change anesthetic management.

Specific Challenges

Anesthesia personnel are frequently called to the emergency department for incoming trauma patients. As a result, we 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.

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 one hour of their injury tend to have better outcomes. For any hope of survival, early intubation with appropriate oxygenation and ventilation, as well as adequate 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. 26.1.

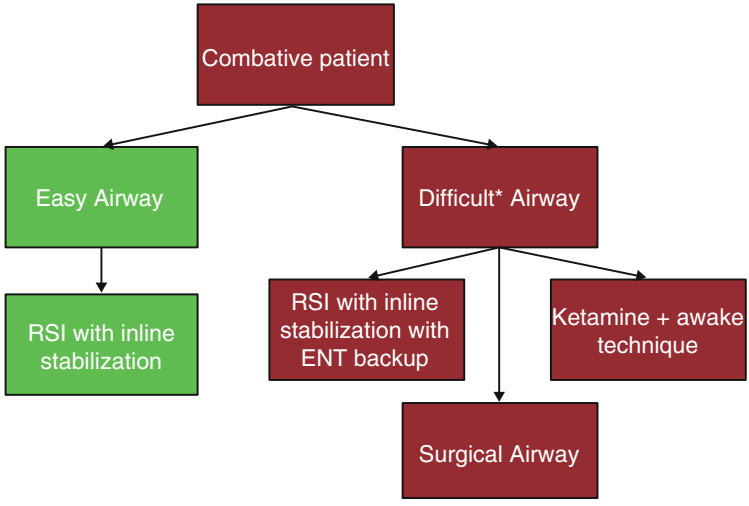

Figure 26.1 Sample plan to manage the airway of a combative trauma patient. *RSI* rapid sequence induction

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 hypoventilation 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 and Acid–Base Analysis). 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

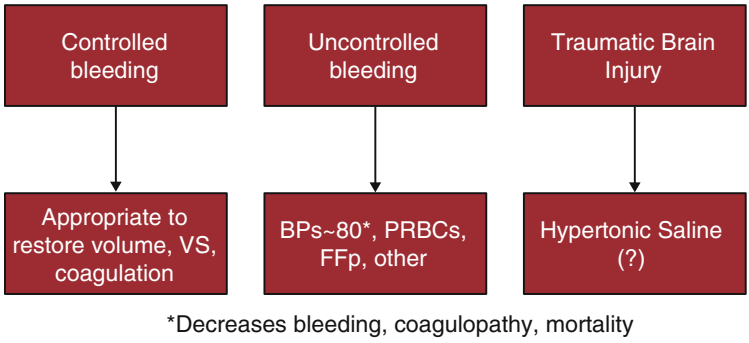

Figure 26.2 **Hypovolemia and acidosis**

Table 26.3 **Trauma blood protocol**

O-neg × 2 units, then
O-pos if
Male
Obviously non-child bearing age
Continue with O-neg

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 ≈70–80 mmHg, although existing head trauma and CPP should be kept in mind (Fig. 26.2).

- **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 (≈$5000/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 and Acid–Base Analysis). Below is a sample Trauma Blood/Massive Transfusion Protocol (Tables 26.3 and 26.4).

Table 26.4 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

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 the technique due to failure rates, cost, or administrative complexity. Neuraxial anesthesia (with or without general anesthesia) has been shown to reduce

Table 26.5 Anesthetic options for orthopedic procedures

Procedure	Anesthetic considerations
Hand surgery	Frequently performed with local anesthesia/sedation only. Also can use Bier or axillary block
Wrist surgery	Infraclavicular block
Elbow surgery	Difficult to anesthetize. Infraclavicular or axillary block *may* be adequate
Shoulder surgery	Interscalene block (±catheter) provides excellent surgical anesthesia and postoperative analgesia
Hip surgery	Spinal/epidural block
Knee surgery	Intra-articular local anesthetics for arthroscopy. Spinal ± continuous femoral block or epidural block for knee replacement
Ankle surgery	Popliteal fossa block (±catheter) provides excellent surgical anesthesia and postoperative analgesia
Foot/toe surgery	Frequently performed with local anesthesia/sedation only. Also can use ankle block

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 26.5 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 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/hypoxemia, 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.

The location of a hip fracture will significantly impact the amount of blood loss that occurs: subcapital < transcervical < neck base < intertrochanteric < subtrochanteric (see Fig. 26.3).

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.

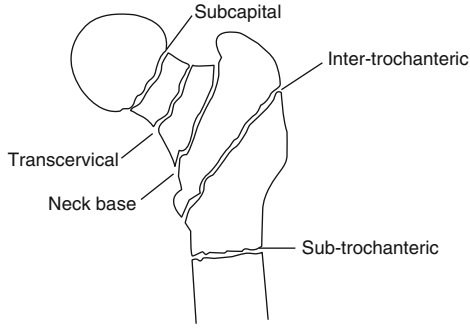

Figure 26.3 Hip fracture locations

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

1. Sell SL, Avila MA, Yu G, Vergara L, Prough DS, Grady JJ, DeWitt DS (2008) Hypertonic resuscitation improves neuronal and behavioral outcomes after traumatic brain injury plus hemorrhage. Anesthesiology 108:873–881

2. Spinella PC, Perkins JG, McLaughlin DF, Niles SE, Grathwohl KW, Beekley AC, Salinas J, Mehta S, Wade CE, Holcomb JB (2008) The effect of recombinant activated factor VII on mortality in combat-related casualties with severe trauma and massive transfusion. J Trauma 64:286–293

3. Hirshberg A, Stein M, Adar R (1997) Reoperation. Planned and unplanned. Surg Clin North Am 77:897–907

4. Mantilla CB, Horlocker TT, Schroeder DR, Berry DJ, Brown DL (2002) Frequency of myocardial infarction, pulmonary embolism, deep venous thrombosis, and death following primary hip or knee arthroplasty. Anesthesiology 96:1140–1146

Part VI

Postoperative Considerations

Chapter 27

Perioperative Acute and Chronic Pain Management

Mark A. Hoeft

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

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(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 27.1). 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. 27.1.

Table 27.1 Classification of peripheral nerves

Fiber class	Diameter (μm)	Myelin	Conduction velocity (m/s)	Innervation	Function
A alpha	12–20	+++	75–120	Afferent to skeletal muscle	Motor and reflexes
A beta	5–12	+++	30–75	Afferent from cutaneous mechanoreceptors	Vibration, light touch and pressure
A gamma	3–6	++	12–35	Efferent to muscle spindles	Muscle tone
A delta	1–5	++	5–30	Afferent pain and thermoreceptors	"Fast", sharp, intense, lancinating pain, touch and temperature
B	<3	+	3–15	Preganglionic sympathetic efferent	Autonomic function
C	0.2–1.5	–	0.4–2.0	Afferent pain and thermoreceptors	"Slow", dull, burning, achy pain, touch, pressure, temperature, postganglionic autonomic

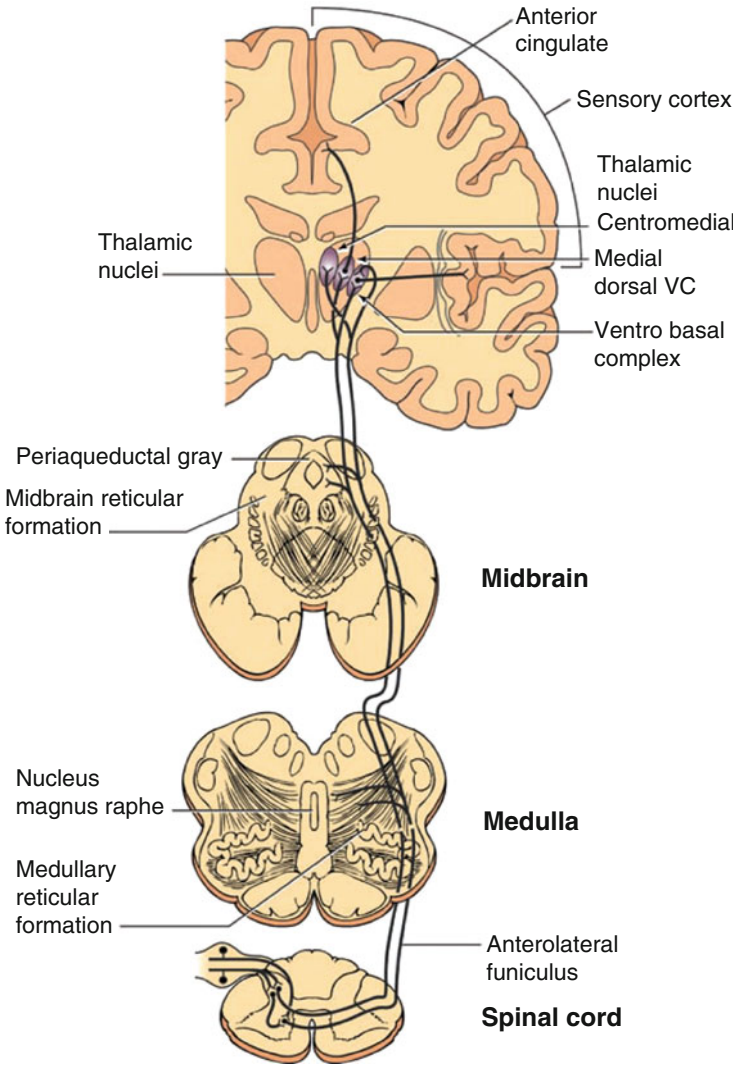

Figure 27.1 Gross anatomy of pain pathways (From Sorkin et al. [5]. Used with permission)

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 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.

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

Table 27.2 General pain types

Nociceptive pain	Normal, acute pain perception evoked by short-lasting noxious stimuli in intact tissue, in the absence of peripheral or central sensitization
Inflammatory pain	Pain following tissue injury but with no neural injury
Neuropathic pain	Pathophysiologic state of pain after neural injury resulting in peripheral and central reorganization

serves as a protective mechanism (Table 27.2). 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, hyperalagesia) 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

Table 27.3 Abnormal pain descriptor definitions

Allodynia	The perception of pain by a stimulus that is not normally painful
Hyperalgesia	The enhanced perception of pain by a normally painful stimulus
Dysesthesia	Abnormal sensations experienced in the absence of stimulation
Paresthesia	An abnormal sensation (e.g. burning, pricking, tickling, or tingling)

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 (Table 27.3).

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 27.4.

Table 27.4 Common interventional pain procedures

Procedure	Target	Mechanism	Indicated pain syndrome
Epidural steroid injection	Nerve root	Injection of steroid to decrease inflammation surrounding the nerve root	Herniated discs, spinal stenosis, foraminal stenosis
Medial branch block	Medial branch of dorsal ramus	Local anesthetic injection	Diagnostic test to determine if the pain is facet mediated
Radiofrequency ablation	Medial branch of the posterior division of the spinal nerve	Coagulative destruction of the medial branch nerve	Therapeutic intervention if medicated pain is the cause of pain after a diagnostic medial branch block is performed
Trigger point injection	Trigger points	Relaxation and lengthening of the muscle fiber	Myofascial pain
Spinal cord simulator	Posterior column of spinal cord	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	Neuropathic pain, angina, peripheral ischemic pain
Intrathecal pumps	Intrathecal space	Decreasing systemic dose of medications such as opioids, thus decreasing side effects	Cancer pain patients
Neurolytic blocks	Celiac plexus, trigeminal ganglion, lumbar sympathetic chain	Destruction of nerve/plexus via phenol, alcohol or RFA	Pallative care patients
Stellate ganglion block/ lumbar sympathetic	Stellate ganglion/ lumbar plexus	Local anesthetic blocking of sympathetic efferent nerves	Complex regional pain syndrome

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.

Common Chronic Pain Medications Classifications

Opioids

The opioids are a diverse classification of mediation that typically provide analgesic effect via actions on the μ, δ, and κ 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 27.5. 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.

Table 27.5 Common oral opioid pharmacodynamics and dosing

Opioids	Half-life	Duration (h)	Equianalgesic oral doses (mg)	Initial dose (mg)	Dosing interval (h)
Codeine	3	3–4	80	30–60	4
Hydromorphone	2–3	2–3	2	2–4	4
Hydrocodone	1–3	3–6	10	5–7.5	4–6
Oxycodone	2–3	3–6	7	5–10	6
Methadone	15–30	4–6	10–20	20	6–8
Morphine	2–3.5	3–4	10	10–30	3–4
Propoxyphene	6–12	3–6	43–45	100	6
Tramadol	6–7	3–6	40	50	4–6

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 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: (Nortiptyline, 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

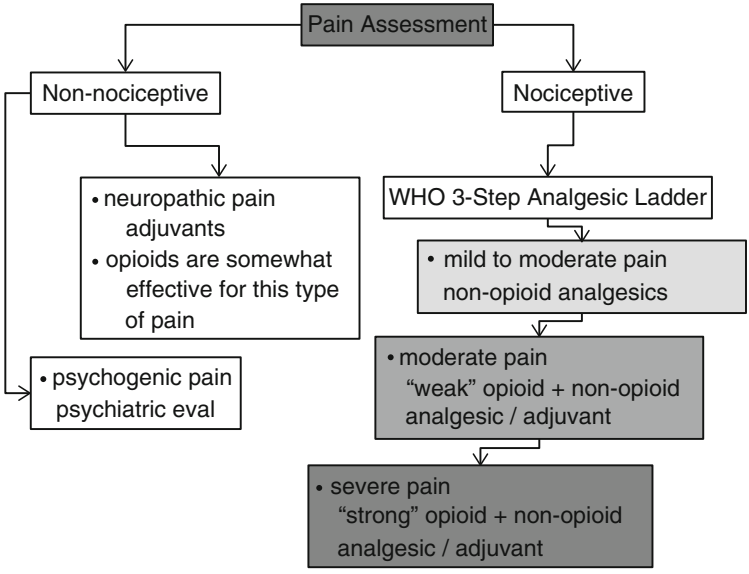

Figure 27.2 Assessment of pain and World Health Organization analgesic ladder

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. 27.2. 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 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 and controversial 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, dry needling, or Botox injections 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 and steroid injections 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 *allodynia*, pain elicited by a normally nonpainful stimulus, and *dysesthesia* and *paresthesia*, abnormal sensations occurring spontaneously or in response to stimulation. You have verified allodynia 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 cutanous evidence of sympathetic excess and disuse atrophy, and she has allodynia. 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 antidepression 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

1. Benzon HT, Rathemell JP, Wu CL, Turk DC, Argoff CE (2008) Raj's practical management of pain, 4th edn. Mosby-Elsevier, Philadelphia

2. Bonica JJ, Loeser JD, Chapman CR, Fordyce WE (2001) The management of pain, 3rd edn. Lippincott, Williams & Williams, Philadelphia

3. Kandel ER, Schwartz JH, Jessell TM (2000) Principles of neural science, 4th edn. McGraw-Hill, New York

4. Longnecker DE, Brown DL, Newman MF, Zapol WM (2007) Anesthesiology, 1st edn. McGraw-Hill, New York

5. Sorkin L et al (1998) Atlas of anesthesia. Current Medicine Group, Philadelphia

Chapter 28

Postoperative Anesthesia Care Unit (PACU) and Common Postoperative Problems

R. Dean Nava Jr, Tarun Bhalla, and Jesse M. Ehrenfeld

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 28.1 shows an example of a typical sign-out an anesthesiologist would give to the PACU staff.

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Table 28.1 Sample PACU sign-out

Preop history	Medications, allergies, past medical history
	Underlying diagnosis
	Premedications
Intra-operative history	Procedure
	Anesthesia type
	Medications & fluids given
	Estimated blood loss, urine output
	Intra-operative events/problems
	Vital sign ranges
Patient status	Airway (preop exam, airway management, ETT position)
	Size, location of lines, catheters and invasive monitors
	Level of consciousness
	Pain level
	Intravascular volume status
	Overall impression
Postop instructions	Acceptable ranges (blood loss, vitals, urine output)
	Potential cardiovascular or respiratory problems
	Labs or diagnostic studies (CXR, ECG) if necessary
	Location and physician contact information

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 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 CO_2 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 $PaCO_2$ >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 dysrhythmias. The late signs usually include hypotension, obtundation, bradycardia, and cardiac arrest. The treatment generally includes supplemental O_2, 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 28.2.

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,

Table 28.2 Causes of hypotension

Decreased preload	Hypovolemia (most common)
	"Third spacing" (fluid sequestration)
	Bleeding
	Wound drainage
	Venodilation due to spinal/epidural anesthesia
	Pericardial tamponade
	Tension pneumothorax
	Air embolism
Left ventricular dysfunction (impaired contractility)	Severe metabolic derangements (acidosis, sepsis, hypoxemia)
	Myocardial infarction
	Volume overload
	Dysrhythmias
Arterial vasodilatation (decreased afterload)	Possible inflammatory response
	Anesthetic related

Table 28.3 Suggested medical therapies for hypertension

Mild to moderate hypertension	Beta blockers (labetalol, esmolol, metoprolol)
	Calcium channel blockers (nicardipine)
	Nitro paste
	Hydralazine
Severe or refractory hypertension (consider intra-arterial BP monitoring)	IV antihypertensive infusions
	Nicardipine
	Nitroglycerine
	Nitroprusside

Table 28.4 Causes of postoperative tachycardia

Noxious stimuli	Pain, anxiety
	Endotracheal tube
	Distended bladder
Physiologic derangements	Acidosis
	Hypoxemia
	Hypotension and hypovolemia
	Hypoglycemia
	Increased intracranial pressure
	Myocardial ischemia
Medications	Beta adrenergic vasopressors
	Dopamine
	Dobutamine
	Bronchodilators
Anesthetics	Ketamine
	Isoflurane

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 28.3 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 28.4 for differential diagnosis of tachycardia.

Signs and symptoms may include hypertension or hypotension and angina. Treatment 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 28.5 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,

Table 28.5 Common causes of postoperative bradycardia

Medications	Neostigmine, drophonium
	Phenylephrine, norepinephrine
	Opioids
	Succinylcholine
	Beta blockers
	Local anesthetics
	Ganglionic blockers
	High spinal/epidural anesthesia
Physical causes	Carotid sinus massage
	Valsalva maneuver
	Gagging
	Rectal exam
	Increased ocular pressure
	Distended bladder
	Stimulation of pharynx
Metabolic derangements	Severe acidemia
	Hypoxemia

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 28.6.

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

Table 28.6 Causes of postoperative mental status changes

Hypoxemia
Metabolic derangements
Cerebral hypoperfusion
Extremes of age
Emotionally significant operations
Presence of intraoperative recall
Scopolamine or atropine
Substance abuse
Pain, nausea, pruritus

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 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 28.7:

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,

Table 28.7 PONV contributing factors

Patient related
Young women
Hiatal hernia
Obesity
History of postoperative nausea
History of motion sickness
Non-smokers have higher risk
Surgical related
ENT, abdominal, gynecologic procedures
Extraocular muscle traction
Middle ear irritation
Peritoneal, intestinal irritation
Dental procedures
Anesthesia related
Gas in stomach due to facemask ventilation
Use of nitrous oxide (controversial)
Use of parenteral opioids
Use of etomidate
Hypotension after spinal/epidural anesthesia
Postop related
Use of parenteral opioids
Postoperative oral fluid intake

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 28.8. 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.

Table 28.8 Commonly used opioids in the PACU

Drg	Duration	Typical bolus dose
Fentanyl	Short acting	25–100 mcg IV
Hydromorphone (Dilaudid)	Intermediate to long duration	0.2–1 mg IV
Meperedine (Demerol)	Intermediate to long duration	50–100 mg IV/SC/IM
Morphine	Intermediate to long duration	2–5 mg IV

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.

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 28.9.

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 28.9 Effects of hypothermia and shivering

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, SpO298 % 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

1. Barash PG, Cullen BF, Stoelting RK (eds) (2006) Clinical anesthesia, 5th edn. Lippincott, Williams and Wilkins, Philadelphia

2. Morgan GE, Mikhail MS, Murray MJ (eds) (2006) Clinical anesthesiology, 4th edn. McGraw-Hill, New York

3. (2000) Practice advisory for the prevention of perioperative peripheral neuropathies: a report by the American Society of Anesthesiologists Task Force on Prevention of Perioperative Peripheral Neuropathies. Anesthesiology 92:1168–1182

4. American Society of Anesthesiologists Task Force on Postanesthetic Care (2002) Practice guidelines for postanesthetic care: a report by the American Society of Anesthesiologists Task Force on Postanesthetic Care. Anesthesiology 96:742–752

5. Gan TJ, Meyers T, Apfel CC et al (2003) Consensus guidelines for managing post-operative nausea and vomiting. Anesth Analg 97:62–71

6. American Society of Anesthesiologists Task Force on Acute Pain Management (2004) Practice guidelines for acute pain management in the perioperative setting: an updated report by the American Society of Anesthesiologists Task Force on Acute Pain Management. Anesthesiology 100:1573–1581

Chapter 29

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 the basic modes of mechanical ventilation
- Review other supportive therapies in the ICU

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.

J.M. Ehrenfeld et al. (eds.), *Anesthesia Student Survival Guide*,
DOI 10.1007/978-3-319-11083-7_29

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:

$$CaO_2 = \left[Hb \times 1.34 \times SaO_2 \right] + \left[PaO_2 \times 0.003 \right]$$

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_2ER) is defined as oxygen consumption divided by oxygen delivery:

$$O_2ER = \left(VO_2 / DO_2 \right) \times 100$$

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 for brief periods during exercise or stress up to a maximum O_2ER of about 70 %. Any further or prolonged 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 (Hb) and hemoglobin saturation (SaO_2) influence the oxygen

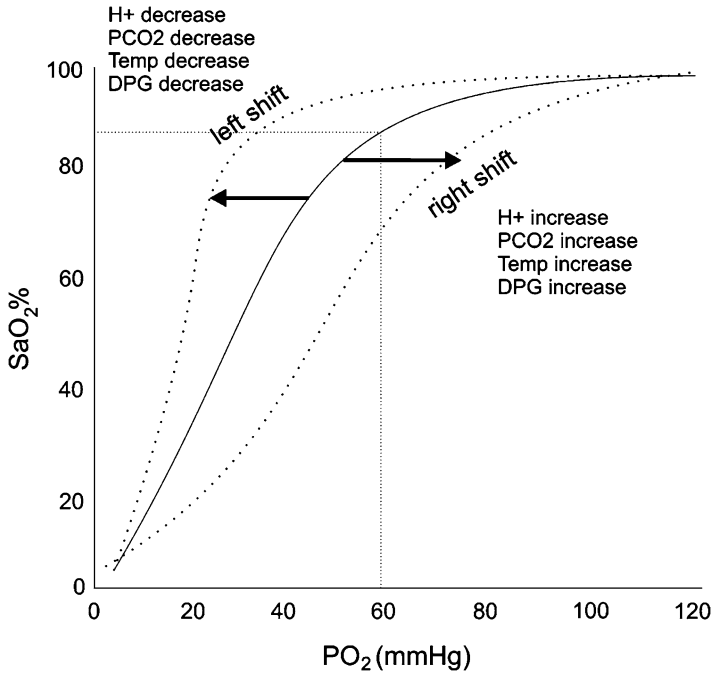

Figure 29.1 Oxyhemoglobin dissociation curve. This curve defines the relationship between partial pressure of oxygen in the blood and the percent of hemoglobin that is saturated with oxygen. The position of this curve is influenced by factors such as H+ concentration, PaCO2, temperature, and 2-3-diphosphoglycerate (DPG) (Image Courtesy J. Ehrenfeld)

content. The relationship between partial pressure of oxygen in the blood and hemoglobin saturation is defined by the oxyhemoglobin dissociation curve (Fig. 29.1). 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 ($ScvO_2$) correlates well with mixed venous blood oxygen saturation (SvO_2) in most circumstances, and can be used to reflect tissue oxygenation. Normal $ScvO_2$ is approximately 70 % (compared to normal SvO_2 of approximately 65 %). Lower than normal $ScvO_2$ or SvO_2 is an indication of poor tissue oxygenation and the need for improved oxygen delivery and perfusion. The advantage of using $ScvO_2$ is that it does not require a pulmonary artery catheter (versus SvO_2 which is drawn from the pulmonary artery).

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). Blood pressure monitoring with a noninvasive cuff may be 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

Table 29.1 Complications associated with arterial cannulation

Complication	Precautions to decrease risk
Hematoma	Avoid multiple needle punctures/attempts Apply pressure if artery punctured
Bleeding	Caution in coagulopathic patients Apply pressure to bleeding site
Thrombosis	Avoid multiple needle sticks Use continuous flush system Avoid prolonged catheterization
Vasospasm	Avoid multiple or traumatic punctures/attempts at cannulation
Air embolism	Caution when flushing catheter
Nerve damage	Avoid sites in close proximity to nerve
Infection	Use sterile technique Avoid prolonged catheterization
Intra-arterial drug injection	Keep venous and arterial lines well-organized, separated, and clearly labeled
Ischemia	Avoid traumatized sites Avoid prolonged catheterization Place pulse oximeter on ipsilateral side to verify perfusion

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 29.1.

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 mmHg) of the blood pressure in patients who are hypovolemic (Fig. 29.2).

Cardiac Output

Recall that global oxygen delivery (DO_2) to the tissues is dependent on the oxygen content of blood (CaO_2) as well as cardiac output (CO). Cardiac output is equal to the product of heart rate (HR) and stroke volume (SV):

$$CO = HR \times SV$$

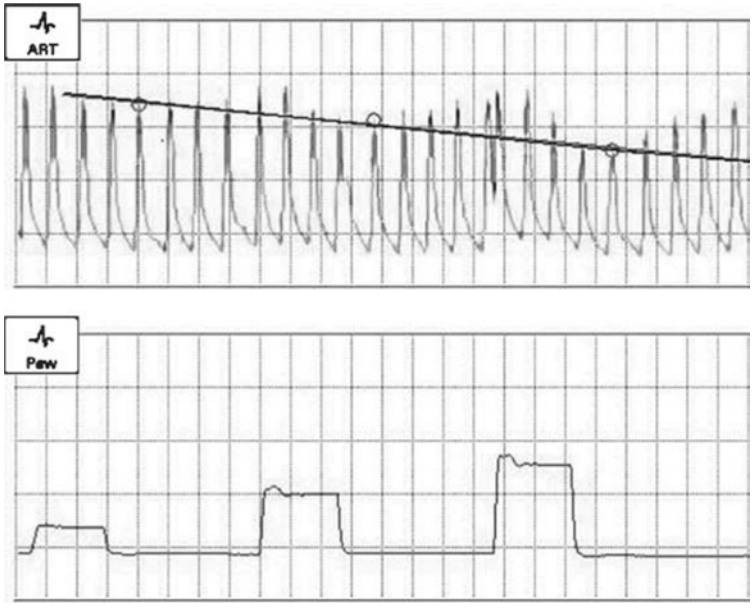

Figure 29.2 Arterial blood pressure tracing showing systolic pressure variation with three consecutive incremental breaths (10, 20, 30 cm H2O) during changes in mechanical ventilation. The line of best fit connects the three respective lowest systolic blood pressures (Used with permission. From Functional Hemodynamic Monitoring by Michael R. Pinsky, Didier Payen. Springer, 2004)

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. 29.3). 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 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

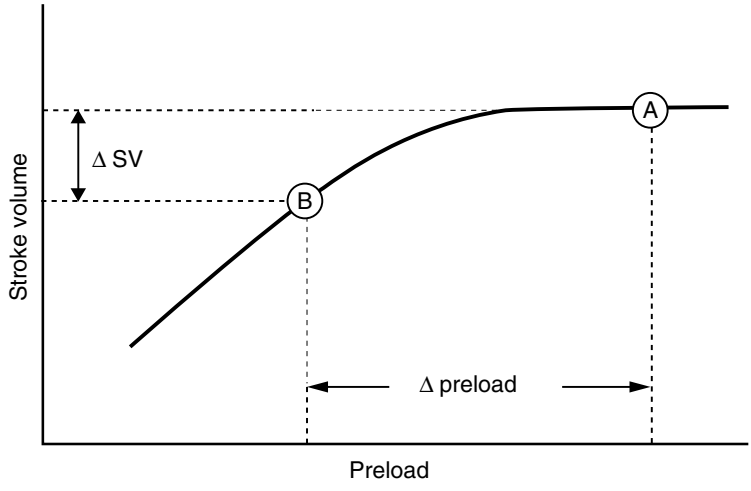

Figure 29.3 Frank-Starling curve. This curve shows the relationship between preload (end-diastolic volume) and stroke volume. Increases in preload lead to increased stroke volume until a point is reached where further increases in end-diastolic volume lead to congestive heart failure (point B to point A) (Used with permission. From Pediatric Critical Care Medicine: Basic Science and Clinical Evidence. Derek S. Wheeler, Hector R. Wong, and Thomas P. Shanley, editors. Springer, 2007)

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 clinically represented by the pulmonary vascular resistance (PVR) and left ventricular afterload is clinically represented by the systemic vascular resistance (SVR). SVR may be calculated from the following equation when cardiac output measurements are obtained:

$$\mathrm{SVR} = \left[\left(\mathrm{MAP} - \mathrm{CVP}\right) / \mathrm{CO}\right] \times 80$$

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 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 mmHg. 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 Chap. 15, IV, Arterial & Central Line and Gastric Tube Placement Techniques). Complications associated with the placement of a central line are presented in Table 29.2. To reduce the number of cannulation attempts and the risk of inadvertent arterial puncture, direct visualization with ultrasound guidance should be used when possible during placement of a central line.

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 (SvO_2) in order to evaluate oxygen balance. Risks associated

Table 29.2 Complications associated with central venous and pulmonary artery catheterization

Complication	Precautions to decrease risk
Hematoma	Avoid multiple needle punctures/attempts Apply pressure if vein or nearby artery punctured
Bleeding/hemorrhage	Caution in coagulopathic patients Apply pressure to bleeding site
Air or thrombotic embolism	Caution with infusions Use head-down tilt and avoid open catheter to air Avoid prolonged catheterization and use continuous flush
Carotid artery puncture/cannulation	Use appropriate landmarks ± sonographic visualization Use small finder needle; transduce pressure to verify venous
Pneumothorax/hemothorax[a]	Use appropriate landmarks Avoid multiple needle sticks No risk with femoral vein Risk with internal jugular < risk with subclavian
Infection/bacteremia/endocarditis	Use strict sterile technique[b] Avoid prolonged catheterization
Nerve trauma	Use appropriate landmarks and avoid sites in close proximity to nerves
Thoracic duct damage/chylothorax	Avoid left subclavian and internal jugular when possible
Complete heart block	Extreme caution placing PAC in patient with LBBB
Cardiac dysrhythmias	Use ECG monitoring while placing catheter and avoid prolonged placement of wire/catheter in atria/ventricles
Pulmonary ischemia/infarction	Do not keep PAC continuously wedged Minimize balloon inflation time
Pulmonary artery rupture Myocardial perforation	Do not over-wedge PAC; avoid balloon hyperinflation Always inflate balloon before advancing catheter, but never inflate balloon against significant resistance Always deflate balloon before withdrawing catheter

PAC **pulmonary artery catheter,** ***LBBB*** **left bundle branch block,** ***ECG*** **electrocardiogram**
[a]**A chest xray should always be performed after catheterization to verify correct positioning and absence of pneumothorax/hemothorax**
[b]**Strict sterile technique includes handwashing, sterile gloves, gown, mask, hat, patient drape, and sterile prep with chlorhexidine**

with PAC placement include those associated with central line placement as well as additional risks (Table 29.2). Table 29.4 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 29.3 shows the four main types of shock and lists examples of each.

Table 29.4 presents the most likely hemodynamic disturbances that are associated with each type of shock.

Table 29.3 Classification of shock and examples

Shock type	Examples
Hypovolemic	Dehydration, hemorrhage
Cardiogenic	Acute myocardial infarction, congestive heart failure
Distributive	Sepsis, anaphylaxis, neurogenic shock
Obstructive	Cardiac tamponade, tension pneumothorax

Table 29.4 Hemodynamic disturbances in shock

Shock type	Central venous pressure or pulmonary capillary wedge pressure	Cardiac output	Systemic vascular resistance
Hypovolemic	Decreased	Decreased	Increased
Cardiogenic	Increased	Decreased	Increased
Distributive	Depends on volume status (initially decreased)	Normal or increased	Decreased
Obstructive	Increased	Decreased	Increased

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 mmHg*. 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 $ScvO_2$ (or SvO_2 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/or inotropes. Many of the vasoactive medications used to treat shock have more than one mechanism of action. Table 29.5 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

Table 29.5 Commonly used vasoactive agents in shock

Agent	Mechanism of action (receptor)
Dopamine	Chronotropy (β1), Inotropy (β1) › Vasoconstriction (α at higher doses)
Dobutamine	Chronotropy (β1), Inotropy (β1) › Vasodilation (β2)
Epinephrine	Chronotropy (β1), Inotropy (β1) › Vasoconstriction (α at higher doses)
Norepinephrine	Vasoconstriction (α) › Chronotropy (β1), Inotropy (β1)
Phenylephrine	Vasoconstriction (α1)
Vasopressin	Vasoconstriction (V1)

source of infection with the early initiation of broad-spectrum antibiotics and if necessary, surgical debridement. An overview of the management of septic shock is presented in Table 29.6.

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 have 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. 29.4). 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.

Table 29.6 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 mmHg (12–15 mmHg if mechanically ventilated)
      $ScvO_2 \geq 70$ % (or $SvO_2 \geq 65$ %)
   (b) Begin with fluid resuscitation if the patient is hypotensive or has elevated lactate >4 mmol/L
      Minimum 30 mL/kg crystalloid
      Consider colloid (albumin) if patient requiring considerable fluid
      Avoid hydroxyethyl starches
      Continue fluid resuscitation as long as patient shows hemodynamic improvement
   (c) Add a vasopressor if the patient is not responding appropriately to fluid resuscitation
      Use an arterial line if vasopressors are required
      Norepinephrine is the first-line vasopressor
      Vasopressin may be added to norepinephrine if needed
      Epinephrine if additional agent needed
   (d) Consider administration of steroids only if the patient has a poor response to fluids and vasopressor therapy
   (e) Consider inotropic support with dobutamine if not meeting hemodynamic goals with above measures (especially if evidence of myocardial dysfunction)
   (f) Consider a blood transfusion if Hb <7 g/dl
2. Diagnosis – try to obtain 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 first hour of hypotension
   (b) Initiate broad-spectrum antibiotics
   (c) Follow cultures daily 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 (see sections “Acute Respiratory Distress Syndrome” and “Supportive Care in the ICU”) – low tidal volume ventilation, glucose management, thromboprophylaxis, ulcer prophylaxis, nutrition

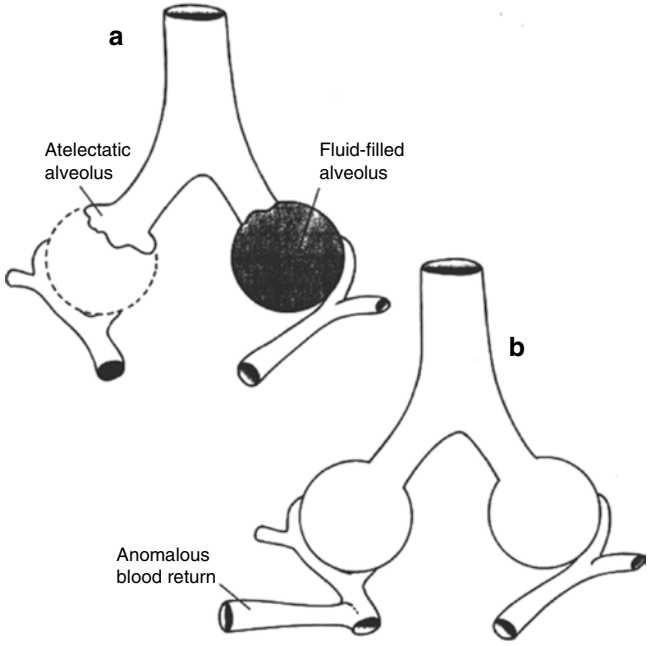

Figure 29.4 Examples of intrapulmonary shunt. (a) Collapsed and fluid-filled alveoli are examples of intrapulmonary shunt. (b) Anomalous blood return of mixed venous blood bypasses the alveolus and thereby contributes to the development of intrapulmonary shunt (Used with permission. From Critical Care Study Guide: Text and Review. Gerard J. Criner and Gilbert E. D'Alonzo, editors. Springer, 2002)

Other causes of hypoxemia include increased dead space (see below), 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 than leads to decreased alveolar minute ventilation. Minute ventilation (Va) is defined by the following equation:

$$\mathrm{Va} = \mathrm{f} \times \left(\mathrm{Vt} - \mathrm{Vd} \right)$$

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) and/or increases in dead space ventilation. Dead space ventilation includes any area of the respiratory tract 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 Respiratory Distress Syndrome (ARDS)

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 and edema. Diagnostic criteria for ARDS have been defined by an international consensus committee and include:

- Acute process (within 1 week of clinical insult)
- Severe hypoxemia ($PaO_2/FiO_2 < 300$ mmHg)
- Bilateral diffuse pulmonary infiltrates seen on chest radiograph
- Evidence of noncardiogenic pulmonary edema

Further, the severity of ARDS is classified by the PaO2/FiO2 ratio as follows: PaO2/FiO2 ratio < 300 is mild ARDS, PaO2/FiO2 ratio < 200 is moderate ARDS, and PaO2/FiO2 ratio < 100 is severe ARDS. Increased severity correlates with increased mortality rates. 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 alternative modes of ventilation, prone positioning, inhaled nitric oxide, and extracorporeal membrane oxygenation. While these therapies can improve oxygenation and provide temporary support, none of them have 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
- airway protection

Commonly Used Modes of Ventilation

Assist-Control Ventilation (Also Known as CMV, 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

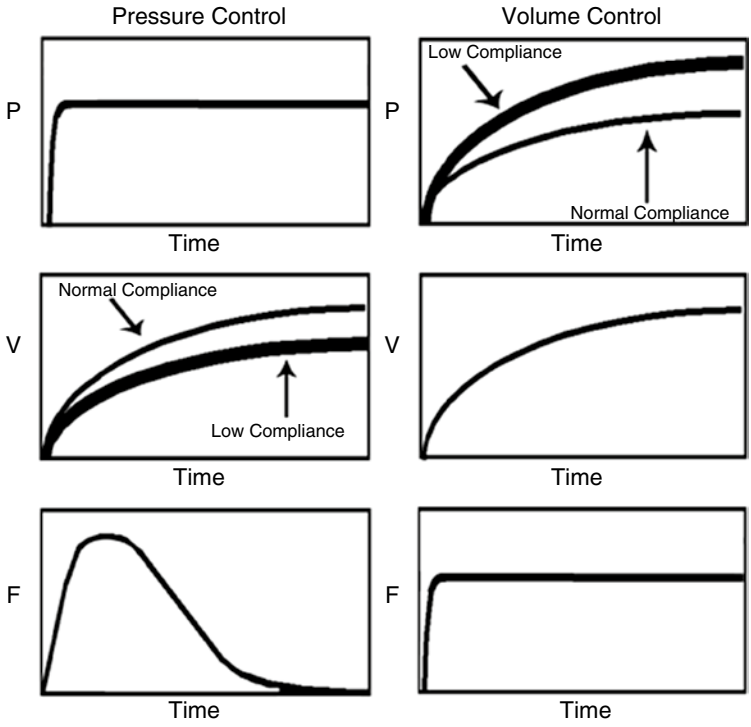

Figure 29.5 Graphical representation of pressure (P), volume (V), and flow (F) versus time for Pressure Control Ventilation and Volume Control Ventilation. Note in Pressure Control, the pressure stays constant as the tidal volume varies based on lung compliance. Note in Volume Control, the flow and volume remain constant while pressure varies based on lung compliance (Used with permission from Wilson WC, Grande CM, Hoyt DB, eds. *Trauma Critical Care*. Informa Healthcare USA, Inc. New York, 2007)

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. Figure 29.5 shows pressure, volume, and flow tracings for pressure control versus volume control.

Intermittent Mandatory Ventilation (IMV)

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 (SIMV) 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 control 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 (FiO_2) 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 H_2O to ensure that plateau pressures remain less than 30 cm H_2O. 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 H_2O 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 without additional pressure support during inspiration. 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. Methicillin-resistant *Staphylococcus aureus* and gram-negative organisms (such as *Pseudomonas aeruginosa*) 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. Guidelines for the management of VAP have been published 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 30–45° for prevention of aspiration
5. Oral hygiene with chlorhexidine rinse for intubated patients
6. Avoidance of inappropriate use of antibiotics
7. Sedation and ventilator weaning protocols

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. Based on the most recent data, the optimal target range for blood glucose in critically ill patients is <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 H_2-receptor antagonist.

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. Many intensive care units now have Palliative Care teams to assist with end-of-life care.

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 to 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 to PACU were normal, but the blood pressure has been progressively declining and the heart rate has been rising since then. Five minutes ago, the patient's blood pressure was 68/40 with 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 and lack of vital signs, 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 by activating the "code" team or other system in place in your particular hospital. (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 (VF). 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 continued 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/or internal hemorrhage as the etiology, as immediate reoperation will be necessary. Continue ACLS until return of spontaneous circulation, after which aggressive fluid administration and vasopressors can temporize until definitive intervention can take place.

Suggested Further Reading

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Part VII

Special Topics

Chapter 30

Professionalism, Safety, and Teamwork

Sheila Ryan Barnett, Stephen D. Pratt, and Jesse M. Ehrenfeld

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.

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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 30.1 represent a synthesis of several definitions of Professionalism.

Table 30.1 Professionalism: essential attributes for anesthesiologists

(1) Competence in the fundamental elements necessary for the safe delivery of anesthesia, including both technical and non technical aspects
(2) Assumes responsibility for the care of individual patients and as such contributes to the well being of society in general
(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
(4) Exhibits the following humanistic qualities including: altruism, accountability, excellence, duty, honor and integrity, and respect for others

For anesthesiology professionalism, at a minimum, implies competence in the knowledge of anesthesia. The basic curricular elements considered necessary for certification in anesthesiology 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 30.2.

For most patients, surgery is a relatively unique event, and one that is surrounded by significant anxiety and trepidation. The way an anesthesiologist

Table 30.2 Etiquette-based anesthesia introductions

(1)	Verify with the nurse and the patient that now is an appropriate time to begin the anesthetic interview and preparation
(2)	Introduce yourself – as a physician. First names can come later
(3)	Look the patient in the eye and shake hands. Introduce yourself to family members – ask their relationship, do ***not*** make assumptions
(4)	Briefly explain your role within the anesthesia team (i.e. a student or resident), name the attending if applicable
(5)	Verify with the patient the surgery that will be occurring
(6)	Begin your discussion regarding the administration of anesthesia

Adapted from Kahn [6]

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 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 44,000–98,000 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

Table 30.3 Common causes of communication failure

Interruptions: Up to 1/3 of communication events in the operating room are interrupted. This can lead to confusion, loss of information or failure to complete the communication
Fear: Medical students, residents and nurses are frequently afraid of being chastised, of offending their superior, or of looking incompetent if they ask questions or communicate the wrong information
Stress/conflict: It has been estimated that open conflict occurs between clinicians in the operating room in about 10 % of cases. This is similar to the rate seen in the cockpit of commercial aircraft. However, these conflicts are resolved about 80 % of the time in the cockpit, versus only 20 % in the operating room
Too much communication: Up to 100 communication events occur per procedure in the operating room and resource nurses must manage an average of 74 communication events per hour
Noise: The average noise level in the operating room is 57 dB, but it can reach much higher

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 30.3.

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 1980s 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.

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Chapter 31

Quality Assurance, Patient and Provider Safety

Arti Ori and Jesse M. Ehrenfeld

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

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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 31.1 ASA Closed Claims Study – most common adverse outcomes

Adverse outcome (N = 7,740)	% of claims	Median payment ($)	Range of payment ($)
Death	29	338,000	353–17,934,000
Nerve damage	19	92,650	394–10,716,000
Permanent brain damage	10	1,216,950	5,950–35,960,000
Airway trauma	7	72,000	34–2,115,000
Eye damage	4	97,600	37–3,335,000
Injury to newborn	3	667,069	3,966–15,822,000
Stroke	3	301,250	7,050–24,966,195
Pneumothorax	3	62,900	465–13,950,000
Back pain	3	26,400	2,240–1,782,500
Headache	3	18,300	884–874,500
Aspiration pneumonitis	3	301,750	573–3,450,000
Myocardial infarction	2	218,000	7,600–1,810,500
Burn, thermal	2	49,995	5,025–844,800
Skin reaction	2	21,788	488–727,500
Awareness	1	37,463	1,940–846,000
Meningitis	1	101,219	4,608–873,000

Table 31.1 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).

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 (Table 31.2). For example, difficult airway management during induction of anesthesia has long been regarded as one of the most challenging issues

Table 31.2 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

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.* 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.

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 as 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 then 90° 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 1970s. She is anxious because her father has told her stories of surgery in the 1950s and 1960s, 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 1950s that have improved safety?

Numerous monitors have been added to the manual blood pressure cuff and finger on the pulse of the mid-twentieth 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 definition of the term). 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 1980s, 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.

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Chapter 32

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.

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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

Advance 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 consent form is not sufficient. 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

1. Beauchamp TL, Childress JF (2001) Principles of biomedical ethics. Oxford University Press, New York

2. Studdert DM et al (2006) Claims, errors, and compensation payments in medical malpractice litigation. N Engl J Med 354:2024–2033

3. Drane JF (1984) Competency to give an informed consent. A model for making clinical assessments. JAMA 252:925–927

Chapter 33

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
- 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 (Figs. 33.1, 33.2, and 33.3).

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 with simulation during your medical training. You have used simulation if you have participated in any form of problem-based learning using paper cases, have practiced suturing on a pig's foot, or have been assessed using standardized patient encounters.

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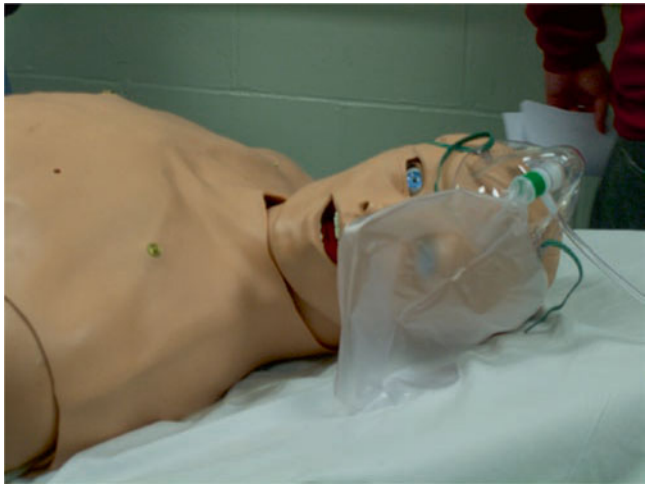

Figure 33.1 A simulation mannequin

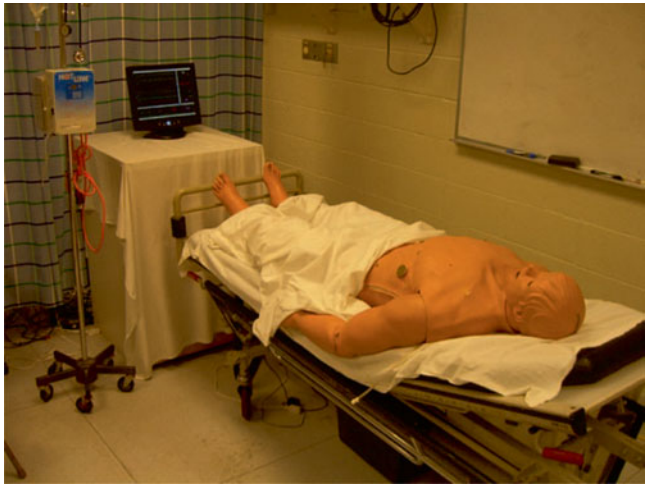

Figure 33.2 A typical set-up of a full-body mannequin on a hospital gurney, with the computer monitor in the background displaying the vitals

Figure 33.3 A view of the ongoing simulation from the control room

Table 33.1 Matrix of categories of simulation

Fidelity	Cognitive	Procedural	Teamwork
Low	Paper cases	Pig foot suturing	Table-top exercise
Medium	Computer case		
High	Full-body mannequin/patient actors	Task trainers	Full-body mannequin
Highest	Virtual reality	Task trainers with haptics (tactile sensation)	Virtual reality

In order to understand simulation better, it is helpful to categorize the 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 33.1 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 higher-fidelity simulations. In addition, recent pressure from both the public 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 1960s for anesthesiologists. It was not until the 1980s 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 2000s, medical simulation has spread to all levels of medical training (undergraduate medical school to continuing medical education) and into many different specialties.

What Is the Evidence behind Medical Simulation?

There are several studies showing improved outcomes after the use of simulation (Cook et al). 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 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 to 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 1970s, 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 "crisis resource management." As a student, you may participate in some crisis resource management scenarios in the simulation laboratory.

Medical training institutions are using 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 laboratory 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 team work 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 features with newly-built or renovated centers, while others may be a mannequin set up in a classroom or closet. Either way, the simulation team works to make the simulation feel realistic to the participants.

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, a 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 an available space with the mannequin and monitor. In this case, one room may hold all three sections: the

mannequin, the control area, and the debriefing area. Before the simulation encounter, you should receive enough information from the simulation staff to understand the clinical environment in which you are practicing, and thus what resources are available to you (Fig. 33.2).

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 controls the voice/responses of the mannequin. This control area may be in direct view of you, in the same room but behind a curtain, or in a separate room separated by one-way mirror or viewed through cameras. Usually, you will not spend time in the control room if you are a learner in the sessions (Fig. 33.3).

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. The actors in the scenarios are there to help orient you to the environment.

At least one instructor will be present to lead the debriefing session after a simulation encounter. This instructor may be a physician, nurse, paramedic, or an educator. During the scenario, the instructor is either in the simulation room or in the control room. While the instructor may be able to help you manage the case, try to use your own knowledge and skills in the scenario rather than depending on the instructor for answers.

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 conversation will focus on that concept. Take advantage of these discussions to ask questions about your performance and fund of knowledge. One of the powerful benefits

of simulation is that it allows the participant to actively obtain knowledge by being actively involved in the case, rather than passively hearing the information in a lecture-style environment.

What Can a Mannequin Do?

Several simulation companies exist, each with a mannequin that has slightly different characteristics. The more expensive mannequins typically are able to portray more complex responses or exam findings. 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 realism of the findings. If you have a question about a specific exam finding, then you can ask either an actor in the room or the instructor to clarify a specific 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 separate 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?

Prior to the start of the simulation, you should ask if there will be a formal assessment of your performance during the encounter. You will receive verbal feedback on how you performed in the debriefing scenario after each simulation, but you may not have formal assessment written as part of your record. If you are being formally assessed, it will be important to ascertain the rater's expectations of your performance. It also is important for you to have a chance to experience the simulation equipment 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. 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. Simulations allow you to challenge yourself and make mistakes, with the opportunity for the practice and feedback that Ericsson suggests.

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. Some encounters will have pre-scenario reading or other preparation; however, most encounters will give you no advance information regarding the case content. Sometimes scenarios are of rare events that you may have not managed in the past. The faculty member observing the case will be able to directly observe your actions and may ask for your unspoken thoughts in the simulation scenario. Not only is your participation in the simulation scenario important to your learning, but also your active participation in the debriefing discussion. 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.

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 and the debriefing experience.

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 clinical reasoning by someone more expert than you. Not many other times in your training when you will be directly observed as you will be in the simulation laboratory.

Case Study

*Note that not all simulation cases may be this complex.

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 with a 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

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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.

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Appendix B

Malignant Hyperthermia

Richard D. Urman and Jesse M. Ehrenfeld

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.

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Presenting Signs and Diagnosis

The most common presenting features of MH include significant, unexplained elevation in expired CO_2, 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

Table B.1 Main clinical features of malignant hyperthermia

Rising $ETCO_2$ and $PaCO_2$
Tachycardia
Tachypnea
Muscle rigidity and masseter spasm
Hemodynamic instability
Cardiac arrhythmias
Increased body temperature
Metabolic acidosis
Hyperkalemia
Myoglobinuria

Table B.2 Treatment of acute malignant hyperthermia

1. Discontinue volatile agents and succinylcholine
2. Call for help
3. Hyperventilate with 100 % oxygen
4. Inform the surgeon and curtail the surgical procedure
5. Initiate treatment with dantrolene (2.5 mg/kg)
6. Administer bicarbonate for metabolic acidosis
7. Actively cool the patient
8. Treat acidosis and hyperkalemia to avoid arrhythmias
9. Follow $ETCO_2$, electrolytes, blood gasses, CK, temperature, and urine output

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.

Suggested Further Reading

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